

The Evolution of Plasticity and Nonplastic Spatial and Temporal Adaptations in the Presence of Imperfect Environmental Cues

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ABSTRACT: A model for the evolution of plasticity is considered in which the phenotype, undergoing stabilizing selection, is modeled as a linear function of an environmental cue correlated with the phenotypic optimum, with the coefficients z_0 and z_1 evolving according to standard quantitative genetic theory. In contrast to previous theoretical models, as the rate of migration between demes or the rate of cyclic fluctuations in the optimum increases, the amount of plasticity $\bar{z}_1$ at equilibrium is shown to increase gradually, in part accounting for the effect of reduced nonplastic adaptation and reaching a maximum equal to the squared correlation between the environmental cue and the phenotypic optimum. Given that information available to the organism is limited, this bias of the expressed phenotype toward the global optimum is still optimal, however, in a certain decision-theoretic sense. When genetic variation in the plastic component of the trait is small so that spatial or temporal differentiation in plasticity is small, the effect of plasticity on nonplastic adaptation is to reduce the effects of variation in the phenotypic optimum by a factor $1 - \bar{z}_1$ only. Information acquisition costs and joint evolution of sensory systems are discussed.

Keywords: plasticity, quantitative genetics, spatial variation, temporal variation, environmental cues, sensory systems.

Spatial variation in selection pressures over small geographic scales or a too-high rate of gene flow can prevent local adaptation from occurring (Slatkin 1985; Barton 1999). Similarly, if temporal fluctuations in selection pressures are too fast, a population will, on average, deviate substantially from and lag behind the optimal phenotype (Lande and Shannon 1996). Phenotypic plasticity has been proposed as a mechanism by which adaptation to variable

environments in space and time can still occur by conditional development of the phenotype in different environments. The norm of reaction of a certain genotype is the relationship between the expressed average phenotype that the genotype would express across a number of environments.

Empirical evidence for adaptive genetic geographic differentiation in many life-history traits (Stearns 1992), as well as adaptive plastic responses (Travis 1994; Via et al. 1995) to different environmental conditions, exist for many species. This suggests that many adaptations in nature, in general, have both plastic and nonplastic components. The joint evolution of plastic and nonplastic adaptations is not well understood, however, but has instead mostly been treated as separate problems. Given that enough genetic variation in plasticity exists, previous theoretical models (Via and Lande 1985; van Tienderen 1997) have predicted that the trait means expressed in different environments will evolve to match completely local optima and that all genetic differentiation between subpopulations will be lost, even when gene flow is limited. To explain the discrepancy between these predictions and empirical data, it has been suggested that plasticity may be slow to evolve because genetic variation in plasticity may be low and that the predicted equilibrium of complete phenotype-environment matching, therefore, is not attained in nature. Another suggestion has been that genetic variation in plasticity may become exhausted, preventing full plasticity from evolving (Via and Lande 1985). However, genotype-environment interactions, equivalent to genetic variation in plasticity, are found in many populations (Falconer 1981).

Other authors have suggested that some cost of plasticity must exist. This implies that an individual with a high degree of plasticity, which happens to express the same phenotype as some other nonplastic individual, on average, experiences a reduction in fitness (van Tienderen 1997). Such costs may be related to the maintenance of plasticity and to the ability to produce different pheno-

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types, information acquisition costs, and costs arising from a higher degree of developmental instability in more plastic individuals. Little empirical evidence for such costs have yet emerged, however (DeWitt 1998; DeWitt et al. 1998; Scheiner and Berrigan 1998).

As noted by Moran (1992), in all organisms, the development of the phenotype does not occur instantaneously but extends over a certain period of time, during which the surrounding environment changes in part in an unpredictable manner. During the same period, the organism is also exposed to episodes of selection. It follows that only the state of the environment at some period in time before major episodes of selection can be important for evolution of plasticity. At this time, some feature of the environmental state picked up by the organism, an environmental cue, determining its further developmental pathway, will never be perfectly correlated with the future selective environment. As pointed out first by Moran (1992) and later by Getty (1996), Gregorius (1997), and de Jong (1999), this has fundamental implications for the evolution of plasticity. Moran (1992) considered a model with two environments, each with its own optimal phenotype, and showed that imperfect environmental cues in contrast to Via and Lande's (1985) model, leads to a reduction in mean fitness of plastic genotypes and only partial phenotype-environment matching.

An important consequence of partial phenotype-environment matching as a result of either costs or cue unreliability is that selection pressures will remain, generating spatial or temporal nonplastic genetic adaptations. Because the level of these two types of adaptations are mutually dependent, the evolution of plasticity and nonplastic differentiation, therefore, in general, have to be understood jointly. This problem, however, has only received limited attention. Lively (1986), using a simple two-patch evolutionarily stable strategy model with discrete morphs, concluded that a mixture of morphs with environmentally and genetically induced development can coexist, given that cues are sufficiently reliable. Scheiner (1998), based on a quantitative genetic simulation model, concluded that a mixed evolutionary outcome with both plastic and nonplastic adaptation will result, even with no apparent explicit costs or cue unreliability. However, as noted by de Jong (1999), because individuals migrate during development in the model of Scheiner (1998), this in effect leads to a form of cue unreliability. More generally, however, as I will argue here, cue unreliability can arise simply from, for example, lack of information in the environment surrounding the organism at the beginning of its development and need not involve any migration.

