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The selective advantage of reaction norms for environmental tolerance

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Abstract

A tolerance curve defines the dependence of a genotype's fitness on the state of an environmental gradient. It can be characterized by a mode (the genotype's optimal environment) and a width (the breadth of adaptation). It seems possible that one or both of these characters can be modified in an adaptive manner, at least partially, during development. Thus, we extend the theory of environmental tolerance to include reaction norms for the mode and the width of the tolerance curve. We demonstrate that the selective value of such reaction norms increases with increasing spatial heterogeneity and between-generation temporal variation in the environment and with decreasing within-generation temporal variation. Assuming that the maintenance of a high breadth of adaptation is costly, reaction norms are shown to induce correlated selection for a reduction in this character. Nevertheless, regardless of the magnitude of the reaction norm, there is a nearly one to one relationship between the optimal breadth of adaptation and the within-generation temporal variation perceived by the organism. This suggests that empirical estimates of the breadth of adaptation may provide a useful index of this type of environmental variation from the organism's point of view.

Introduction

Eighty years ago, Woltereck (1909) developed the concept of a reaction norm. He studied the variation in helmet length for several clones of *Daphnia* in the laboratory. By changing one environmental factor, e.g., food conditions, while keeping all other environmental conditions as constant as possible, he obtained a "phenotype curve" from measures of the mean phenotype of this trait. Performing measurements for all possible combinations of environmental parameters would result in an enormous number of such phenotype curves. Woltereck called the totality of such curves for a character its specific reaction norm. He also tried to define the genotype as all of its inherited specific reaction norms. In practice, however, only a small fraction of phenotype curves can be measured, which he called partial reaction norms.

We will refer to Schmalhausen's (1949) reaction norm which is equivalent to a single phenotype curve in the sense of Woltereck. Roughly speaking, a reaction norm describes how the expected phenotype of a given genotype responds to environmental conditions. The concept of reaction norms is described in detail by de Jong (1990). Because of the inherent stochasticity of biochemical and developmental pathways, we expect variation in phenotypes even under absolutely constant external conditions. Therefore, the frequent assumption (e.g., Gregorius, 1977) that a phenotype is totally determined by its genotype and its environment is only approximately true, but seems to be reasonable for mean phenotypic values.

Reaction norms of specific traits are related to the concept of phenotypic plasticity (e.g., Stearns and Koella, 1986) and can obviously vary within and between species (Bradshaw, 1965; Via, 1984; Schlichting, 1986; Scheiner and Lyman, 1989). But little is known about the extent to which reaction norms are adaptive. It is conceivable that they are sometimes nonadaptive by-products of environmental responses of developmental, physiological or biochemical properties.

Our purpose is to investigate the extent to which an adaptive reaction norm can enhance a genotype's fitness. The analysis of this problem involves an application of our earlier work on environmental tolerance, which describes the response of a genotype's mean fitness over an environmental gradient (Lynch and Gabriel, 1987). We assume that organisms are able to modify their phenotype in response to some environmental cues during development. By exploiting appropriate reaction norms for phenotypic traits, the environment tolerance of an organism should be enhanced. Under a "jack-of-all-trades is a master-of-none" scenario, reaction norms can be particularly advantageous since they provide genotypes with a means of fine-tuning to the immediate environment without the associated cost of maintaining broadly adapted phenotypes. We will be concerned with the extent to which general features of environmental variation favor the evolution of reaction norms as well as with the secondary evolutionary consequences of reaction norms for the breadth of adaptation.

Theoretical framework

Our model for environmental tolerance (Lynch and Gabriel, 1987) was developed for asexual species, but most of the conclusions are valid under sexual reproduction as well (Gabriel, 1988). The environmental scale is assumed to be a property, such as temperature, which is independent of population density. The tolerance curve of an individual over the environmental gradient is described by two parameters: 1) the individual's optimal environmental state (at which fitness is maximized), and 2) the breadth of adaptation, which is a measure of the width of the tolerance curve, i.e., of the degree of generalism of the organism. The scale of environmental states is assumed to be chosen such that the tolerance curve is Gaussian. It is also assumed that an increased breadth of adaptation is costly, i.e., there is a tradeoff between breadth of adaptation and fitness in the optimum environment.

We showed earlier (Lynch and Gabriel, 1987) that the optimal environmental state is expected to evolve to the long-term average value of the environment. The optimal breadth of adaptation depends on the spatial heterogeneity of the environment perceived by the organism as well as on the temporal environmental variance within and between generations. Development noise, which causes phenotypic values of the mode and the breadth to deviate from their genetic expectations, complicates the theory. Developmental noise for the optimum environmental state effectively broadens the tolerance curve, while developmental noise for the breadth actually narrows it. Such effects have an influence on the optimal genotype value for the breadth of adaptation.

In this paper, we expand the theory of environmental tolerance by assuming that a shift in the mode and/or breadth of the tolerance curve during development can be used to enhance fitness. By this means, an organism that expects to live in a colder environment than normally encountered would shift its thermal optimum to a lower value; or, when the expected temporal variability of the environment is larger than the long-term expected value, then the phenotypic breadth of adaptation would be increased compared to its genotypic value. (As a point of reference, we define the genotypic values to be the expected phenotypes expressed in the long-term average environment.) Such forecasting of the environment may be rendered possible by environmental cues encountered during development. Information on the expected environment may also be transferred by maternal effects. We do not have reversible physiological acclimation in mind. Rather we assume that the phenotypic values are fixed for life after the developmental period. Thus, in this new version of our model the phenotypic values of the two tolerance curve parameters are functions of the genotypic values, of the random developmental noise during development, and of superimposed predictable changes due to reaction norms of quantitative characters contributing to fitness.

The model

We follow the notation of Lynch and Gabriel (1987) (see Table 1). For phenotypes, z_1 is the location of the mode (i.e., the optimal environmental state), and z_2 is equivalent to the "variance" of the individual's tolerance curve. We will refer to the square-root of this variance as the breadth of adaptation. Letting ϕ be the actual environmental state, the fitness of an individual is given by

$$w = (2\pi z_2)^{-1/2} \exp\left(-\frac{(z_1 - \phi)^2}{2z_2}\right). \qquad (1)$$

Table 1. A glossary of variables used in the text

z	phenotypic value
g	genotypic value
r	reactive deviation
e	developmental noise
b	boundary of a reaction norm
ϕ	environmental state
V	variance
Subscripts:	
1	mode of the tolerance curve
2	variance of the tolerance curve
s	spatial
tw	temporal, within-generation
tb	temporal, between-generation
opt	optimal
$\sqrt{z_2}$	phenotypic breadth of adaptation
$V_{\phi..} = V_{\phi s} + V_{\phi tb}$	

Implicit in the use of Equation (1) is a tradeoff between breadth of adaptation and fitness in the optimum environment – for fixed z_1, w declines with increasing z_2.

