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A QUANTITATIVE GENETIC THEORY OF LIFE HISTORY EVOLUTION[1]

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Abstract. Dynamic models of quantitative (polygenic) characters are more generally applicable in the analysis of life history evolution than are static optimization methods or one and two locus genetic models. A dynamic theory of life history evolution is derived by synthesizing population demography with quantitative genetics. In a population under weak selection with a nearly stable age distribution, the relative fitness of individuals with a particular life history phenotype can be approximated as an average of age-specific relative fecundity and mortality rates, weighted respectively by the present productivity and future reproductive value of each age-class. An adaptive topography is constructed showing that, with phenotype- and age-specific fecundity and mortality rates constant in time, evolution of the mean life history maximizes the intrinsic rate of increase of a population. However, the rate and direction of evolution in response to selection are strongly influenced by genetic correlations among characters. Negative genetic correlations among major components of fitness are often obscured phenotypically by positive environmental correlations, but commonly constitute the ultimate constraint on life history evolution, as illustrated by artificial selection experiments. Methods are suggested for measuring selective forces and evolutionary constraints that affect life history characters in natural populations.

Key words: adaptive topography; age distribution; evolutionary dynamics; fitness; genetic constraints; life history; natural selection; population demography; quantitative characters; reproductive value.

INTRODUCTION

Models of life history evolution are usually based on optimization methods, which attempt to predict the equilibrium state(s) of a population by maximizing some measure of fitness subject to certain constraints (reviewed by Stearns 1977). The use of optimization depends not only on correct identification of a quantity maximized by evolution and an appropriate set of constraints, but also on the assumption that sufficient time and suitable genetic variations have occurred for the population to reach an optimum (Cody 1974, Maynard Smith 1978, Lewontin 1979). Many of the difficulties with static models of evolutionary processes, including optimization, game theory, and similar techniques, can be overcome by dynamic models that incorporate genetic mechanisms of evolutionary change and indicate the appropriate evolutionary constraints.

Without a dynamic theory it may be difficult or impossible to identify a quantity maximized by evolution. For example, Maynard Smith (1978) asserts that life history evolution maximizes the expected number of offspring in an individual's lifetime. Dynamic models of gene frequency change (Fisher 1958, Charlesworth 1980) indicate, however, that in a constant environment life history evolution maximizes the growth rate of a population. Under some types of density-dependent selection, evolution maximizes the population size itself (Fisher 1958:45–48, Charlesworth 1980:Chap. 4). Frequency-dependent selection, immigration, mutation, changing environments, and random genetic drift can substantially decrease the mean fitness in a population (Wright 1949, 1969:Chap. 5, Fisher 1958:Chap. 6, Lande 1980*b*, 1981). Even when some measure of fitness is maximized, the possible existence of multiple stable equilibria (Wright 1932, 1949, Simpson 1953, Schaffer and Rosenzweig 1977) casts further doubt on the general utility of optimization methods in analyzing life history evolution.

Life history is here broadly defined to include not only the age-specific fecundity and mortality rates, but the entire sequence of changes through which an organism passes in its development from conception to death. The morphological, physiological, and behavioral characters of organisms which determine their age-specific fecundity and mortality schedules are generally influenced by many genes of small effect and by environmental effects (Falconer 1960, Wright 1968:Chap. 15, 1978:Chap. 8). This creates severe measurement problems, since it is usually impossible or impractical to distinguish individual genes influencing particular quantitative characters. The theory of gene frequency evolution therefore cannot be directly applied to classical evolutionary problems concerning whole organisms and their phenotypes. Thus, there is a need for a statistical theory of phenotypic evolution that distills the essential features of complex genetic systems into relatively few variables that can be easily estimated from phenotypic measurements. The foundations of such a theory, known as quantitative ge-

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netics, were established by R. A. Fisher (1918) and Sewall Wright (1921).

