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QUANTITATIVE GENETICS AND THE EVOLUTION OF REACTION NORMS

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Abstract.—We extend methods of quantitative genetics to studies of the evolution of reaction norms defined over continuous environments. Our models consider both spatial variation (hard and soft selection) and temporal variation (within a generation and between generations). These different forms of environmental variation can produce different evolutionary trajectories even when they favor the same optimal reaction norm. When genetic constraints limit the types of evolutionary changes available to a reaction norm, different forms of environmental variation can also produce different evolutionary equilibria. The methods and models presented here provide a framework in which empiricists may determine whether a reaction norm is optimal and, if it is not, to evaluate hypotheses for why it is not.

Key words.—Genetic constraints, heterogeneous environments, infinite-dimensional traits, optimization, quantitative traits, reaction norm.

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A reaction norm describes the phenotypes that a genotype can produce across a range of environments (Woltereck, 1909; Johannsen, 1909, 1911). Ever since Schmalhausen (1949) introduced the concept of reaction norms to modern studies of evolution, biologists have viewed the set of phenotypic responses by a trait to environmental variation as a metacharacter that can be molded by selection. Two views have developed that place different emphases on the factors dominating the evolution of reaction norms. The first highlights the importance of fitness optimization in the evolution of reaction norms (Gause, 1947; Schmalhausen, 1949; Lerner, 1954; Waddington, 1957; Bradshaw, 1965). The second underscores the significance of evolutionary constraints in preventing organisms from responding optimally in every environment (MacArthur, 1961; Levins, 1968; Huey and Hertz, 1984). Using a quantitative genetic perspective, this paper examines how selection and constraints can interact to determine the outcome of reaction norm evolution. This framework also provides methods for determining empirically whether observed reaction norms are selectively optimal or evolutionarily constrained.

Previous workers who have stressed the role of optimization recognized that selection favors different reaction norms under different conditions. If selection favors organisms that adjust their phenotypes in response to the environments they inhabit, then phenotypic plasticity is favored (Gause, 1947; Bradshaw, 1965). Bradshaw (1965), for example, suggests that plasticity in the timing of germination may be advantageous because of the success realized by seeds that develop under favorable conditions. Alternatively, organisms that maintain constant developmental pathways under variable conditions may be selectively favored. In this case, homeostatic reaction norms are advantageous (Schmalhausen, 1949; Lerner, 1954; Waddington, 1957).

Those who emphasize the importance of evolutionary constraints agree that selection favors any organism that can produce an optimal phenotype in every environment it encounters. At the same time, they observe that organisms often fail to respond optimally in every environment. For instance, Huey and Hertz (1984) showed that lizards are unable to maximize sprint speed over all temperatures even though it would be advantageous for them to do so. Suboptimal performance, it is argued, results from the presence of evolutionary constraints.

These constraints are realized through fitness trade-offs within the norm of reaction. Increased adaptation to one set of environ-

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ments can only be achieved at the cost of decreased adaptation to other environments. Consequently, "a jack-of-all-trades is the master of none" (MacArthur, 1961) because adapting to a broad range of environments will often cause a loss of fitness in any single environment. This view leads to a hydraulic metaphor: as selection molds a reaction norm, the trade-offs cause the area under it to act like an incompressible fluid (Fig. 1, upper panel). A conservation principle of this sort is used in several models for the evolution of reaction norms (Levins, 1968; Huey and Slatkin, 1976; Lynch and Gabriel, 1987).

While it is commonly assumed that fitness trade-offs within reaction norms guide their evolution, the evidence for such trade-offs is inconsistent (Huey and Hertz, 1984; Huey and Kingsolver, 1989). The results from two experiments selecting for tolerance to high temperatures illustrate this with an interesting contrast. Dallinger (1887) selected on flagellates over seven years and was able to increase their tolerance from 18°C to 70°C. Trade-offs were demonstrated by his finding that the selected population was no longer able to survive at the initial temperature (analogous to the upper panel in our Fig. 1). Bennett et al. (1990), using *E. coli*, also selected for tolerance to high temperatures. After 200 generations, the growth rate at high temperature (42°C) was increased by 7%. Unlike Dallinger, however, they found no evidence for trade-offs: the lines selected to high temperatures also grew faster at the original temperature (37°C) than did unselected controls. There is thus no evidence for the existence of trade-offs within the norm of reaction in this study (analogous to the lower panel in our Fig. 1).

If adaptation to one environment does not necessarily sacrifice adaptation to another, what prevents populations from enhancing their performance across all environments? One possibility is that there are fitness trade-offs involving characters other than performance across the environmental gradient under study (Huey and Hertz, 1984). In *Phlox drummondii*, for example, a phenotypic response that increases total weight will, at the same time, decrease the efficiency of flower production (Schlichting, 1986 p. 675). A central question for those who advocate the importance of trade-offs is: How commonly is evolution constrained by fitness trade-offs within the reaction norm?

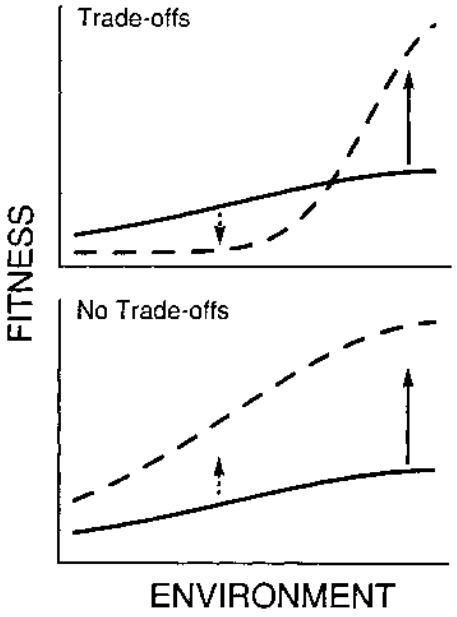

FIG. 1. Comparison of the effects of fitness trade-offs versus no trade-offs. Solid curves indicate initial fitness curves. With trade-offs (upper panel), fitness increases realized over some environments (solid arrow) result in decreases over others (broken arrow). With no trade-offs (lower panel), fitness increases over some environments (solid arrow) do not require loss of fitness in others (broken arrow).

To resolve the influences of optimality and constraint in molding reaction norms, methods are needed to determine whether observed reaction norms are optimal. If they are not, we would like to determine whether the constraints may be attributed to trade-offs that occur within reaction norms or to other factors.

Two approaches have been used in previous theoretical studies of the evolution of reaction norms. The first treats environmental variation as a continuous variable (such as temperature) and uses optimization to predict the reaction norm favored by selection. Huey and Slatkin (1976) follow this approach in a study of the evolution of lizard thermoregulation. They focus on the costs and benefits of thermoregulation when environments vary temporally within generations. Lynch and Gabriel (1987) likewise

use optimization in analyzing a model of the evolution of environmental tolerance in temporally and spatially variable environments. Both of these studies assume a priori specific evolutionary trade-offs by postulating that the area under the reaction norm is evolutionarily fixed (see Fig. 1). Stearns and Koella (1986) also use optimization in their study of reaction norms for size and age at maturity. They also assume constraints a priori, in this case a specific trade-off between the two traits. Like all optimality models, these studies are based on functional constraints that are not explicitly related to measurable genetic parameters, and they do not provide a way to predict the evolution of a reaction norm when it is not at equilibrium (see Charlesworth, 1990).

The second approach considers discrete environments (such as the host species of a herbivorous insect) and assumes a quantitative-genetic basis for the inheritance of the reaction norm. A character's expression in two (or more) different environments can be viewed as two (or more) genetically correlated characters (Falconer, 1952). Via and Lande (1985) use this idea to formulate quantitative-genetic models for the evolution of a trait that is expressed differently in two environments [see Via (1987) for more environments].

The quantitative-genetic models have several advantages over the optimization models. They predict evolutionary trajectories in addition to equilibria, are based on measurable parameters of inheritance, explicitly account for between-individual variation, and do not assume a priori the existence of fitness trade-offs. This approach thus provides a natural framework for empirical study of adaptation and constraint in the evolution of reaction norms. A limitation of previous quantitative-genetic models is that they do not apply to continuous forms of environmental variation (such as temperature), unlike the optimization models.

In this paper we integrate the strengths of both previous approaches by extending the quantitative-genetic approach to reaction norms that vary as a function of a continuous environmental variable. Because the phenotype produced in each environment can be viewed as a different character and since there is a continuum of environmental states, these types of reaction norms represent *infinite-dimensional characters*. We describe a basic quantitative-genetic model for the evolution of infinite-dimensional characters introduced by Kirkpatrick and Heckman (1989) and show how it may be applied to the evolution of continuous reaction norms.

Reaction norms are selected in response to environmental variation. We investigate reaction norm evolution under several forms of spatial and temporal environmental heterogeneity. Our analyses highlight how the presence or absence of genetic constraints affects evolution. First, two models of spatial heterogeneity, soft and hard selection, are presented. Second, we consider evolution when the environment varies temporally within a generation and when the environment fluctuates between generations but is constant within them. Following these analyses, we present simulation results that illustrate how the forms of environmental variation and genetic variation can influence evolutionary trajectories and even the equilibria that are reached. Finally, we discuss how the quantities that appear in the models can be empirically estimated and used to test hypotheses regarding the roles of selection and constraints in influencing the evolution of reaction norms.

