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The evolution of phenotypic plasticity in spatially structured environments: implications of intraspecific competition, plasticity costs and environmental characteristics

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Abstract

We model the evolution of reaction norms focusing on three aspects: frequency-dependent selection arising from resource competition, maintenance and production costs of phenotypic plasticity, and three characteristics of environmental heterogeneity (frequency of environments, their intrinsic carrying capacity and the sensitivity to phenotypic maladaptation in these environments). We show that (i) reaction norms evolve so as to trade adaptation for acquiring resources against cost avoidance; (ii) maintenance costs cause reaction norms to better adapt to frequent rather than to infrequent environments, whereas production costs do not; and (iii) evolved reaction norms confer better adaptation to environments with low rather than with high intrinsic carrying capacity. The two previous findings contradict earlier theoretical results and originate from two previously unexplored features that are included in our model. First, production costs of phenotypic plasticity are only incurred when a given phenotype is actually produced. Therefore, they are proportional to the frequency of environments, and these frequencies thus affect the selection pressure to avoid costs just as much as the selection pressure to improve adaptation. This prevents the frequency of environments from affecting the evolving reaction norm. Secondly, our model describes the evolution of plasticity for a phenotype determining an individual's capability to acquire resources, and thus its realized carrying capacity. When individuals are distributed randomly across environments, they cannot avoid experiencing environments with intrinsically low carrying capacity. As selection pressures arising from the need to improve adaptation are stronger under such extreme conditions than under mild ones, better adaptation to environments with low rather than with high intrinsic carrying capacity results.

Introduction

Phenotypic plasticity – the ability of a genotype to produce alternative phenotypes according to the environment that it experiences – might be an adaptive response to environmental variability (Schmalhausen, 1949; Levins, 1963, 1968; Bradshaw, 1965). During the last two decades, empirical evidence that phenotypic plasticity meets all the conditions required for it being selected for has accumulated: (i) phenotypically plastic genotypes can have fitness advantages relative to non-plastic ones in both animals (Travis, 1994) and plants (Schlichting, 1986; Sultan, 1987); (ii) plasticity may be genetically controlled (Schlichting & Pigliucci, 1993, 1995; Pigliucci, 1996); and (iii) additive genetic variation exists for phenotypic plasticity (Schlichting, 1986; Sultan, 1987; Scheiner, 1993).

Appreciating that phenotypic plasticity can be selected for immediately begs the question about the genetic and

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ecological conditions that promote or prevent its evolutionary emergence. This question has been intensively investigated during recent years, mainly through theoretical approaches. Except for a few multilocus and gametic studies (Zhivotovsky *et al.*, 1996; Scheiner, 1998), the majority of models for the evolution of phenotypic plasticity belong to just two groups: quantitative genetics models (e.g. Via & Lande, 1985, 1987; van Tienderen, 1991; Gomulkiewicz & Kirkpatrick, 1992; Gavrilets & Scheiner, 1993a,b) and optimality models (e.g. Stearns & Koella, 1986; Houston & McNamara, 1992; Kawecki & Stearns, 1993; Sasaki & de Jong, 1999). Quantitative genetics models are mainly meant to investigate the implications of genetic constraints on the evolution of reaction norms resulting from the lack of genetic variance or from adverse genetic correlations, whereas optimality models primarily focus on the ecological conditions promoting the evolution of phenotypic plasticity.

These two frameworks have enabled important, complementary insights into the evolution of phenotypic plasticity. Yet, three crucial issues have received little attention so far:

1. Most models do not consider interactions between individuals, like competition or predation, as potential selective forces for the evolution of phenotypic plasticity. This is mainly due to the particular difficulty of including nonlinear population dynamics in quantitative genetics and optimality models. Indeed, via individual interactions, populations become part of their own environment, which in turn affects their dynamics and selective pressures, generating an eco-evolutionary feedback (Maynard-Smith, 1982; Brown & Vincent, 1988; Metz *et al.*, 1992; Kawecki, 1993; Heino *et al.*, 1998). Such feedback typically results in density-dependent population dynamics and frequency-dependent selection, and implies that whether or not an individual with a given phenotype is selected for depends on the other phenotypes present in the population. It is thus essential to be able to account for nonlinear population dynamics and the resulting frequency-dependent selection pressures in order to include realistic interactions between individuals in models of phenotypic plasticity evolution. One notable exception accounting for density dependence is a study by Sasaki & de Jong (1999). However, their model does not incorporate frequency-dependent selection, because density regulation is assumed to be uniform across phenotypes.

2. Costs of phenotypic plasticity, which are supposed to originate from the differential expenses incurred by fixed and plastic developments, are rarely considered. Yet such costs are expected to act as major constraints for the evolution of plastic phenotypes (see DeWitt *et al.*, 1998 for details). First steps towards exploring the impact of costs on the evolution of phenotypic plasticity were taken by van Tienderen (1991, 1997), Moran (1992) and Léon (1993). These studies focused on maintenance costs, i.e. on expenses incurred by maintaining the potential for being plastic (DeWitt *et al.*, 1998). However, plastic organisms can also incur other types of cost (DeWitt *et al.*, 1998), like production costs. Costs of this latter type are those production costs paid by a plastic genotype actually producing a given phenotype in excess to those incurred by a fixed genotype producing the same phenotype. Production costs of phenotypic plasticity are expected to induce selection pressures on reaction norms that are qualitatively different from those caused by maintenance costs. (The different types of costs are discussed in more detail below.)

3. Except for the multilocus model of Zhivotovsky *et al.* (1996), evolutionary implications of the detailed characteristics of environmental heterogeneity have not yet received much attention. Different frequencies for the occurrence of environmental types, differential resource availability in these environments and varying sensitivities to maladaptation are all likely to play specific and important roles in shaping reaction norms, especially when considering individual interactions such as competition or predation. For example, when an environmental type is infrequent or offers low resource availability, at first glance, only a small degree of adaptation would be expected to evolve for this type. In contrast, when an environmental type occurs frequently or offers extensive resources, higher investments towards an adapted phenotype could be expected (Kawecki & Stearns, 1993; Zhivotovsky *et al.*, 1996).

The purpose of this paper is to extend and integrate the first steps undertaken in these three directions by van Tienderen (1991, 1997), Moran (1992), Léon (1993), Sasaki & de Jong (1999) and Zhivotovsky *et al.* (1996). With this aim, we present a model for the evolution of phenotypic plasticity driven by intraspecific competition for resources in a spatially structured environment and do account for density- and frequency-dependent selection, for different types of plasticity costs, and for different characteristics of environmental heterogeneity.

Modelling approach

In this study, we characterize phenotypic plasticity through reaction norms. A reaction norm is defined as the function that quantifies the systematic profile of phenotypes a genotype produces across a given range of environments (Schmalhausen, 1949). We then model the evolution of phenotypic plasticity through the evolutionary trajectories of reaction norms, based on adaptive dynamics theory (Dieckmann & Law, 1996; Metz *et al.*, 1996; Dieckmann, 1997; Geritz *et al.*, 1998). At the expense of genetic detail, but closely based on the underlying population dynamics, this approach (just like evolutionary game theory) analyses the course of natural selection through invasion processes. Considering a population consisting of resident individuals with a given reaction norm, we investigate whether or not mutant

individuals with a new reaction norm can invade and spread in that population. The probability of invasion by a mutant is determined directly from its population dynamics, using invasion fitness of the mutant (Metz *et al.*, 1992; Rand *et al.*, 1994; Ferrière & Gatto, 1995). We then describe the long-term evolution of reaction norms as sequences of invasion events in the course of which selectively advantageous mutants replace residents. This perspective enables us to derive the selection pressures acting on reaction norms from the specific ecological scenarios characterized by the population dynamics at hand, and to describe evolutionary transient states as well as equilibria. Most importantly, invasion fitness can be extracted from nonlinear population dynamics and, thus, allows accounting for all types of density dependence and frequency-dependent selection pressures that can arise from particular individual interactions.

To reflect the costs of phenotypic plasticity in our model, we follow DeWitt *et al.* (1998) in distinguishing two elementary types of cost. First are maintenance costs, which measure expenses for forming and maintaining the sensory systems and the regulatory machinery required by plastic development, in excess of those necessary for a fixed development. Simplifying DeWitt *et al.*'s classification, we assume that the costs of acquiring information about the environment and the detrimental consequences of developmental instability potentially incurred from plasticity also belong in this category. All these costs are envisaged as contributing to the price an organism has to pay for its potential of being phenotypically plastic. By contrast, a second type of plasticity cost is expected to result from the actual investments an individual with a potential for plasticity has to make in order to produce the specific phenotype that is induced by the type of environment it is exposed to. These expenses are referred to as production costs and are only incurred if and when a phenotype is expressed. Notice that all fixed genotypes also incur production costs, referred to as direct production costs (Scheiner & Berrigan, 1998). Production costs of phenotypic plasticity, by contrast, are those production costs paid by a plastic genotype to produce a given phenotype in excess of the direct production costs incurred by a fixed genotype producing the same phenotype.