In our earlier work, the phenotypic values z_1 and z_2 were distributed approximately normally around their genetic expectations (g_1 and g_2) with variances of V_{E1} and V_{E2}, such that

$$z_i = g_i + e_i,$$

where $i = 1$ or 2, with e_i being the deviation due to developmental noise. In our approach, V_{E1} and V_{E2} are independent of both the genotype and the environmental state. The developmental noise is most easily thought of as the phenotypic variance of a clonal population in a constant external environment. At least in principle, the four variables g_1, g_2, V_{E1}, and V_{E2} can be separated and estimated from experimental data by a maximum-likelihood approach (Gabriel, 1987).

In this paper we will extend our earlier theory by assuming that the expression of genotypic values depends in a specific way on the environment experienced by the individual during development (or by its mother). This modification of the phenotype is controlled such that

$$z_i = g_i + e_i + r_i. \tag{2}$$

(We omit the subscript i in the following if equations or conclusions are valid for both characters $i = 1$ and 2.) In contrast to the random variable e, r is a fixed response determined by the environment that the organism perceives during development.

Equation (2) might, of course, be correct only as a first approximation. For example, with genotype × environment interaction, the phenotype could be a nonadditive function of the genotypic value, developmental noise, and reaction norm. We are interested mainly in predicting the possible qualitative influence of the reactive deviation, and, therefore, such complications are not discussed further.

Under ideal conditions, when the organism is perfectly capable of predicting the future environment and adjusting to it phenotypically, the sum of the genotypic value and the expected reactive deviation would be equal to the optimal phenotypic value, i.e.,

$$r = z_{opt} - g.$$

The organism would then have a phenotypic value $z = z_{opt} + e$. Without any genetic component in such a character, the only variation would be due to developmental noise. This is, of course, biologically unrealistic for most characters. Because we are interested in at least partially genetically determined characters, we have to assume that the reactive deviations are limited. As a first approach, we will assume a reaction norm with a nearly linear response for small deviations of the genotypic value from the optimal phenotype and with a smooth approach to a maximal reactive deviation in extreme environments (Figure 1).

We will try to minimize the introduction of new parameters by letting the reaction norm depend only on the boundary condition b (with $|r| < b$) and the difference between genotypic value and the optimal phenotypic value z_{opt}. This difference may be measured in units of the boundary value b such that

$$d = \frac{|z_{opt} - g|}{b}. \tag{3}$$

The reactive deviation is assumed to have the sigmoid form

$$r = \pm b[1 - \exp(-d)] \tag{4}$$

where the ± sign is determined by the sign of $(z_{opt} - g)$. The graph of this reaction norm is shown in Figure 1 together with the unbounded reaction norm. Appendix I gives the optimal phenotypic values z_1 and z_2 for an organism that is able to forecast the mean and variance of the environmental states that it will encounter in its life. Generally, the optimal breadth depends on the mode of the tolerance curve, on the average environmental state, and on the temporal variance of the environment.

Figure 2 illustrates qualitatively the modifications of a tolerance curve that can be induced via reaction norms. Let us assume that the environment differs considerably from the genotypic mode g_1 of the tolerance curve. Without a reaction norm, the parameters of the tolerance curve would be fixed to suboptimal values. A reaction norm for the mode alone shifts the tolerance curve towards the expected environment; with an unbounded reaction norm, the expected mode of the tolerance curve would coincide with the environmental state (Figure 2a). Now assume that the organism has a reaction norm for the breadth of its tolerance curve but not for the position of the optimum. Then it could adjust its breadth to gain maximal fitness (Figure 2b).

We assume three independent random processes determining the actual environmental state experienced by an individual. As in Lynch and Gabriel (1987), all members of the population experience the same within-generation temporal variance $V_{\phi tw}$, but the mean value of the environment differs between individuals because of spatial variability $V_{\phi s}$. The mean environment also fluctuates between generations with variance $V_{\phi tb}$.

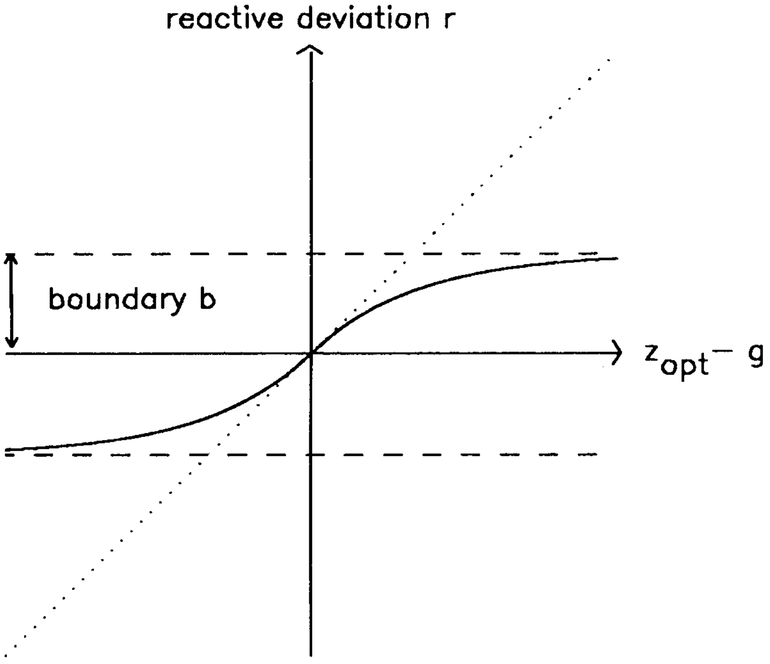

Fig. 1. The reactive deviation r depends upon the difference between the genotypic value g of a trait and the optimal phenotypic value z_{opt} which is assumed to be predictable from environmental cues. The dotted line represents an unbounded reaction norm, such that the expected phenotypic value is always the optimum. The sigmoid curve is a reaction norm for a case of limited phenotypic plasticity (boundaries $\pm b$). The reactive deviation $|r|$ is always smaller than b so that the phenotypic value is near its optimum only for small deviations of g from z_{opt}. For larger deviations, r approaches the boundary value.

The optimal *genotypic* values are obtained by maximizing long-term clonal fitness over three successive averages: 1) the geometric average over the temporal variation within a generation, 2) the arithmetic average over the spatial distribution of environmental states, and 3) the geometric average over the temporal variation between generations. Geometric averages are taken over time since the fitness components (such as day-to-day survivorships) are multiplicative within generations, and population growth between generations is a multiplicative process. The spatial variation is averaged arithmetically since all members of a cohort are contemporaneous. (For the mathematical formulation, see Appendix II.)

It is intuitively clear that the optimal g_1 value is equal to the long-term average environmental state. The optimal g_2 depends on the spatial heterogeneity and on the temporal variability within and between generations. The case in which there are no reaction norms for z_1 or z_2 has been addressed in Lynch and Gabriel (1987), and we give some simple approximations in Appendix III, along with some asymptotic results for the cases in which the reaction norms are unbounded.