The Models

Consider the reaction norm of a trait, denoted by $_{\delta}(\cdot)$, that varies as a function of a continuous environmental variable. For example, $_{\delta}(x)$ could represent lizard sprint speed capacity at temperature x (see Hertz et al., 1988). The population mean reaction norm is denoted $\bar{\delta}$ so that the mean phenotype expressed in environment x is simply $\bar{\delta}(x)$. No assumptions are made regarding the particular form of $\bar{\delta}$. In fact, $\bar{\delta}$ need not even be continuous. It might, for example, represent expression of cannibalistic and omnivorous tadpole morphs as a function of a pond's chemical cues (Pfennig, 1989). Our goal is to determine the evolutionary change in $\bar{\delta}$.

We assume that the reaction norm of an individual can be represented by the sum of two (square-integrable) functions. The

first function is the additive-genetic component inherited from the individual's parents and the second is attributable to nonadditive effects including dominance, developmental noise, and other within-environment effects (Falconer, 1981; Via and Lande, 1985; Lynch and Gabriel, 1987). These two components are defined to be statistically independent of one another and are assumed normally distributed on an appropriate scale, as is standard in quantitative genetics (Falconer, 1981; Bulmer, 1985; see below). The normality assumptions may often provide reasonable approximations to a more complicated reality. The models also assume that generations are nonoverlapping, that inheritance is autosomal, and that effects due to random genetic drift, mutation, epistasis, and recombination are all negligible compared to selection.

Normal distributions of functions, termed Gaussian stochastic processes, are natural extensions of multivariate normal distributions. In the same way that a multivariate normal process is characterized by a mean vector and a covariance matrix (Bulmer, 1985), a Gaussian process is characterized by a mean function and a covariance function (Doob, 1953). For our models of reaction norms, a Gaussian process of additive-genetic effects is characterized by a mean function $\bar{z}(\cdot)$ and a covariance function $G(\cdot, \cdot)$. In particular, $G(x, y)$ is the additive-genetic component of the covariance between the phenotype $z(x)$ that an individual would express in environment x, and the phenotype $z(y)$ that it would express in environment y. Similarly, $G(x, x)$ is the additive-genetic component of variance for $z(x)$. The patterns of additive-genetic variation summarized by G result from changes in gene expression (Paterson et al., 1991) and physiological activity of gene products (Hochachka and Somero, 1984) in response to changes in the environment. If $G(x, y)$ is positive then genes which increase $z(x)$ will tend to increase $z(y)$, while if $G(x, y)$ is negative genes which increase $z(x)$ will tend to decrease $z(y)$. The additive-genetic variance $G(x, x)$ will be positive or zero depending on whether or not there is a heritable component to the variability in $z(x)$.

The evolutionary response to selection on reaction norms is described by the equation

$$\Delta\bar{z}(x) = \int G(x, y)\beta(y)\, dy, \qquad (1)$$

where $\Delta\bar{z}(\cdot)$ is the evolutionary change in the mean reaction norm after one generation of selection and $\beta(\cdot)$ is the selection gradient function (Kirkpatrick and Heckman, 1989). Integration is taken over the set of environments. This equation extends the familiar multiple characters matrix equation $\Delta\bar{\mathbf{z}} = \mathbf{G}\boldsymbol{\beta}$ (Lande, 1979).

The selection gradient function, $\beta(\cdot)$, describes the forces of directional selection on reaction norms (Lande and Arnold, 1983; Kirkpatrick, 1988). For example, a positive value of $\beta(x)$ indicates that selection favors values of $z(x)$ that are larger than average, holding all other traits $z(y)$ constant. When $\beta(x) = 0$, selection has no tendency to change the mean value of $z(x)$.

Once the selection gradient is determined for a particular form of selection, the resulting evolutionary change in $\bar{z}$ over a single generation can be determined via Equation (1) even if the additive-genetic covariance function changes between generations. In this paper, we do not model the evolution of genetic covariance functions; rather, we will assume G to have been empirically determined (as described in the Discussion). This simplifies our analyses considerably but may restrict their applicability to intermediate time scales and relatively small evolutionary changes in the mean. Recent work by Barton and Turelli (1987), Turelli (1988), and Turelli and Barton (1990) shows that evolution of a population's *mean* phenotype can often be accurately predicted for tens to hundreds of generations by a Gaussian model which assumes constant additive-genetic variances, even when the actual genetics are non-Gaussian and have nonconstant additive-genetic variances.

Although we assume G is given, β must still be determined. Two approaches can be taken. The first is to measure the selection gradient function directly. This requires data on the phenotypes and fitnesses of individuals, but no further information about the ecological setting of the population is necessary. These methods are described in the

Discussion. An alternative approach is to make assumptions regarding the forms of environmental variation that the population experiences, and then deduce the selection gradient function that would result. We pursue this approach now in order to illustrate several general principles of reaction norm evolution.

When selection is frequency-independent, there is a direct relation between the strength of selection acting on a trait and the effect that a change in the mean of that trait will have on the population's mean fitness (Wright, 1942, 1969; Lande, 1976, 1979). In the simplest case of a single trait, the selection gradient is equal to the gradient (i.e., the derivative) of the logarithm of the population's mean fitness with respect to the mean of that trait. This result extends in a natural way to cases involving multiple traits by using the gradient (i.e., the partial derivatives) of $\ln \overline{W}$ with respect to the mean of each trait (Lande, 1979). The value of the selection gradient for each trait thus reflects the effect that a small change in the population's mean for that trait has on the population's fitness, holding all other traits constant. For infinite-dimensional traits such as reaction norms, this rule continues to hold (Gomulkiewicz and Beder, unpubl. data). The value of $\beta(x)$, the selection gradient in environment x, measures the impact of a small change in $\bar{\delta}$, the mean phenotype expressed in environment x, on the logarithm of population mean fitness. This can be calculated by applying the infinite-dimensional gradient operator $\nabla_{\bar{\delta}(x)}$ to the logarithm of the population's mean fitness, $\ln \overline{W}$. That is,

$$\beta(x) \equiv \nabla_{\bar{\delta}(x)}[\ln \overline{W}]. \qquad (2)$$

In practice, $\nabla_{\bar{\delta}(x)}[\ln \overline{W}]$ is calculated using the same method of partial differentiation that is used to determine the selection gradient vector in conventional models of quantitative characters (see Appendix A and below). Technically, $\nabla_{\bar{\delta}(\cdot)}[\ln \overline{W}]$ is a functional derivative (Courant and Hilbert, 1953).

How a reaction norm evolves depends on the genetic variation present as well as the form of selection it experiences. The additive genetic covariance function G can fall into either of two categories; these have major evolutionary implications. If genetic variation is available for all conceivable evolutionary changes in $\bar{\delta}$ then G is said to be *nonsingular.* (Mathematically, a covariance function is nonsingular if integral transforms with kernal G can be inverted; see Kirkpatrick and Heckman, 1989). When G is nonsingular, the mean reaction norm will evolve until directional selection ceases (that is, $\beta(x) = 0$ for all x). If there is a reaction norm that maximizes fitness locally, an equilibrium will be reached when $\bar{\delta}$ reaches this optimum.

Alternatively, G may be *singular,* meaning that no additive genetic variation exists for certain kinds of evolutionary changes in the shape of $\bar{\delta}$. In this case, the population's mean reaction norm may stop evolving even when directional selection on it persists (that is, $\beta(x) \neq 0$ for some or all x). Thus the population can reach an evolutionary equilibrium when the mean reaction norm is not at the optimum, although evolution still maximizes fitness to the extent that it can (Via and Lande, 1985). In this situation, any adaptive evolutionary change in one part of the reaction norm necessarily produces maladaptive change in other parts (see Fig. 1 top). That is, fitness trade-offs occur within the reaction norm. For these reasons, we use the terms "singular genetic covariance function," "genetic constraints," and "trade-offs" synonymously. Both empirical analyses and theoretical considerations (Kirkpatrick and Lofsvold, 1989, unpubl. data) suggest that genetic constraints may be common for infinite-dimensional traits. Constraints may eventually be altered or disappear entirely with changes in the genetic structure of the population (Turelli, 1988; Charlesworth, 1990). Their presence will nevertheless affect the evolutionary outcome in the short term, and may do so for evolutionarily long time scales as well. We are therefore compelled to consider the possibility of constraints in the evolution of reaction norms.

Classes of Traits

There are two classes of traits whose reaction norms are of general interest (Futuyma and Moreno, 1988). At one extreme are

traits referred to as *labile* (Schmalhausen, 1949). An individual's phenotype for a labile trait adjusts rapidly to changes in the environment due to physiological and behavioral responses by the organism. One example of a labile trait is the locomotory performance of poikilotherms as a function of their body temperature (Huey and Bennett, 1987; Huey and Kingsolver, 1989). At the other extreme are traits that are termed *nonlabile*. The degree of expression of a nonlabile trait depends on the environment experienced during a sensitive period of development. Thereafter, it is fixed so that only one of the possible phenotypes in an individual's reaction norm is expressed during its lifetime. In a classic experiment with *Drosophila melanogaster*, Waddington (1953) induced development of a crossveinless wing condition by exposing pupae to heat shock. Wing venation is thus an example of a nonlabile trait. We also classify as nonlabile any trait that can only be expressed at one stage of ontogeny, such as age at maturity (see Trexler, 1989).

Forms of Environmental Heterogeneity

Reaction norms result from the interaction of organisms with their environments. In a uniform habitat, the environmental contribution to the phenotypes of individuals is constant and so reaction norms are not expressed or selected. Reaction norms do experience selection, however, when the environment varies in either space or time.

