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CONSTRAINTS ON PHENOTYPIC EVOLUTION

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Abstract.—Constraints on phenotypic evolution can take a variety of forms. Constraints can arise from inheritance, selection, development, and design limits. Contemporary visions of the evolutionary process often focus on one or two of these varieties and ignore the others. A unifying framework that considers all four major varieties of constraint is emerging within the discipline of quantitative genetics. I attempt to sketch that emerging framework and summarize recent efforts toward unification. Although couched in the technical language of quantitative genetics, the ongoing search for a common framework promises a rapprochement among the approaches of optimality theorists, population geneticists, and developmental biologists.

This article has two aims. The first is to review the rapidly expanding literature dealing with constraints on phenotypic evolution. The second is to briefly discuss some aspects of constraints as they affect and are affected by behavioral evolution. No attempt is made to review the empirical literature on genetic variances and covariances for behavioral traits, a subject treated in two recent books (Hahn et al. 1990; Boake 1993).

Evolutionary constraints are restrictions or limitations on the course or outcome of evolution. Discussions of evolutionary constraint are often difficult to follow because of a failure to distinguish among underlying concepts. In particular, it is useful to distinguish between genetic, selective, functional, and developmental constraints. Definitions of these varieties of constraint and their interrelations are discussed in the sections that follow.

It is also useful to recognize four key properties of constraints: source, strength, consequence, and persistence (Maynard Smith et al. 1985). By source I mean the most proximate causes of constraint—for example, statistical distributions of allelic effects in the case of genetic constraint, relationships among ontogenetic precursors in the case of developmental constraints. Strength is an attribute that can sometimes be measured in statistical or mathematical terms (e.g., variance, covariance, regression slope, first and second derivatives). The consequences of a constraint are ultimately on evolutionary process and outcome. The consequences may be direct or mediated through other kinds of constraint. Persistence refers to the stability of a constraint over evolutionary time and can be assessed by longitudinal or comparative studies.

AN OVERVIEW OF THEORY

A current challenge is to find a unifying framework that accounts for all four basic properties of genetic and other kinds of constraint. Progress has been made,

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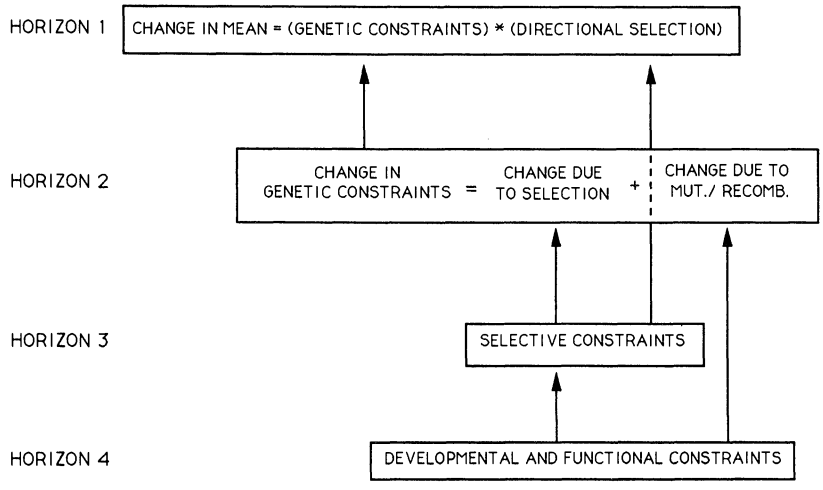

FIG. 1.—A framework for the main theoretical connections between genetic, selective, developmental, and functional constraints. Immediate consequences are shown by *arrows*.

but we still do not have a conceptual framework that serves all purposes equally well. The most tangible progress has been made by building on the statistical framework provided by the field of quantitative genetics, the study of the inheritance of traits affected by many loci. A quantitative genetic framework does a good job of describing the strength and short-term consequences of genetic constraints. It has been less successful in dealing with the sources, long-term consequences, and persistence of genetic constraints. Much theoretical work in the last decade has focused on correcting these deficiencies. To understand both the triumphs and shortcomings of this recent work, a brief historical summary is useful. For a fuller account of historical developments in quantitative genetics, see Hill (1984*a*, 1984*b*).

The theoretical structure provided by quantitative genetics consists of four horizons, shown diagrammatically in figure 1, with the oldest horizon on the top and the most recent on the bottom. The main message in the diagram is that selective, developmental, and functional constraints exert their evolutionary effects by affecting genetic constraints.

We may define *genetic constraints* as the pattern of genetic variation and covariation for a set of traits. The most useful definition of genetic variation and covariation has proved to be the additive genetic variances and covariances that describe resemblance between parents and offspring and that consequently enable us to predict responses to selection. In theoretical work these variances and covariances are often arranged in a so-called G-matrix in which the row and column labels refer to particular phenotypic attributes or traits (table 1). Thus, the elements on the main diagonal are genetic variances, and the other elements are genetic covariances. The heritability of a trait is simply its standardized additive genetic variance: $h_i^2 = G_{ii}/P_{ii}$, where G_{ii} and P_{ii} refer, respectively, to the

TABLE 1

THE ESTIMATED ADDITIVE GENETIC VARIANCE-COVARIANCE MATRIX FOR A BRISTLE COUNT AND FOUR MEASUREMENTS IN AN EXPERIMENTAL POPULATION OF *DROSOPHILA MELANOGASTER*

Trait	BB	TX	WL	WW	TB
BB. Bristle number	.9370	.0042	−.0119	−.0022	.0492
TX. Thorax length	.0042	.4844	.2367	.3713	.1868
WL. Wing length	−.0119	.2367	.3238	.3696	.1564
WW. Wing width	−.0022	.3713	.3696	1.0241	.2511
TB. Tibia length	.0492	.1868	.1564	.2511	.3733

SOURCE.—Wilkinson et al. 1990. Reprinted by permission.

NOTE.—The estimates are based on an analysis of 181 sets of parents and six offspring from a line selected for large thorax length.

additive genetic variance and phenotypic variance of the ith trait. The genetic correlation between two traits is simply a standardized genetic covariance: $G_{ij}/\sqrt{G_{ii}\,G_{jj}}$, where G_{ij} is the additive genetic covariance between the ith and jth traits. The magnitudes of the genetic variances and covariances (or their standardized analogues) measure the strength of genetic constraint.

The pattern of constraints embodied in the **G**-matrix can be visualized by transforming it to a diagonal form. The elements on the main diagonal (eigenvalues) of the transformed **G**-matrix are the genetic variances for trait combinations; the off-diagonal elements are all zero. The rows of the transformation matrix (eigenvectors) give the trait combinations or the new axes in multivariate space. Such diagonalized **G**-matrices can be used to identify directions in phenotypic space that are most genetically constrained in the sense that they have the least genetic variance. Examples of diagonalization of **G**-matrices using principal component analysis are given elsewhere (Gale and Eaves 1972; Arnold 1981; Atchley et al. 1981; Cheverud 1982; Leamy and Cheverud 1984).

The First Horizon: Response of the Mean to Selection

The first horizon consists of a family of equations for predicting the change in trait means from one generation to the next. The univariate member of the family was in place by the 1930s (see, e.g., Lush 1937). This earliest version says that the mean of a single trait will be shifted across generations by an amount equal to the product of heritability and the force of directional selection ($\Delta\bar{z} = h^2S$). This version may be called the breeder's equation, because its principal use is to predict how much improvement to expect in some attribute when deliberate selection is practiced on that attribute. The view that comes from the breeder's equation is that genetic constraint, expressed as heritability, acts as a simple brake on evolutionary change. Under perfect inheritance the full force of selection is translated into evolutionary change, but in a world of imperfect inheritance only a fraction of the selective force is translated into change.