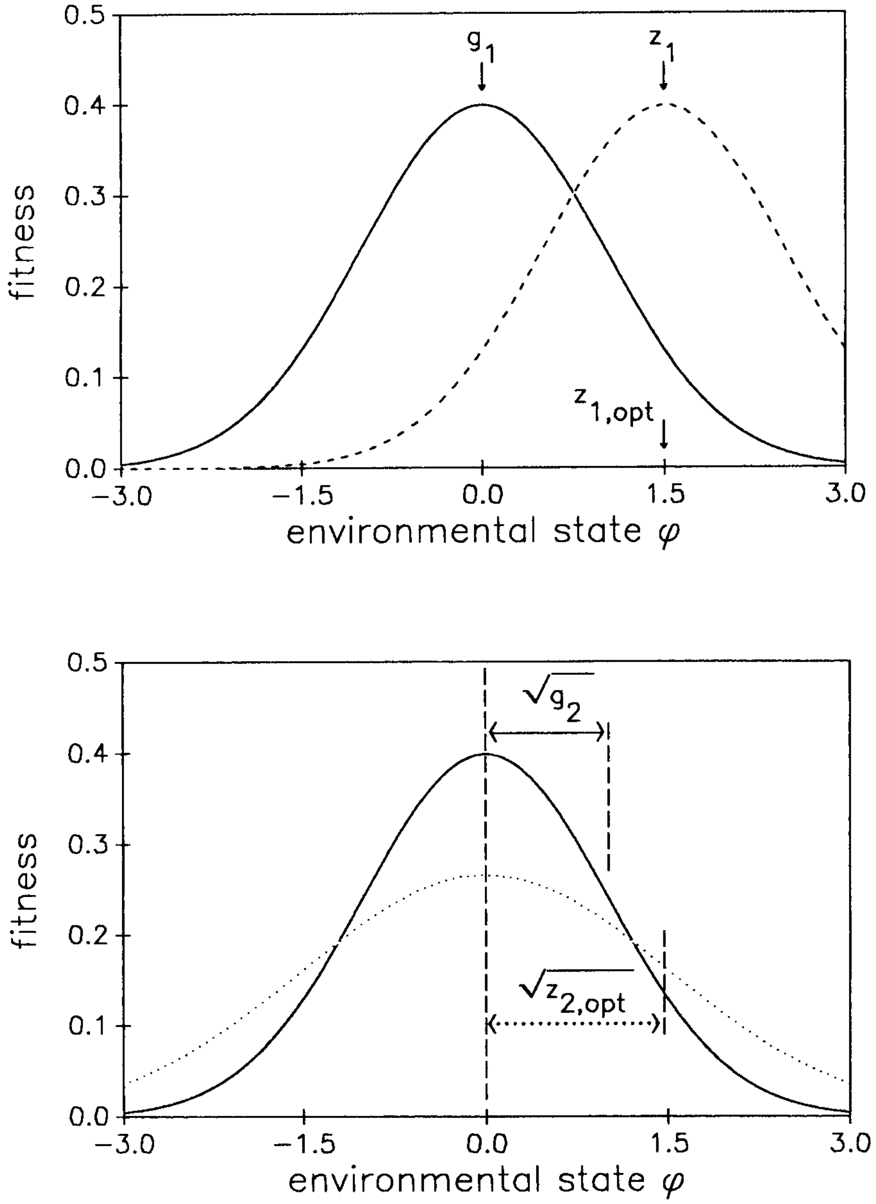

Fig. 2. Optimal modifications of a tolerance curve via reaction norms for an organism that is able to forecast the environment. The long-term average environmental state is $\phi = 0$, but the realized environmental state for the illustrated individual is $\phi = 1.5$. **a)** Shift of a tolerance curve (broken line) due to an unbounded reaction norm for the environmental optimum. z_1 is the phenotypic value, and g_1 is the genotypic value. **b)** Shift of a tolerance curve due to a reaction norm for the breadth of adaptation. $z_2 = z_{2,opt}$ maximizes fitness at environmental state $\phi = 1.5$. The genotypic value for the breadth is defined to be $\sqrt{g_2}$, whereas $\sqrt{z_2}$ is the realized phenotypic value.

Results

Our primary focus will be on the selective value of reaction norms, that arises when organisms are able to shift their tolerance curve parameters to the appropriate optima for the remainder of their lives. Assuming they are adaptive, reaction norms in the tolerance curve parameters must enhance fitness if there is spatial or between-generation variance of the environmental state. The gain in fitness must increase with increasing boundaries to the reaction norm, converging to the maximal selective advantage with an unbounded reaction norm (Figure 3a).

In effect, a reaction norm for a tolerance curve parameter is equivalent to a reduction in the influence of spatial and temporal variance of the environment on fitness. Thus, since generalism is assumed to be costly in our model, a secondary advantage of a reaction norm for z_1 is the reduced need to maintain a large breadth of adaptation. An organism with an unbounded reaction norm in z_1 has an optimal breadth of adaptation which is only a function of the within-generation temporal variance in the environment (Appendix III). Thus, with an increasing boundary in the reaction norm for z_1, the optimal g_2 converges to the value expected at the observed $V_{\phi tb}$ but for $V_{\phi tb} = V_{\phi s} = 0$. This is demonstrated in Figure 3b.

A reaction norm in z_2 also selects for a reduction in the optimal g_2 value. However, the effect is small relative to that induced by the reaction norm for the mode (dotted line in Figure 3b). The maximum selective advantage of the reaction norm in z_1 cannot be enhanced by an additional reaction norm in z_2 (see Appendix IV), since the mode of the tolerance curve will always coincide with the spatially determined environmental state, and a change in the breadth of adaptation cannot improve fitness any further (Figure 4). Even if the reaction norm in z_1 is bounded, a reaction norm in z_2 is only slightly advantageous, unless the bounds on z_1 are very stringent relative to $\sqrt{V_{\phi s} + V_{\phi tb}}$. The dotted line in Figure 3a indicates that the fitness advantage for simultaneous reaction norms in z_1 and z_2, for the case in which the boundary b (breadth of reaction norm) is the same for both characters, is almost identical to that for a reaction norm for z_1 alone.

Figures 4 and 5 compare the optimal breadths of adaptation and their selective advantages (ratio of fitness with reaction norm to fitness without reaction norm) for unbounded and bounded reaction norms in z_1 and/or z_2 for various spatial and temporal variances of the environment. The boundary for the reaction norm in z_1 is taken to be half the optimal breadth of adaptation without a reaction norm; the boundary for z_2 is 0.284 times that same optimum z_2. (This value for b makes the fitness effect of a reaction norm on z_2, for this particular example, comparable to that for the reaction norm in z_1.)