Here, I extend the conceptual framework introduced by Moran (1992) and develop a quantitative genetic model for the evolution of plasticity under stabilizing selection.

The model, similar to that of Scheiner (1998), encompasses both nonplastic adaptation to either spatial or temporal environmental variability and evolution of plasticity. Fitness depends on the expressed phenotype only, that is, no intrinsic costs of plasticity are assumed. Several new simple analytic quantitative results, extending those of Scheiner (1998) and de Jong (1999), are derived, showing how much plastic and nonplastic adaptation evolves for the general case of intermediate rates of migration or intermediate rates of temporal fluctuations in the phenotypic optimum. It is also shown how the evolution of reaction norms relates to standard decision theory in the presence of uncertainty (e.g., Berger 1985) and possible extensions of the model involving coevolution of sensory systems are discussed.

The Model

The following model is considered. The state of the population in a single deme is given by distribution of two quantitative traits, z_0 and z_1 . At some early developmental stage, each individual obtains an environmental cue, $\hat{\theta}$, from its surrounding environment. The cues obtained by different individuals are assumed to be stochastically independent, conditional on the phenotypic optimum θ in the deme and have variance $\text{var}(\hat{\theta} | \theta) = \sigma_{\hat{\theta}}^2$. This variance may be a result of the fact that information available in the surrounding environment may be limited but may also be as a result of inefficiencies in the sensory system used by the organism in interpreting available information. In addition, as noted by de Jong (1999), this variance may arise from migration between the different "microhabitats" of development and selection. Without loss of generality, based on a simple scaling argument, it follows that we can assume that the cues are unbiased, that is, $E(\hat{\theta}) = \theta$. The phenotypes of each individual is given by

$$P = z_0 + z_1 \hat{\theta} + E, \quad (1)$$

where E denotes random effects of the environment not interacting with z_1 . The above first-order polynomial representation of the character state in the presence of different environmental cues $\hat{\theta}$ is, as pointed out by de Jong (1995) and Via et al. (1995), equivalent to representing the character state in different environments as separate traits, as in Via and Lande (1985). It must be noted, however, that the phenotype P of some individual, given $\hat{\theta}$, is independent of the actual state of the selective optimum θ in the deme (which is unknown to each individual). This is in contrast to the models of, for example, Via and Lande (1985), Gomulkiewicz and Kirkpatrick (1992), and de Jong

(1995), where the expressed phenotype is implicitly assumed to depend directly on the local optimum.

Fitness depends on individual phenotypes only and falls off quadratically from the optimum:

$$w(P) = 1 - \frac{s}{2}(P - \theta)^2. \quad (2)$$

Assuming that selection and other evolutionary forces are weak and that a large number of loci influence the trait (Bulmer 1980), single demes will remain approximately in linkage equilibrium and the genetic variances σ_0^2 and σ_1^2 , and the covariance between z_0 and z_1 will remain approximately constant. The state of the population is then completely characterized by the means $\bar{z}_0$ and $\bar{z}_1$. In what follows, for mathematical convenience, it will be assumed that the genetic covariance between z_0 and z_1 is 0. This may seem like a strong assumption; individual genes invariably have pleiotropic effects on a large number of traits to a large or small extent (Lande 1982), resulting in genetic covariance between different traits. Summing variances and covariances of effects on z_0 and z_1 over individual loci, however, covariances contributed by individual loci will, for appropriate transformations of the axis system, to some extent average out. Note that, for example, in Via and Lande's (1985) representation—a simple linear transformation of the representation used here—the assumption of zero covariance translates to equal genetic variances of the genotypic values expressed in each environment of their two-deme model (corresponding to, say, $\hat{\theta} = \pm 1$), and $\sigma_0^2 > \sigma_1^2$ translates to a positive covariance between the character state expressed in each deme.

Substituting (1) into (2) and taking expectations with respect to z_0 , z_1 , θ , and E , we find that the mean fitness for a given optimum θ is given by

$$\begin{aligned} \bar{w}(\bar{z}_0, \bar{z}_1) = 1 - \frac{s}{2} [(\bar{z}_0 + z_1\theta - \theta)^2 + \sigma_0^2 \\ + \sigma_1^2(\theta^2 + \sigma_\theta^2) + \bar{z}_1^2\sigma_\theta^2 + \sigma_E^2], \end{aligned} \quad (3)$$

where σ_E^2 denotes the variance of environmental effects E .