Finally, we consider a complex spatially structured environment to which reaction norms must adapt by accounting for three fundamental characteristics of environmental heterogeneity: the frequency at which different types of environment occur, the quality and abundance of their resources described by their carrying capacity, and the sensitivity to phenotypic maladaptation in each type of environment.

We begin this study by deriving the population dynamics for phenotypically plastic organisms that compete for resources, from which we extract the invasion fitness determining the probability that a mutant can invade. In the next section, we then describe the evolutionary trajectories of reaction norms and assemble the resulting insights about the evolution of phenotypic plasticity. In the last section, we evaluate the results presented in the light of previous work and highlight a number of promising directions for future research.

Population dynamics and invasion criterion

Reaction norms and environmental heterogeneity

We consider organisms that express a reaction norm p across a range of environmental types e. Each type e is characterized by its frequency of occurrence $o(e)$ and an intrinsic carrying capacity $k(e)$ that measures the abundance and quality of its resources. The phenotype expressed in environmental type e, denoted as $p(e)$, determines the efficiency $E_p(e)$ with which an individual can acquire and/or utilize resources in this particular type of environment. One can think here of any morphological trait linked to the gathering or handling of resources, like beak size in birds or root length for plants, but also of any physiological character linked to the assimilation of food resources, like digestive enzymes. For each environmental type e, a matching phenotype $m(e)$ exists that brings this efficiency up to 1 according to the following Taylor expansion,

$$E_p(e) = 1 - s(e)[p(e) - m(e)]^2, \qquad (1)$$

such that a perfectly matched reaction norm $p = m$ would give an individual maximal efficiency in every environmental type. The matching phenotypes $m(e)$ can be interpreted as the collection, over the different environmental types e, of maximally adapted specialist phenotypes. Here $s(e)$ measures the sensitivity to phenotypic maladaptation in environmental type e, i.e. as $s(e)$ increases, the loss in terms of efficiency of resource utilization due to a not perfectly matched phenotype, $p(e) \neq m(e)$, increases. Sensitivity to phenotypic maladaptation is, together with the frequency of occurrence of environmental types and their intrinsic carrying capacity, the third characteristic of environmental heterogeneity considered in our analysis.

In order to study evolution of the reaction norm p, we model evolutionary trajectories as sequences of substitutions $p \rightarrow p'$, where a resident population with reaction norm p is invaded and then replaced by a selectively advantageous mutant with reaction norm p'. We use invasion fitness f, defined as the long-term per capita growth rate of a rare mutant arising in a resident population that has reached is population dynamical attractor (Metz *et al.*, 1992; Rand *et al.*, 1994; Ferrière & Gatto, 1995): individuals with reaction norm p' can invade into resident populations with reaction norm p only if their invasion fitness is positive, $f_{p',p} > 0$. In order

to derive invasion fitness, in the following we specify the population dynamics of the organisms considered and detail how the reaction norm p influences the population growth rate.

Population dynamics

We consider situations in which the reaction norm p affects competition for resources between individuals via the efficiency of resource utilization. We describe the contribution of individuals living in environments of type e to the total population growth rate by a Lotka–Volterra competition model and assume that some costs of phenotypic plasticity C_p decrease this contribution. With $n_p(e, t)$ measuring the density of individuals with reaction norm p living in environmental type e at time t, the net contribution of these individuals to the total population growth rate is given by

$$n_p(e,t)\left\{r\left[\frac{1-L_p(e,t)}{k_p(e)}\right]-C_p\right\}, \qquad (2)$$

where the intrinsic growth rate r is independent of the environmental type e, $k_p(e)$ is the realized carrying capacity of individuals with reaction norm p living in an environment of type e, and $L_p(e, t)$ is the strength of competition experienced by these individuals at time t.

Then, the dynamics of the total population density $n_p(t)$ of individuals with reaction norm p at time t are obtained by summing the local contributions over all environmental types e, which leads to

$$\frac{\mathrm{d}n_p(t)}{\mathrm{d}t}=\int n_p(e,t)\left\{r\left[\frac{1-L_p(e,t)}{k_p(e)}\right]-C_p\right\}\mathrm{d}e. \qquad (3)$$

Assuming that individuals with reaction norm p choose to settle in an environment of type e with probability $d_p(e)$, such that $n_p(e, t) = n_p(t)d_p(e)$, we finally obtain

$$\frac{\mathrm{d}n_p(t)}{\mathrm{d}t}=n_p(t)\left\{r\int\left[\frac{1-L_p(e,t)}{k_p(e)}\right]d_p(e)\,\mathrm{d}e-C_p\right\}. \qquad (4)$$

The distribution of individuals across environmental types has two implications. First, offspring are not constrained to stay in the same environmental type as their parent and are free to move to any environmental type e with a probability $d_p(e)$. This process is critical for the evolution of phenotypic plasticity in the sense that it couples the local population dynamics across environments and thus ensures that we do not consider a collection of isolated local populations evolving on their own. Secondly, the probability distribution d_p can be interpreted in two ways. Considering individuals that are free to move during their lifetime, $d_p(e)$ measures the proportion of its lifetime that an individual with reaction norm p spends in environmental type e. In contrast, if we focus on individuals that spend their entire life in the same environmental type, $d_p(e)$ is the proportion of individuals with a reaction norm p that settle in environmental type e. Due to these two possible interpretations, our model applies both to situations in which phenotypes are labile during the lifetime of organisms as well as to situations in which they are fixed.

In the following sections, we detail the different components of eqn (4), in particular the dependence of intraspecific competition on the efficiency of resource utilization and the costs of phenotypic plasticity, and finally derive invasion fitness.

Resource competition

We allow for symmetric as well as asymmetric competition. In environmental type e, an individual with reaction norm p' exerts competition on an individual with reaction norm p at a strength measured by the competition coefficient $A_{p,p'}(e)$. For an individual living in environmental type e the probability density to encounter a competitor with reaction norm p' is given by the density of individuals with reaction norm p' living in environmental type e, $n_{p'}(e, t)$, divided by the frequency at which that environmental type occurs, $o(e)$. The total strength of competition, $L_p(e, t)$, experienced by an individual with reaction norm p in environmental type e is then obtained as the sum over the coefficient of competition of reaction norms p', weighted by the probability $n_{p'}(e, t)/o(e)$,

$$L_p(e,t)=\int A_{p,p'}(e)\frac{n_{p'}(e,t)}{o(e)}\,\mathrm{d}p'. \qquad (5)$$

We assume that higher resource utilization efficiency in a given environmental type gives two advantages to an organism: an improved competitive ability in case of asymmetric competition and/or an increase in the amount of accessible resource in that environmental type. This flexibility is incorporated by allowing both the competition coefficient $A_{p,p'}(e)$ and the realized carrying capacity $k_p(e)$ to depend on resource utilization efficiency $E_p(e)$.

First, we describe the competition coefficient affecting an individual with reaction norm p that competes with an individual with reaction norm p' in environmental type e as

$$A_{p,p'}(e)=\frac{2}{1+\exp[4a\Delta E_{p,p'}(e)]}, \qquad (6)$$

where the constant a determines the degree of competitive asymmetry and $\Delta E_{p,p'}(e) = E_p(e) - E_{p'}(e)$ is the difference in resource utilization efficiency between competitors with reaction norms p and p'. The case $a = 0$ describes symmetric competition, for which the competition coefficient does not depend on this difference. By contrast, $a > 0$ describes asymmetric competition, in which case the competition coefficient is a decreasing sigmoid function of the competitors' difference in resource utilization efficiency (Fig. 1a). This behaviour allows for situations in which two individuals encountering each other as they search for resources experience unequal effects of competition. If the