An individual can experience temporal heterogeneity through two causes. First, the habitat it occupies may change. Second, spatial variation is translated into temporal variation if the individual moves over distances that are larger than the size of the environmental patches. From an evolutionary standpoint, this results in the same pattern of selection as is experienced by sessile organisms in a changing habitat, and we therefore treat both situations as temporal variation. Our discussions of spatial variation, in contrast, refer to situations in which the sizes of environmental patches are large relative to the movements of individuals, that is, when the spatial "grain" of the environment (Levins, 1968) is coarse. Each patch may encompass many individuals or may be inhabited by only a single individual. In the following two sections, we consider selection on reaction norms generated by spatial and temporal variation.

Spatial Variation

Selection will act on a reaction norm when individuals are distributed over environmental patches that affect the expression of a trait or when different patches favor different values for the trait. Our models of spatial heterogeneity assume that the relative frequencies of different patch types remain constant from generation to generation. Individuals are distributed randomly among patches, which implies that there is no habitat choice. Mating is random among all the different patches so that each patch is actually a subpopulation within an interbreeding population. This assumption implies that the dispersal distances of selected adults or their gametes greatly exceeds the size of the patch in which they were selected. While the level of gene exchange among the patches assumed here is extreme, the techniques we introduce can be modified to model more restricted migration patterns, including habitat selection.

The environment in each patch is assumed to be constant during each generation. This implies that within a patch, expression of both labile and nonlabile traits is constant. Any distinction between nonlabile or labile traits in these models of spatial heterogeneity is therefore moot (see Caswell, 1983).

Genetic models of evolution in patches distinguish between two forms of population regulation, termed hard and soft selection (Wallace, 1968; Christiansen, 1975). Under hard selection, the contribution of a particular patch to the gamete pool is proportional to the mean fitness of individuals inhabiting the patch (Dempster, 1955). This occurs when the strength of density-dependent population regulation within patches is weak. For example, seeds in certain plant populations may be sparsely dispersed over various patches. Mortality selection then produces different numbers of adults in each patch depending upon the viabilities of the zygotes dispersed to it. Under soft selection, the contribution of gametes from each type of patch is fixed and independent of the mean fitness of its inhabitants (Levene, 1953). Soft

selection operates when strong density-dependence within each patch determines the total number of individuals that survive there, regardless of the genotypes of the initial inhabitants. This would be the case if, say, the number of adult plants supported in a patch was limited by access to sunlight. If juveniles can grow only in spaces vacated by deaths, then the number of adults surviving in each patch would be independent of the genotypes of the residents. Holsinger and Pacala (1990) discuss the types of traits that are expected to be soft and hard selected.

In the following models of hard and soft selection, we let $f(x)$ denote the frequency of environmental condition x. It is assumed that an individual inhabiting an environmental state x expresses phenotype $_\delta(x)$ throughout its lifetime and has fitness $W(_\delta, x)$. Finally, the mean fitness of individuals inhabiting environment x is denoted by $\overline{W}(x)$.

Soft Selection

Given soft selection and random dispersal of gametes, individuals selected within environments ranging from x to $x + dx$ contribute a fraction $f(x)dx$ of the offspring to the next generation. The evolutionary change in the mean reaction norm due to selection is found using Equation (1) with selection gradient

$$\beta(x) = \nabla_{\delta(x)}\left[\int f(y) \ln \overline{W}(y)\, dy\right]. \quad (3)$$

Equation (3) is found by extending the multiple characters result (Via and Lande, 1985) to infinite dimensions (Appendix A). The integration is taken over environments.

According to the definition (2), the integral in brackets on the right hand side of (3) represents the mean log fitness for the whole population. The population mean fitness under soft selection, $\overline{W}_{\text{soft}}$, is thus the geometric mean fitness (Ewens, 1979 p. 293):

$$\overline{W}_{\text{soft}} = \exp\left[\int f(y) \ln \overline{W}(y)\, dy\right]. \quad (4)$$

Consequently, Equation (3) can be written as

$$\beta = \nabla_{\bar{\delta}}[\ln \overline{W}_{\text{soft}}]. \quad (5)$$

The geometric mean fitness (4) defines an adaptive topography for evolution. Along with the additive-genetic covariance function G, this adaptive topography determines the rate and direction of evolution of the mean reaction norm. When the selection gradient function $\beta(x)$ is equal to zero for all x, the mean fitness in each environment is locally maximized by this form of selection (Wright, 1969; Via and Lande, 1985).

We now present a specific example to illustrate the form of a selection gradient function under soft selection. Consider stabilizing selection that favors the phenotype $\theta(x)$ in environment x. If x represents ambient temperature, then $_\delta(x)$ might represent the body temperature that a lizard maintains at that ambient temperature and $\theta(x)$ would be the optimal body temperature. Weak stabilizing selection can be approximated by a Gaussian fitness function with optimum $\theta(x)$:

$$W(_\delta, x) \propto \exp\left\{-\frac{[_\delta(x) - \theta(x)]^2}{2\omega^2(x)}\right\}, \quad (6)$$

where $\omega^2(x)$ is inversely related to the strength of stabilizing selection in environment x. We will assume that $\omega^2(x)$ is much larger than the phenotypic variance of $_\delta(x)$. The selection gradient function for soft selection may be found using the indirect method discussed in Appendix A, which shows that

$$\beta(x) \approx f(x)\frac{\theta(x) - \bar{\delta}(x)}{\omega^2(x)}. \quad (7)$$

This result reveals that the strength of selection in environment x is proportional to the difference between the expressed mean and optimum, $\theta(x) - \bar{\delta}(x)$, the relative frequency of x, $f(x)$, and the strength of stabilizing selection, $1/\omega^2(x)$.

According to (7), the selection gradient vanishes when the mean reaction norm coincides with the optimum, i.e., $\bar{\delta}(x) = \theta(x)$ for all x. This means that the optimum reaction norm is an evolutionary equilibrium. Furthermore, $\bar{\delta}(x)$ will always reach $\theta(x)$ whenever there are no genetic constraints (G is nonsingular). When G is singular, however, the reaction norm will generally not be able to evolve to the optimum. The pop-

ulation's mean is constrained to evolve within a subset (technically, a manifold) of all possible reaction norms. This set of "evolutionarily accessible" reaction norms (Kirkpatrick and Lofsvold, unpubl. data) is determined by the population's initial $\bar{\delta}$ and $\mathcal{G}$ (provided the additive genetic covariances remain approximately constant). Once determined, $\bar{\delta}$ and $\mathcal{G}$ specify a unique equilibrium reaction norm whose location can be calculated directly (Kirkpatrick and Lofsvold, unpubl. data). While the mean reaction norm will generally not be optimal at equilibrium, it is the reaction norm within the set of accessible reaction norms that maximizes the population's mean fitness. Implications of this type of constrained equilibrium are illustrated by the simulations presented below.

Hard Selection

The distinguishing feature of hard selection is that the contribution by a patch to the population's gamete pool depends on the mean fitness of the individuals that inhabit the patch. Individuals occupying environments in the range $(x, x + dx)$ contribute a proportion $[\overline{W}(x)/\overline{W}_{\text{hard}}]f(x)dx$ of the offspring, where

$$\overline{W}_{\text{hard}} = \int \overline{W}(x)f(x)\,dx \qquad (8)$$

is the arithmetic mean fitness for the entire population. By extending the multivariate result (Via and Lande, 1985) to infinite dimensions (Appendix A) and using (2), it can be shown that the selection gradient for hard selection is

$$\beta(x) = \nabla_{\bar{\delta}(x)}\,[\ln \overline{W}_{\text{hard}}]. \qquad (9)$$

Comparing (9) with (5), we see that hard and soft selection are distinguished by the type of mean fitness that determines the effects of selection. For hard selection, this mean fitness is arithmetic (8) while for soft selection it is geometric (4). These mean fitnesses also determine the adaptive topographies for the corresponding one-locus models (Dempster, 1955; Li, 1955).

When there is weak Gaussian stabilizing selection within each environment (Eq. 6), the selection gradient function under hard selection is

$$\beta(x) \approx \left[f(x)\frac{\overline{W}(x)}{\overline{W}_{\text{hard}}}\right]\frac{\theta(x) - \bar{\delta}(x)}{\omega^2(x)}. \qquad (10)$$

This expression may be found by applying the same procedures used to obtain (7) (Appendix A).

In the absence of genetic constraints ($\mathcal{G}$ nonsingular), Equation (10) implies that the population will always evolve to the optimum reaction norm θ, as it will under soft selection. When genetic constraints are present, however, it is difficult to fully analyze the possible evolutionary outcomes. Unlike the case of soft selection, hard selection results in a selection gradient that is a nonlinear function of $\bar{\delta}$, which prevents us from solving analytically for the equilibrium. Simulation can be used, however, to find the equilibrium for any particular case of interest. The equilibrium that is reached depends on the population's initial reaction norm, as is also true for soft selection. In contrast to soft selection, we have found examples in which there is more than one locally stable equilibrium within the set of evolutionary accessible reaction norms specified by $\mathcal{G}$ and the initial mean reaction norm $\bar{\delta}$. This further highlights the importance of a population's history to its ultimate fate: if two populations diverge in their mean reaction norms and are then subject to identical forms of hard selection, they may evolve to different equilibria even if the same set of reaction norms are evolutionarily accessible to both. The role of history can also be an important factor in the evolution of populations that experience temporal fluctuations, which is our next topic.

Temporal Variation

We now consider models for the evolution of reaction norms when environments vary temporally. While natural populations encounter temporal variation on all scales, we will simplify our discussion considerably by focussing on two extremes. The first occurs when variation is within generations so that individuals experience a sequence of environments within their lifetimes. When treating within-generation temporal variation, we will assume that every individual experiences the same distribution of envi-

ronments (which may or may not be encountered in the same sequence) and that this distribution is the same in each generation. This situation may roughly correspond to the thermal variation experienced by long-lived poikilothermic vertebrates. The second temporal scale we consider occurs between generations. Our major simplification in this case is the assumption that individuals within the same generation all experience the same fixed environment. This model might represent, albeit roughly, the type of thermal variation encountered by alternate generations of multivoltine insects.