The inadequacies of the breeder's equation, even for practical applications, were appreciated by the early 1940s (see, e.g., Hazel 1943). When selection is exerted on a particular trait, that trait may respond to selection, but so may other

traits. These secondary or correlated responses to selection may be undesirable and must be accounted for in the breeding program. The solution, which remains today, was to expand the concept of genetic constraint to include connections between traits. The solution was rooted in observations of the following kind. When a poultry flock is selected for increased egg production, production tends to increase, but body size decreases (Dickerson 1955). The Mendelian basis for such observations can be derived using the algebraic model independently introduced by Weinberg (1910), Fisher (1918), and Wright (1921). (For a translation of Weinberg's article by K. Meyer, see Hill 1984*a*.) Resemblance between parents and offspring in the same trait can be ascribed to variance in additive genetic values for the trait (additive genetic variance) (Fisher 1918). Correspondence between one trait in parents and another trait in offspring can be ascribed to covariance in additive genetic values (additive genetic covariance). For example, a negative genetic covariance between egg production and body size helps explain nonintuitive responses to selection in chickens (Gyles et al. 1955). Thus, by the early 1940s we had the ingredients for a multivariate view of genetic constraints. Over the next few decades the multivariate view of genetic constraints focused on the practical problem of improving domestic animals and plants. During this period it was largely ignored by evolutionary biologists.

The most relevant conceptual advance in the practical realm was the development of selection indexes. The breeder has multiple objectives, and so the problem is to devise a selection program that will give the best results across the board. The standard approach, rooted in the work of Smith (1936) and Hazel (1943), is to devise a weighted sum of traits, the index, upon which selection is practiced. To find the index that gives the best aggregate genetic response to selection, one needs to know both the genetic and phenotypic variances and covariances of the traits as well as the economic value of each trait (Hazel 1943). By the late 1970s a general solution had been achieved using the convenient notation of matrix algebra (Young and Weiler 1960; Magee 1965; Yamada 1977).

These later articles also provided a solution to the inverse problem of retrospective selection analysis, which arises when observed responses to a selection program do not match expectations. In these circumstances, it is desirable to estimate the selection that was actually imposed and compare it with the selection that was supposedly imposed. The ingredients in such a retrospective analysis are the phenotypic covariances of the traits with the index, as well as the phenotypic and genetic variances and covariances of the traits. If we move beyond the purely practical concerns of the authors in question and equate the selection index with fitness, then their equations (see, e.g., Yamada 1977, eqq. [3b] and [11]) converge on those later used by evolutionary biologists to predict responses to multivariate natural selection (Lande 1979). This connection, however, is only obvious with the clarity of hindsight. In summary, solutions to the problems of imposing multivariate selection and analyzing that selection in retrospect were characterized by increasing sophistication in statistical techniques and multivariate characterizations of genetic constraints during the period 1936–1979. Meanwhile, application of quantitative genetics in evolutionary biology remained

stalled at the level of the univariate breeder's equation, despite expository efforts by Robertson (1955) and Falconer (1960).

The application of multivariate concepts of selection and genetic constraint to evolutionary problems was made in a pathbreaking article by Lande (1979). The problem posed by Lande was how to predict the genetic response of a population when natural selection acts simultaneously on multiple traits. The ingredients of the solution consisted of multiple genetic constraints, encapsulated in a **G**-matrix, and a multivariate characterization of selection, a vector of selection gradients, $\boldsymbol{\beta}$. More precisely, the change in means of each of a set of traits from one generation to the next was shown to be the matrix product of **G** and $\boldsymbol{\beta}$, $\Delta\bar{\mathbf{z}} = \mathbf{G}\boldsymbol{\beta}$. The selection gradients were derived as partial derivatives of mean fitness with respect to average trait values in the population. The derivation of multivariate response to selection followed the tradition in quantitative genetics of using the regression of offspring values on the phenotypes of their parents as a launching point. What was new in Lande (1979), aside from the application to multivariate evolutionary problems, was the formal characterization of selection as a gradient on an adaptive landscape. This gradient view of selection provided a bridge to Wright's adaptive landscape for a field of gene frequencies, as well as the starting point for a host of evolutionary models, and it will be discussed here as the third horizon in the classification scheme. Thus, by the late 1970s the first horizon in the conceptual scheme consisted of a multivariate version of the breeder's equation that is useful for predicting response to natural selection.

The Second Horizon: Genetic Constraints and Their Evolutionary Persistence

The second horizon in the diagram (fig. 1) deals with evolution of the genetic constraints themselves. The equation in the top horizon tells how much the mean of each trait will shift from one generation to the next. To apply the equation to successive generations, one must know whether the system of genetic constraints (i.e., the **G**-matrix) has changed. Many evolutionary biologists are familiar only with the early argument, which traces to Fisher (1930), that directional and stabilizing selection will progressively erode the genetic variance of a quantitative trait and eventually eliminate all heritable variation. Lande (1976*a*) argues that the genetic variance of a trait affected by many loci will enjoy appreciable input of mutation each generation. Consequently, under weak stabilizing selection, a balance will be struck between loss due to selection and input from mutation so that appreciable genetic variance might be maintained at equilibrium. Later, analogous models and arguments were advanced for the maintenance of genetic covariances between traits (Lande 1980*a*, 1984). In response, Turelli (1984, 1985, 1986) argues that the amount of genetic variance or covariance maintained at equilibrium might be minuscule or appreciable, depending on what distributional assumptions are made about mutational input. Turelli (1988) argues that the issue cannot be settled on theoretical grounds and has appealed for further empirical work. The important point is that by the mid-1980s a formal equation for evolutionary change in genetic constraints was advanced that took into account both selection and mutation. I shall take up the theoretical aspects of change in genetic constraints in the

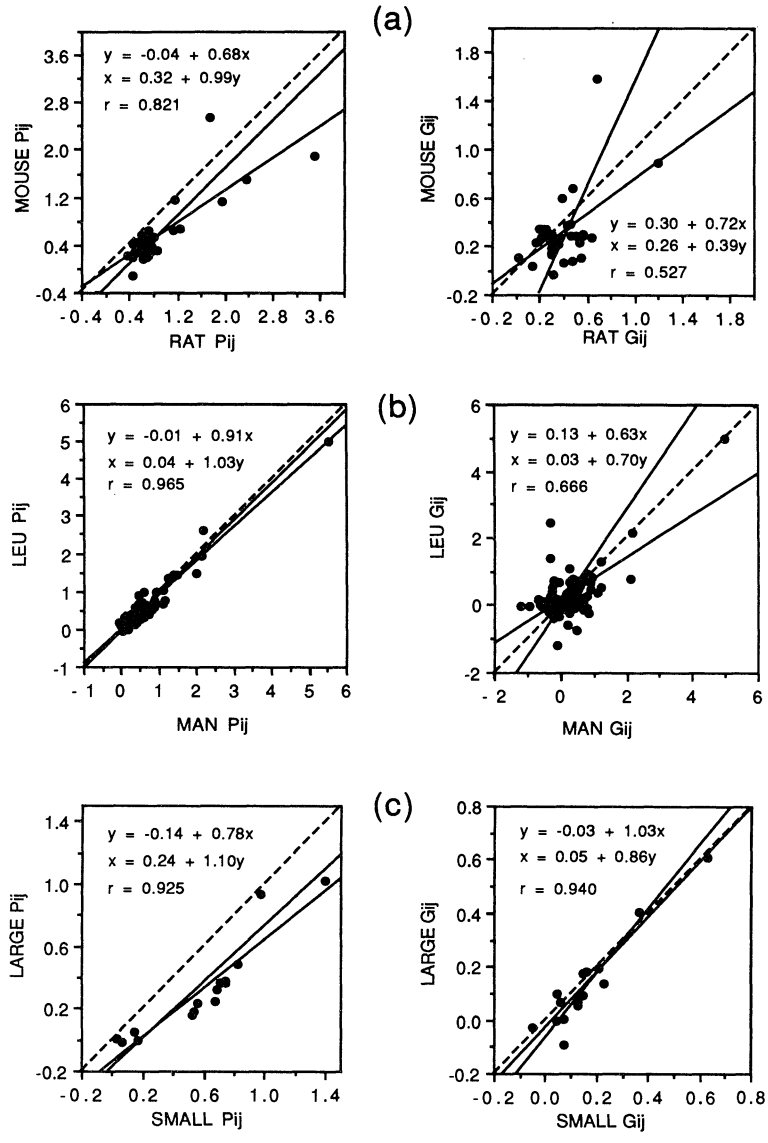

FIG. 2.—Comparative studies of phenotypic (*left*) and additive genetic (*right*) variance-covariance matrices. The scatterplots show matrix elements from one population plotted against corresponding matrix elements from the other population. Sample sizes for phenotypic matrices are denoted n (number of individuals) and for genetic matrices are denoted N (number of families). The *solid lines* are least-squares regression lines. The *dashed lines* show the alignment of points expected under matrix identity. *a,* Matrix comparisons for random-bred mice (*Mus musculus;* $n = 707$, $N = 108$) and selected lines of domestic rats (*Rattus norvegicus;* $n = 522$, $N = 54$). The matrices are based on eight pelvic measurements (yielding 36 data points in the plots) (data from Kohn and Atchley 1988). *b,* Matrix comparisons for white-footed mice (*Peromyscus leucopus;* n = ca. 315, $N = 105$) and deer mice (*Peromyscus maniculatus nebrascensis;* n = ca. 273, $N = 91$). The matrices are based on 15 skull and body measurements (yielding 120 data points in the plots) (data from Lofsvold 1986). Estimates of genetic variance in the right-hand plot were constrained by the relation-

next section after I introduce a characterization of selective constraints. For the moment, I will concentrate on the empirical aspect of the second horizon.