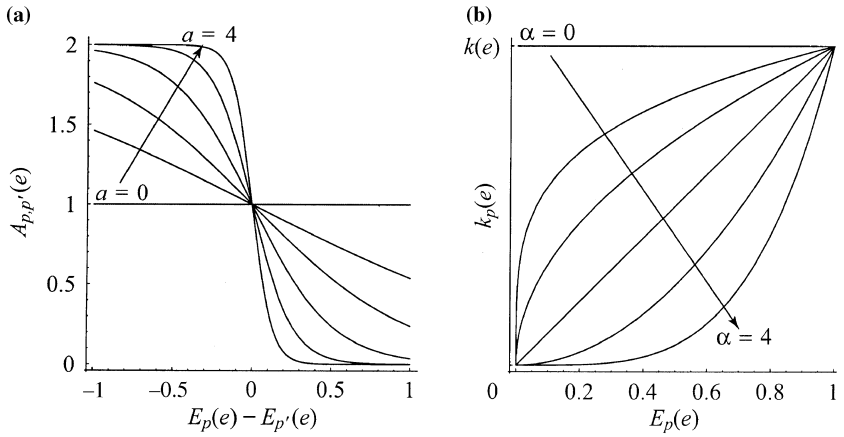

Fig. 1 Efficiency of resource utilization and intraspecific competition for resources. An individual's efficiency of resource utilization determines its competitive ability when encountering another individual, as well as its realized carrying capacity. (a) Competition coefficient [$A_{p,p'}(e)$]. When competition is symmetric ($a = 0$), the coefficient of competition does not depend on the efficiencies of resource competition of the two competitors, $E_p(e)$ and $E_{p'}(e)$. When competition is asymmetric ($a > 0$), the competition coefficient becomes a decreasing sigmoid function of the difference between the resource utilization efficiencies of the two competitors, $E_p(e) - E_{p'}(e)$: the competitor with the higher efficiency has an advantage over the other one. (b) Realized carrying capacity [$k_p(e)$]. When the realized carrying capacity is independent of the efficiency of resource utilization ($\alpha = 0$), individuals have access to the total amount of resources present in the environment, $k_p(e) = k(e)$. When the realized carrying capacity depends on the efficiency of resource utilization ($\alpha > 0$), individuals have only access to the proportion $E_p^{\alpha}(e)$ of resources present in the environment.

individual with reaction norm p possesses lower resource utilization efficiency than its competitor with reaction norm p', it will then lose a high amount of resource to its opponent, thus suffering from a more pronounced decrease in its per capita growth rate than its competitor. In contrast, if $E_p(e)$ is higher than $E_{p'}(e)$, the individual with reaction norm p gathers a large amount of resource at the expense of its competitor, thus experiencing only a small decrease in its per capita growth rate.

Secondly, we describe the realized carrying capacity of an individual with reaction norm p living in environmental type e as

$$k_p(e) = k(e)E_p^{\alpha}(e), \tag{7}$$

where α allows for adjusting the impact of the resource utilization efficiency $E_p(e)$ on the realized carrying capacity. For $\alpha = 0$, $k_p(e)$ is independent of resource utilization efficiency, and is equal to the intrinsic carrying capacity $k(e)$ of the environmental type considered. By contrast, for $\alpha > 0$, the realized carrying capacity increases with $E_p(e)$, describing situations in which the amount of resource an individual has access to is determined by its efficiency in gathering or utilizing the particular type of resource (Fig. 1b).

Costs of phenotypic plasticity

Costs of phenotypic plasticity are incurred at the level of the reaction norm. One can draw a parallel here with the quantitative genetics models of van Tienderen (1991, 1997), in which costs of plasticity are defined at the genotypic level because phenotypes expressed in different environments encountered by different individuals having the same genotype may all contribute to the costs of that particular genotype.

Following van Tienderen (1991, 1997), we measure costs of phenotypic plasticity relative to a cost-free reaction norm $\bar{p}$. Because costs of phenotypic plasticity are defined relative to fixed development (DeWitt *et al.*, 1998), this cost-free reaction norm $\bar{p}$ is 'flat', $\bar{p}(e) = \bar{p}$, corresponding to an organism exhibiting no plasticity at all. Then, we define the costs of phenotypic plasticity across environmental types for a reaction norm p as proportional to its variance around the cost-free reaction norm $\bar{p}$,

$$C_p = c \int [p(e) - \bar{p}]^2 \frac{d_p^{\beta}(e)}{\left|d_p^{\beta}\right|} \, \mathrm{d}e, \tag{8}$$

where the constant c scales the intensity of costs, and the parameter β is explained below. The quadratic dependence in the integrand is again motivated by a Taylor expansion. One can set the cost-free reaction norm $\bar{p}$ to any particular fixed value depending on the specific biological problem at hand. In this study, two reasons led us to set $\bar{p}$ equal to the mean phenotype along the reaction norm p, which is given by

$$\bar{p} = \int p(e) \frac{d_p^{\beta}(e)}{\left|d_p^{\beta}\right|} \, \mathrm{d}e. \tag{9}$$

First, we consider the cost-free reaction norm to be a developmental property of plastic organisms: $\bar{p}$ is genetically coded and is the fixed phenotype that an

individual can reach through cost-free development. Secondly, it can be shown that, for any particular reaction norm p, the cost-free reaction norm $\bar{p}$ that minimizes C_p is given by the mean phenotype along p. Then, it is quite natural to assume that natural selection on plastic organism swiftly results in the evolution of development so as to set the cost-free reaction norm $\bar{p}$ equal to the one that minimizes the costs of plasticity.

Cost contributions from different phenotypes $p(e)$ are weighted in the variance, eqn (8) and mean eqn (9), by the frequency distribution of individuals across environmental types, $d_p(e)$, taken to the power β. Notice that the frequency distribution of individuals across environmental types e is also the frequency at which the different phenotypes $p(e)$ are expressed. Thus, the exponent β conveniently allows us to distinguish between maintenance and production costs of phenotypic plasticity. Maintenance costs are paid for the potential of being plastic. These costs must be paid independently of whether or not a particular phenotype is produced, and therefore independently of the frequency distribution $d_p(e)$. Maintenance costs are thus accounted for by setting $\beta = 0$; the cost-free reaction norm is then simply the mean of phenotypes along the reaction norm, $\bar{p} = \int p(e)\,\mathrm{d}e$. By contrast, production costs are incurred when a given phenotype is actually produced, and must be paid as often as phenotypes are expressed. Thus, a linear dependence of the costs on $d_p(e)$, $\beta = 1$, characterizes production costs. The cost-free reaction norm is then the mean realized phenotype across all types of environment, $\bar{p} = \int p(e)d_p(e)\,\mathrm{d}e$. The range $0 < \beta < 1$ allows for different mixtures between maintenance and production costs. In these cases, division by $|d_p^\beta| = \int d_p^\beta(e)\,\mathrm{d}e$ is needed to normalize the distribution d_p^β. Note that, as highlighted by previous authors (DeWitt *et al.*, 1998; Scheiner & Berrigan, 1998), production costs of phenotypic plasticity are only paid by plastic genotypes, whereas direct production costs are incurred by both plastic and fixed genotypes producing similar phenotypes. Direct production costs are not included in our model because, being incurred by both plastic and fixed genotypes, they are not expected to constrain the evolutionary emergence of phenotypic plasticity relative to fixed development. The absence of plasticity production costs for fixed genotypes is reflected in our model by the fact that the cost-free reaction norm is set flat and at the mean expressed phenotype. A fixed genotype with a flat reaction norm will thus have a mean expressed phenotype that reduces its plasticity production cost to zero. Therefore, plasticity production costs as we defined them are just those production costs incurred by a plastic genotype in excess of the direct production costs incurred by a fixed genotype when both produce the same phenotype. The difference between maintenance and production costs is illustrated in Fig. 2.