If selection is weak, the rate of evolution in $\bar{z} = (\bar{z}_0, \bar{z}_1)$ in continuous time is in general given by

$$\frac{d\bar{z}}{dt} = \mathbf{G}\nabla\bar{w}, \quad (4)$$

where $\mathbf{G}$ is the genetic variance-covariance matrix and $\nabla\bar{w} = (d\bar{w}/d\bar{z}_0, d\bar{w}/d\bar{z}_1)^T$ (Lande 1982, eq. [11a]). Using the assumption that there is no covariance between z_0 and z_1 , and differentiating (3) with respect to $\bar{z}_0$ and $\bar{z}_1$, we find that the rate of evolution in each trait is given by

$$\frac{d\bar{z}_0}{dt} = -s\sigma_0^2(\bar{z}_0 + \bar{z}_1\theta - \theta), \quad (5)$$

$$\frac{d\bar{z}_1}{dt} = -s\sigma_1^2[\theta(\bar{z}_0 + \bar{z}_1\theta - \theta) + \sigma_\theta^2\bar{z}_1]. \quad (6)$$

If stabilizing selection is the only evolutionary force acting, and there is no temporal variation in θ , it follows that $\bar{z}_0$ and $\bar{z}_1$ will evolve to the optimum:

$$\bar{z}_0 = \theta, \bar{z}_1 = 0, \quad (7)$$

obtained by setting the left-hand side of (5) and (6) equal to 0.

Thus, in a completely isolated deme with no temporal variation in the optimum, stabilizing selection on the phenotype and cue unreliability leads to evolution against plasticity and evolution toward local adaptation.

Spatial Variation

With spatial variation in the optimum θ and migration between demes, migration will locally pull the means in some particular deme i , $\bar{z}_{0i}$ and $\bar{z}_{1i}$, toward the mean of the immigrants and will prevent evolution to the above optimum (7) from occurring. Here, I assume that the pattern of migration takes the form of Wright's (1943) island model, that is, the total population consists of a large number of demes $i = 1, 2, \dots, \infty$ and migration occurs via one common migrant pool so that immigrants entering different demes all have the same mean. Let $\bar{\bar{z}}_0$ and $\bar{\bar{z}}_1$ denote the global traits means across all demes when $\bar{z}_{0i}$ and $\bar{z}_{1i}$ in all demes are in balance between migration and stabilizing selection. Let θ_i denote the optimum in deme i and assume that the distribution of θ_i across demes is symmetric about 0. From this symmetry, it follows that $\bar{\bar{z}}_0 = 0$.

Mixing of different populations by migration will, in general, generate linkage disequilibrium and inflate the local genetic variances (and covariances), but this effect will be small provided that migration is slow relative to the rate of recombination (Via and Lande 1985; J. Tufto, unpublished manuscript). The rate of change in the trait means in a particular deme i with optimum θ_i is then given by

$$\frac{d\bar{z}_{0i}}{dt} = -m(\bar{z}_{0i} - 0) - s\sigma_0^2[\bar{z}_{0i} - (1 - \bar{z}_{1i})\theta_i], \quad (8)$$

$$\frac{d\bar{z}_{1i}}{dt} = -m(\bar{z}_{1i} - \bar{\bar{z}}_1) - s\sigma_1^2[\theta_i(\bar{z}_{0i} + \bar{z}_{1i}\theta_i - \theta_i) + \sigma_\theta^2\bar{z}_{1i}]. \quad (9)$$

The equilibrium solution for $\bar{z}_{0i}$ and $\bar{z}_{1i}$ in some deme i

and for the global mean plasticity $\bar{z}_1$, accurate as long as the variance in the optimum θ_i between demes, σ_θ^2 , or genetic differentiation in plasticity between demes is small, is given in appendix A. A plot of the solution for an example set of parameter values is shown in figure 1. As shown also by Scheiner (1998), a single level of plasticity does not evolve. Instead, more plasticity evolves in the more extreme demes for large positive or negative values of θ_i . The nonplastic component of the trait follows the phenotypic optimum but with a much shallower slope, also leveling off in the more extreme and plastic demes. The mean expressed phenotype, however, closely tracks the phenotypic optimum with only a slight difference in slope.

It will also be helpful to look at some analytic solutions in a number of limiting cases. These solutions can be expressed as functions of the compound parameters

$$\mu = \frac{m}{s\sigma_0^2}, \quad (10)$$

and

$$\gamma = \frac{\sigma_1^2}{\sigma_0^2}, \quad (11)$$

the rate of migration relative to the rate of response to selection in $\bar{z}_{0i}$ and the genetic variance in z_{1i} , the plasticity, relative to the genetic variance in z_{0i} , respectively.