The persistence of genetic constraints is an empirical issue. In principle, genetic variances and covariances could change considerably from generation to generation because they are functions of underlying gene frequencies (Falconer 1989). The issue is whether the **G**-matrix actually changes during evolutionary excursion of the multivariate mean phenotype. Stability has been assessed by selection experiments and comparative studies. The two kinds of studies offer slightly different perspectives on the issue of stability in nature. A selection experiment can tell us whether and how much the **G**-matrix changes under a known selection regime. To extrapolate the results to the natural world, one needs to know how the imposed selection compares with selection in nature. A comparative study can tell whether and how much the **G**-matrix has changed under natural selection regimes and over long time intervals. But, without a companion study of selection, a comparative study of **G**-matrices cannot diagnose the selective causes of change. Only a handful of studies have used one or the other technique to compare large **G**-matrices that are estimated from 100 or more families in each sample (Atchley et al. 1981; Lofsvold 1986; Kohn and Atchley 1988; Wilkinson et al. 1990).

The comparison of covariance matrices, such as **G**-matrices, can be viewed as a series of nested hypotheses: matrix identity (equality of corresponding elements), matrix proportionality (the elements in one matrix are a scalar multiple of elements in the other matrix), and similarity in principal component structures (Flury 1987). The first two hypotheses can be visualized by making scatterplots such as those shown in figure 2. Under matrix identity the points should be arrayed on a 45° line. Under matrix proportionality the points should be arrayed on some other line that passes through the origin. The optimistic view of results from the best studies is that **G**-matrices, as well as the **P**-matrices, from divergent lines or taxa are proportional and sometimes virtually identical. The pessimistic view is that some plots show an uncomfortably large amount of scatter, with some matrix elements showing considerable divergence. Note that the sample sizes for the phenotypic matrices plotted in figure 2 are 3–10 times larger than the sample sizes for genetic matrices. Thus, the higher correspondence of phenotypic matrices (r = 0.82–0.96 vs. r = 0.53–0.94 for genetic matrices) suggests that an appreciable amount of scatter in the scatterplots for genetic matrices represents errors of estimation.

Although there are encouraging signs that the constraints imposed by **G**-matrices may be evolutionarily persistent, much remains to be done. Clearly, we need more data if we are to determine which traits show stable genetic variances and covariances and which do not, and on what time scale. An empirical challenge

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ship $h^2 \leq 1.0$. *c*, Matrix comparisons for two lines of *Drosophila melanogaster* after 23 generations of selection for large (n = 870, N = 145) and small (n = 762, N = 127) thorax size. The matrices are based on four body measurements and one bristle count (yielding 15 data points in the plots) (data from Wilkinson et al. 1990).

is to move from comparisons of pairs of taxa to multiple comparisons on a known phylogeny so that we can trace the evolution of **G**-matrices. Also, statistical techniques for comparison of **G**-matrices need to be perfected (Turelli 1988; Shaw 1991).

Constancy or proportionality of **G**-matrices opens the door to several novel modes of data analysis. We can reconstruct the net forces of directional selection that have produced differentiation in the phenotypic means of a pair of sister taxa (Lande 1979; Price et al. 1984; Schluter 1984; Price and Grant 1985; Arnold 1988; Lofsvold 1988; Turelli 1988). We can also test the pattern of among-taxa variation and covariation in phenotypic means against the pattern expected under multivariate drift (Lande 1979; Lofsvold 1988). In addition, we may be able to determine the roles of genetic constraints and among-taxa covariance in selection in producing interspecific covariation in means and allometry (Felsenstein 1988; Zeng 1988). Equations underlying the last two exercises are predicated on star phylogenies and need to be extended to the case of arbitrary branching sequences.

The Third Horizon: Selective Constraints and Their Consequences

Selective constraints arise from the ecological relationships between the population and its environment (Endler 1986) and also from interactions between the parts of an organism (Riedl 1979; Cheverud 1984). Selective constraints exert their effects on evolution directly under the guise of directional selection and indirectly under the guise of stabilizing (nonlinear) selection that affects genetic constraints (fig. 1). The direct and indirect effects of selective constraints can be visualized using the idea of an adaptive landscape. More exactly, the strength and pattern of selective constraints can be measured as the slope and curvature of an adaptive landscape.

An adaptive landscape is simply the relationship between average fitness (or its logarithm) and average trait values. The most familiar adaptive landscape for a single trait is a curve that is bowed downward. Such a curve contracts the phenotypic variance of the trait and is said to exert stabilizing selection if the population mean is near the peak of the curve. The critical selection parameter for this effect is the curvature of the landscape (Lande and Arnold 1983). The stronger the curvature, the greater the contraction in variance and the stronger the stabilizing effect. If we generalize this kind of landscape to two trait dimensions, we have a hill. In three trait dimensions, we have a spheroid. But, as we move to two or more dimensions, we need to consider the orientation of the hill, spheroid, and so forth. The selection parameters describing orientation, like the measures of curvatures, are second derivatives of fitness with respect to pairwise trait products. These orientation parameters are measures of so-called correlational selection. They describe effects on the phenotypic (and genetic) covariances of traits (Phillips and Arnold 1989). The set of curvature and orientation parameters can be conveniently arranged in a $\boldsymbol{\gamma}$-matrix, with coefficients of stabilizing selection on the main diagonal and coefficients of correlational selection elsewhere. The $\boldsymbol{\gamma}$-matrix can be estimated in a natural or experimental population by curvilinear regression of relative fitness on trait values and products of trait values (Lande and Arnold 1983). The important points are that we have a formal

theory relating multivariate curvature of the adaptive landscape to changes in the **G**-matrix and that we have statistical techniques for estimating those curvatures.

One useful equation from the second horizon relates the change in genetic constraints to the curvature and slope of the adaptive landscape and to mutation and recombination:

$$\Delta \mathbf{G} = \mathbf{G}(\boldsymbol{\gamma} - \boldsymbol{\beta}\boldsymbol{\beta}^{T})\,\mathbf{G} + \mathbf{U},$$

where $\Delta\mathbf{G}$ is the change in the **G**-matrix from one generation to the next, **G** is the additive genetic variance-covariance matrix before selection, the term in parentheses is the curvature of the adaptive landscape evaluated at the population's phenotypic mean, and **U** is a matrix describing mutational and recombination contributions to genetic variances and covariances (Lande 1980*a;* Phillips and Arnold 1989). The first term on the right represents the change in **G** within a generation due to selection (before mutation and recombination). The matrix of genetic constraints, **G**, evolves toward an equilibrium pattern that is a compromise between patterns imposed by selection and mutation (Lande 1980*a*). The pattern of stabilizing selection, however, will evidently play the dominant role in shaping the **G**-matrix unless pleiotropic input from mutation is strong. At equilibrium, $\Delta\mathbf{G} = \mathbf{0}$ and there is no directional selection ($\boldsymbol{\beta} = \mathbf{0}$). Rearranging the equation, we find that $-\hat{\mathbf{G}}\boldsymbol{\gamma}\hat{\mathbf{G}} = \mathbf{U}$, where $\hat{\mathbf{G}}$ is the equilibrium **G**-matrix. Solutions to this equation indicate that the pattern of the equilibrium **G**-matrix for two traits generally corresponds to the orientation of the adaptive landscape, which is described by the coefficients of stabilizing and correlational selection, $\boldsymbol{\gamma}$. Sokal (1978) and Cheverud (1982, 1984) have argued for such a correspondence but perhaps make the case stronger than it actually is. The correspondence between equilibrium patterns of selection and genetic constraint is strong in the special case considered by Cheverud (1984) in which there are no pleiotropic mutational inputs, or such inputs cancel, so that all off-diagonal elements in **U** are zero. In the face of strong pleiotropic inputs from mutation, however, the equilibrium pattern of genetic constraint can differ considerably from the pattern imposed by selection. Nevertheless, the theoretical results from the second and third horizons give hope that estimates of selection can illuminate the evolution of genetic constraints.