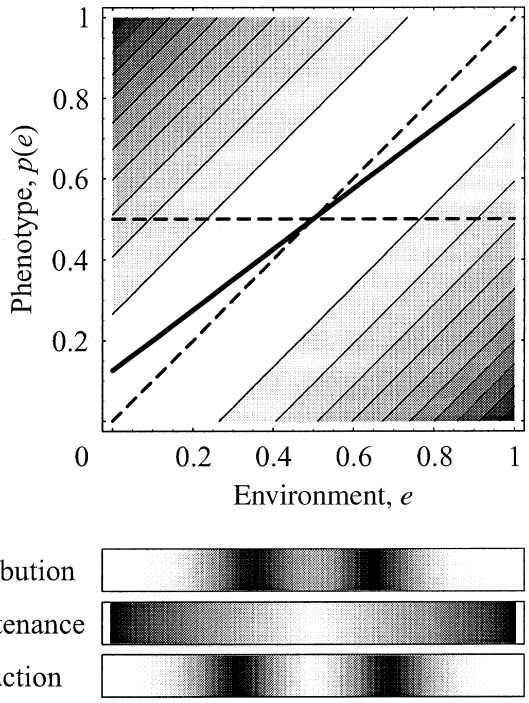

Fig. 2 Maintenance and production costs of phenotypic plasticity. As in all subsequent figures, the upper panel depicts the isoclines of competitive efficiency $E_p(e)$ for all environmental types e and for all possible phenotypes $p(e)$ (shades of grey, with white corresponding to highest efficiency), the curve through all matching phenotypes $m(e)$ (sloped dashed line), the evolutionarily singular reaction norm $p^*(e)$ (thick continuous curve) and the corresponding cost-free reaction norm $\bar{p}^*$ (horizontal dashed line). For all examples, environmental types e vary between 0 and 1 and matching phenotypes are given by $m(e) = e$. Here, the three greyscale bars below the upper panel depict the distribution of individuals along the environmental gradient, as well as the differential contributions of all phenotypes $p(e)$ to maintenance and production costs (black indicates highest values). The top bar depicts the assumption that the distribution of individuals is peaked at two environmental types, 0.35 and 0.65, $d_p = 0.5N(0.35, 0.085) + 0.5N(0.65, 0.085)$. The middle bar illustrates that maintenance costs are independent of this distribution: these costs simply increase with the difference between plastic phenotypes $p(e)$ and the cost-free phenotype $\bar{p}$. Thus, maintenance costs are primarily accrued for extreme phenotypes $p(e)$. The bottom bar highlights that, by contrast, production costs do depend on the distribution of individuals: as these cost are only incurred when a phenotype $p(e)$ is expressed, they are primarily accrued for phenotypes produced in frequently encountered types of environment.

For the sake of simplicity, we scaled the environmental types e between 0 and 1. If one wanted to consider environmental types on a different interval, it would be necessary to divide the variance and the mean in eqns (8) and (9) by the length of this interval.

With the ecological setting thus fully specified, we can now turn to the invasion fitness of a mutant with reaction

norm p' arising in a monomorphic resident population consisting of individuals with reaction norm p.

Invasion fitness

Invasion fitness characterizes the fate of a rare mutant in a resident population that has reached its population dynamical attractor. For the sake of simplicity, we concentrate on stable equilibrium attractors. A stable equilibrium $\hat{n}_p$ for a monomorphic resident population with reaction norm p is reached when $\mathrm{d}\hat{n}_p(t)/\mathrm{d}t = 0$. This yields

$$\hat{n}_p = \frac{r - C_p}{r|d_p^2/(ok_p)|}, \tag{10}$$

with $|d_p^2/(ok_p)| = \int d_p^2(e)/[o(e)k_p(e)]\,\mathrm{d}e$ being the average proportion of the intrinsic growth rate r lost per encountered competitor.

As mutants with reaction norms p' are initially rare, their population density can be neglected when considering density regulation. According to eqn (5), the strength of competition $L_{p'}(e, t)$ experienced by a mutant thus only depends on the resident density $\hat{n}_p$, $L_{p'}(e, t) = A_{p',p}(e)d_p(e)\hat{n}_p(t)$. Recasting eqn (4) accordingly, the population dynamics of a mutant with reaction norm p' that appears in a resident population with reaction norm p at equilibrium are given by

$$\frac{\mathrm{d}n_{p'}(t)}{\mathrm{d}t} = n_{p'}(t)\left\{\int r\left[\frac{1 - A_{p',p}(e)d_p(e)\hat{n}_p}{o(e)k_{p'}(e)}\right]d_{p'}(e)\,\mathrm{d}e - C_{p'}\right\}. \tag{11}$$

The invasion fitness of a mutant p' relative to a resident p is then obtained as the per capita growth rate of the mutant,

$$f_{p',p} = \int r\left[\frac{1 - A_{p',p}(e)d_p(e)\hat{n}_p}{o(e)k_{p'}(e)}\right]d_{p'}(e)\,\mathrm{d}e - C_{p'}. \tag{12}$$

For the sake of simplicity, we restrict our attention in this study to situations in which individuals distribute randomly across environmental types. Such a distribution strategy applies to organisms that have no selective control over their local habitat and, consequently, distribute across environmental types e according to the frequency $o(e)$ at which these types occur (more sophisticated distribution strategies can readily be analysed within the formal framework established here and will be investigated in a separate study). Then, substituting $d_p(e) = o(e)$ and eqn (10) in eqn (12) gives

$$f_{p',p} = r - (r - C_p)\frac{\int A_{p',p}(e)o(e)/k_{p'}(e)\,\mathrm{d}e}{|o/k_p|} - C_{p'}. \tag{13}$$

This result shows that invasion fitness (and thus selection) is frequency-dependent, as it is affected by the resident reaction norm. We can now determine which mutants with reaction norm p' arising in a population of residents with reaction norm p can invade, as they must fulfill the condition $f_{p',p} > 0$. According to eqn (13), this occurs when

$$\frac{r - C_{p'}}{\int A_{p',p}(e)o(e)/k_{p'}(e)\,\mathrm{d}e} > \frac{r - C_p}{\int o(e)/k_p(e)\,\mathrm{d}e}, \tag{14}$$

i.e. when the ratio between the intrinsic growth rate, diminished by the costs of phenotypic plasticity, and the average proportion of growth rate lost per encountered competitor is higher for the mutant than for the resident. Everything else being equal, this may be achieved by reducing the costs of plasticity, by improving competitive ability, or by increasing the access to resources. However, each of these three possibilities cannot be achieved without affecting the others, as they all depend on the reaction norm: improving competitive ability or access to resources requires increasing plasticity and thus increasing its costs and vice versa. Mutant invasions, and hence the evolutionary trajectories of reaction norms, will then depend on a balance between competitive advantages conferred by phenotypic plasticity and the related costs.

Evolution of reaction norms

Selection gradient and evolutionary trajectories

The invasion fitness obtained in eqn (13) allows us to describe the evolutionary trajectories of reaction norms as sequences of substitutions during which residents are replaced by mutants having positive invasion fitness, $f_{p',p} > 0$. For any phenotypic trait, the expected rate of such sequences of phenotypic substitutions is proportional to the selection gradient, the derivative of invasion fitness with respect to trait of the mutant (Dieckmann, 1994; Dieckmann *et al.*, 1995; Dieckmann & Law, 1996). The selection gradient also gives the direction of evolution relative to the current value of the trait: it will be positive if an increase in the value of the trait is selectively advantageous, and negative if that increase is unfavourable. It is helpful to realize that the proportionality between the rate of evolutionary change and the selection gradient also applies in models of quantitative genetics (Lande, 1982). The analysis below is thus not restricted to evolutionary changes through sequences of invasion and substitution (Kirkpatrick & Heckman, 1989; Gomulkiewicz & Kirkpatrick, 1992).

To obtain the selection gradient of an infinite-dimensional trait, like a reaction norm, one has to consider the functional definition of a derivative,

$$g_p(e) = Df_p(e) = \lim_{\varepsilon\to 0}\frac{f_{p+\varepsilon\delta_e,p} - f_{p,p}}{\varepsilon} = \frac{\partial}{\partial\varepsilon}f_{p+\varepsilon\delta_e,p}\bigg|_{\varepsilon=0}, \tag{15a}$$

where $\delta_e(e') = \delta(e - e')$ is Dirac's delta function peaked at e. Applying this definition to the invasion fitness function given in eqn (13) (see Appendix for details), we obtain the selection gradient for a reaction norm p,

$$g_p(e) = w_m(e)[m(e) - p(e)] + w_{\bar{p}}(e)[\bar{p} - p(e)], \qquad (15b)$$

with the coefficients $w_m(e)$ and $w_{\bar{p}}(e)$ given further below [eqn (16b)]. Then, under the assumption that mutations affecting different points of the reaction norm occur independently, the selection gradient allows us to describe the expected evolutionary changes in the current shape of the reaction norm p. Specifically, $g_p(e)$ determines, for any environmental type e, the direction of evolution of the expressed phenotype $p(e)$: it is positive if an increase in the phenotypic value $p(e)$ is advantageous and negative if such a change is unfavourable.

The resulting selection gradient offers two insights. First, it is composed of two qualitatively different selective pressures. One of them points towards the matching phenotype, increasing with the distance between the currently expressed phenotype and the matching phenotype $m(e) - p(e)$ and weighted by the coefficient $w_m(e)$. The other one is directed to the cost-free reaction norm, increasing with the distance between the currently expressed phenotype and the cost-free phenotype $\bar{p} - p(e)$ and weighted by the coefficient $w_{\bar{p}}(e)$. This result confirms the insight, obtained from the invasion fitness, that the evolution of reaction norms depends on a balance between increasing competitive ability and/or the amount of accessible resources via an increase of plasticity on the one hand, and decreasing the costs of plasticity via a decrease in plasticity on the other. The second interesting point is that evolution in one environmental type is not independent from evolution in the others. This is because the cost-free reaction norm is the mean phenotype along the reaction norm as defined in eqn (9): $\bar{p} = \int p(e) d_p^{\beta}(e)/|d_p^{\beta}|\,\mathrm{d}e$. This implies that any evolutionary change in the phenotype expressed in a particular environmental type will affect the cost-free reaction norm and thus the evolution of the phenotypes expressed in other environmental types. Costs of plasticity can thus be seen as imposing trade-offs between the phenotypes expressed in different environmental types.