Labile traits are capable of responding plastically to environmental heterogeneity so that when there is within-generation variation, any individual may express a number of phenotypic states. Nonlabile traits, however, are fixed whether or not environmental conditions change. So even if we assume a constant form of fitness, $W(\bar{z}, x)$, labile and nonlabile traits require separate treatments when environments x vary within generations. In contrast, labile and nonlabile traits may be treated in the same way in our models of between-generation variation because the environment is assumed to be constant within each generation.

Within-Generation Variation

We now present models that can be used to investigate the evolution of reaction norms when environments fluctuate within generations. As mentioned before, within-generation heterogeneity affects labile and nonlabile traits differently. We present first a model for the evolution of labile traits and then a treatment of nonlabile traits.

Within-generation temporal variation will cause a labile trait to be expressed in different ways by a single individual. We will focus on a labile trait that has completed development and changes solely in response to environmental fluctuations. If the environment is changing continuously, the phenotype of a labile trait is constantly being adjusted (e.g., the adjustment of pupil size to changing light conditions). To specify the set of phenotypes that are expressed and selected within a generation, we need to describe how the environment varies. Suppose that the environmental condition at time t is given by x_t. By assumption, all individuals experience the same environmental conditions and consequently, the phenotype expressed by a member with reaction norm $\bar{z}$ at time t is $\bar{z}_t = \bar{z}(x_t)$.

To focus discussion we present a particular case of continuous within-generation temporal variation and Gaussian stabilizing selection where the selective optimum and (instantaneous) width in environment x are $\theta(x)$ and $v^2(x)$ respectively. This could approximate, for example, selection by predation on lizard sprint speed if predator activity is mediated by temperature. Appendix B contains derivations of the selection gradient for both labile and nonlabile traits when the environment varies within a generation. Applying those results to the present case shows that the selection gradient under weak selection is (approximately)

$$\beta(x) \approx \frac{T(x)[\theta(x) - \bar{z}(x)]}{v^2(x)}, \qquad (11)$$

where $T(x)$ indicates the time spent in environment x. Note that $v^2(x)$ has units of time (see Appendix B). The logarithm of the population mean fitness $\overline{W}$ corresponding to Equation (11) is defined by

$$\ln \overline{W} \approx -\frac{1}{2} \int \frac{T(x)[\theta(x) - \bar{z}(x)]^2}{v^2(x)}\, dx. \qquad (12)$$

Given that there are no genetic constraints, the mean reaction norm will evolve along the adaptive topography (12) to the optimum θ as intuition might predict. Expressions (11) and (12) show that the rate at which the optimum is approached in environment x depends on both the width, $v^2(x)$, and duration, $T(x)$, of selection in that environment. As in the previous cases, if the genetic covariance function is singular, then the optimal responses may not evolve.

We now turn to the evolution of nonlabile traits that experience within-generation environmental heterogeneity. Nonlabile traits are fixed once their development is complete. Although the details do not add substantively to our present discussion, for completeness we present in Appendix B a model for the evolution of nonlabile characters that are subjected to varying selection within generations. The major conclusion is

that, with no genetic constraints, mean reaction norms for nonlabile characters will evolve along an adaptive topography to an optimal "compromise" that accounts for the intensities and durations of within-generation fluctuations in selection. This optimal compromise will generally not be attained if genetic constraints are present.

Within-generation and between-generation temporal variation share the property that variability is encountered sequentially. Unlike the case of between-generations fluctuations (see below), however, the order in which variation is encountered within a generation need not affect the course of evolutionary change. In this respect, selection that varies within a generation is more similar to spatial variation. What sets within-generation heterogeneity apart is that, in a single generation, it can affect different parts of the reaction norm of a labile trait. It is only this form of environmental variability that selects, in every generation, lability itself.

Between-Generation Variation

In the preceding models, the population as a whole experiences a variety of environmental conditions each generation. In our model of between-generation variation, by contrast, the entire population is subjected to a single environment per generation. A consequence of this is that a population's course of evolution depends upon the sequence of environments it encounters. This differs from evolution under the models of spatial and within-generation temporal heterogeneity in which evolutionary trajectories each follow a regular course along an adaptive topography. Because of its dependence on the actual sequence of environments, an analysis of a general model for between-generation variation would have limited application. Our goal in this section is simpler: to present a basic model which investigators can adapt for the study of biologically interesting cases.

Assume that in generation n, a population encounters the environmental condition x_n. For instance, x_n might be the average photoperiod. A population member with reaction norm δ expresses the phenotype $\delta(x_n)$ and has fitness $W_n(\delta, x_n)$. To simplify notation, we assume that the form of selection is constant in time so that fitness in generation n is represented simply by $W(\delta, x_n)$. We also assume that phenotypes not expressed in environment x_n have no effect on the population mean fitness. The population mean fitness at generation n, $\overline{W}_n$, thus equals the mean fitness within environment x_n, that is, $\overline{W}_n = \overline{W}(x_n)$. It is shown in Appendix A that the selection gradient function in generation n is

$$\beta_n(y) = \nabla_{\bar{\delta}(y)} [\ln \overline{W}_n]. \tag{13}$$

The logarithm of the population mean fitness in generation n, $\overline{W}_n$, is defined by

$$\ln \overline{W}_n = \int \delta(y - x_n) \ln \overline{W}(y)\, dy, \tag{14}$$

where $\delta(\cdot)$ is the Dirac delta function. The Dirac delta function is defined such that

$$\int \delta(x - y)h(y)\, dy = \begin{cases} h(x) \text{ if } x \text{ is in the} \\ \text{domain of integration} \\ 0 \text{ otherwise} \end{cases} \tag{15}$$

(see e.g., Dettman, 1969). In our case, $h(y) = \ln \overline{W}(y)$ and therefore $\int \delta(x - y) \ln \overline{W}(y)\, dy = \ln \overline{W}(x)$ if x is in the range of environments.

As an example, consider the selection gradient function in generation n associated with Gaussian stabilizing selection (Eq. 6). It is shown in Appendix A that the selection gradient is, on the assumption of weak selection,

$$\beta_n(y) \approx \delta(y - x_n) \frac{\theta(x_n) - \bar{\delta}(x_n)}{\omega^2(x_n)}. \tag{16}$$

Equation (16) indicates that selection in generation n directly acts only on that part of the reaction norm which is expressed in environment x_n. That is, $\beta_n(y) = 0$ for every y different from x_n. The evolutionary response to selection in generation n is (using Eqs. 1, 15, and 16):

$$\Delta\bar{\delta}_n(u) = \int G(u, y)\beta_n(y)\, dy \approx G(u, x_n) \frac{\theta(x_n) - \bar{\delta}(x_n)}{\omega^2(x_n)}. \tag{17}$$

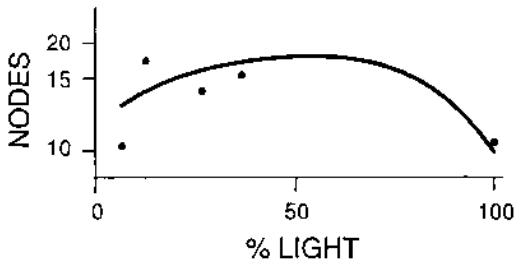

FIG. 2. Semi-log plot of mean node numbers (solid points) observed for *A. theophrasti* raised under five light environments. Curve shows the estimated population mean reaction norm determined by fitting fourth degree Legendre polynomials to the data. (For the rationale behind choosing Legendre polynomials, see Kirkpatrick et al., 1990). Overfitting the five data points was avoided by first interpolating data points using natural cubic splines (Press et al., 1988) and then performing a least-squares fit of the polynomials to the interpolated data.

From (17), it is clear that the only genetic component influencing the evolution of the trait's expression in environment u is the additive-genetic covariance between $_{\beta}(u)$ and $_{\beta}(x_n)$.

A feature unique to between-generation variation is that the selection gradient in any generation depends upon which environment is encountered (Eqs. 13 and 14). Each evolutionary trajectory depends on a particular sequence or history of environments. One consequence of this dependency is that two identical populations initiated at different times will in general follow different evolutionary paths. This contrasts with the evolutionary trajectories resulting from spatial and within-generation temporal heterogeneity which are, as we have seen, unaffected by the time at which evolution is initiated. Note, however, that if the probabilities of encountering different environments in any generation are fixed, then both population means will eventually reach a common stochastic equilibrium at which the probability of observing a particular mean phenotype in any generation is fixed (see also Gavrilets and Scheiner, 1991*a*).

SIMULATIONS

We now highlight our main theoretical findings with a series of simulations, based on a real data set, which illustrate reaction norm evolution under soft and hard selection. The first simulation shows how genetic constraints can prevent the mean reaction norm from evolving to its optimum shape under realistic conditions. The next two sets of simulations demonstrate some of the ways that the form of selection (soft versus hard) can effect evolutionary trajectories and how the modes of selection can interact with genetic constraints to determine evolutionary equilibria.