Although we can solve for the equilibrium **G**-matrix, solving for the evolutionary path of the **G**-matrix (its dynamics) is a difficult problem. As the phenotypic mean evolves on the adaptive landscape, the pattern of selection changes, and consequently the rate and direction of evolution in the **G**-matrix change. Those changes in **G** can in turn alter the rate and direction of change in phenotypic mean. However, coupled equations from the second and third horizons can be used to numerically trace the evolution of the **G**-matrix. Via and Lande (1987) have used this approach in a model for the evolution of phenotypic plasticity. In that model, even large displacements of the mean from an adaptive peak produce only small and transient changes in the **G**-matrix.

The long-term consequences of selective constraints on evolutionary outcome have been explored in a series of models for phenotypic evolution. These models assume constant patterns for genetic and selective constraints (i.e., an invariant

G-matrix and a constant adaptive landscape). Even though the adaptive landscape is constant, as the population mean moves on the landscape, it experiences changing selection pressures. Typically, an adaptive landscape with a single peak is modeled (Lande 1980*b;* Via and Lande 1985), but some models with two peaks have also been constructed (Felsenstein 1979; Kirkpatrick 1982; Slatkin 1984; Lande 1986; Slatkin and Kirkpatrick 1987; Charlesworth and Rouhani 1988).

Evolutionary models with constant adaptive landscapes and **G**-matrices provide some generalizations about the effects of selective constraints. First, the phenotypic mean of the population tends to evolve in an uphill direction on the adaptive landscape (Lande 1979). When selection is frequency-independent and the landscape is Gaussian, the population mean equilibrates on an adaptive peak. Bürger (1986), however, modeled evolution on a landscape that was an ascending ridge with increasingly steep flanks. He found that the population would equilibrate on the ridge crest when stabilizing selection reached a critical level. Second, frequency-dependent selection generally causes the population to equilibrate some distance downslope from an adaptive peak (Lande 1976*b,* 1980*b*). Finally, when the number of selective constraints is less than the number of genetic constraints so that some genetically variable traits are selectively neutral, there is a collection of possible equilibrium points (a line, plane, or hyperplane) rather than a single equilibrium point (Lande 1981; Lande and Arnold 1985). In the case of mating preferences and sexually selected traits, for example, the evolutionary outcome is changed dramatically depending on whether selection acts on mating preferences (Arnold 1987; Pomiankowski et al. 1991).

The most general effect of genetic covariances or unequal genetic variances is to cause curved evolutionary trajectories (Lande 1980*a;* Via and Lande 1985). The population mean evolves on a straight path toward an adaptive peak only in the special case of zero genetic covariances and equal genetic variances. When genetic covariances are nonzero and/or genetic variances are unequal, evolution proceeds rapidly in some directions but only very slowly in other directions (fig. 3). The consequence is a curved evolutionary trajectory toward the adaptive peak with a slow final approach in a direction for which there is little genetic variation. When the landscape has two peaks and the population mean is situated in a critical boundary region, genetic covariance can determine the peak toward which the population evolves (Bürger 1986; Slatkin and Kirkpatrick 1987).

The Fourth Horizon: Developmental and Functional Constraints and Their Consequences

A promising recent approach is to view genetic constraints as arising from underlying constraints (the lowest horizon in fig. 1). The search for order in genetic constraints has been pursued by students of morphological inheritance and by students of life-history evolution. Both schools seek a manageable theory for the evolution of constraints that is cast in terms of underlying, proximate causes. The goal of both schools is to move beyond the earlier attitude of viewing genetic constraints as parameters that must be estimated on a case-by-case basis. The constraints of the morphological school are usually developmental. The func-

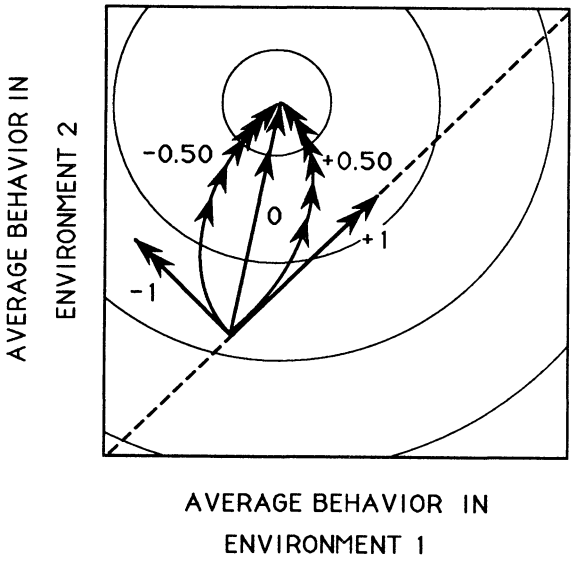

FIG. 3.—The Via-Lande model for the evolution of phenotypic plasticity. At the start of each generation, individuals disperse with equal probability into one of two environments and then spend their entire life in that environment. The *contour lines* show mean fitness as a function of average phenotype (behavior) in each environment. There is an adaptive peak near the top center of the plot. Populations with no behavioral plasticity would lie on the *dashed line*. Starting from the same behavioral average with no plasticity, evolutionary trajectories are shown for five populations with different values for genetic correlation in behavior. *Arrows* show evolutionary change in the mean at about 50 generation intervals (after Via and Lande 1985, fig. 3*C*).

tional constraints of the life-history school deal with limitations on trait values, but the constraints are not described in developmental terms.

Developmental constraints are limitations on the set of possible developmental states and their morphological expressions (Atchley 1987). Developmental constraints arise from ontogenetic processes, especially from their ordering in time. Many authors have argued that developmental processes and constraints have important evolutionary consequences (Gould 1977; Alberch et al. 1979; Riedl 1979; Bonner 1982; Wake 1991). One difficulty in evaluating the evolutionary consequences of developmental constraints is that most arguments have been cast in nongenetic terms. We can ask, however, How does development impose genetic constraints (Atchley 1987; Wagner 1989; Atchley and Hall 1991)?

One perspective on the genetic constraints that are imposed by development is gained by following the **G**-matrix through ontogeny. This approach has been explored by students on postnatal mammalian development for nearly three decades (reviewed in Atchley 1984). (For an introduction to an analogous tradition in the behavioral sciences, see Hahn et al. 1990.) A breeding design is conducted to produce individuals of known relationship, and then a set of measurements

(mass, head length, tail length, etc.) is made on each individual at a series of ages. The many genetic variances and covariances that are estimated in such a study can be displayed in a variety of tabular and graphic formats. For the present discussion, it is useful to imagine them assembled in a single developmental **G**-matrix. The matrix includes genetic variances and covariances at each age, as well as genetic covariances between traits measured at different ages.

Developmental **G**-matrices commonly show regularities in their elements. These regularities reflect genetic constraints that are imposed by developmental processes such as growth. For example, the genetic variance of a particular trait may increase with age (Cheverud et al. 1983*a*). Another common pattern is for the genetic correlation between the same trait measured at different ages (e.g., between head length at different ages) to be high when the two ages are close (e.g., ages 1 and 2) and to decline progressively as the two ages become more disparate (e.g., ages 1 and 10) (Cheverud et al. 1983*b;* Cheverud and Leamy 1985). Such regularities in the developmental **G**-matrix apparently reflect a greater communality of underlying processes for traits expressed at adjoining ages as opposed to widely separated ages. Atchley (1984) discusses how such regularities can be related to a model of compensatory (targeted) growth.