Outcomes of reaction norm evolution: trading perfect matching against cost avoidance

The evolution of reaction norms p eventually stops when the selection gradient g_p vanishes. We refer to these outcomes as evolutionary singularities (Metz *et al*., 1996) and denote them by p^*. If we assume that second-order terms in $p(e)$ around the matching phenotype $m(e)$ and around the cost-free reaction norm $\bar{p}$ are negligible, solutions of $g_{p^*} = 0$ can be determined analytically (see Appendix),

$$p^*(e) = \frac{w_m(e)}{w_m(e) + w_{\bar{p}}(e)} m(e) + \frac{w_{\bar{p}}(e)}{w_m(e) + w_{\bar{p}}(e)} \bar{p}^*, \qquad (16a)$$

where $\bar{p}^*$ is the cost-free reaction norm associated with the singular reaction norm p^* (see the Appendix for a description of how $\bar{p}^*$ is determined without knowing p^*). The coefficients $w_m(e)$ and $w_{\bar{p}}(e)$ are the same as those that weigh the two different selective pressures in the selection gradient, eqn (15b), and are given by

$$w_m(e) = \frac{2(\alpha + 2a) r o(e) s(e)}{k(e)|o/k_{p^*}|} \quad \text{and} \quad w_{\bar{p}}(e) = \frac{2c o^{\beta}(e)}{|o^{\beta}|}, \qquad (16b)$$

where $|o^{\beta}|$ and $|o/k_{p^*}|$, respectively, are defined analogously to the constants $|d_{p^*}^{\beta}|$ and $|d_{p^*}^2/(o k_{p^*})|$ introduced above. Numerical checks for the accuracy of our approximate solution confirm a relative precision of the order of 10^{-4}, whatever the scale of the phenotype p^*.

As already suggested by the selection gradient, eqns (15a) and (15b), the evolutionarily singular reaction norm, eqns (16a) and (16b), results from a balance between two selective forces. The first one is directed to the matching phenotype $m(e)$. It receives an absolute weight $w_m(e)$ and results from the benefits of an increase in efficiency of resource utilization through phenotypic adaptation. This component purely arises from local density- and frequency-dependent selection, because the benefits of a higher efficiency of resource utilization translate into better competitive ability and higher realized carrying capacity. This result highlights how the evolution of phenotypic plasticity can be driven by individual interactions such as intraspecific competition for resources. In contrast, the second component originates from the costs of phenotypic plasticity and pushes the reaction norm towards the cost-free reaction norm $\bar{p}^*$, receiving an absolute weight $w_{\bar{p}}(e)$.

In addition to the local conditions described by $o(e)$, $k(e)$ and $s(e)$, several nonlocal parameters affect the weights of these two components. Without costs of phenotypic plasticity, $c = 0$, the weight $w_{\bar{p}}(e)$ vanishes, resulting in a perfectly matched singular reaction norm, $p^* = m$. Indeed, it can be shown that, under such conditions, evolution stops when evolutionary changes in the reaction norm can no longer improve resource utilization efficiency in any type of environment, $\partial E_p / \partial p|_{p = p^*} = 0$. As the intensity c of costs increases, the singular reaction norm is gradually flattened towards the cost-free reaction norm $\bar{p}^*$ (Fig. 3). Finally, when costs become so large ($c \to \infty$) that the relative weight towards the matching phenotype $m(e)$ vanishes, $w_m/(w_m + w_{\bar{p}}) \to 0$, evolution results in the cost-free reaction norm, $p^* = \bar{p}^*$, i.e. in the removal of all plasticity. In contrast, a strong impact α of the efficiency of resource utilization on the realized carrying capacity or a high degree of competitive asymmetry a both increase the selection pressures arising from the competitive interactions between individuals and, thus, strengthen the component involving the matching phenotype (Fig. 3). Likewise, a high intrinsic population growth rate r enables individuals to withstand higher costs of phenotypic plasticity and therefore also shifts the singular reaction norm towards the matching phenotypes

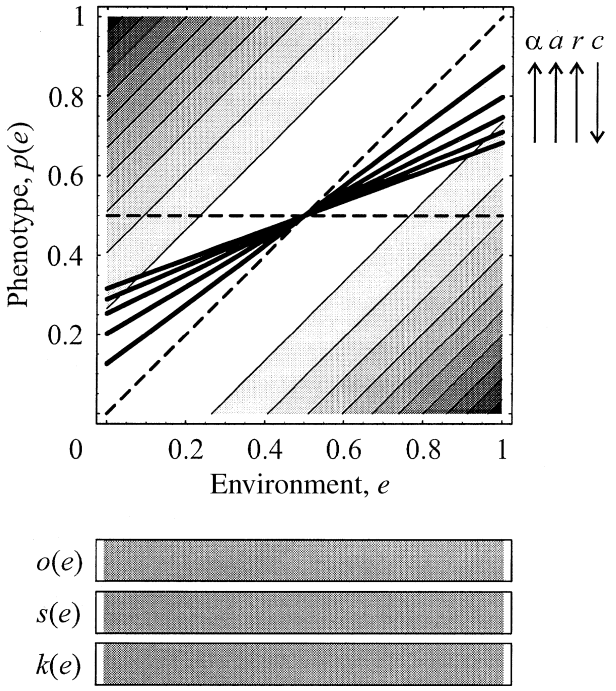

Fig. 3 Effects of global ecological factors. As in all subsequent figures, the three characteristics of environmental heterogeneity are shown by greyscale bars below the upper panel. These bars depict, for each environmental type, its frequency $o(e)$ (top bar), the sensitivity to maladaptation $s(e)$ (middle bar) and its intrinsic carrying capacity $k(e)$ (bottom bar, for all bars black indicates highest values). Here, all three characteristics of environmental heterogeneity are uniform across environmental types, $o(e) = s(e) = k(e) = 1$, and costs of phenotypic plasticity are mixed, $\beta = 0.5$. The upper panel shows that matching of the singular reaction norm (thick continuous lines) increases with three global ecological factors: the dependence of the realized carrying capacity on resource utilization efficiency (α), the asymmetry of competition (a) and the intrinsic growth rate (r). Conversely, for increasing costs of phenotypic plasticity (c) the reaction norm is pushed towards the cost-free phenotype $\bar{p}^*$ (horizontal dashed line).

(Fig. 3). However, if $|o/k_{p^*}|$ (the average proportion of the intrinsic growth rate r lost per encountered competitor) increases, the selection pressure towards the matching phenotype decreases, which flattens the singular reaction norm towards the cost-free reaction norm. Finally, we note that the singular reaction norm p^* is invariant under parameter variations that leave the quantity $(\alpha + 2a)r|o^\beta|/(c|o/k_{p^*}|)$ unchanged. This makes it plain that one cannot infer from a reaction norm observed in the field details of the ecological setting that caused its evolution.

The generality of the result in eqns (16a) and (16b) should be appreciated. First, a huge variety of different ecological settings is described by this result: allowing for variations between plasticity affecting realized carrying capacities and/or competitive abilities (α), competition being symmetric and/or asymmetric (a), and plasticity implying maintenance and/or production costs (β), and, in addition, featuring three freely adjustable characteristics of environmental heterogeneity (o, k, and s). How all these factors jointly determine the shape of evolving reaction norms, by influencing the weights in eqn (16b), is rather complex and could not have been unveiled through less formal reasoning. Secondly, even when other kinds of density or frequency dependence and other ways of quantifying plasticity costs are considered, the form of eqn (16a) stays unchanged (results not shown). This means that also in such generalized situations the evolutionarily singular reaction norm is shaped by the balance between perfect matching and cost avoidance as quantified in eqn (16a). The only requirement is that local density-dependent vital rates must depend on the distance between the expressed and the matching phenotype, and that plasticity costs must depend on the distance between the expressed and the cost-free phenotype. Then, only the weights $w_m(e)$ and $w_{\bar{p}}(e)$, eqn (16b), change according to the specific functions used for describing these two dependences.

Maintenance and production costs of phenotypic plasticity: are evolutionarily singular reaction norms better matched to frequent environmental types?