The simulations are based on data collected for a previous study of velvetleaf (*Abutilon theophrasti*) reaction norms. The data set, graciously provided to us by K. Garbutt (West Virginia University), describes the expression of node number under five levels of light transmission. Data were collected using a maternal half-sib experimental design (described in Garbutt and Bazzaz, 1987). Garbutt employed standard quantitative genetic techniques to compute the means, variances, and additive-genetic covariances (Falconer, 1981; also see Discussion). Maternal effects are ignored in our simulations to simplify analysis. From this data, we estimated the mean and covariance functions using techniques described in Kirkpatrick et al. (1990). These functions are displayed in Figures 2 and 3. We caution that large statistical uncertainties are associated with these estimates; the quantitative conclusions in this section should be interpreted accordingly. This section is intended solely to illustrate qualitatively the theoretical concepts discussed above as well as to indicate the feasibility of gathering and analyzing the necessary data.

There are shapes characteristic of a genetic covariance function that determine the ways in which mean reaction norms may be deformed during evolution. These characteristic deformations are called eigenfunctions. Along with an initial mean reaction norm, they determine the set of evolutionary accessible reaction norms. Eigenfunctions are the infinite-dimensional extensions of the eigenvectors of a matrix (see, e.g., Kirkpatrick and Heckman, 1989). Associated with each eigenfunction is an eigenvalue that is proportional to the additive-genetic variance available for the changes represented by the eigenfunction. The eigenfunction for the largest eigenvalue (0.022) of the covariance function is shown

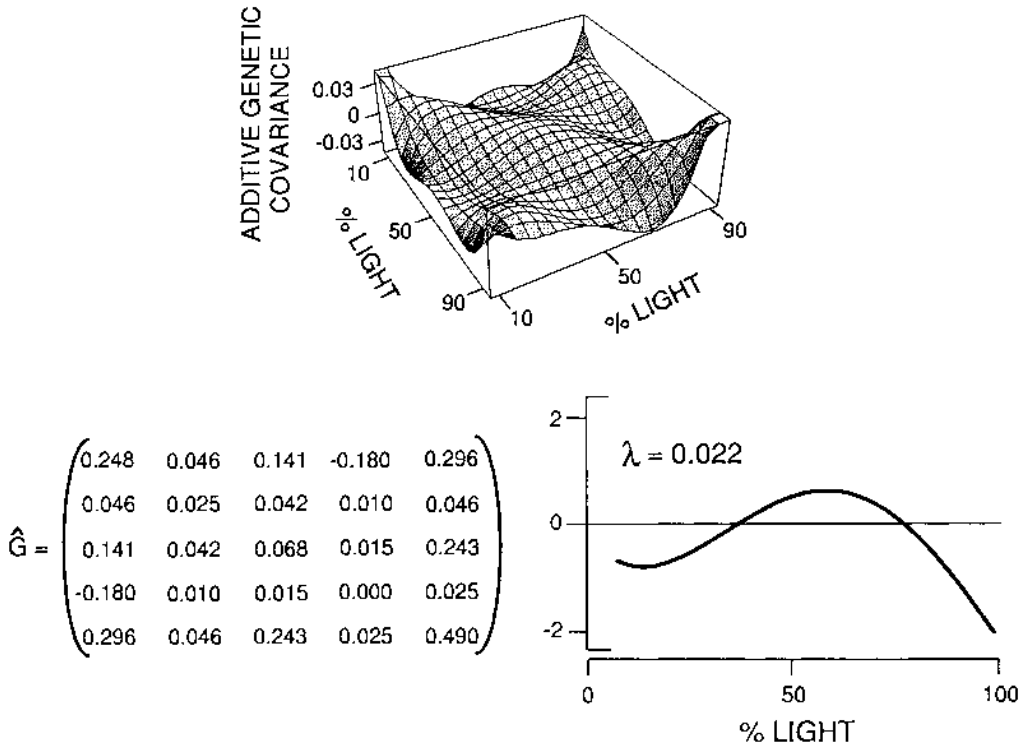

FIG. 3. $\hat{G}$: estimated additive genetic covariances for expression of log node number under 7%, 13%, 17%, 27%, and 100% transmitted light in *A. theophrasti*. Upper panel: additive genetic covariance function, $\mathcal{G}(x, y)$, for log node number over light levels. To avoid an overfit of $\mathcal{G}$ to $\hat{G}$, data were interpolated with bicubic splines (Press et al., 1988). Fourth degree Legendre polynomials were fit to the interpolated data and the estimate was "squeezed" following procedures described in Kirkpatrick et al. (1990). Lower right: eigenfunction associated with the largest eigenvalue of $\mathcal{G}$. Remaining eigenvalues are 0.010, 0.010, 0.009, and 0.

in Figure 3. This eigenfunction represents changes in the mean reaction norm that evolve most rapidly. The estimated covariance function also has three other nonzero eigenvalues (0.01, 0.01, and 0.009) and one zero eigenvalue. The zero eigenvalue indicates that the estimated covariance function is singular and thus that genetic constraints may be present. Because of the large uncertainties associated with these estimates, we cannot be confident that the genetic constraints are real as opposed to a sampling artifact. While we are unable to prove statistically the existence of constraints, our primary purpose in this section is to illustrate evolution in cases where constraints do exist. We thus treat the constraints as though they are real and invite more rigorous studies.

Our simulations assume weak stabilizing selection for node number. This form of selection is plausible since reproductive effort is reduced for plants that have either too few nodes (due to reduced vegetation and hence insufficient energy uptake) or too many nodes (because of the energy diverted to growth). In each simulation, evolution begins from the observed mean reaction norm (Fig. 2). For simplicity, we assume that additive-genetic covariances are constant through time. Finally, we set the distribution of environments (light levels) equal to a uniform distribution for convenience. This might, however, crudely approximate the conditions for a population of *A. theophrasti* inhabiting the understory of a corn field, which is one of its natural habitats. Identical parameters are used for hard and soft selection cases.

The first set of simulations illustrates how

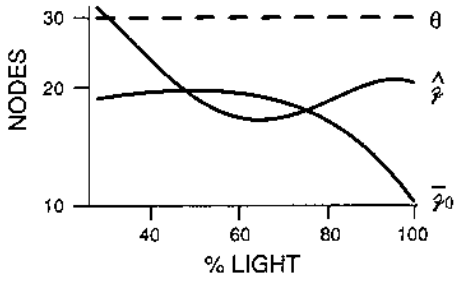

FIG. 4. Semi-log plot of equilibrium reaction norm for node numbers over light states, $\hat{\beta}$, reached by evolution under both hard and soft selection in the presence of genetic constraints. Additive-genetic covariance function used is that shown in Figure 3. Initial mean reaction norm, $\bar{\beta}_0$, is the observed mean reaction norm (Fig. 2). Dashed line is the optimum reaction norm, θ.

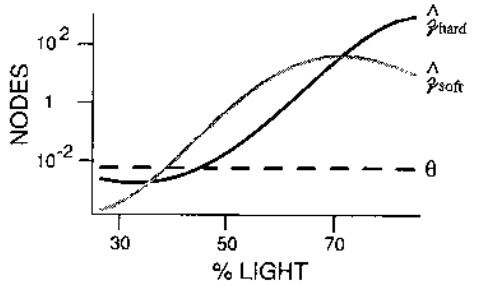

FIG. 5. Semi-log plot of equilibrium mean reaction norms obtained under soft selection, $\hat{\beta}_{soft}$, and hard selection, $\hat{\beta}_{hard}$, in the presence of genetic constraints. Dashed line is the optimum reaction norm $\theta(x)$. Other functions and parameters are as in Figure 4.

genetic constraints can affect evolutionary equilibria. We imagine that selection favors thirty nodes under all light conditions, that is, $\theta(x) = 30$ nodes for all feasible light levels x. The evolutionary equilibria reached under hard and soft selection (which are visually indistinguishable) are displayed in Figure 4. This figure shows how far from optimal the mean reaction norm may be when genetic constraints are present. We remark that the shape of the equilibrium reaction norm shown here depends as much on the initial mean reaction norm as it does on the additive-genetic covariance function.

The above theory shows that soft and hard selection have different adaptive topographies and, consequently, may produce different evolutionary trajectories and equilibria. Our next two simulations highlight these points. Due to the nature of these particular data, hard and soft selection produce dramatically distinct trajectories and equilibria only under biologically unrealistic conditions. Because it is possible that differences would be more pronounced for other data sets, we qualitatively illustrate some of the possibilities by assuming (unrealistically) that the optimal reaction norm is the constant function $\theta(x) \approx 0.0067$ for all x.

Figure 5 shows how soft and hard selection can produce different evolutionary equilibria when genetic constraints are present. Note once again that genetic constraints prevent reaction norms from evolving to the optimal shape under both modes of selection. However, the equilibrium reaction norms reached under these modes of selection are strikingly different even though both simulations were started from the same mean reaction norm (Fig. 2) and selection favors the same optimum in both cases. The difference is a consequence of the distinctive way that each adaptive topography (see Eqs. 4 and 8) interacts with the given pattern of genetic constraints.

A final set of simulations illustrates how evolutionary rates can differ under soft and hard selection when there are no genetic constraints. As we have seen, absence of constraints implies a nonsingular covariance function. To satisfy this requirement, we constructed a hypothetical additive-genetic covariance function which has the same eigenfunctions as the estimated covariance function, but is such that all eigenvalues are equal to the largest found from the data ($\lambda = 0.022$).

The simulation results appear in Figure 6. As expected, the mean reaction norm evolves to its optimum under both soft selection and hard selection when there are no genetic constraints. Comparing the reaction norms for hard and soft selection at intermediate times shows that each mean reaction norm follows a different evolutionary trajectory even though the same equilibrium is eventually reached. These differences are, once again, due to the distinct adaptive topographies associated with hard and soft selection (Eqs. 4, 8). These simulations thus demonstrate that, even in the absence of genetic constraints, evolutionary

rates may differ under distinct modes of selection.