First, for small μ ($m \ll s\sigma_0^2$), $\bar{z}_{0i}$ (A4) simplifies to

$$\bar{z}_{0i} \approx \theta_i \left[1 - \left(\frac{\theta_i^2}{\sigma_\theta^2} + 1 \right) \mu \right]. \quad (12)$$

Thus, for no migration, $\bar{z}_{0i}$ is at first at the optimum θ_i but is then pulled away from the equilibrium as μ increases. This effect is counteracted by an increase in plasticity with increasing migration, which, again, for small μ , evolves to

$$\bar{z}_{1i} \approx \frac{\theta_i^2}{\sigma_\theta^2} \mu. \quad (13)$$

It may be noted that more plasticity evolves in the more extreme demes; that is, $\bar{z}_{1i}$ reaches higher values in demes with large negative or positive θ_i , a result also shown by Scheiner (1998) using simulations.

The increase in plasticity (13) is not sufficient to counteract fully the effect of reduced local nonplastic adaptation, however, which can be seen by evaluating the expectation of the phenotype in deme i , P_i . In a deme with optimum θ , this expectation is

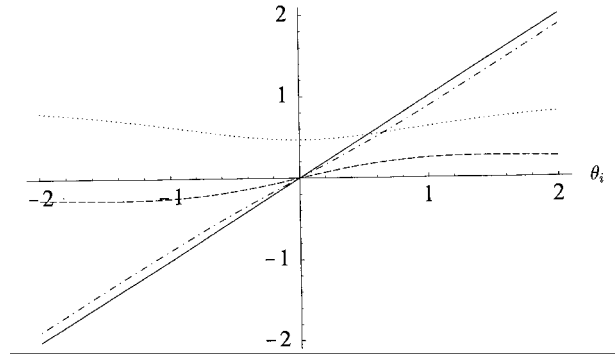


Figure 1: Numerical example of the approximate solution (A4) and (A5) under the island model form of spatial variation, with $\mu = m/(s\sigma_0^2) = 0.5$, $\gamma = \sigma_1^2/\sigma_0^2 = 1$, $\sigma_\theta = 0.5$, and $\sigma_\theta = 1$. Each curve represents the equilibrium state in different demes as a function of different local optima θ_i . For these parameter values, only a small amount of genetic differentiation in the nonplastic component of the trait $\bar{z}_{0i}$ (dashed line) evolves. Instead, the plasticity $\bar{z}_{1i}$ (dotted line) evolves to a value of about 0.7 with a slight drop in plasticity around $\theta = 0$ and a slight increase in plasticity in extreme demes (with large negative or positive values of θ). This allows the expected phenotype $E(P_i) = \bar{z}_{0i} + \bar{z}_{1i}\theta_i$ (dashed-dotted line) to closely track the spatial variation in optimum θ_i (solid line).

$$E(P_i) = \bar{z}_{0i} + \bar{z}_{1i}\theta_i \approx \theta_i(1 - \mu), \quad (14)$$

that is, $E(P_i)$ is pulled away from the local to the global optimum $E(\theta_i) = 0$ by a factor depending on the rate of migration relative to the rate of response to selection. For small μ , this decrease in “total” local adaptation is independent of the variance of environmental cues, σ_θ^2 .

It must be noted that if the environmental cue is perfectly correlated with the environmental optimum ($\sigma_\theta^2 = 0$), then approximation (14) based on (12) and (13) no longer holds. Instead, all genetic differentiation is lost and the equilibrium $\bar{z}_{0i} = 0$ and $\bar{z}_{1i} = 1$ evolves in all demes, with perfect matching between the phenotype and the optimum ($E(P_i) = \theta_i$), as in Via and Lande (1985).

As shown by consideration of the limiting case of a small rate of migration (13) and the general solution (fig. 1), the amount of plasticity that eventually evolves in each deme, $\bar{z}_{1i}$ becomes a function of the local optimum θ_i , that is, a single value of $\bar{z}_{1i}$ maintained in the whole population does not exist. If there is a limited amount of genetic variance in plasticity, however, ($\gamma = \sigma_1^2/\sigma_0^2$ small), migration will, once an equilibrium eventually is reached, prevent the above adaptive dependency on θ from evolving. This can be seen by taking the limit $\gamma \rightarrow 0$ of expression (A5), which becomes independent of θ_i and simplifies to

$$\bar{z}_{1i} = \bar{z}_1 = \frac{\sigma_\theta^2}{(1 + 1/\mu)\sigma_\theta^2 + \sigma_\theta^2}. \quad (15)$$

In the same limit, the nonplastic component (A4) can be written

$$\bar{z}_{0i} = \frac{\theta_i}{1 + \mu} (1 - \bar{z}_1). \quad (16)$$

From this expression, we see that the presence of plasticity in this limit has a particularly simple effect on the nonplastic component of the trait in that it reduces genetic differentiation in $\bar{z}_{0i}$ from the case without any plasticity (the fraction in the above expression) by a factor $1 - \bar{z}_1$. This can also be seen directly by rewriting the expression for the rate of evolutionary change in $\bar{z}_{0i}$ (8), such that $\bar{z}_{1i}$ and θ_i appear together in the compound parameter $(1 - \bar{z}_{1i})\theta_i$.