The maximum selective advantage of reaction norms depends strongly on the relation between the components of environmental variance (Figures 5 and 6). (Here it is assumed that g_1 and g_2 are optimized with respect to the environment.) Generally, it increases with $V_{\phi tb}$ and $V_{\phi s}$ but decreases with $V_{\phi tw}$. The reasons for this can be understood more deeply by referring to the mathematical treatment in Appendices III and IV. In essence, the spatial and between-generation environmental deviations are fixed during development, while within-generation temporal

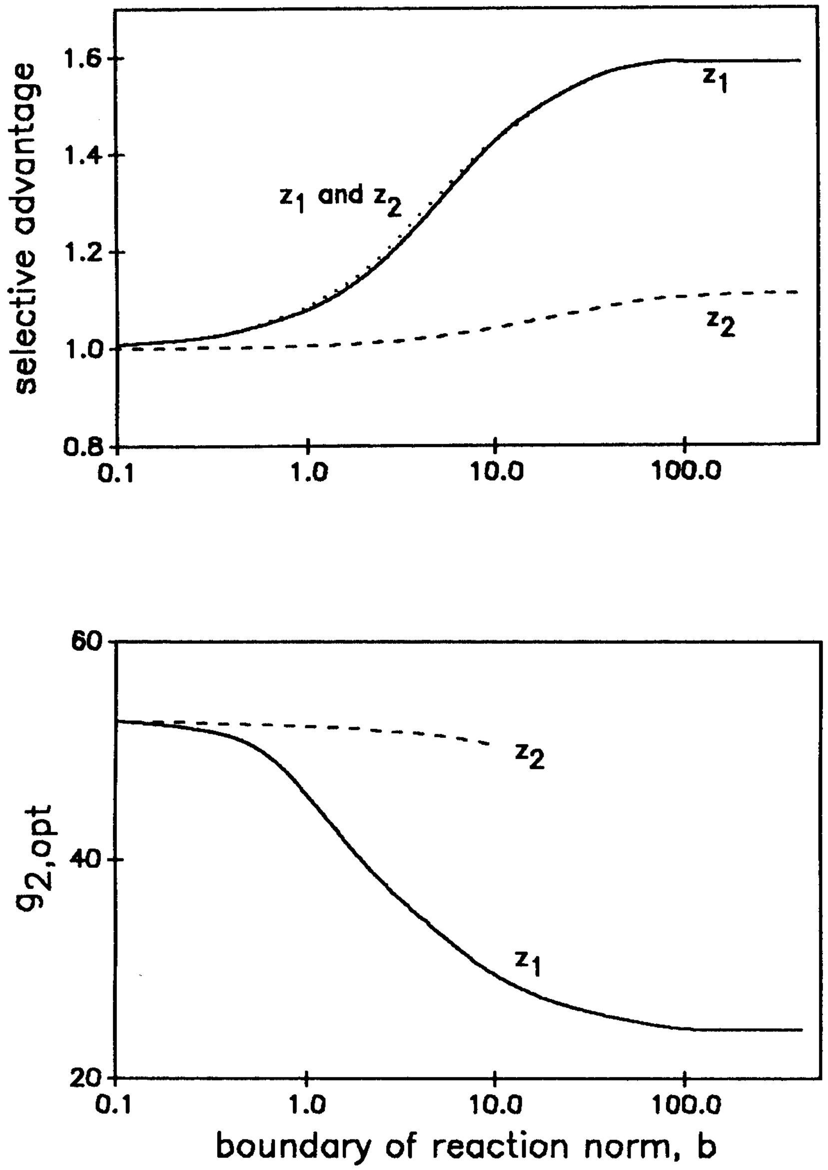

Fig. 3. a) Selective advantage (ratio of long-term geometric mean fitnesses with and without reaction norms) of reaction norms in z_1 and z_2 as a function of the boundary of the reaction norm. Solid line - reaction norm only in z_1; broken line - reaction norm only in z_2; dotted line simultaneous reaction norms in z_1 and z_2. **b)** Reduction of the optimal genotypic value for the breadth of adaptation depending on the boundary of the reaction norm. The effect of reaction norms in z_1 and z_2 are given by the solid and broken lines. For large boundary values for the reaction norm of z_2, the optimal genotypic value g_2 is meaningless since fitness is essentially independent of g_2. In this example, $V_{\phi th} = V_{\phi tw} = V_{\phi s} = 25$.

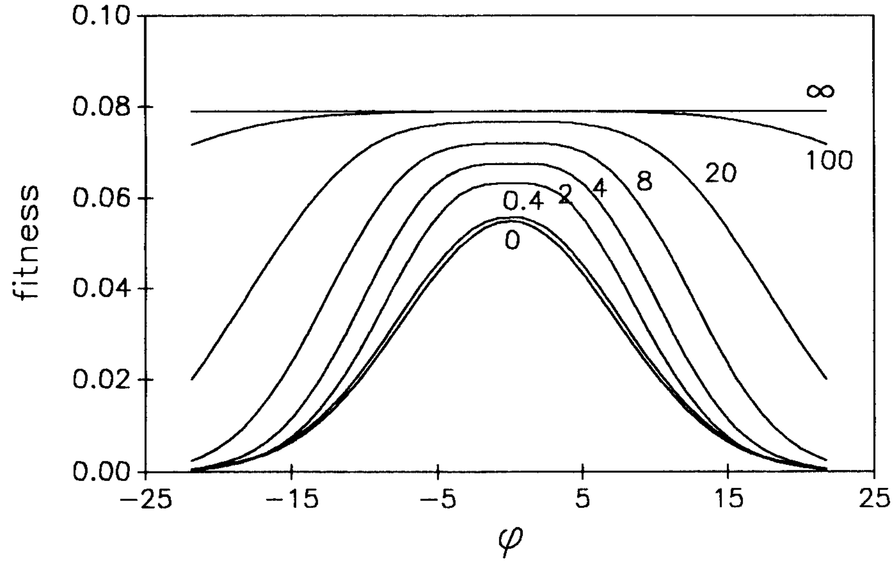

Fig. 4. Change in the tolerance curve due to a reaction norm in z_1. Numbers in the figure give the boundary b of the reaction norm. The curves are calculated for $V_{\phi s} = V_{\phi tw} = V_{\phi tb} = 25$. g_2 is assumed to take on its optimal value for this type of environment.

variance is a continuous source of noise throughout life. Figure 7 gives approximate values for the maximum selective advantage as a function of $V_{\phi..}/V_{\phi tw}$, where $V_{\phi..} = V_{\phi s} + V_{\phi tb}$. The values are exact predictions for $V_{\phi..} = V_{\phi s}$ (where $V_{\phi tb} = 0$) and are otherwise good approximations provided $V_{\phi tb} < -v_{\phi tw}$. The selective advantage for a reaction norm in z_1 is always larger than that for a reaction norm in z_2. The dotted line gives the selective advantage for the hypothetical case in which there is a reaction norm in z_1 but g_2 is constrained to the value which is optimal without a reaction norm. Such a comparison serves to demonstrate the selective advantage of readjusting the breadth of adaptation with increasing $V_{\phi..}/V_{\phi tw}$.