Discussion

Quantitative genetics provides a natural framework in which to study many kinds of traits by providing an interface between theoretical and empirical methods. We have extended the quantitative genetic approach to reaction norms by adopting a recently introduced model for infinite-dimensional traits. Individuals are represented by a function describing the phenotypes that are expressed across a continuum of environmental conditions. This model has been used to study the evolution of reaction norms under several forms of selection that involve two patterns of spatial variation (hard and soft selection) and two modes of temporal variation (within and between generations).

One general conclusion that emerges from these analyses is that patterns of genetic variation for the reaction norms in a population may affect their evolutionary trajectories and equilibria. Regardless of the mode of selection, a reaction norm may be prevented from reaching its optimal shape if genetic variation for some changes in its form are absent in the population. Furthermore, interactions between the mode of selection and these constraints can influence the final equilibrium that is reached. To illustrate this point, we simulated the evolution of a reaction norm for the velvetleaf (*Abutilon theophrasti*) under hypothetical scenarios involving hard selection and soft selection. Even when the initial and optimal reaction norms are identical in the two cases, the evolutionary rates and end points can differ.

Whether constraints of this sort are important in the evolution of reaction norms in nature is, of course, a question that can only be answered empirically. Our models suggest the data and analyses that would be appropriate to address this issue. The first type of information needed regards the form of selection acting on the population, the second involves quantifying genetic variation available for evolutionary changes in the reaction norm.

The hypothesis that a population's mean reaction norm is optimal can be tested by measuring the selection gradient function β.

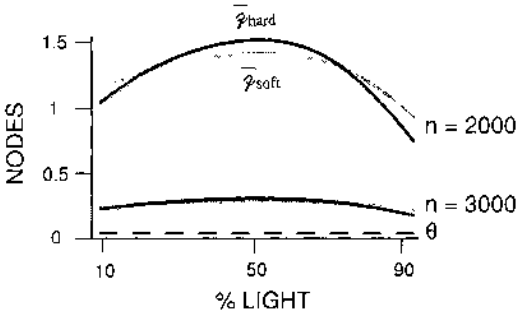

FIG. 6. Evolutionary trajectories of mean reaction norms under soft selection (gray curve) and hard selection (black solid curve) with no genetic constraints. Reaction norms are shown at generations n = 2,000 and 3,000 of evolution. The optimum (dashed line) is attained at equilibrium under both hard and soft stabilizing selection. The genetic covariance function was constructed to admit maximal additive genetic variance (λ = 0.022) for all eigenfunctions. Other functions and parameters are as in Figure 4.

The hypothesis is confirmed if $\beta(x) = 0$ for every environment x. The selection gradient function can be obtained using standard methods to estimate the selection gradient in each of several environments (i.e., values of x) (see Lande and Arnold, 1983; Arnold and Wade, 1984*a*, 1984*b*; Mitchell-Olds and Shaw, 1987). Although the number of environments in which data should be taken depends on how rapidly β and $\bar{z}$ change, data from four or five environments are likely to be sufficient for many studies. These values can then be interpolated to estimate the continuous selection gradient function $\beta(x)$ (see Kirkpatrick et al., 1990).

If the selection gradient is nonzero over some range of environments, this implies that the mean reaction norm is not at an optimum. There are several possible causes. The first is that there are trade-offs within the reaction norm. This hypothesis can be tested by analyzing the additive-genetic covariance function G. To estimate G, an additive-genetic covariance matrix for expression of the trait within and across several environments is obtained using standard quantitative-genetics methods. Labile traits are easiest to work with because the phenotype of each individual can be measured in every environment. This allows the additive-genetic covariances between character states to be estimated by standard

methods (Falconer, 1981; Bulmer, 1985). Nonlabile traits require a more indirect approach in which covariances are estimated with data taken from relatives (as in the *Abutilon* example discussed earlier; see Garbutt and Bazzaz, 1987); methods are discussed by Yamada (1962) and Via (1984). Once an estimate of an additive-genetic covariance matrix is obtained, the continuous additive-genetic covariance function is found by interpolation using the techniques described by Kirkpatrick et al. (1990).

Estimates of the mean and covariance functions are inevitably based on measurements taken from a finite number of environments. A different approach that can be taken to accommodate this restriction is to approximate the infinite-dimensional reaction norm by its values at a finite number of environments, that is, treat it as a multivariate trait. It has been shown (Kirkpatrick and Heckman, 1989), however, that infinite-dimensional methods provide more efficient empirical descriptions of infinite-dimensional traits than multivariate techniques. Infinite-dimensional descriptions are also more valuable for predicting the evolution of infinite-dimensional traits.

The existence of evolutionary constraints within the reaction norm can be tested by determining if G is singular. Methods for this analysis (including confidence interval construction and hypothesis testing) are given in Kirkpatrick et al. (1990) and Kirkpatrick and Lofsvold (1989, unpubl. data). If the additive-genetic covariance function is found to be singular, this implies that some evolutionary changes in the mean reaction norm are not possible. Definitive evidence that these constraints are preventing the reaction norm from evolving to its optimum would be obtained by inserting the estimates for β and G into Equation (1) and showing that no evolutionary change will result under this pattern of selection.

If directional selection is observed but within-reaction norm constraints are not found, other hypotheses are suggested. Evolution of the reaction norm may be constrained by genetic correlations with other traits. (These correlations may themselves depend on the environments in which they are measured; Gebhardt and Stearns, 1988; de Jong, 1989, 1990; Scheiner et al., 1991; Stearns et al., 1991.) For example, by maximizing sprint speed at all temperatures, lizards may sacrifice endurance performance (Huey and Hertz, 1984) or clutch size (Vitt and Congdon, 1978). This hypothesis can be tested by extending the genetic analysis outlined above to multiple traits (see Kirkpatrick, 1988).

Another hypothesis to account for an apparently suboptimal reaction norm is the presence of undetected physiological costs. We distinguish two types of costs that can affect reaction norms. The first, which we call "expression costs," are associated with a trait's expression in a specific environment, for example the metabolic demand of thermoregulation at a particular temperature (Huey and Slatkin, 1976). The second, which we call "maintenance costs," are costs associated with maintaining the capacity to respond plastically to a range of environments, for example the energy involved in developing and maintaining sweat glands. Van Tienderen (1991) was the first to consider formally the evolutionary consequences of maintenance costs.

The presence of costs raise two issues. The first is whether the evolution of the mean reaction norm can be correctly predicted it the costs are known. The models described above can in fact be modified to accommodate both expression costs and maintenance costs (Gomulkiewicz and Kirkpatrick, unpubl. data). The qualitative results that emerge are similar to what has already been seen. In particular, the mean reaction norm will evolve so as to maximize the population's fitness. Whether or not an optimum can be reached depends on the presence or absence of genetic constraints.

The second question is whether selection on the reaction norm generated by costs is correctly measured by the empirical program outlined above. In the case of expression costs, the answer is yes. Because the fitness effects of expression costs are restricted to individual environments, the procedure described earlier will give a valid estimate of the selection gradient function. Selection generated by maintenance costs, in contrast, will not be correctly measured. Additional information is needed, specifi-

cally data on the fitness effects of the unexpressed portions of a reaction norm as well as the expressed portions (Van Tienderen, 1991). Given these data, methods for estimating the selection gradient can be modified to accommodate maintenance costs (Gomulkiewicz and Kirkpatrick, unpubl. data). If these data are unavailable, then a selection analysis can lead to the incorrect conclusion that the population is not at a fitness maximum (Van Tienderen, 1991). We anticipate, however, that unless maintenance costs are large, they will not have a substantial impact on the equilibrium reaction norm.

There are a variety of additional reasons why a reaction norm might not lie at its evolutionary optimum, including mutation pressure, gene flow, and the possibility that it will evolve to the optimum if given enough time. These hypotheses, however, are generic to all kinds of traits and have not been suggested as particularly important in the evolution of reaction norms.

Much of the work on reaction norms has developed around the concept of phenotypic plasticity. Phenotypic plasticity (or stability) is generally defined in terms of some measure of across-environment phenotypic variability. A variety of methods have been proposed to describe and quantify phenotypic plasticity (reviews in Freeman, 1973; Lin et al., 1986; Schlichting, 1986). Simple measures include the range, variance, and coefficient of variation of phenotypic means over environments (e.g., Falconer, 1990). Other common measures, based on linear regression analyses of phenotypes over environmental indices, include the regression coefficient (e.g., Finlay and Wilkinson, 1963; Jinks and Connolly, 1973; Falconer, 1981; Bierzychudek, 1989) and the residual variance (Eberhart and Russell, 1966). Scheiner and Goodnight (1984) used a two-way analysis of variance on genotypes and environments, and defined plasticity to be the fraction of the total phenotypic variance due to environmental variance and genotype-environment interaction (see also Scheiner and Lyman, 1989). More complicated descriptions of reaction norms have also been used, for example, two-line and quadratic regressions of phenotypes on environments (Jinks and Pooni, 1979, 1988; Pooni and Jinks, 1980).

Although different in many details, these previous approaches are similar in that they reduce all the data about the reaction norms in a population to one or two statistics. This kind of simplification is useful for many descriptive and comparative purposes. Their limitation, however, is that they do not have sufficient information to predict the evolution of the reaction norm or to analyze the underlying cause of an equilibrium (Lewontin, 1974; Gupta and Lewontin, 1982; Sultan, 1987; van Noordwijk, 1989). These are exactly the capabilities that are needed to answer many of the questions that have been posed by evolutionary biologists who study phenotypic plasticity and reaction norms. It is in this context that the infinite-dimensional approach developed here may be useful.