As already noted, a small genetic variance in plasticity will slow down the rate of approach to equilibrium. In addition, it will also, on average, lead to slightly larger deviation of the expected phenotype from the local optimum. This can be seen by considering the load component arising from this deviation locally, $L_m(\theta_i) = s/2(\bar{z}_{0i} + \bar{z}_{1i}\theta_i - \theta_i)^2$. Substituting the solution (A4) and (A5) into this expression, assuming that θ_i follows a normal distribution, and approximating the global mean of this load component by

$$\bar{L}_m = L_m(0) + \frac{1}{2} L_m''(0) \text{var}(\theta_i) + \frac{1}{24} L_m'''(0) E(\theta_i^4),$$

we find that the global mean, for small errors in the environmental cues σ_θ^2 , is given by

$$\bar{L} = \frac{s\sigma_\theta^4(1 + \mu - 6\sigma_\theta^2\gamma)}{2(1 + \mu)\sigma_\theta^2}, \quad (17)$$

which, as expected, decreases with increasing genetic variance in plasticity γ .

Finally, consider the limit of a large rate of migration relative to strength of selection. We then essentially have panmixia, with individuals encountering different environmental optima θ at random. In this limit, as $\mu \rightarrow \infty$, both means (A4) and (A5) become independent of θ (i.e., all genetic differentiation is lost) and simplify to

$$\bar{z}_{0i} = 0, \quad (18)$$

$$\bar{z}_{1i} = \frac{\sigma_\theta^2}{\sigma_\theta^2 + \sigma_\theta^2}. \quad (19)$$

It can be noted that the above limiting value for $\bar{z}_1$ is the square of the correlation between θ and $\hat{\theta}$ if θ is regarded as stochastic in the above sense. The expected phenotype in a deme with optimum θ becomes

$$E(P_i) = \frac{\sigma_\theta^2}{\sigma_\theta^2 + \sigma_\theta^2} \theta_i. \quad (20)$$

Thus, unless $\sigma_\theta^2 = 0$, the expected expressed phenotype will differ from the local optimum and will be shifted toward the global mean optimum $E(\theta) = 0$. This last result (19) is identical to that of de Jong (1999).

Temporal Variation

Consider a single deme with a temporally varying optimum $\theta(t) = \sqrt{2}\sigma_\theta \sin(2\pi t/T)$, that is, a sine wave with period T and amplitude $\sqrt{2}\sigma_\theta$, as in Lande and Shannon (1996). This choice of amplitude makes the variance of $\theta(t)$ equal to σ_θ^2 , if t is regarded as a random uniformly distributed variable.

Substituting this pattern of temporal variability into (5) and (6), the evolutionary change in the means becomes

$$\frac{d\bar{z}_0}{dt} = -s\sigma_0^2 \left[\bar{z}_0 - (1 - \bar{z}_1) \sqrt{2}\sigma_\theta \sin \frac{2\pi t}{T} \right], \quad (21)$$

$$\begin{aligned} \frac{d\bar{z}_1}{dt} = & -s\sigma_1^2 \left\{ \sqrt{2}\sigma_\theta \sin \frac{2\pi t}{T} \right. \\ & \times \left[\bar{z}_0 - (1 - \bar{z}_1) \sqrt{2}\sigma_\theta \sin \frac{2\pi t}{T} \right] + \sigma_\theta^2 \bar{z}_1 \left. \right\}. \end{aligned} \quad (22)$$

The differential equations (21) and (22) have no simple general analytic solution. An example numerical solution is shown in figure 2.

An approximate asymptotic solution can be found, however, if we assume that the genetic variance in the plasticity, σ_1^2 , is small. Plasticity will then, again, be slow to evolve, but once asymptotic limit cycles have been attained, the mean plasticity $\bar{z}_1$ can be taken as constant because fluctuations in $\bar{z}_1$ will be small (see fig. 2). As shown in appendix B, in this limit, the mean plasticity in the population will asymptotically fluctuate around

$$\bar{z}_1 = \frac{\sigma_\theta^2}{1 + \left(s\sigma_0^2 T / 2\pi \right)^2 \sigma_\theta^2 + \sigma_\theta^2}. \quad (23)$$

In the same limit, the nonplastic component of the trait $\bar{z}_0$ will, asymptotically, follow a sine wave with period and lag equal to solution for the case without plasticity (Lande and Shannon 1996; see app. B) but with the amplitude reduced by a factor $1 - \bar{z}_1$.