Inheritance, selection, and evolution of developmental trajectories can also be modeled with continuous functions (Kirkpatrick 1988; Kirkpatrick and Heckman 1989; Kirkpatrick et al. 1990). A character that changes with age can be viewed as a continuous function of age (an infinite-dimensional trait) rather than a measurement made at a series of landmark ages. In the infinite-dimensional framework, the **G**-matrix becomes a *G*-function. By decomposing the *G*-function into its eigenvalues and eigenfunctions, we can determine possible directions of evolutionary change in growth trajectories for which there is little or no genetic variation. In other words, we can identify the directions of evolutionary change that are most genetically constrained.

Another way to find a bridge between development and genetic constraints is to construct a model for the relationship between two traits and their developmental precursors and examine the model's statistical consequences. The relationship between precursor and trait constitutes the developmental constraint. Perhaps the easiest consequence to model is the correlation that arises when two traits develop from a common precursor. For example, Riska (1986) used standard expressions for the variance and covariance of variables that are the sums or products of other variables to dissect the correlations expected under various simple developmental processes. To use such models directly, we would need to assess the phenotypic and genetic variances and covariances of embryonic traits and developmental processes, as well as the statistics of end points. However, the quantitative genetics of embryos is a virtually unexplored realm.

Slatkin (1987) and Wagner (1989) have extended Riska's approach by constructing models for the evolution of developmentally coupled traits. Slatkin (1987) presents a modeling framework for the evolution of the developmental processes that underlie ontogenetic endpoints (adult traits). Slatkin solved for the phenotypic and genetic covariation of the end points in terms of the covariation

of the rates and timing of the underlying developmental processes. Assuming that selection acts directly on the adult traits and only indirectly on developmental processes, Slatkin is able to model the evolution of ontogeny as a correlated response to selection on adult traits. Wagner (1989) constructs his model on the premise that pleiotropic effects of a gene are constrained by the developmental framework in which it is expressed. The system of developmental constraints is assumed to be evolutionarily constant, and mutational input is superimposed on that system. Wagner is able to show that the equilibrium **G**-matrix is a function of per-locus mutation rates, developmental constraints, and the curvature of the adaptive landscape.

The cause of genetic constraint is also a contemporary issue among students of life-history evolution (Partridge and Sibly 1991). An important bridge between life-history evolution and quantitative genetics was provided by the insight that life-history trade-offs can be described by genetic covariances. If we wish to predict the short-term response of life-history traits to selection, it is the genetic covariances between traits rather than the more accessible phenotypic covariances that most directly affect the prediction (Lande 1982; Reznick 1985). In recent years the focus has shifted to the issue of whether a trade-off inevitably implies a negative genetic covariance and to the connection between functional and genetic constraints. In other words, the focus has shifted from the upper to the lower horizons in figure 1. Although this recent literature is directed at life-history evolution, its lessons have broader implications.

Van Noordwijk and de Jong (1986) make the important observation that variation in processes of resource acquisition can mask an underlying trade-off. The argument is easiest to understand by analogy with a problem in household economics. Within any given household there is likely to be a trade-off between the amount of money that can be spent on a car versus the home. But because households differ in income, when we look at the correlation between car and home expenditures across all households, the correlation may be positive rather than negative. More generally, variation in acquisition of resources can mask a trade-off between resources allocated to two competing alternatives (fig. 4). For example, the correlation between reproduction and subsequent survival rate may be positive rather than negative simply because individuals vary in the total amount of energy that can be devoted to these two functions. Houle (1991) casts the argument in genetic terms and explores its consequences. The bottom line is that a simple correlation is not an infallible indicator of underlying trade-offs. Interestingly, this point was appreciated by some empiricists and circumvented using partial correlation (Stewart 1979) before the general theoretical argument was advanced. The van Noordwijk–de Jong model, like Riska's (1986) models of developmental constraint, partitions the covariance between traits into the variation and covariation of underlying traits or processes. Limits on the values of trait combinations, which will be considered next, are a stronger form of constraint.

Functional constraints are limitations on values of traits or of trait combinations. The limitations are imposed by time, energy, or the laws of physics. In models of life-history equilibria, functional constraints take such forms as "total

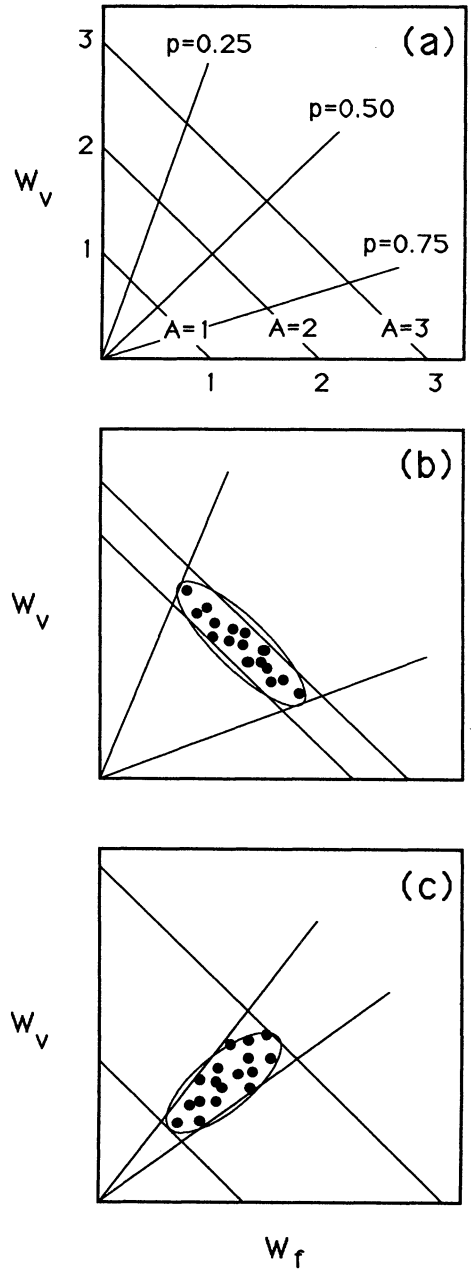

FIG. 4.—The van Noordwijk–de Jong model for covariance between two life-history traits, w_f and w_v, in terms of variation in underlying processes of acquisition and allocation. *a*, Acquisition, A, and allocation, p, constrain the values of viability, w_v, and fecundity, w_f, because $w_f = pA$ and $w_v = (1 - p)A$. Acquisition and allocation are independent random variables, but they determine the covariance between w_v and w_f. *b*, Large variation in allocation and little variation in acquisition result in a negative covariance between w_v and w_f. *c*, Large variation in acquisition and little variation in allocation results in a positive correlation between w_v and w_f.

lifetime fecundity cannot exceed a fixed constant'' or ''there is a convex curve relating viability to fecundity'' (Charnov 1989; Charlesworth 1990). Functional constraint is another label for the trade-off curves that are used in optimization models in behavioral ecology (Stephens and Krebs 1986; Mangel and Clark 1988). Under some rather strong assumptions, the genetic variances and covariances for a set of traits can be deduced from functional constraints (Charnov 1989; Charlesworth 1990). The approach parallels Wagner's (1989) model of developmental constraints.

Some general statements can be made about equilibrium genetic variances and covariances when a set of traits is bound together by functional constraints. A rendition of the Charnov-Charlesworth argument is given in figure 5 for a case involving only two traits that are bound together at equilibrium by a convex trade-off. When multiple traits are considered, genetic covariances are no longer a simple mapping of the underlying trade-off curves (Charlesworth 1990). Furthermore, negative genetic covariances are not an inevitable outcome; some genetic covariances can be positive (Pease and Bull 1988; Charlesworth 1990). In the case of multiple life-history traits, the most general conclusion that has been reached is that at least one pair of traits will show a negative genetic covariance at equilibrium (Lande 1982; Charlesworth 1990). Note, however, that these arguments (e.g., fig. 5) may not apply if there is substantial curvature of the adaptive landscape. In that case, the population may equilibrate at an adaptive peak well inside the boundary imposed by a constraint.

Price and Schluter (1991) and Schluter et al. (1991) provide a different perspective on the genetic constraints affecting life-history evolution. In the simplest rendition of the Price-Schluter argument (fig. 6), the two primary components of fitness, fecundity and viability, each exert stabilizing selection on a single morphological trait but select toward different optima. The model assumes maintenance of genetic variance for the morphological trait. Genetic variance and covariance of fecundity and viability, and hence genetic variance for total fitness, are a linear function of directional selection on the morphological trait and its genetic variance. As the morphological trait evolves, the components of directional selection on it change, and consequently the genetic variances and covariances of the life-history traits change in a simple pattern (fig. 6). In the Price-Schluter model an evolving morphological trait constrains genetic variation in life history, while in the Charnov-Charlesworth model functional limits on life history play that role. Despite their apparent differences, the two types of models yield similar predictions about the evolutionary genetics of life history.