Several previous studies have developed genetic models for the evolution of a continuous reaction norm. Lynch and Gabriel (1987) studied a model for the reaction norm of total fitness. Their model assumes that fitness is a Gaussian function of the environmental state, and the reaction norm evolves through changes in the mean and variance of this function. Thus the shape of all possible reaction norms are assumed to follow Gaussian curves and evolution is restricted to two evolutionary "degrees of freedom," specifically the reaction norm's mean and variance. Gavrilets (1986) and Gavrilets and Scheiner (1991*a*, 1991*b*) proposed models in which the reaction norm is a polynomial of the environmental state, and derived equations for the evolution of linear and quadratic reaction norms. The genetic model discussed in this paper can roughly be thought of as an extension of these earlier models to cases in which no prior assumption is made about the current or future shape of the reaction norm; rather, constraints are deduced from the data.

Our models of the evolution of mean reaction norms complement the numerous ecological investigations of adaptation in heterogeneous environments (e.g., Van Valen, 1965; Levins, 1968; Roughgarden, 1972; Antonovics, 1976; Caswell, 1983; Garbutt et al., 1985). Measures of population level

phenotypic variability used in those studies, such as niche width, can be derived from our descriptions of reaction norms and their distributions. Alternatively, these studies describe types of spatial and temporal variability that can be incorporated in our models of reaction norm evolution. In addition, the additive-genetic covariances required for our models may ultimately be explained by research on the role played by genotype-environment interaction in determining components of genetic variances and covariances (Gavrilets, 1986; Gimelfarb, 1986; Gregorius and Namkoong, 1986; Via and Lande, 1987; Gillespie and Turelli, 1989).

The previous work most similar to ours is Via and Lande's (1985) model for the expression of a trait in two discrete environments. The present work complements theirs by extending the quantitative genetic model to a continuum of environments. We have, however, highlighted somewhat different aspects of the probable outcome of evolution. Via and Lande (1985) emphasized the effects that patterns of genetic variation have on trajectories and rates of approach to an evolutionary optimum when the optimum will eventually be reached.

This paper has focussed, in contrast, on the importance of genetic constraints in preventing evolutionary optimization. Moreover, we suggest that such constraints may be common. The basis for this view lies in the extreme number of the traits considered here. When more than two traits are under selection, constraints on their evolution can exist even when all of the pairwise genetic correlations are less than unity (Dickerson, 1955; Via and Lande, 1985; Via, 1987). Roughly speaking, the larger the number of traits, the easier it is for the genetic covariance matrix to be singular. When dealing with reaction norms that vary in response to a continuous environmental cue, the effective number of traits under selection is infinite and the possibility of constraints is maximized. Analyses of growth trajectories, which are another type of infinite-dimensional trait, suggest that constraints may be common in the evolution of high-dimensionality phenotypes (Kirkpatrick and Lofsvold, 1989, unpubl. data). A major challenge is to determine whether such constraints have actually played an important part in determining the outcome of reaction norm evolution.

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Appendix A

We provide here some rules of association that may be used for extending multivariate character results to infinite-dimensional characters and demonstrate their application for several of the models presented in the text. The first rule is that functions are the infinite-dimensional extensions of vectors and matrices: the extension of the vector $\mathbf{z} = (z_1, z_2, \ldots, z_n)$ is a univariate function, $_{\delta}(x)$, while that of a matrix $\mathbf{G} = \{G_{ij}\}$ is a bivariate function, $G(x, y)$. A second rule is that integration is the infinite-dimensional extension of summation. This implies, for example, that the infinite-dimensional extension of the vector product $\mathbf{y}^{\top}\mathbf{z} = \sum_i y_i z_i$ is the integral $\int y(x)_{\delta}(x)\,dx$ and the extension of the matrix-vector product $\mathbf{Gz} = \left\{\sum_j G_{ij}z_j,\ i = 1, \ldots, n\right\}$ is the function $\int G(x, y)_{\delta}(y)\,dy$. Finally, consider the solution $\mathbf{z} = \mathbf{G}^{-1}\mathbf{b}$ to the equation $\mathbf{Gz} = \mathbf{b}$ (assuming $\mathbf{G}$ is invertible). The infinite-dimensional extension, denoted $G^{-1}b(x)$, is the solution to the invertible integral equation $\int G(x, y)_{\delta}(y)\,dy = b(x)$ (Kirkpatrick and Heckman, 1989).

Any correct application of these rules to multiple characters results does not constitute a proof for infinite-dimensional characters but rather represents a practical method of obtaining results. These results can be rigorously justified under the biologically mild restriction that functions be square-integrable (Kirkpatrick and Heckman, 1989). For the purposes of this paper, the most convenient application of these rules is in obtaining selection gradient functions for infinite-dimensional characters from their multiple characters counterparts. A direct, rigorous computation of the infinite-dimensional selection gradient, while producing the same selection gradient function as the indirect method of extension, requires techniques and results from the calculus of variations and Gaussian processes theory (Gomulkiewicz and Beder, unpubl. data). We now illustrate how to apply these rules and methods by analyzing particular examples of soft selection and between-generation variability.

Consider first a case of soft selection in which the fitness $W(_{\delta}, x)$ of individuals inhabiting environment x are given by Equation (6). Corresponding to the phenotypic functions $_{\delta}(\cdot)$ and $\theta(\cdot)$ are the vectors $\mathbf{z} = \{z_1, z_2, \ldots, z_n\}^{\top}$ and $\theta = \{\theta_1, \theta_2, \ldots, \theta_n\}^{\top}$ respectively, and the Gaussian fitness function of a discrete reaction norm $\mathbf{z}$ in environment i is

$$W(\mathbf{z}, i) \propto \exp\left\{-\frac{[z_i - \theta_i]^2}{2\omega_i^2}\right\}. \tag{A1}$$

Assuming that $\mathbf{z}$ is multivariate normally distributed with mean vector $\bar{\mathbf{z}} = \{\bar{z}_1, \bar{z}_2, \ldots, \bar{z}_n\}$ and phenotypic variance P_{ii} in environment i, and that the frequency of environment i is f_i, Via and Lande (1985) show that the selection gradient vector, β, has i-th component

$$[\beta]_i \approx f_i \frac{\theta_i - \bar{z}_i}{\omega_i^2} \tag{A2}$$

assuming weak selection, $\omega_i^2 \gg P_{ii}$. According to the above rules, the functional extension of this result is Equation (7). A similar procedure can be used to deduce Equation (10) from the fitness function (6) in the case of hard selection.

We now consider between-generation temporal variation. To obtain the general selection gradient function $\beta_n(\cdot)$ in generation n, we begin with the corresponding multivariate normal model. Since the multivariate model requires a discrete set of environments, assume that the environmental state encountered in generation n has index i_n. The selection gradient vector, β_n, must be zero for all environmental conditions except the one indexed by i_n, where it assumes the value of the selection gradient in environment i_n. That is,

$$[\beta_n]_j = \begin{cases} \dfrac{\partial \ln \widetilde{W}(i_n)}{\partial \bar{z}_{i_n}} & \text{if } j = i_n \\ 0 & \text{otherwise.} \end{cases}$$

By writing the logarithm of the population mean fitness in generation n as

$$\ln \overline{W}_n = \sum_j \delta_{i_n j} \ln \overline{W}(j) \tag{A3}$$

(δ_{kl} is the Kronecker delta: $\delta_{kk} = 1$, $\delta_{kl} = 0$ if $k \neq l$), the complete selection gradient vector may be computed with the vector gradient operator $\nabla_{\bar{z}}$:

$$\beta_n = \nabla_{\bar{z}}[\ln \overline{W}_n]. \tag{A4}$$

To obtain the infinite-dimensional extensions of (A3) and (A4), given by Equations (14) and (13) respectively, note that the infinite-dimensional extension of δ_{ij} in (A3) is the Dirac delta function (see Eq. 15). As a concrete example, consider Gaussian stabilizing selection (6). Using (A1) and the one-character selection gradient (Lande, 1976), the selection gradient vector in generation n for the associated multiple-characters model, assuming weak selection, is seen to have components

$$[\beta_n]_j \approx \delta_{i_n j} \frac{\theta_{i_n} - \bar{z}_{i_n}}{\omega_{i_n}^2}. \tag{A5}$$

The infinite-dimensional extension of this result appears in Equation (16).

Appendix B

In this section, we present a simple model of within-generation Gaussian selection for the evolution of the reaction norm of a labile trait and derive the selection gradient. We begin with a model in which within-generation fluctuating selection acts on a general multivariate quantitative trait. We assume that no selection occurs until the trait of interest has completed development; any subsequent phenotypic changes occur solely in response to environmental fluctuations. We first consider environments that fluctuate at discrete time points within a generation and from there generalize to continuous change. The results are then adapted for multivariate reaction norms and extended

to reaction norms over continuous environments. Last, the selection gradient for a nonlabile trait subjected to within-generation fluctuating selection is determined.