From (23) it can be seen that if the rate of response to selection in the nonplastic component of the trait, $s\sigma_0^2$, is small, or equivalently, if there are fast fluctuations in the

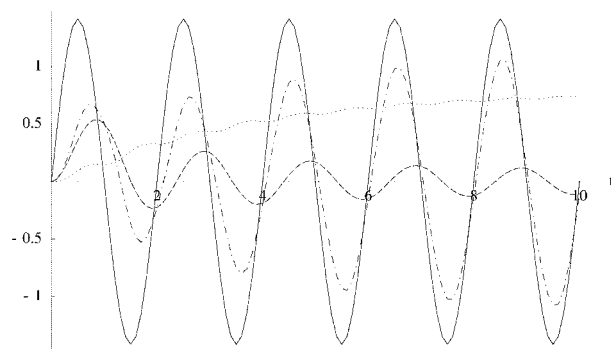


Figure 2: Numerical solution of (21) and (22) obtained using function NDSolve in Mathematica (Wolfram 1996) with $s = 1$, $\sigma_0 = 1$, $\sigma_1 = 0.5$, $\sigma_2 = 0.5$, $\sigma_3 = 1$, $T = 2$, and initial conditions $\bar{z}_0(t=0) = 0$ and $\bar{z}_1(t=0) = 0$, that is, no initial plasticity. The nonplastic component $\bar{z}_0(t)$ of the phenotype (dashed line) initially adapts to the moving optimum $\theta(t)$ (solid line) but lags behind by about 0.5 time units. The plasticity $\bar{z}_1(t)$ (dotted line) evolves more gradually to an asymptotic value of about 0.7. The rate of increase is largest at those point in time when there is a large negative or positive difference between the expected phenotype $E(P(t))$ (dashed-dotted line) and the optimum $\theta(t)$, creating fluctuations in $\bar{z}_1$ with frequency twice that of the environmental fluctuations driving the dynamics. Also note that, as the amount of plasticity increases, the expected phenotype (dashed-dotted line) matches the optimal phenotype more closely and the lag decreases.

environmental optimum (T small), then the situation again, essentially, is that individuals encounter different environmental condition at random, preventing adaptation to present temporal conditions, and the same limiting value regarding spatial variation with high rate of migration, (19), is reached.

From (23) we also see that for slow fluctuations in $\theta(t)$ (T large) or a high rate of response to selection in $\bar{z}_0$, the long-term average plasticity tends to 0, unless perfect environmental cues are available ($\sigma_2^2 = 0$).

Discussion

Under either spatial or temporal variation in the phenotypic optimum, the analysis here has shown that only partial phenotype-environment matching evolves, given that environmental cues are only partially correlated with the selective optimum. This means that selection pressures remain, generating geographic or temporal differentiation in the nonplastic component of the trait. As either the rate of migration or rate of temporal fluctuations in the optimum increases, more plasticity evolves and temporal and spatial differentiation in the nonplastic component of the trait is gradually lost. This is a new result differing from the results of Via and Lande (1985) and van Tienderen (1997), who, for a model with two discrete environmental

optima, concluded that perfect matching and no genetic differentiation should eventually evolve in the absence of any costs, even when the rate of migration is small.

The results derived here extend those of Scheiner (1998) and de Jong (1999). Scheiner (1998), based on a quantitative genetic clinal simulation model with no costs and no explicit cue unreliability reached qualitatively similar conclusions (e.g., Scheiner 1998, fig. 1) to the results presented here (e.g., fig. 1). He argued that it is the presence of different types of loci, affecting either the slope and the height of the norm of reaction, that is responsible for a mixed evolutionary outcome with partial plasticity and genetic differentiation. However, as shown here (16) and elsewhere (Via and Lande 1985), no differentiation evolves if environmental cues are completely reliable, as long as genetic variation is not exhausted. As noted by de Jong (1999), the mixed evolutionary outcome of Scheiner's (1998) model is a result of migration between the environment of development and selection, implicitly producing a form of cue unreliability. The analysis presented here also extends de Jong's (1999) result, that the slope of the reaction norm equals the squared correlation between the environment of development and selection, equivalent to the limiting case of a high rate of migration or fast fluctuations in the environmental optimum (19).

In the general case, however, that is, for moderate rates of migration or fluctuations in the environmental optimum, both plastic and nonplastic adaptations will evolve. In the case of small temporal or spatial variation in plasticity, a simple new result is that the presence of plasticity reduces the selective forces acting on the nonplastic component of the trait arising from variation in the optimum θ by a factor $1 - \bar{z}_1$. This result should be useful in studies of geographic (or temporal) genetic differentiation, in providing a link between the rate of migration, local selection pressures, genetic differentiation, the evolved level plasticity, and the quality of environmental cues. For example, in a study of elongation as a shade avoidance response in velvetleaf (*Abutilon theophrasti* L.), Weinig (2000) showed that both light-quality cues and population origin had significant effects on elongation response, indicating the importance of both plastic and nonplastic adaptation. Also, as predicted by the model considered here ([13] and [A5]), some genetic differentiation also in plasticity were found, with the more extreme demes being more plastic. Evidence for such geographic variation in the norm of reaction, possibly consistent with the above predictions, exists also for many other species of plants (reviewed by Travis 1994).