BEHAVIOR AS A BUILDER AND BREAKER OF GENETIC CONSTRAINTS

In the preceding overview we have seen a trend toward seeking the causes of constraints in terms of lower-level constraints. Formal theory is developing more or less rapidly along these lines, and many aspects of behavioral evolution can be profitably viewed in this telescoping framework. In other words, when evolving behavior is viewed as a responder to constraints, a considerable amount of formal theory is available to us. Other aspects of behavioral evolution, however, do not

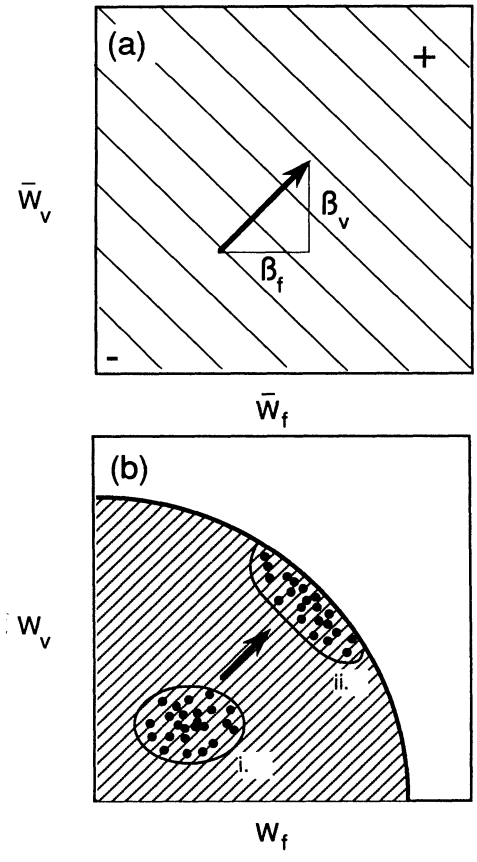

FIG. 5.—The Charnov-Charlesworth model for equilibrium genetic covariance for a single pair of life-history traits. *a*, Two life-history traits, fecundity and viability, with means $\bar{w}_f$ and $\bar{w}_v$ are under perpetual directional selection (*arrow*) for increasing values. In other words, the adaptive landscape slopes upward toward the upper right corner. Contours for average total fitness are shown. *b*, The *heavy, curved line* shows the trade-off curve that describes the functional constraint between the two traits, w_f and w_v. The constraint is such that w_f and w_v can only take values that lie inside the curve, in the *cross-hatched region*. Because w_f and w_v are always under directional selection for higher values, the population will tend to evolve outward from a position such as *i*, so that at equilibrium it lies on the trade-off curve, for example, at *ii*. Perpetual directional selection has the effect of pushing the bivariate distribution of genetic values up against the curve so that a negative genetic covariance prevails at equilibrium.

fit so comfortably into the framework we have surveyed. In particular, the evolution of some behaviors will affect the evolution of constraints. Such effects, in which behavior acts as a builder and breaker of constraints, would constitute downward arrows in figure 1. Such effects do not challenge or threaten our framework, but behavior viewed as a builder and breaker of constraints has received less attention than behavior as a simple responder.

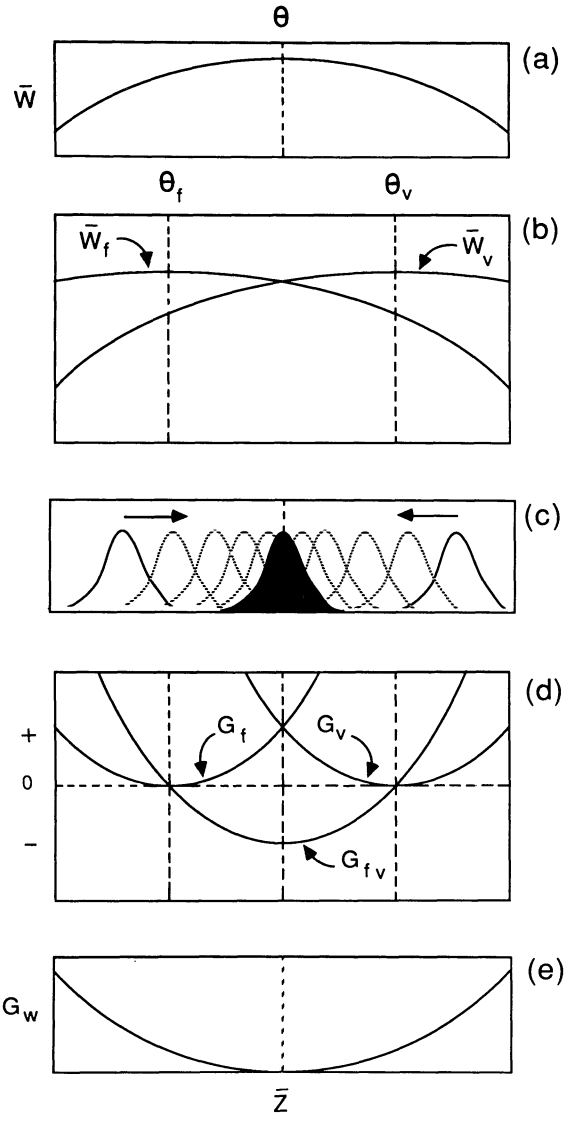

FIG. 6.—The Price-Schluter model for the evolution of genetic variance and covariance of two fitness components in response to the evolution of the mean of a morphological trait, $\bar{z}$. *a,* The morphological trait is under stabilizing selection toward an intermediate optimum, θ. *b,* With respect to fecundity the trait is selected toward an optimum, θ_f, that differs from the optimum under viability selection, θ_v. *c,* The mean of the morphological trait and its phenotypic distribution (shown as normal curves) evolve toward an equilibrium position under the overall optimum, θ. *d,* Because the two fitness components, fecundity and viability, are a function of directional selection on z and its genetic variance, the genetic variances of fecundity and viability, G_f and G_v, and their genetic covariance, G_{fv}, change as $\bar{z}$ evolves. *e,* Because total fitness is a function of its two components, its genetic variance, G_w, also changes as $\bar{z}$ evolves.

Behavior as a Builder of Constraints

Behaviors that affect the choice of mates and habitat have a special impact on population genetics. They can affect correlations between alleles at different loci and build genetic covariance through linkage disequilibrium. Such genetic covariance is fragile in the sense that it decays rapidly if selection is relaxed (Crow and Kimura 1970). But if selection is persistent, behaviors involved in mate and habitat choice may be capable of promoting genetic covariance that will in turn have important evolutionary consequences. Likewise, when optimal morphology or physiology varies with behavior, selection may promote genetic covariance between behavior and other phenotypic attributes.

Mating preferences within a population may help maintain a stable genetic covariance between the behavior of one sex and a sexually selected trait in the other sex (Lande 1981). Thus, mating preferences can create a genetic constraint that can in turn profoundly affect their evolution.

Correlational selection that favors certain combinations of behavior and morphology (or physiology) may be capable of creating genetic covariances by promoting linkage disequilibrium. Students of host plant polymorphism have argued that such selection may build genetic covariance between behaviors that function in host plant recognition and traits that affect efficiency in using host plants (Via 1991). Brodie (1989, 1991) has argued that correlational selection may favor certain combinations of coloration and antipredator behaviors and has provided evidence for both correlational selection and genetic covariance in a polymorphic population of garter snakes.

Behavior as a Breaker of Constraints

Some kinds of behavior will have the effect of breaking prevailing constraints. Behavior that promotes acquisition of resources is one example, and learning is another. Undoubtedly there are many others.

In the van Noordwijk–de Jong (1986) model, two compartments for allocation show a negative covariance when there is little variation in acquisition of resources. Variation in behaviors that promote acquisition of resources and time should be capable of breaking this kind of constraint. What limits variation in behavior that could rescue populations from constraints and limits on resource acquisition?