Assume that immediately following development, multivariate phenotypes $\mathbf{z}$ are multivariate normally distributed with mean vector $\bar{\mathbf{z}}$ and covariance matrix $\mathbf{P}$. The time interval between the end of development and the end of the generation is denoted by $[0, \mathcal{T}]$. If Gaussian selection occurs with optimum vector θ_i and width matrix $\mathbf{W}_i$ at discrete epochs τ_i ($\tau_0 = 0, \tau_1, \ldots, \tau_N = \mathcal{T}$) during $[0, \mathcal{T}]$, the distribution of phenotypes remains multivariate normal. The mean vector $\mathbf{m}_i$ and covariance matrix $\mathbf{P}_i$ satisfy the following equations at time τ_i, $i \geq 1$ (Felsenstein, 1977):

$$\begin{cases} \mathbf{P}_i = (\mathbf{W}_{i-1}^{-1} + \mathbf{P}_{i-1}^{-1})^{-1} \\ \mathbf{m}_i = (\mathbf{W}_{i-1}^{-1} + \mathbf{P}_{i-1}^{-1})^{-1} \\ \qquad \cdot (\mathbf{W}_{i-1}^{-1}\theta_{i-1} + \mathbf{P}_{i-1}^{-1}\mathbf{m}_{i-1}) \end{cases} \tag{B1}$$

The solution satisfying the initial conditions $\mathbf{P}_0 = \mathbf{P}$, and $\mathbf{m}_0 = \bar{\mathbf{z}}$ is ($i \geq 1$)

$$\begin{cases} \mathbf{P}_i = \left(\mathbf{P}^{-1} + \sum_{j=0}^{i-1} \mathbf{W}_j^{-1}\right)^{-1} \\ \mathbf{m}_i = \left(\mathbf{P}^{-1} + \sum_{j=0}^{i-1} \mathbf{W}_j^{-1}\right)^{-1} \\ \qquad \cdot \left(\sum_{j=0}^{i-1} \mathbf{W}_j^{-1}\theta_j + \mathbf{P}^{-1}\bar{\mathbf{z}}\right). \end{cases} \tag{B2}$$

An immediate consequence of (B2) is that the order in which environments are encountered plays no role in determining the final mean vector or covariance matrix.

Now consider the selection gradient under fluctuating selection. First, the selection differential, $\mathbf{s}$, is calculated as

$$\begin{aligned} \mathbf{s} &= \mathbf{m}_N - \bar{\mathbf{z}} \\ &= \left(\mathbf{P}^{-1} + \sum_{i=0}^{N-1} \mathbf{W}_i^{-1}\right)^{-1} \\ &\quad \cdot \left(\sum_{i=0}^{N-1} \mathbf{W}_i^{-1}[\theta_i - \bar{\mathbf{z}}]\right). \end{aligned} \tag{B3}$$

Define the matrix $\tilde{\mathbf{W}}$ by

$$\tilde{\mathbf{W}}^{-1} = \sum_{i=0}^{N-1} \mathbf{W}_i^{-1}$$

and the vector $\tilde{\theta}$ by

$$\begin{aligned} \tilde{\theta} &= \sum_{i=0}^{N-1} [\mathbf{W}_i\tilde{\mathbf{W}}^{-1}]^{-1}\theta_i \\ &= \tilde{\mathbf{W}} \sum_{i=0}^{N-1} \mathbf{W}_i^{-1}\theta_i. \end{aligned}$$

By utilizing the relationship $\beta = \mathbf{P}^{-1}\mathbf{s}$ (Lande and Arnold, 1983), the selection gradient takes the form

$$\beta = (\tilde{\mathbf{W}} + \mathbf{P})^{-1}(\tilde{\theta} - \bar{\mathbf{z}}). \tag{B4}$$

These results are general for arbitrarily strong selection. The Gaussian genetic model on which we rely, however, will generally require that selection is weak. Specifically, the requirement is that the eigenvalues of $\tilde{\mathbf{W}}$ are much larger than those of $\mathbf{P}$ (Lande, 1980). When this condition holds, (B2) shows that $\mathbf{P}_N \approx \mathbf{P}_0 = \mathbf{P}$. In other words, the effect of selection on the phenotypic variance will be negligible. Under these conditions (B4) is approximated as

$$\beta \approx \tilde{\mathbf{W}}^{-1}(\tilde{\theta} - \bar{\mathbf{z}}). \tag{B5}$$

From (B5) it can been seen that the population evolves along an adaptive topography which is determined by the population mean fitness $\overline{W}$ whose logarithm is defined approximately and to within a constant by the quadratic form

$$\ln \overline{W} \approx -\tfrac{1}{2}(\tilde{\theta} - \bar{\mathbf{z}})^{\top}\tilde{\mathbf{W}}^{-1}(\tilde{\theta} - \bar{\mathbf{z}}). \tag{B6}$$

We next consider selection on multivariate reaction norms of labile traits. Let the vector $\bar{\mathbf{z}}$ represent a mean reaction norm over a finite set of environments so that at epoch 0, the k-th component $\bar{\mathbf{z}}_k$ is the average phenotype expressed in environment k. Selection in environment k has no direct effect on the unexpressed phenotypes so that at a time τ_i in which environment k is encountered, the selection width matrix $\mathbf{W}_i^{-1}$ has only one nonzero component: its k-th diagonal element $[\mathbf{W}_i^{-1}]_{kk} = 1/\omega_i^2$. (Note, $[\mathbf{A}]_{ij}$ denotes the ij-th component of a matrix $\mathbf{A}$ and $[\mathbf{x}]_k$ denotes the k-th element of a vector $\mathbf{x}$). In order to keep the strength of selection finite over the interval $[0, \mathcal{T}]$, we set $1/\omega_i^2 = s(\tau_i)\mathcal{T}/N$. Here, $s(\tau_i)$ is the instantaneous strength of selection at epoch τ_i. Let

$$s_k(\tau_i) = \begin{cases} s(\tau_i) & \text{if environment} \\ & k \text{ encountered at time } \tau_i. \\ 0 & \text{otherwise} \end{cases}$$

That is, $s_k(\tau_i)$ is a function which indicates the instantaneous strength of selection at time τ_i on the phenotype that is expressed in the k-th environment. Under these conditions, $\tilde{\mathbf{W}}^{-1}$ is a diagonal matrix whose k-th diagonal element is the sum $\sum_{i=0}^{N-1} s_k(\tau_i)\mathcal{T}/N$. As the partitions of the interval $[0, \mathcal{T}]$ become finer (i.e., N increases), these sums converge to the integral $\int_0^{\mathcal{T}} s_k(t)\,dt$. Note also that $[\mathbf{W}_i^{-1}\theta_i]_k = [\theta_i]_k s_k(\tau_i)\mathcal{T}/N$. Denoting $[\theta_i]_k$ by $\theta_k(\tau_i)$, we have

$$\begin{aligned} \left[\sum_{i=0}^{N-1} \mathbf{W}_i^{-1}\theta_i\right]_k &= \sum_{i=0}^{N-1} \theta_k(\tau_i)s_k(\tau_i)\frac{\mathcal{T}}{N} \\ &\to \int_0^{\mathcal{T}} \theta_k(t)s_k(t)\,dt \\ &\quad \text{as } N \to \infty. \end{aligned} \tag{B7}$$

The approximate selection gradient β with continuous selection is thus given by (B5) where the matrix $\tilde{\mathbf{W}}$ has jk-th entry

$$[\tilde{\mathbf{W}}]_{jk} = \frac{\delta_{jk}}{\int_0^{\mathcal{T}} s_k(t)\,dt} \tag{B8}$$

and

$$\tilde{\theta}_k = \frac{\int_0^{\mathcal{T}} \theta_k(t)s_k(t)\,dt}{\int_0^{\mathcal{T}} s_k(t)\,dt}. \tag{B9}$$

We remark that the cumulative strength of selection over the interval $[0, \mathcal{T}]$ will be small if $s_k(t)$ is small for all k.

We can now find the selection gradient function for the reaction norm of a labile character. An appropriate extension to infinite dimensions (Appendix A) shows that under weak selection the selection gradient function is (from [B5])

$$\beta \approx w^{-1}(\theta - \bar{\theta}) \tag{B10}$$

where, from (B8) and (B9),

$$w(x, y) = \frac{\delta(x - y)}{\int_0^{\mathcal{T}} s(x, t)\,dt} \tag{B11}$$

and

$$\tilde{\theta}(x) = \frac{\int_0^{\mathcal{T}} \theta(x, t)s(x, t)\,dt}{\int_0^{\mathcal{T}} s(x, \tau)\,d\tau}. \tag{B12}$$

The bivariate function $s(x, t)$ indicates the instantaneous strength of selection in environment x at time t. For the example in the text, $1/v^2(x)$ is the instantaneous strength of selection in environment x. In this case, Equation (B11) may be written as

$$w(x, y) = \frac{\delta(x - y)v^2(x)}{T(x)} \tag{B13}$$

where $T(x)$ is the total time out of $\mathcal{T}$ spent in x. Combining Equations (B10)–(B13) with our assumption that the optimum depends solely on the environment [i.e., $\theta(x, t) = \theta(x)$ for all t] yields the selection gradient function in Equation (11). Similar considerations show that Equation (12) is the infinite-dimensional version of Equation (B6).

Finally, we determine the selection gradient for a nonlabile character subjected to fluctuating Gaussian selection. This case is handled exactly as above except that phenotypes are fixed and constantly selected throughout the generation interval $[0, \mathcal{T}]$. The environmental variable x now refers to the condition under which the nonlabile character developed. Repeating the above derivation mutatis mutandis shows that the selection gradient for a nonlabile character that develops in environment x and undergoes weak variable Gaussian selection with optimum $\theta_x(t)$ and instantaneous strength $s_x(t)$ is

$$\beta(y) \approx \delta(y - x)\frac{\tilde{\theta}_x - \bar{\theta}(x)}{w_x}, \tag{B14}$$

where

$$w_x = \frac{1}{\int_0^{\mathcal{T}} s_x(t)\,dt} \quad \text{and}$$

$$\tilde{\theta}_x = \frac{\int_0^{\mathcal{T}} \theta_x(t)s_x(t)\,dt}{\int_0^{\mathcal{T}} s_x(\tau)\,d\tau}.$$

The phenotype $\tilde{\theta}_x$ represents an optimal compromise in environment x that will evolve provided there are no genetic constraints.