If one defines costs of plasticity as an additional reduction in fitness of the more plastic genotypes acting independently of phenotypic stabilizing selection, say, a reduction in fitness by a factor proportional to the ex-

ponentiated squared deviation of z_1 from the amount of plasticity incurring minimum cost, then only partial phenotype-environment matching will evolve (van Tien-deren 1997). It should be noted that this form of cost, by definition, implies that selection acts directly on the genotypic value of the underlying plasticity trait and not only on the expressed phenotype as in the model considered here. It can also be noted that if one follows van Tien-deren's (1997) definition, used in the classification of DeWitt et al. (1998), then unreliable cues are not a form of cost but rather another form of limitation arising from stochasticity in the environment. Because little yet is known about the magnitude of costs of plasticity, as defined by van Tien-deren (1997), and the variability of environmental cues, it is possible that both forms of limitations can be important for the evolution of plasticity. However, if unreliable environmental cues, as one might expect, are associated with environmental variability in general, then unreliable cues should be an ubiquitous factor limiting the evolution of plasticity in cases where plasticity is in fact needed, that is, in variable environments. While intrinsic costs of plasticity may still be important, such costs would not be necessary to explain imperfect phenotype-environment matching.

Although imperfect environmental cues, like costs of plasticity, can lead to imperfect phenotype-environment matching, the bias of the phenotype toward the global optimum $\theta = 0$, is in a certain sense still beneficial. Consider the case of panmixia (μ large) or fast fluctuations in the environmental optimum (T small) so that individuals encounter different optima essentially at random. If we, as suggested by Getty (1996), think of the problem as a signal detection problem or, more generally, as a Bayesian decision-theoretic problem (Berger 1985), the "decision" would be in this case what phenotype P to develop; the "data" would be the environmental cue θ , which is used by the organism to "estimate" an unknown "parameter"; the environmental optimum θ . With the distribution of θ between demes used as the "prior" for the "parameter" θ , it is easy to verify that the decision P maximizing expected "utility" (fitness) indeed is the one evolving as a result of natural selection (20). Thus, imperfect phenotype-environment matching is not necessarily suboptimal; given that information available to an individual at the beginning of its development is limited, relying also on past experience about the environment accumulated through natural selection into its genes, is optimal in the above decision-theoretic sense. A similar interpretation of the constant environment case without migration (7) can also be made; the nonplastic genotype evolving in this situation corresponds to using a prior probability density with all its probability mass concentrated in the local value

of θ so that the posterior for θ then only depends on the prior, and environmental cues are ignored.

Another cost, related to limitations associated with imperfect environmental cues, may arise from information acquisition. Several authors have suggested that this may give rise to a cost of plasticity (DeWitt et al. 1998). While information acquisition, that is, some perhaps initially simple mechanism by which the organism senses and interprets information in its surrounding environment (some primitive sensory system) is necessary for evolution of plasticity to occur, there is no reason a priori that the precision of information acquisition, say, the square of the correlation between θ and $\hat{\theta}$, ρ^2 , if regarded as a quantitative trait in itself, should be positively genetically correlated with traits for plasticity and that, say, directional selection toward small values of ρ^2 (arising from a cost of information acquisition), therefore, should lead to indirect selection for reduced plasticity.

Given that there is genetic variation in both traits, plasticity and the precision of information acquisition ρ^2 —once ρ^2 becomes sufficiently large—should evolve together, however, because increased values of ρ^2 should increase fitness when the information acquired is used by a plastic genotype. Conversely, as shown here, increased precision should lead to an increase in the optimum amount of plasticity. It is not clear, however, how such a coevolutionary process can initially get started because no initial correlation between the environmental optimum θ and the obtained cue $\hat{\theta}$ ($\rho^2 = 0$) always would lead to selection against plasticity. Similarly, if there initially is no plasticity in the population, ρ^2 will be a selectively neutral trait, making the joint initial state of no correlation ($\bar{\rho} = 0$) and no plasticity ($\bar{z}_1 = 0$) an evolutionary saddle point. If relatively small genetic changes are sufficient for sensory systems to begin to evolve as suggested by some evidence (Hodos and Butler 1997), and if information acquisition costs are small for small ρ^2 , genetic drift should be easily sufficient to start the process, however.

The approach here has been to base the analysis on the simplest possible model for the underlying genetic basis of the traits z_0 and z_1 , the infinitesimal model (Bulmer 1980). Furthermore, complications arising from linkage disequilibrium generated by migration and selection have been ignored so that the genetic variances remain constant and the state of the population can be characterized by the trait means only. Some authors have expressed dissatisfaction with this approach when applied to the evolution of plasticity in particular (e.g., Schlichting and Pigliucci 1995) and have argued that identification of individual regulatory genes must be an integral component of any satisfactory scenario for the evolution of plasticity (Pigliucci 1996). This critique, however, can be made of applications of quantitative genetics to any evolutionary

problem (Barton and Turelli 1989). Essentially, one reason why the quantitative genetic approach (and more probabilistic statistical models in general) is needed is that our state of knowledge about the underlying genetic basis of most traits (like our knowledge about the details of many other phenomena) is and will continue to be incomplete. Also, even though new methods for mapping quantitative trait loci can produce some of the information from which explicit Mendelian multilocus simulations of gene frequency evolution can be made, analytic mathematical treatment producing general results will seldom be possible unless a very small number of loci are involved (Barton and Turelli 1989). Although any quantitative genetic model

must be seen as an approximation of the genetic basis of the trait in question, sometimes involving some genes of large effects (Orr and Coyne 1992), the quantitative genetic approach remains a simple and tractable starting point for building an understanding of phenotypic evolution (Lande 1982) including the evolution of plasticity.