When behavior is not modified by maturation or experience, the same behavior is performed in all circumstances. Consequently, when the fitness consequences of a behavior change from moment to moment or from season to season, the best invariant behavior is a compromise between fluctuating optima in behavioral states. Temporally invariant behavior is constrained in the sense that there is perfect phenotypic (and presumably genetic) serial autocorrelation. Learning can break this type of constraint. In other words, learning represents an escape from the constraint of serial autocorrelation in behavior. We could probably model the evolution of learning and its constraint-breaking effects using the infinite-dimensional format recently introduced by Kirkpatrick and his colleagues (Kirkpatrick 1988; Kirkpatrick and Heckman 1989; Kirkpatrick et al. 1990).

SUMMARY

Consideration of constraint is one of the most active areas in evolutionary biology, as evidenced by an ongoing explosion of literature. Constraints on evolution have been profitably viewed from many different perspectives. My approach has been not to challenge any of these diverse perspectives but to seek their connections. I see genetics as the connecting principle because it provides the machinery for predicting the attributes of the next generation from those of its predecessor. Quantitative genetic theory emerges as the particular organizing principle for viewing evolutionary constraints because it deals with the inheritance and evolution of polygenic, continuously distributed traits that figure in discussions of phenotypic evolution. The formal theory of quantitative genetics and its ongoing extensions can be conveniently grouped into a series of levels or horizons. The first two horizons are models for the evolution of the first two genetic moments of trait distributions (means and variance-covariances). Selective, developmental, and functional constraints exert their evolutionary effects via these two horizons. Consequently, quantitative genetics provides a formal mechanism for evaluating the short-term consequences of selective, developmental, and functional constraints.

Quantitative genetic theory does not make predictions about the long-term persistence of evolutionary constraints. The persistence of genetic and other kinds of constraint can be evaluated with comparative studies, but relatively little work has been pursued along these lines. The evolutionary persistence of adaptive landscapes, for example, is virtually unexplored. Thus, quantitative genetics alone is not a sufficient beacon to guide evolutionary studies. It needs to be combined with ecological, developmental, functional, and phylogenetic perspectives.

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LITERATURE CITED

Alberch, P., S. J. Gould, G. Oster, and D. Wake. 1979. Size and shape in ontogeny and phylogeny. Paleobiology 5:296–317.

Arnold, S. J. 1981. Behavioral variation in natural populations. I. Phenotypic, genetic and environmental correlations among chemoreceptive responses to prey in the garter snake, *Thamnophis elegans*. Evolution 35:489–509.

———. 1987. Quantitative genetic models of sexual selection: a review. Pages 283–315 *in* S. C. Stearns, ed. The evolution of sex and its consequences. Birkhäuser, Basel.

———. 1988. Quantitative genetics and selection in natural populations: microevolution of vertebral numbers in the garter snake *Thamnophis elegans*. Pages 619–636 *in* B. S. Weir, E. J. Eisen, M. M. Goodman, and G. Namkoong, eds. Proceedings of the Second International Conference on Quantitative Genetics. Sinauer, Sunderland, Mass.

Atchley, W. R. 1984. Ontogeny, timing of development, and genetic variance-covariance structure. American Naturalist 123:519–540.

———. 1987. Developmental quantitative genetics and the evolution of ontogenies. Evolution 41: 316–330.

Atchley, W. R., and B. K. Hall. 1991. A model for development and evolution of complex morphological structures. Biological Reviews of the Cambridge Philosophical Society 66:101–157.

Atchley, W. R., J. J. Rutledge, and D. E. Cowley. 1981. Genetic components of size and shape. II. Multivariate covariance patterns in the rat and mouse skull. Evolution 35:1037–1055.

Boake, C. R. P. 1993. Quantitative genetics studies of the evolution of behavior. University of Chicago Press, Chicago (in press).

Bonner, J. T. 1982. Evolution and development. Springer, Berlin.

Brodie, E. D., III. 1989. Genetic correlations between morphology and antipredator behavior in natural populations of the garter snake *Thamnophis ordinoides*. Nature (London) 342:542–543.

———. 1991. Functional and genetic integration of color pattern and antipredator behavior in the garter snake *Thamnophis ordinoides*. Ph.D. diss. University of Chicago, Chicago.

Bürger, R. 1986. Constraints for the evolution of functionally coupled characters: a nonlinear analysis of a phenotypic model. Evolution 40:182–193.

Charlesworth, B. 1990. Optimization models, quantitative genetics, and mutation. Evolution 44: 520–538.

Charlesworth, B., and S. Rouhani. 1988. The probability of peak shifts in a founder population. II. An additive polygenic trait. Evolution 42:1129–1145.

Charnov, E. L. 1989. Phenotypic evolution under Fisher's fundamental theorem of natural selection. Heredity 62:113–116.

Cheverud, J. M. 1982. Phenotypic, genetic, and environmental morphological integration in the cranium. Evolution 36:499–516.

———. 1984. Quantitative genetics and developmental constraints on evolution by selection. Journal of Theoretical Biology 110:155–171.

Cheverud, J. M., and L. J. Leamy. 1985. Quantitative genetics and the evolution of ontogeny. III. Ontogenetic changes in correlation structure among live-body traits in randombred mice. Genetical Research 46:325–335.

Cheverud, J. M., L. J. Leamy, W. R. Atchley, and J. J. Rutledge. 1983*a*. Quantitative genetics and the evolution of ontogeny. I. Ontogenetic changes in quantitative genetic variance components in randombred mice. Genetical Research 42:65–75.

Cheverud, J. M., J. J. Rutledge, and W. R. Atchley. 1983*b*. Quantitative genetics of development: genetic correlations among age-specific trait values and the evolution of ontogeny. Evolution 37:895–905.

Crow, J. F., and M. Kimura. 1970. An introduction to population genetics theory. Harper & Row, New York.

Dickerson, G. E. 1955. Genetic slippage in response to selection for multiple objectives. Cold Spring Harbor Symposia on Quantitative Biology 20:213–224.

Endler, J. A. 1986. Natural selection in the wild. Princeton University Press, Princeton, N.J.

Falconer, D. S. 1960. Introduction to quantitative genetics. Ronald, New York.

———. 1989. Introduction to quantitative genetics. 3d ed. Longman, New York.

Felsenstein, J. 1979. Excursions along the interface between disruptive and stabilizing selection. Genetics 93:773–795.

———. 1988. Phylogenies and quantitative characters. Annual Review of Ecology and Systematics 19:445–471.

Fisher, R. A. 1918. The correlation between relatives on the supposition of Mendelian inheritance. Transactions of the Royal Society of Edinburgh 52:399–433.

———. 1930. The genetic theory of natural selection. Clarendon, Oxford.

Flury, B. K. 1987. A hierarchy of relationships between covariance matrices. Pages 31–43 *in* A. K. Gupta, ed. Advances in multivariate statistical analysis. Reidel, Dordrecht.

Gale, J. S., and L. J. Eaves. 1972. Variation in wild populations of *Papaver dubium*. V. The application of factor analysis to the study of variation. Heredity 29:135–149.

Gould, S. J. 1977. Ontogeny and phylogeny. Harvard University Press, Cambridge, Mass.

Gyles, N. R., G. E. Dickerson, G. B. Kinder, and H. L. Kempster. 1955. Initial and actual selection in poultry. Poultry Science 34:530–539.

Hahn, M. E., J. K. Hewitt, and N. D. Henderson. 1990. Developmental behavior genetics: neural, biometric, and evolutionary approaches. Oxford University Press, Oxford.

Hazel, L. N. 1943. The genetic basis for constructing selection indexes. Genetics 28:476–490.

Hill, W. G. 1984*a*. Quantitative genetics. Pt. 1. Explanation and analysis of continuous variation. Van Nostrand Reinhold, New York.

———. 1984*b*. Quantitative genetics. Pt. 2. Selection. Van Nostrand Reinhold, New York.

Houle, D. 1991. Genetic covariance of fitness correlates: what genetic correlations are made of and why it matters. Evolution 45:630–648.

Kirkpatrick, M. 1982. Quantum evolution and punctuated equilibria in continuous genetic characters. American Naturalist 119:833–848.

———. 1988. The evolution of size in size-structured populations. Pages 13–28 *in* B. Ebeman and L. Persson, eds. The dynamics of size-structured populations. Springer, Berlin.