Discussion

We have extended the theory of environmental tolerance (Lynch and Gabriel, 1987) as one approach to establishing a theory on the evolution of reaction norms. Reaction norms are common, and it seems reasonable to assume that they are often adaptive. The approach taken here is that the reaction norms for subsidiary morphological, behavioral, and physiological characters are molded evolutionarily to maximize the geometric mean fitness of a genotype in the face of spatial and temporal variation in the environment. Thus although we have not examined specific traits, the theory in this paper is applicable to any kind of environmentally induced (irreversible) switches in development.

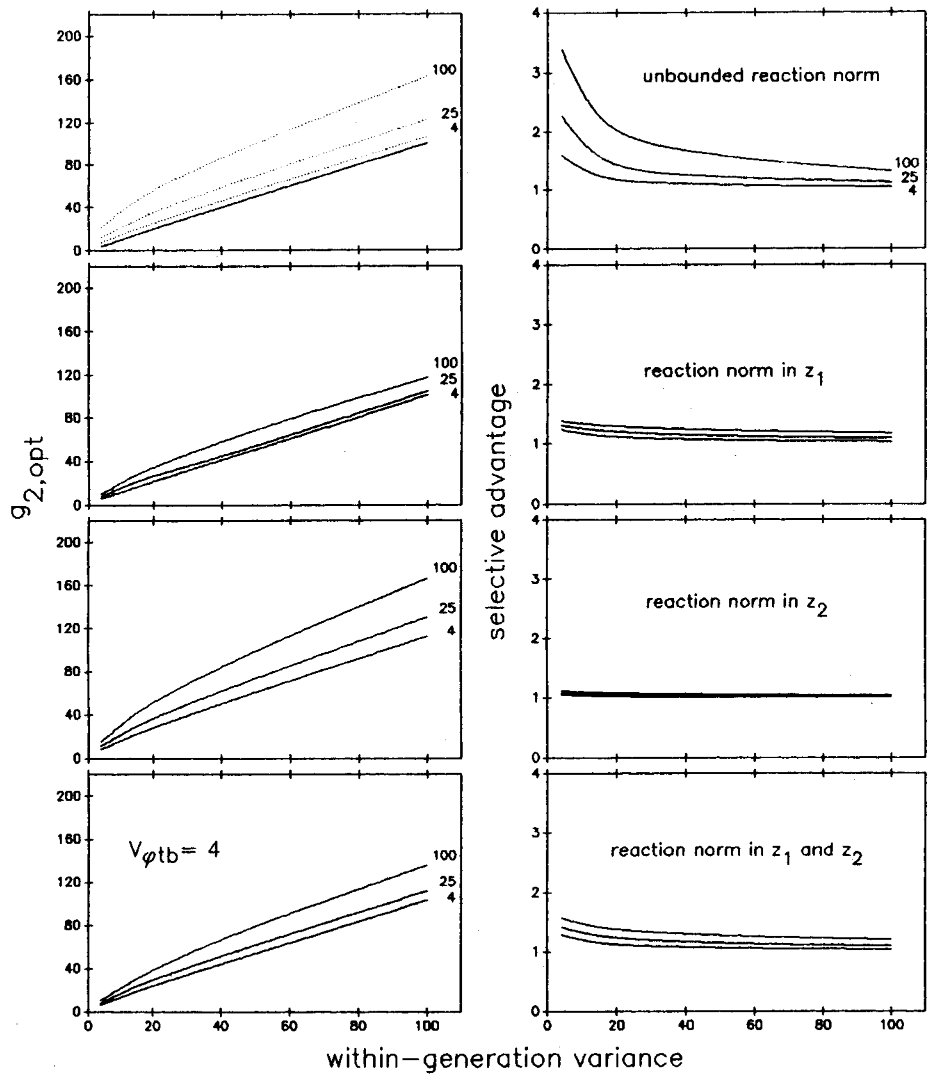

Fig. 5. Optimal breadth of adaptation and the selective advantage of bounded and unbounded reaction norms as a function of the within-generation temporal variance and of the spatial variance of the environment (indicated as numbers in the figures) at a low level of between-generation temporal variance ($V_{\phi tb} = 4$). The dotted lines in the upper left figure represent the optimal g_2 values in the absence of reaction norms. Except in the first row, the reaction norms are bounded, as described in the text.

Although the exact form of the reaction norm that we utilized was adopted for analytical tractability, most reaction norms are probably bounded due to the physical/chemical constraints on living systems. However, reaction norms may be constrained in another way. The procurement of accurate information on the mean and variance of the environment may require an extension of the developmental