The selection pressures induced by maintenance and production costs of phenotypic plasticity, $co^\beta(e)[\bar{p} - p(e)]$ [see Appendix and eqn (15b)], sharply differ in their dependence on the frequency distribution o of the different environmental types. For maintenance costs, $\beta = 0$, the selection pressure does not involve this frequency distribution and only depends on the distance between the expressed phenotype and the cost-free reaction norm, then simply given by the absolute mean of phenotypes, $\bar{p} = \int p(e)\,\mathrm{d}e$. In contrast, for production costs, $\beta = 1$, the selection pressure is directly proportional to the frequency of occurrence of environmental types, $co(e)[\bar{p} - p(e)]$, and also indirectly depends on that frequency through the cost-free reaction norm, which is then the mean 'realized' phenotype, $\bar{p} = \int p(e)o(e)\,\mathrm{d}e$. The direct effect reflects the simple fact that the costs associated with the production of a phenotype $p(e)$ are paid as frequently as this phenotype is produced. The interpretation of the indirect effect is subtler. Due to the fact that we defined the cost-free reaction norm as the one that minimizes the costs of phenotypic plasticity, it is adjusted such that it balances the direct effect. Production costs associated with a frequent phenotype $p(e)$ are paid frequently (direct effect), but at the same time, the phenotype $p(e)$ receives a high weight $o(e)$ in determining $\bar{p}$. This shifts the cost-free reaction norm $\bar{p}$ closer to that phenotype, reducing the difference $p(e) - \bar{p}$ and, accordingly, diminishing the selective pressure towards $\bar{p}$. This finding is in accordance with our interpretation of the cost-free reaction norm as a developmental property of plastic organisms: we would

expect development to evolve such as to produce a cost-free reaction norm which is close to the phenotypes most commonly produced.

Of course, these differences in selection pressures have repercussions for the singular reaction norm, eqns (16a) and (16b). For pure maintenance costs, $\beta = 0$, the frequency distribution o of environmental types only affects the component directed at the matching phenotype $m(e)$. Consequently, the singular reaction norm is better matched in the environmental types that are frequent. By contrast, for pure production costs, $\beta = 1$, the frequency distribution of environmental types is involved in both components, the one pointing towards the matching phenotypes and the one pointing towards the cost-free reaction norm, in such a way that the two effects cancel. In that case, the reaction norm is no longer shaped by the frequencies of the different environmental types, and frequent and infrequent environmental types have an equivalent impact on its evolution. To illustrate this effect, we present in Fig. 4 different singular reaction norms evolving in an environmental setting where environmental types have a nonuniform frequency of occurrence (intermediate types are more frequent) for β varying between 0 (pure maintenance costs) and 1 (pure production costs). In the former case, a closely matched phenotype evolves for intermediate environmental types, as these are frequent, whereas obviously no net fitness advantage is to be gained by adjusting the expressed phenotype in extreme types, as these are rare. As β increases and costs of plasticity thus become mixed, the reaction norm straightens and the effect of the frequency of environmental types on the shape of the reaction norm dwindles. For pure production costs, the frequency of occurrence of environmental types no longer shapes the reaction norm. In particular, infrequent types also induce adjustment of the expressed phenotype.

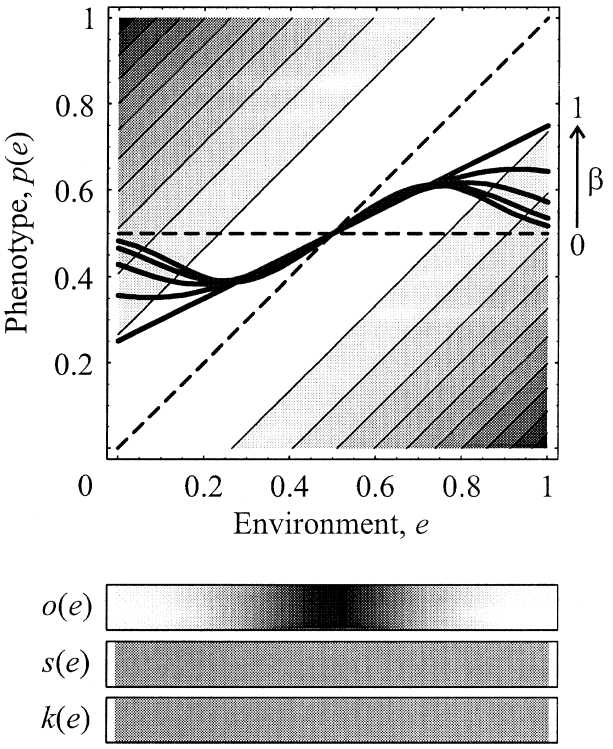

Fig. 4 Interaction between the type of costs of phenotypic plasticity and the frequency of environmental types. Here, as for the subsequent examples, the ratio r/c, the asymmetry of competition a, and the exponent α are all kept at 1. In this example, the sensitivity to maladaptation and the intrinsic carrying capacity are uniform, $s(e) = k(e) = 1$. We assume that intermediate environmental types are more frequent, such that the frequency of environmental types follow a normal distribution peaked at $e = 0.5$, $o = N(0.5, 0.2)$. In the upper panel, five evolutionarily singular reaction norms are shown, corresponding to costs of phenotypic plasticity varying from pure maintenance costs, $\beta = 0$, to pure production costs, $\beta = 1$, with steps of 0.25 in between.

Struggle for life: better matching under high sensitivity to phenotypic maladaptation and in environments with low intrinsic carrying capacity

In contrast with the frequency distribution, the impact of the sensitivity to phenotypic maladaptation $s(e)$ and of the intrinsic carrying capacity $k(e)$, representing the quality or quantity of resources in the different environmental types, do not depend on the type of cost of phenotypic plasticity.

A high sensitivity to phenotypic maladaptation $s(e)$ favours the evolution of a better matching in environmental type e, eqns (16a) and (16b). Indeed, when $s(e)$ is high, the costs of imperfect matching, $p(e) \neq m(e)$, in terms of lost resource utilization efficiency increase, which of course strengthens the selection pressure towards the matching phenotype. Figure 5 shows the evolutionarily singular reaction norm evolving in an environmental setting where the sensitivity of maladaptation varies according to environmental types (sensitivity increases with environmental types). For environmental types on the far left, sensitivity is weak and, consequently, the reaction norm evolves so as to lie close to the cost-free reaction norm $\bar{p}^*$. For environmental types more towards the right, the sensitivity to maladaptation increases significantly, which is illustrated by the narrowing of the isoclines of resource utilization efficiency (depicted by different shades of grey) that are shaped by the matching phenotype and the sensitivity to maladaptation. In this range of environmental types, the reaction norm is moulded on these isoclines and lies closer and closer to the matching phenotype. Indeed, as sensitivity to maladaptation increases, it becomes more and more critical to express a matched phenotype, as the costs of maladaptation in terms of lost efficiency of resource utilization increase.

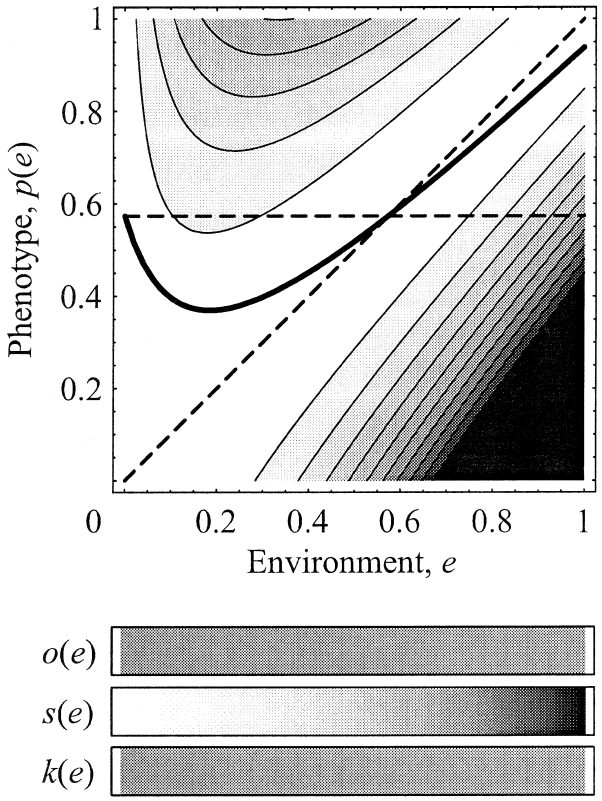

Fig. 5 Effect of the sensitivity to maladaptation. In this example, the intrinsic carrying capacity and the frequency of environmental types are uniform, $k(e) = o(e) = 1$, and costs of phenotypic plasticity are mixed, $\beta = 0.5$. We assume that the sensitivity to maladaptation increases with environmental type e, $s(e) = 2e$.