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APPENDIX A

Approximate Solution under Spatial Variation

Setting the left-hand sides of (8) and (9) equal to 0 and solving the resulting two linear equations for $\bar{z}_{0i}$ and $\bar{z}_{1i}$, we obtain an expression for the equilibrium plasticity in each deme i as a function $\bar{z}_{1i}(\theta_i, \bar{z}_1)$ of the global mean plasticity, $\bar{z}_1$, the local optimum θ_i , and the other parameters of the model. The global mean plasticity by definition satisfies

$$\bar{z}_1 = E\bar{z}_{1i}(\theta_i, \bar{z}_1), \quad (\text{A1})$$

where the expectation is taken with respect to θ_i . This equation, with the above solution substituted in for $\bar{z}_{1i}(\theta_i, \bar{z}_1)$, determines $\bar{z}_1$ but cannot in general be solved analytically. An approximate solution of (A1) can be found, however, if we use the approximation

$$\bar{z}_1 = \bar{z}_{1i}(E\theta, \bar{z}_1) + \frac{1}{2}\bar{z}_{1i}''(E\theta, \bar{z}_1)\text{var}(\theta_i), \quad (\text{A2})$$

which will be accurate provided that $\text{var}(\theta_i)$ or the genetic differentiation between demes in local plasticity is small. Substituting the second derivative of $\bar{z}_{1i}(\theta, \bar{z}_1)$ (from the above preliminary solution) evaluated at $\theta = 0$ into (A2) and solving for $\bar{z}_1$, we find that

$$\bar{z}_1 = \frac{\mu(\mu + \gamma\sigma_\theta^2)\sigma_\theta^2}{\gamma\sigma_\theta^4 + \mu\sigma_\theta^2(1 + \gamma\sigma_\theta^2) + \mu^2(\sigma_\theta^2 + \sigma_\theta^2)}, \quad (\text{A3})$$

where the compound parameters μ and γ are given by (10) and (11) in the main text.

Substituting (A3) back into the original solutions for $\bar{z}_{0i}$ and $\bar{z}_{1i}$, we find that, in a deme with optimum θ_i ,

$$\bar{z}_{0i} = \frac{(1 + \mu)\sigma_\theta^2(\mu + \gamma\sigma_\theta^2)^2\theta_i}{[\mu(1 + \mu + \gamma\theta_i^2) + (1 + \mu)\gamma\sigma_\theta^2][(1 + \mu)\sigma_\theta^2(\mu + \gamma\sigma_\theta^2) + \mu^2\sigma_\theta^2]}, \quad (\text{A4})$$

$$\bar{z}_{1i} = \frac{\mu[(1 + \mu)\gamma\theta_i^2\sigma_\theta^2(\mu + \gamma\sigma_\theta^2) + \mu(\mu(1 + \mu + \gamma\theta_i^2) + (1 + \mu)\gamma\sigma_\theta^2)\sigma_\theta^2]}{[\mu(1 + \mu + \gamma\theta_i^2) + (1 + \mu)\gamma\sigma_\theta^2][(1 + \mu)\sigma_\theta^2(\mu + \gamma\sigma_\theta^2) + \mu^2\sigma_\theta^2]}. \quad (\text{A5})$$

APPENDIX B

Approximate Solution under Temporal Variation

Let $\bar{\bar{z}}_1$ denote the long-term asymptotic average of $\bar{z}_1$. From (21) it can be noted that an approximately constant value of $\bar{z}_1 = \bar{\bar{z}}_1$, in effect, will reduce the amplitude of θ by a factor of $1 - \bar{\bar{z}}_1$. Thus, for a given value of $\bar{\bar{z}}_1$, using Lande and Shannon's (1996) equation (4), the solution for $\bar{z}_0$ can be written in the parameterization used here,

$$\bar{z}_0 = \zeta \sqrt{2\sigma_\theta} (1 - \bar{\bar{z}}_1) \sin\left(\frac{2\pi t}{T} - \beta\right), \quad (\text{B1})$$

where $\zeta = s\sigma_0^2 / \sqrt{s^2\sigma_0^4 + (2\pi/T)^2}$ and $\beta = \arccos(\zeta)$.

Substituting (B1) and $\bar{\bar{z}}_1$ into (22), noting that $d\bar{z}_1/dt$ integrated over one period must be equal to 0 when the population is at equilibrium, and solving the resulting equation for $\bar{\bar{z}}_1$, we obtain (23).

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