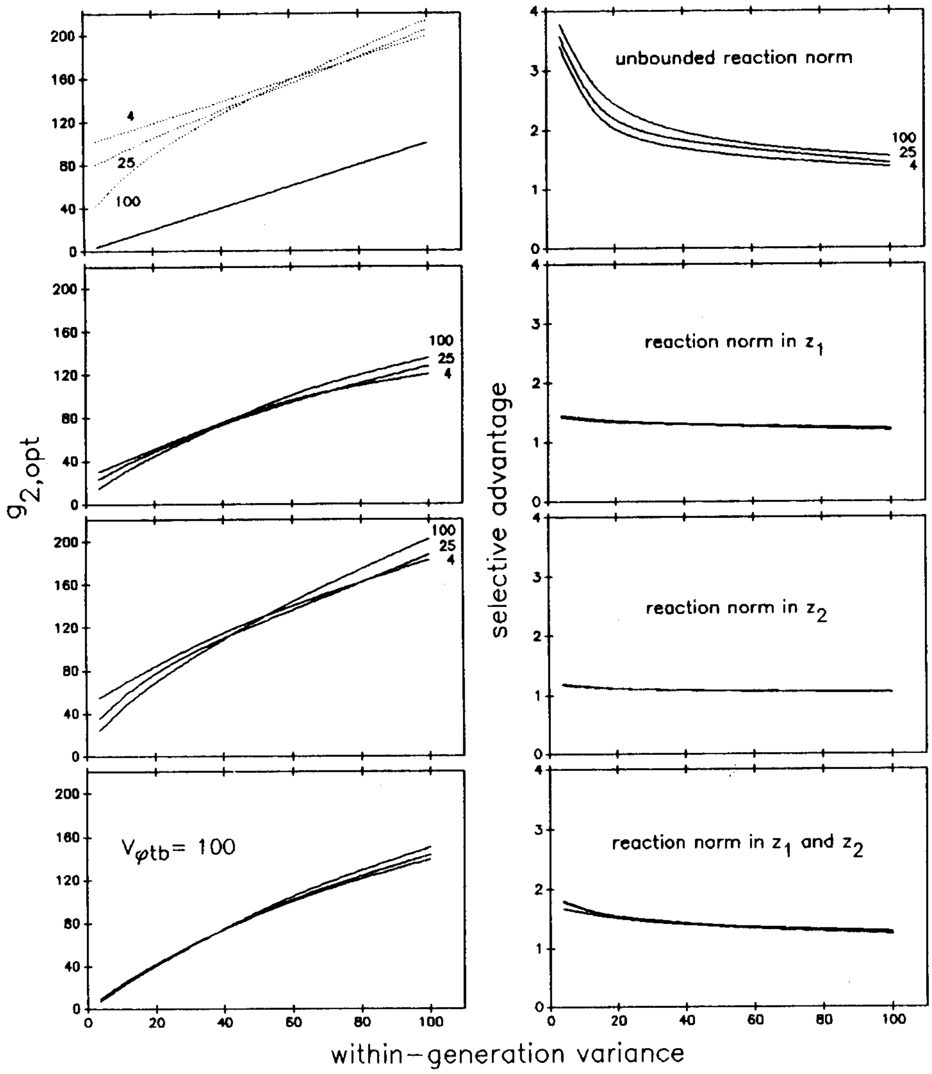

Fig. 6. The same as Figure 5 but with a high level of between-generation variance ($V_{\phi tb} = 100$).

period, and the cost to fitness of such a postponement of maturation will eventually outweigh the advantages of the reaction norm.

Two factors that we have not considered could reduce the selective advantage of reaction norms. First, developmental noise for the tolerance curve parameters, which we assumed to be of negligible importance, will reduce their heritability, and hence their response to selection. Second, developing individuals will almost always have less than the perfect knowledge of their future environment that we have assumed.

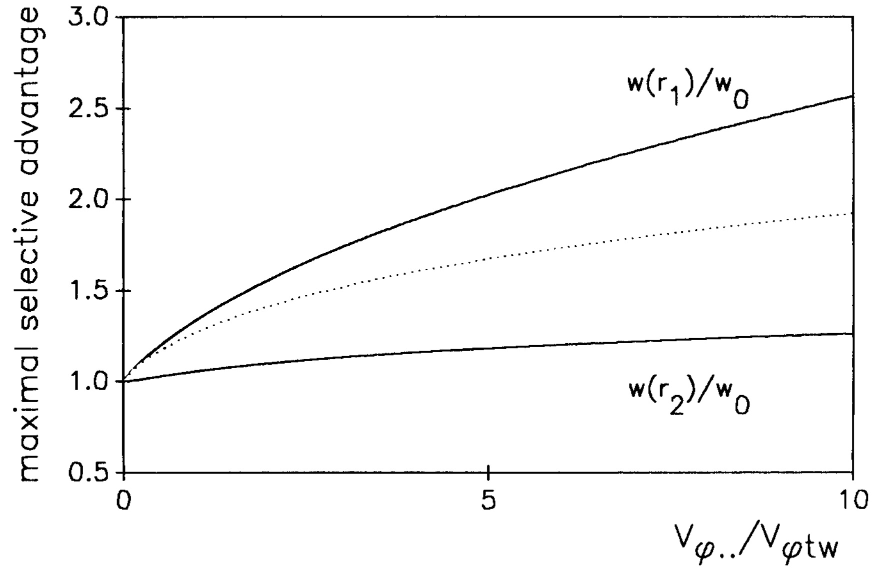

Fig. 7. The maximum selective advantage of reaction norms in z_1 and z_2 as a function of the relation between the environmental variances ($V_{\phi s}$ spatial, $V_{\phi tw}$ within generations, $V_{\phi tb}$ between generations), assuming unbounded reaction norms. The dotted line gives the selective advantage of a reaction norm in z_1 assuming no correlated response in the genetic value for the breadth of adaptation, i.e. g_2 retains the value which is optimal without a reaction norm. $V_{\phi ..} = V_{\phi s} + V_{\phi tb}$.

Our results show, in a fairly general fashion, that any reaction norm that moves the mode of the tolerance curve towards the current environmental state will be selected for more strongly than a reaction norm that modifies the breadth of adaptation. Thus, under an adaptational hypothesis, we anticipate that appropriate empirical studies will demonstrate more pronounced reaction norms for the position of the mode than for the width of the tolerance curve.

A second prediction that emerges from the theory is that a strong reaction norm for the mode of the tolerance curve will induce correlated selection for a smaller breadth of adaptation. In the absence of a reaction norm for the mode, a certain level of breadth of adaptation has to be maintained so that the genotype can remain viable in the range of environments to which it is exposed. In our model, this is an expensive strategy because genotypes obtain a broad level of adaptation (and correspondingly, relatively high fitness in extreme environments) only at the expense of fitness in their optimum environment. This need to maintain a broad tolerance curve can be avoided when there is a reaction norm in the mode. Thus, we predict a negative correlation between the magnitude of a reaction norm in the mode and the breadth of adaptation.

In our earlier work that did not employ reaction norms, we deomonstrated that the realized breadth of adaptation for a genotype, which is a function of V_{E1} and V_{E2} as well as g_2 is expected to take on values close to $V_{\phi tw}$ under an adaptational hypothesis (Lynch and Gabriel, 1987). The work presented above (Figures 5 and 6) indicates that this conclusion even extends to cases in which there are strong reaction norms for the mode and/or breadth of adaptation. This suggests the interesting possibility that genotype-specific measures of the breadth of adaptation may be correlated with the average within-generation temporal variation perceived by individuals.

Our results suggest that selection for strong reaction norms for fitness-related characters is most likely to occur in environments with low levels of within-generation but high levels of between-generation and spatial components of environmental variance. This seems to be consistent with the high degree of phenotypic plasticity often seen in planktonic organisms and in multivoltine insects. It is also consistent with the high levels of plasticity in most terrestrial plants, which experience high levels of spatial heterogeneity relative to more motile organisms, which can average out their spatial environment.

Our work does not address explicitly the issue of genotype × environment interaction, since we have not investigated the ways in which polygenic mutation and possibly the patterns of spatial and temporal variation in the environment might interact to maintain genetic variation for tolerance curve parameters within populations. We simply point out that genotype-specific tolerance curves must induce genotype × environment interaction provided they are not parallel.

We have assumed that the genotypic values g_1, g_2, and b are not mutually constrained by pleiotropic effects. However, it is conceivable that the reaction norm boundary b is genetically coupled to the breadth of adaptation $\sqrt{g_2}$. If a genotype is equipped to live in extreme environments via a high g_2, the boundary for the reaction norm in z_1 may be relatively high as well. Only minor changes in behavior, biochemistry, and/or physiology may be required to shift the mode of the tolerance curve. On the other hand, a low value of g_2 may imply that major (possibly unattainable) qualitative changes in the phenotype would be required for adaptation to extreme environments via a reaction norm. However, as we have noted above, a high b for the reaction norm in the mode of the tolerance curve may induce correlated selection for a lower g_2. Thus, the problem is by no means clear cut.