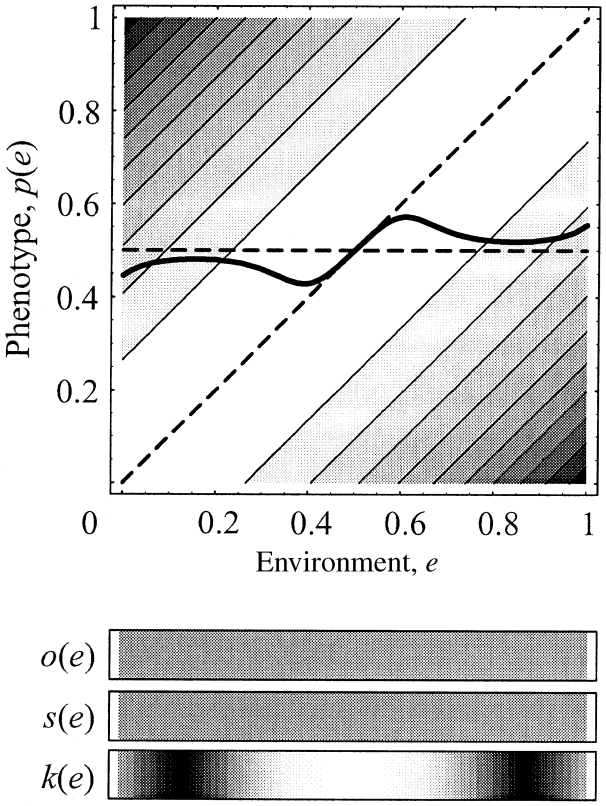

Fig. 6 Effect of differential resource abundance across environmental types. In this example, the sensitivity to maladaptation and the frequency of environmental types are uniform, $s(e) = o(e) = 1$, and costs of phenotypic plasticity are mixed, $\beta = 0.5$. We assume that extreme environmental types offer abundant resources, whereas intermediate ones offer little resources, as described by a bimodal intrinsic carrying capacity, $k = N(0.125, 0.077) + N(0.875, 0.077)$.

Equations (16a) and (16b) also show that the component of the selection pressure directed towards the matching phenotype decreases with the intrinsic carrying capacity of an environmental type, implying that matching will be weak in environmental types with high intrinsic carrying capacity, whereas better matching will evolve in environmental types with low intrinsic carrying capacity. Figure 6 presents the evolutionarily singular reaction norm that evolves in an environmental setting where resource abundance is heterogeneous across environmental types (extreme environmental types to the left and right offer abundant resources, i.e. high intrinsic carrying capacity, whereas intermediate ones offer little resources, i.e. low intrinsic carrying capacity). The evolutionary outcome may then seem surprising: the singular reaction norm lies close to the matching phenotype for environmental types that offer little resources, whereas, for rich environmental types, it lies close to the cost-free reaction norm. This striking result originates from the fact that, in our model, the evolution of reaction norms is driven by competition for resources and that the distribution of individuals across environmental types is random. When the intrinsic carrying capacity is high, competition between individuals is weak so that individuals that express a maladapted phenotype can still acquire some resources. In contrast, when the intrinsic carrying capacity is low, the intensity of competition between individuals increases and, under such extreme conditions, it becomes critical to express a well-adapted phenotype that allows taking advantage of the little resources available. In addition to this, as individuals have no selective control of the environmental type in which they live (random distribution), they cannot avoid or escape from poor environments. They must, therefore, be well adapted to the environmental conditions encountered there in order to ensure a minimal resource intake.

Discussion

The potential advantage of plastic organisms for dealing with environmental heterogeneity has been acknowledged very early (Schmalhausen, 1949; Levins, 1963,

1968; Bradshaw, 1965) and conceptual research to predict the conditions that would favour its evolution began in the 1960s (Bradshaw, 1965; Levins, 1968). Yet, despite considerable attention to the matter during the last two decades, some key issues have remained under-investigated. In this study, we have proposed a model for the long-term evolution of phenotypic plasticity that aimed at extending current knowledge in three particular directions. First, we have focused on the evolution of reaction norms under density dependence and frequency-dependent selection arising from interactions between individuals, like competition for resources. Secondly, to better appreciate the importance of costs of phenotypic plasticity, we have distinguished between maintenance and production costs, showing that these generate very different selective pressures. Finally, we have highlighted the importance of characteristics of environmental heterogeneity by systematically examining the evolutionary impact of the frequency of occurrence of the different environmental types, of the quality and quantity of their resources, and of the sensitivity to maladaptation in these environments.

Individual interactions, frequency-dependent selection and reaction norm evolution

Most previous theoretical studies considered the evolution of phenotypic plasticity for cases in which plastic organisms were not affected by conspecifics or by interactions with other species. Consequently, selective pressures preventing or promoting the evolution of phenotypic plasticity were mainly restricted to those arising from abiotic environmental factors or, when of biotic origin, did not account for the potential impact of plastic organisms on their environment (affecting, for instance, the availability of resources consumed by the plastic organism). However, it is well known that individual interactions can be primary determinants of phenotypic plasticity, and many experimental studies focused on cases of plasticity in which interacting individuals alter their phenotype in response to interactions with other individuals, like, for instance, in the case of competition for light between plants or for predator-induced defence in some animal species. In a salient review, Agrawal (2001) gives an almost exhaustive list of individual interactions that can trigger plastic processes, categorizing them according to five main groups: competition, mutualism, predation (for animals), parasitism/herbivory and food quality (prey items).

Focusing on resource competition, but without being restricted to it (see last section of the Discussion), our study emphasizes how such interactions between individuals can drive the evolution of phenotypic plasticity and mould the shape of the resulting reaction norms. Including individual interactions greatly enhances the realism and scope of models for the evolution of phenotypic plasticity. The reason why earlier models of reaction norm evolution did not focus on such interactions is two-fold. First, optimality models cannot account for the nonlinear population dynamics typically arising from individual interactions involving frequency-dependent selection, thus excluding cases in which selective pressures depend on the phenotype of the other individuals present (Meszéna *et al.*, 2001). Secondly, quantitative genetics studies neglected the ecological complexity arising from intraspecific interactions by focusing on analyses of genetic effects affecting the evolution of phenotypic plasticity (Pigliucci & Schlichting, 1997). One exception is the model by Sasaki & de Jong (1999) which was used to investigate the effects of density dependence on the evolution of phenotypic plasticity. Yet, this model did not account for frequency-dependent selection (Heino *et al.*, 1998), as the interactions between individuals were not influenced by their expressed phenotype, but only by the total number of competitors.

Costs of phenotypic plasticity and their interaction with the frequency of environmental types

Although costs of phenotypic plasticity are frequently mentioned as potential constraints for the evolution of phenotypic plasticity (e.g. Via & Lande, 1985; Schlichting, 1986; Gomulkiewicz & Kirkpatrick, 1992; Scheiner, 1993; Pigliucci, 1996; DeWitt *et al.*, 1998), only three theoretical studies have examined some of their evolutionary consequences (van Tienderen, 1991; Moran, 1992; Léon, 1993), and experimental attempts to evaluate these costs in animals (DeWitt, 1998; Scheiner & Berrigan, 1998; Relyea, 2002) and in plants (Tucic *et al.*, 1998; Donohue *et al.*, 2000; Dorn *et al.*, 2000; van Kleunen *et al.*, 2000; Agrawal *et al.*, 2002; Steinger *et al.*, 2003) have been undertaken only very recently. The general importance of plasticity costs remains to be determined, as the three studies on animals found no or almost no costs of plasticity, whereas four of six experiments on plants found unequivocal evidence for such costs, and a fifth one found weaker evidence. However, all these studies concentrated on only two very specific kinds of phenotypic plasticity: all animal studies dealt with predator-induced defence and five of six experiments on plants dealt with shade avoidance in plants. This narrow focus makes any general conclusions uncertain. Accordingly, we suggest that the range of plastic phenomena experimentally investigated so far is currently not wide enough to corroborate or refute the ubiquity of costs of plasticity in nature.

Including plasticity costs in our model, the reaction norm evolves as a compromise between a perfectly matched reaction norm and a flat reaction norm located at the cost-free phenotype. These results extend those of van Tienderen (1991), who, focusing on plasticity maintenance costs and on organisms facing two discrete environments, gained the same qualitative insight.