Kirkpatrick, M., and N. Heckman. 1989. A quantitative genetic model for growth, shape, reaction norms, and other infinite-dimensional characters. Journal of Mathematical Biology 27: 429–450.

Kirkpatrick, M., D. Lofsvold, and M. Bulmer. 1990. Analysis of inheritance, selection and evolution of growth trajectories. Genetics 124:979–993.

Kohn, L. A. P., and W. R. Atchley. 1988. How similar are genetic correlation structures? Data from mice and rats. Evolution 42:467–481.

Lande, R. 1976*a*. The maintenance of genetic variability by mutation in a polygenic character with linked loci. Genetical Research 26:221–235.

———. 1976*b*. Natural selection and random genetic drift in phenotypic evolution. Evolution 30: 314–334.

———. 1979. Quantitative genetic analysis of multivariate evolution, applied to brain:body size allometry. Evolution 33:402–416.

———. 1980*a*. The genetic covariance between characters maintained by pleiotropic mutations. Genetics 94:203–215.

———. 1980*b*. Sexual dimorphism, sexual selection, and adaptation in polygenic characters. Evolution 34:292–305.

———. 1981. Models of speciation by sexual selection on polygenic traits. Proceedings of the National Academy of Sciences of the USA 78:3721–3725.

———. 1982. A quantitative genetic theory of life history evolution. Ecology 63:607–615.

———. 1984. The genetic correlation between characters maintained by selection, linkage and inbreeding. Genetical Research 44:309–320.

———. 1986. The dynamics of peak shifts and the pattern of morphological evolution. Paleobiology 12:343–354.

Lande, R., and S. J. Arnold. 1983. The measurement of selection on correlated characters. Evolution 37:1210–1226.

———. 1985. Evolution of mating preference and sexual dimorphism. Journal of Theoretical Biology 117:651–664.

Leamy, L., and J. M. Cheverud. 1984. Quantitative genetics and the evolution of ontogeny. II. Genetic and environmental correlations among age-specific characters in randombred house mice. Growth 48:339–353.

Lofsvold, D. 1986. Quantitative genetics of morphological differentiation in *Peromyscus*. I. Test of the homogeneity of genetic covariance structure among species and subspecies. Evolution 40:559–573.

———. 1988. Quantitative genetics of morphological differentiation in *Peromyscus*. II. Analysis of selection and drift. Evolution 42:54–67.

Lush, J. L. 1937. Animal breeding plans. Iowa State College Press, Ames.

Magee, W. T. 1965. Estimating response to selection. Journal of Animal Science 24:242–247.

Mangel, M., and C. W. Clark. 1988. Dynamic modeling in behavioral ecology. Princeton University Press, Princeton, N.J.

Maynard Smith, J., R. Burian, S. Kauffman, P. Alberch, J. Campbell, B. Goodwin, R. Lande, D. Raup, and L. Wolpert. 1985. Developmental constraints and evolution. Quarterly Review of Biology 60:265–287.

Partridge, L., and R. Sibly. 1991. Constraints in the evolution of life histories. Philosophical Transactions of the Royal Society of London B, Biological Sciences 332:3–13.

Pease, C. M., and J. J. Bull. 1988. A critique of methods for measuring life history trade-offs. Journal of Evolutionary Biology 1:293–303.

Phillips, P. C., and S. J. Arnold. 1989. Visualizing multivariate selection. Evolution 43:1209–1222.

Pomiankowski, A., Y. Iwasa, and S. Nee. 1991. The evolution of costly mate preferences. I. Fisher and biased mutation. Evolution 45:1422–1430.

Price, T. D., and P. R. Grant. 1985. The evolution of ontogeny in Darwin's finches: a quantitative genetic approach. American Naturalist 125:169–188.

Price, T., and D. Schluter. 1991. On the low heritability of life-history traits. Evolution 45:853–861.

Price, T. D., P. R. Grant, and P. T. Boag. 1984. Genetic changes in the morphological differentiation of Darwin's ground finches. Pages 49–66 *in* K. Wohrmann and V. Loeschcke, eds. Population biology and evolution. Springer, Berlin.

Reznick, D. 1985. Costs of reproduction: an evaluation of the empirical evidence. Oikos 44:257–267.

Riedl, R. 1979. Order in living organisms. Wiley, New York.

Riska, B. 1986. Some models for development, growth, and morphometric correlation. Evolution 40:1301–1311.

Robertson, A. 1955. Selection in animals: synthesis. Cold Spring Harbor Symposia on Quantitative Biology 20:225–229.

Schluter, D. 1984. Morphological and phylogenetic relations among the Darwin's finches. Evolution 38:921–930.

Schluter, D., T. D. Price, and L. Rowe. 1991. Conflicting selection pressures and life history tradeoffs. Proceedings of the Royal Society of London B, Biological Sciences 246:11–17.

Shaw, R. G. 1991. The comparison of quantitative genetic parameters between populations. Evolution 45:143–151.

Slatkin, M. 1984. Ecological causes of sexual dimorphism. Evolution 38:622–630.

———. 1987. Quantitative genetics of heterochrony. Evolution 41:799–811.

Slatkin, M., and M. Kirkpatrick. 1987. Extrapolating quantitative genetic theory to evolutionary problems. Pages 283–293 *in* M. D. Huetel, ed. Evolutionary genetics of invertebrate behavior. Plenum, New York.

Smith, H. F. 1936. A discriminant function for plant selection. Annals of Eugenics 7:240–250.

Sokal, R. R. 1978. Population differentiation: something new or more of the same? Pages 215–239 *in* P. F. Brussard, ed. Ecological genetics: the interface. Springer, Berlin.

Stephens, D. W., and J. R. Krebs. 1986. Foraging theory. Princeton University Press, Princeton, N.J.

Stewart, J. R. 1979. The balance between number and size of young in the live-bearing lizard *Gerrhonotus coeruleus*. Herpetologica 35:342–350.

Turelli, M. 1984. Heritable genetic variation via mutation-selection balance: Lerch's zeta meets the abdominal bristle. Theoretical Population Biology 25:138–193.

———. 1985. Effects of pleiotropy on predictions concerning mutation-selection balance for polygenic traits. Genetics 111:165–195.

———. 1986. Gaussian versus non-Gaussian genetic analyses of polygenic mutation-selection balance.

Pages 607–626 *in* S. Karlin and E. Nevo, eds. Evolutionary processes and theory. Academic Press, New York.

———. 1988. Phenotypic evolution, constant covariances, and the maintenance of additive genetic variance. Evolution 42:1342–1347.

van Noordwijk, A. J., and G. de Jong. 1986. Acquisition and allocation of resources: their influence on variation in life history tactics. American Naturalist 128:137–142.

Via, S. 1991. The genetic structure of host plant adaptation in a spatial patchwork: demographic variability among reciprocally transplanted pea aphid clones. Evolution 45:827–852.

Via, S., and R. Lande. 1985. Genotype-environment interaction and the evolution of phenotypic plasticity. Evolution 39:505–522.

———. 1987. Evolution of genetic variability in a spatially heterogeneous environment: effects of genotype-environment interaction. Genetical Research 49:147–156.

Wagner, G. P. 1989. Multivariate mutation-selection balance with constrained pleiotropic effects. Genetics 122:223–234.

Wake, D. B. 1991. Homoplasy: the result of natural selection or evidence of design limitations? American Naturalist 138:543–567.

Weinberg, W. 1910. Weitere Beiträge zur Theorie der Verebung. Archiv für Rassen- und Gesellschafts-Biologie 7:35–49.

Wilkinson, G. S., K. Fowler, and L. Partridge. 1990. Resistance of genetic correlation structure to directional selection in *Drosophila melanogaster*. Evolution 44:1990–2003.

Wright, S. 1921. Systems of mating. I. The biometric relations between parent and offspring. Genetics 6:111–123.

Yamada, Y. 1977. Evaluation of the culling variate used by breeders in actual selection. Genetics 86:885–899.

Young, S. S. Y., and H. Weiler. 1960. Selection for two correlated traits by independent culling levels. Journal of Genetics 57:329–338.

Zeng, Z.-B. 1988. Long-term correlated response, interpopulation covariation, and interspecific allometry. Evolution 42:363–374.