An unresolved issue concerns parameter estimation. In our previous paper, we showed how the determinants of a genotype's tolerance curve (g_1, g_2, V_{E1}, V_{E2}) might be estimated by a maximum-likelihood procedure if the conditional phenotype distributions of z_1 and z_2 are approximately normal. This would require that the genotype be propagated clonally and that lifetime fitness be assayed for a large number of individuals grown at different points over the environmental gradient. The need to assume normality and the large sample size requirements do not inspire confidence that the fundamental determinants of tolerance curves can be established empirically, and the possibility of reaction norms for z_1 and/or z_2 raises even more difficulties.

Thus, the best that one can probably accomplish in tolerance curve analysis is the identification of the general form of the function for different genotypes and the magnitude of the noise around it, keeping in mind that any specific function might be a product of many different combinations of the subsidiary traits. Some indication as to whether reaction norms exist for z_1 and/or z_2 might be obtained by raising groups of individuals in different environments through development, and then evaluating whether the form of the tolerance curve changes as a function of the juvenile environment.

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References

Bradshaw, A. D. 1965. Evolutionary significance of phenotypic plasticity in plants. Adv. Genetics 13: 115–155.

Gabriel, W. 1987. The use of computer simulation to evaluate the testability of a new fitness concept, pp. 82–88. *In* D P. F. Möller (ed.) Systems analysis of biological processes. Advances in Systems Analysis. Vol. 2. Vieweg, Braunschweig.

Gabriel, W. 1988. Quantitative genetic models for parthenogenetic species, pp. 73–82. *In* G. de Jong (ed.) Population genetics and evolution. Springer, Berlin.

Gregorius, H.-R. 1977. The genotype × environment-to-phenotype relationship. Theor. Appl. Genet. 49: 156–176.

Jong, G. de 1990. Quantitative genetics of reaction norms. J. evol. Biol. 3: 447–468.

Lynch, M., and W. Gabriel. 1987. Environmental tolerance. Amer. Nat. 129: 283–303.

Scheiner, S. M., and R. F. Lyman. 1989. The genetics of phenotypic plasticity. I. Heritability. J. evol. Biol. 2: 95–108.

Schlichting, C. D. 1986. The evolution of phenotypic plasticity in plants. Ann. Rev. Ecol. Syst. 17: 667–693.

Schmalhausen, I. I. 1949. Factors of evolution: the theory of stabilizing selection. Blakiston. Philadelphia, PA.

Stearns, S. C., and J. C. Koella. 1986. The evolution of phenotypic plasticity in life-history traits: predictions of reaction norms for age and size at maturity. Evolution 40: 893–913.

Via, S. 1984. The quantitative genetics of polyphagy in an insect herbivore. II. Genetic correlations in larval performance within and among host plants. Evolution 38: 896–905.

Woltereck, R. 1909. Weitere experimentelle Untersuchungen über Artveränderung, speziell über das Wesen quantitativer Artenunterschiede bei Daphniden. Verh. Dtsch. Zool. Ges. 1909: 110–172.

Appendices

I. The optimal phenotypic values

For the Gaussian fitness function $w = (2\pi z_2)^{-1/2} \exp(-c^2/2z_2)$ with $z_2 \neq 0$ and c^2 independent of z_2, w is maximized with respect to z_2 at $z_2 = c^2$. Therefore, we obtain from Equation (1) for a fixed environmental state $\phi \neq z_1$,

$$z_{2,opt} = (z_1 - \phi)^2 . \tag{A1}$$

More generally, the optimal phenotypic values depend on the temporal variability within generations. If ϕ is temporally distributed around the mean ϕ_s, with variance $V_{\phi tw}$, one has to maximize the geometric mean fitness:

$$\left[\prod_{i=1}^{n} (2\pi z_2)^{-1/2} \exp\left(-\frac{(z_1-\phi_i)^2}{2z_2}\right)\right]^{1/n} = (2\pi z_2)^{-1/2} \exp\left(-\sum_{i=1}^{n} \frac{(z_1-\phi_i)^2}{2nz_2}\right) \tag{A2}$$

Letting $\overline{\phi^2} = V_{\phi tw} + \phi_s^2$, with $n \to \infty$,

$$c = \sum_{i=1}^{n} \frac{(z_1-\phi_i)^2}{n} = z_1^2 - 2z_1\phi_s + \overline{\phi^2} = (z_1-\phi_s)^2 + V_{\phi tw}\,. \tag{A3}$$

Thus,

$$z_{2,opt} = (z_1-\phi_s)^2 + V_{\phi tw}\,. \tag{A4}$$

It is obvious from Equation (A2) that the optimal z_1 is equal to ϕ_s. Thus, if the reaction norm in z_1 is perfect,

$$z_{2,opt} = V_{\phi tw}\,. \tag{A5}$$

II. The optimal genotypic values

The environmental state experienced by an individual at any point in time and space can be written as

$$\phi = \phi_E + \delta_s + \delta_{tw} + \delta_{tb} \tag{A6}$$

with ϕ_E being the long-term average environment and δ_s, δ_{tw}, and δ_{tb} being the stochastic spatial (s) and temporal deviations within (w) and between (b) generations. These deviations have the respective variances $V_{\phi s}$, $V_{\phi tw}$, and $V_{\phi tb}$ and expectations equal to zero.

The long-term geometric mean fitness of a genotype with properties (g_1, g_2) is

$$w(g_1, g_2) = \lim_{T\to\infty}\left(\prod_{t=1}^{T} w(g_1, g_2, t)\right)^{1/T} \tag{A7}$$

where $w(g_1, g_2, t)$ is the mean genotypic fitness in generation t obtained after integrating over the developmental noise, the reactive deviations contributing to the phenotype, and the spatial and temporal components of variability of the environmental state. For an individual with phenotype (z_1, z_2) living at the average environmental state $\phi_s = \phi_E + \delta_s + \delta_{tb}$, it follows from (A2) and (A3) that

$$w(z_1, z_2 \mid \phi_s) = (2\pi z_2)^{-1/2} \exp\left(-\frac{(z_1-\phi_s)^2 + V_{\phi tw}}{2z_2}\right) \tag{A8}$$

(after Lynch and Gabriel, 1987). When averaged over the distribution of z_1 and z_2 conditional on g_1 and g_2, and over the spatial variance, Equation (A8) yields $w(g_1, g_2, t)$.