Elaborating on van Tienderen's findings, we distinguished between plasticity maintenance and production costs (DeWitt *et al.*, 1998). Maintenance costs are defined as expenses for maintaining the potential for being plastic and thus are independent of the frequency at which different phenotypes are expressed. In contrast, plasticity production costs are the production costs that a plastic genotype producing a given phenotype pays in excess to the direct production costs incurred by a fixed genotype producing the same phenotype. This difference in the nature of costs critically affects whether the frequency distribution of environmental types shapes the singular reaction norm. Maintenance costs allow the frequency at which the different types of environment occur to mould the reaction norm, with better matching arising in frequent environmental types, whereas production costs do not. This latter result is rather counterintuitive. All previous studies accounting for the frequency distribution of environmental types predicted better adaptation in frequent environments (e.g. Via & Lande, 1985; van Tienderen, 1991; Kawecki & Stearns, 1993; Zhivotovsky *et al.*, 1996). However, these investigations did not account for production costs. As production costs are incurred as often as a given phenotype is produced, they actually balance the increased selective pressure for adaptation to frequent environments.

Heterogeneity in sensitivity to maladaptation and resource abundance

Sensitivity to maladaptation can be seen as capturing the strengths of local selective pressures pointing towards the matching phenotype in the different environmental types, thus evidently resulting in better matching in environmental types where this sensitivity is high. Sensitivity to maladaptation is therefore equivalent to the local sensitivity of fitness (Charlesworth, 1980; Caswell, 1989) or the strength of selection as used in quantitative genetics models (Via & Lande, 1985; van Tienderen, 1991, 1997; Gomulkiewicz & Kirkpatrick, 1992); it is also similar to the intensity of within-niche selection in the model by Zhivotovsky *et al.* (1996). Of course, in models detailing the ecology of the organisms considered in each environmental type, the sensitivity function should be directly derived from the underlying ecology, rather than given *a priori* as in the present study.

A more striking result is that reaction norms evolve better matching for environments that are poor in resources, i.e. exhibit low intrinsic carrying capacity. This finding agrees with conclusions drawn by Sasaki & de Jong (1999), who found better adaptation under hard selection in low-productivity (sink) environments. By contrast, Kawecki & Stearns (1993) and Zhivotovsky *et al.* (1996) predicted better matching to be favoured in rich environments. This originates from the fact that, in the models by Kawecki & Stearns, and by Zhivotovsky *et al.*, the amount of accessible resource is independent of the plastic traits. Then, rich environments simply produce more individuals than poor ones, which in turn promote the evolution of better adaptation to the rich ones. However, in our model, the plastic trait also determines how much of the intrinsically available resources are accessible to competitors by determining their realized carrying capacity, as well as their competitive ability in pairwise contests. The selective pressure to maximize realized carrying capacity and competitive ability is obviously much higher in extreme environments with low intrinsic carrying capacity than in those with high intrinsic carrying capacity, which eventually leads to better adaptation to the former.

Assumptions of the model and promising directions for future research

Several assumptions of the model are worth highlighting and discussing. We traded off genetic details against ecological realism. Therefore, genetic constraints that could prevent the reaction norm from reaching the equilibrium as determined by the selection gradient, like the lack of additive genetic variance or genetic correlations reflecting trade-offs between the reaction norm and other life history traits, are not considered. However, costs of phenotypic plasticity as we defined them can be seen as including a trade-off across phenotypes expressed in different environmental types, which critically constrains the evolution of reaction norms. The cost-free reaction norm in our model corresponds to a nonplastic organism expressing a fixed phenotype, and can be regarded as a developmental property of the organism: it is the phenotype plastic genotypes can reach through cost-free development. We defined this cost-free phenotype as the mean phenotypic value along the reaction norm. This definition implies that the evolution of different points of the reaction norm is coupled: an evolutionary change in the phenotype expressed in a particular environment changes the cost-free phenotype and therefore has repercussions for the evolution of phenotypes expressed in other environmental types. This reflects the widespread idea that no organism is able to achieve the best possible adaptation in every environment and that a plastic organism is therefore like a 'jack of all trades but master of none' (Levins, 1968; Via & Lande, 1985; van Tienderen, 1991; Gomulkiewicz & Kirkpatrick, 1992). Of course, the cost-free reaction norm could be set to any other arbitrary value according to the specific eco-evolutionary problem at hand, but we were interested here in the general evolutionary emergence of phenotypic plasticity relative to fixed development.

Our focus on resource competition and the assumption of quadratic plasticity costs in our model are not critical for the qualitative results. As a matter of fact, these stay qualitatively unchanged whatever the density dependence and the cost function used, as long as these depend on the distance between the expressed and the

matching phenotype and between the expressed and the cost-free phenotype, respectively. This means, in particular, that the qualitative insights obtained are valid not only for types of resource competition other than Lotka–Volterra competition, but also for other types of individual interactions such as predation or sexual selection.

The two most surprising results of our study critically depend on the assumption that individuals distribute randomly across environments. First, production and maintenance costs differ in whether they are weighted by the frequency at which the different phenotypes are expressed. As individuals are randomly distributed, this leads to an interaction between the types of cost of phenotypic plasticity and the frequency of environmental types. Secondly, better adaptation evolves in environments with low intrinsic carrying capacity, where competition pressure is stronger, because individuals cannot avoid these environments. Random distribution, however, only applies to a certain range of organisms: examples are sessile organisms, like plants or bivalve molluscs, or organisms that have no or limited skills for selecting their habitat. In contrast, many other living beings exhibit more elaborate behaviour (like foraging, habitat selection, or selective dispersal) that are bound to change the frequency at which the different environmental types are encountered and, therefore, modify the evolutionary outcomes predicted in this study. We can think, for instance, of organisms that are able to assess the availability of resources across environments and that preferentially settle in environments with high intrinsic carrying capacity. In this case, preliminary results show that individuals evolve better adaptation for rich environments. This illustrates, as has been suggested recently (Zhivotovsky *et al.*, 1996; Scheiner, 1998), that the distribution strategy of individuals may have important repercussions for the evolution of reaction norms. A further possibility arises when distribution behaviour and reaction norm evolve jointly. For instance, individual behaviour could evolve from random distribution to more sophisticated mechanisms of habitat selection so as to avoid poor environments, allowing, in turn, the reaction norm to evolve from better adaptation to environments with low intrinsic carrying capacity to better adaptation to environments with high intrinsic carrying capacity. The consideration of joint evolution of phenotypic plasticity and distribution strategy therefore opens up exciting avenues for understanding the adaptation of organisms to heterogeneous environments. We will analyse these extensions in a subsequent study.

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Appendix

Selection gradient

Applying the definition of a functional derivative, eqn (15a), to the invasion fitness given by eqn (13), we obtain the selection gradient. It results from a selection pressure $w_m(e)[m(e) - p(e)]$ related to local adaptation and a selection pressure $w_{\bar{p}}(e)[\bar{p} - p(e)]$ related to the costs of phenotypic plasticity,

$$g_p(e) = Df_p(e) = w_m(e)[m(e) - p(e)] + w_{\bar{p}}(e)[\bar{p} - p(e)],$$

with

$$w_m(e) = 2\frac{r - C_p}{|o/k_p|}\ \frac{o(e)s(e)}{k(e)}\left[\frac{\alpha}{E_p^{\alpha+1}(e)} + \frac{2a}{E_p^{\alpha}(e)}\right],$$

and

$$w_{\bar{p}}(e) = 2c\frac{o^{\beta}(e)}{|o^{\beta}|}.$$

Approximations

The singular reaction norm p^* is determined by setting the selection gradient equal to zero, $g_{p^*} = 0$. This equation can be solved analytically if we neglect second-order terms in $p(e)$ around $m(e)$ and $\bar{p}$. We thus obtain the approximated selection gradient,

$$g_p(e) = \frac{2r(\alpha + 2a)}{|o/k_p|}\ \frac{o(e)s(e)}{k(e)}\left[m(e) - p(e)\right] + 2c\frac{o^{\beta}(e)}{|o^{\beta}|}\left[\bar{p} - p(e)\right].$$

Computation of the constants

With

$$I_1 = \int \frac{w_m(e)o^{\beta}(e)m(e)}{w_m(e) + w_{\bar{p}}(e)}\,\mathrm{d}e,$$

$$I_2 = \int \frac{w_{\bar{p}}(e)o^{\beta}(e)}{w_m(e) + w_{\bar{p}}(e)}\,\mathrm{d}e,$$

and with p^* given by eqns (16a) and (16b), $\bar{p}^*$ and $|o/k_{p^*}|$ are determined numerically by solving the following system of equations,

$$\bar{p}^* = \frac{I_1}{|o^{\beta}| - I_2} \quad \text{and} \quad |o/k_{p^*}| = \int \frac{o(e)}{k(e)E_{p^*}^{\alpha}(e)}\,\mathrm{d}e.$$

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