III. Approximations of mean fitness

Although the optimal value of g_1 is always equivalent to the long-term average environmental state, evaluation of the optimal g_2 often requires numerical computation (Lynch and Gabriel, 1987). We now show that some useful approximations can be obtained if the developmental noise is assumed to be negligible ($V_{E1} = V_{E2} \simeq 0$). We will also assume that g_1 is optimized, although violations of this assumption involve only minor modifications (Lynch and Gabriel, 1987). Without any loss of generality, we assume a scale has been chosen such that the long-term average environmental state ϕ_E is zero.

No reaction norm

Letting $g_2 = z_2$ (since e_2 is assumed to be zero) and $\phi_s = \delta_{tb} + \delta_s$ in (A8) and integrating over the spatial states of the environment

$$w(g_1, g_2, t) = (2\pi V_{\phi s})^{-1/2} \int w(g_1, g_2 \mid \phi_s) \exp\left(-\frac{\delta_s^2}{2V_{\phi s}}\right) d\delta_s .$$

After taking the geometric mean over generations, this yields

$$w(g_1, g_2) = [2\pi(V_{\phi s} + g_2)]^{-1/2} \exp\left[-\left(\frac{V_{\phi tw}}{2g_2} + \frac{V_{\phi tb}}{2(V_{\phi s} + g_2)}\right)\right]. \qquad \text{(A9)}$$

It is possible to calculate the optimum g_2 from (A9), but it requires cubic equations with rather complicated solutions. Further simplification is possible if the spatial and between-generation environmental variances can be treated as approximately additive,

$$V_{\phi..} = V_{\phi s} + V_{\phi tb} . \qquad \text{(A10)}$$

Setting $V_{\phi tb} = 0$ and replacing $V_{\phi s}$ by $V_{\phi..}$ in Equation (A9)

$$w(g_1, g_2) = [2\pi(V_{\phi..} + g_2)]^{-1/2} \exp\left(-\frac{V_{\phi tw}}{2g_2}\right). \qquad \text{(A11)}$$

This approximation is quite good, as justified by computer simulation, if the between-generation variance is moderate compared to the others. From this expression, the optimal g_2 is found to be

$$g_{2,opt} = \frac{V_{\phi tw}}{2}\left[1 + \left(1 + \frac{4V_{\phi..}}{V_{\phi tw}}\right)^{1/2}\right]. \qquad \text{(A12)}$$

The approximate maximum long-term mean fitness without a reaction norm, w_0, is obtained by substituting (A12) into (A11).

Unbounded reaction norm in z_1

An unbounded reaction norm in z_1 implies that $z_1 = \phi_s$ in (A8). Fitness is then independent of the spatial and between-generation components of environmental variance. The optimum g_2 is equal to $V_{\phi tw}$ as noted in (A5), and the expected fitness of the optimal genotype is simply,

$$w(r_1) = (2\pi e V_{\phi tw})^{-1/2}. \tag{A13}$$

Unbounded reaction norm in z_2

In this case, (A8) reduces with (A4) to

$$w(g_1, g_2 \mid \phi_s) = [2\pi e(V_{\phi tw} + \phi_s^2)]^{-1/2} \tag{A14}$$

The expected fitness of the optimal genotype then becomes

$$\begin{aligned} w(r_2) &= (2\pi V_{\phi ..})^{\;1/2} \int w(g_1, g_2 \mid \phi_s) \exp\left(-\frac{\phi_s^2}{2V_{\phi ..}}\right) d\phi_s \\ &= \frac{K_0[V_{\phi tw}/4V_{\phi ..}]}{2\pi(eV_{\phi ..})^{1/2}} \exp\left(\frac{V_{\phi tw}}{4V_{\phi ..}}\right) \end{aligned} \tag{A15}$$

where K_0 is the modified Bessel function of the second type (Hankel's function).

IV. Maximum selective advantages of the reaction norms

Dividing Equation (A13) by w_0, we obtain the maximum selective advantage for a reaction norm for the mode of the tolerance curve,

$$\frac{w(r_1)}{w_0} = \left(\frac{V_{\phi ..}}{V_{\phi tw}} + \frac{\alpha}{2}\right)^{1/2} \exp\left(\frac{1}{\alpha} - \frac{1}{2}\right). \tag{A16}$$

where $\alpha = 1 + (1 + 4V_{\phi ..}/V_{\phi tw})^{1/2}$. Equation (A13) assumed that g_2 was optimized in coevolution with the reaction norm for z_1. If instead, we assume that g_2 remains fixed at the optimum expected in the absence of a reaction norm as given by Equation (A12), then

$$\frac{w(r_1 \mid g_2)}{w_0} = \left(1 + \frac{2V_{\phi ..}}{\alpha V_{\phi tw}}\right)^{1/2} \tag{A17}$$

(This formula is used to calculate the dotted line in Figure 6.)

The maximum selective advantage for a reaction norm for the breadth of adaptation is

$$\frac{w(r_2)}{w_0} = (2\pi)^{-1/2} \cdot K_0[V_{\phi tw}/4V_{\phi ..}] \cdot \left(1 + \frac{\alpha V_{\phi tw}}{2V_{\phi ..}}\right)^{1/2} \exp\left(\frac{1}{\alpha} - \frac{1}{2} + \frac{V_{\phi tw}}{4V_{\phi ..}}\right). \tag{A18}$$

Equation (A16) shows that as $V_{\phi ..}$ becomes much smaller than $V_{\phi tw}$, $\alpha \rightarrow 2$, and the selective advantage of the reaction norm for z_1 disappears because $w(r_1)/w_0 \rightarrow 1$. The same is true for the reaction norm in z_2. Hankel's function can be approximated by $K_0[V_{\phi tw}/4V_{\phi ..}] \simeq (2\pi V_{\phi ..}/V_{\phi tw})^{1/2}$ for $V_{\phi tw}/4V_{\phi ..} > 10$ so that (A18) converges to Equation (A16), leading to $w(r_2)/w_0 \rightarrow 1$ as $V_{\phi ..}/V_{\phi tw} \rightarrow 0$.

The maximum selective advantage of a reaction norm in z_1 is always greater than that for one in z_2, i.e. $w(r_1) > w(r_2)$. From Equations (A13) and (A15),

$$\frac{w(r_1)}{w(r_2)} = \frac{(2\pi V_{\phi ..}/V_{\phi tw})^{1/2}}{K_0[V_{\phi tw}/4V_{\phi ..}]\exp[V_{\phi tw}/4V_{\phi ..}]}. \tag{A19}$$

This function increases monotonically with $V_{\phi ..}/V_{\phi tw}$ and converges to 1 as $V_{\phi ..}/V_{\phi tw} \rightarrow 0$.

If there are unbounded reaction norms simultaneously in z_1 and z_2, the maximum selective advantage is given by Equation (A16). This follows from Equation (A13), because when the reaction norm for z_1 is unbounded, the optimal g_2 leads to a fitness that cannot be increased any further by a reaction norm in z_2.

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