Quantitative genetics has been applied mainly in practical animal and plant breeding programs (Lush 1945, Falconer 1960, Hanson and Robinson 1963) but has been largely ignored by naturalists and ecologists. The evolutionary implications of animal and plant breeding and artificial selection experiments were clearly realized by Darwin (1859, 1876). He understood that correlated variation of individual characters in a population implies that when man or nature selects to change one character, an evolutionary response will occur not only in the selected character but also in characters correlated with it.

In large outcrossing populations almost any simple character will respond to artificial selection (Falconer 1960:Chap. 10, Lewontin 1974:86–94). Advocates of optimization rely on this fact to suggest that genetic variation is generally available to evolve rapidly whatever is selected for in nature (e.g., Maynard Smith 1978, Oster and Wilson 1979:Chap. 8). However, natural selection acts simultaneously on many characters, and these cannot evolve independently because of genetic correlations between them caused by pleiotropy and linkage disequilibrium. Pleiotropic (manifold) effects of a mutant gene usually result from a primary biochemical or developmental perturbation that directly affects certain characters, and may cause compensatory changes in other traits later in development (Caspari 1952, Grüneberg 1963). Pleiotropy is a nearly ubiquitous property of mutations that affect quantitative characters in higher organisms (Gaul 1961, Wright 1968:60–61). Stable linkage disequilibrium (nonrandom combination of alleles at different loci in gametes) is produced by tight linkage of genes that interact to produce an adaptive phenotype (Lewontin 1974), as may occur for character complexes governed by families of related genes that originated partly as tandem duplications (Ohno 1970, MacIntyre 1976). The genetic correlations among characters due to pleiotropy and linkage disequilibrium influence both the rate and direction of evolution in response to selection. Negative genetic correlations between major components of fitness can prevent any net progress, even though genetic variance exists for each component trait. This is a frequent limitation on artificial selection in large populations (Dickerson 1955, Falconer 1960:Chaps. 19, 20).

In a dynamic model of life history evolution, all types of constraints must be incorporated in the multivariate pattern of selection or in the phenotypic and genetic variance-covariance structures of the characters. Thus developmental or energetic constraints must appear as a lack of phenotypic and/or genetic variance for certain character combinations (i.e., negative correlations between the component traits), while functional or ecological constraints are incorporated in the low fitness of certain phenotypes. Quantitative genetics provides a mechanistic theory of phenotypic evolution that can be integrated with other fields of study, such as development and ecology. The present paper illustrates this point by combining quantitative genetics with population demography to construct a dynamic theory of life history evolution.

The use of simplifying assumptions allows the formulation of a tractable analytical model which may serve as a basis for understanding more complex situations. Assuming weak selection and a nearly stable age distribution, the concept of individual fitness is defined for species with overlapping generations. Methods are suggested for estimating the forces of directional selection on correlated life history characters, and an adaptive topography is derived for phenotypic evolution in an age-structured population.

Variance and covariance of life-history characters

Populations are assumed to be either monoecious, or dioecious with the same pattern of age-specific selection on both sexes and no sexual dimorphism. The life history of an individual is represented by a column vector of quantitative characters, some or all of which may be age specific.

$$\mathbf{z} = (z_1, \ldots, z_n)^{\mathsf{T}},$$

where $^{\mathsf{T}}$ denotes matrix transposition. As can often be arranged by a simple scale transformation, usually logarithmic, the joint distribution of life history characters in a population of unselected individuals is assumed to be multivariate normal (Gaussian), with mean $\bar{\mathbf{z}}$ and variance-covariance matrix P. Either artificial selection experiments or observations on phenotypic correlations among relatives can be used to partition P into additive genetic and environmental (plus nonadditive genetic) components (Kempthorne 1957, Mode and Robinson 1959, Falconer 1960). These methods are directly applicable to characters of individuals measured at different ages. If there are no genotype-environment correlations or interactions, the life history phenotype of an individual can be written as the sum of an additive genetic (or breeding) value $\mathbf{x}$ and an environmental deviation $\mathbf{e}$, with $\mathbf{z} = \mathbf{x} + \mathbf{e}$. Denoting the additive genetic and environmental variance-covariance matrices as G and E, respectively, it follows that

$$\mathrm{P} = \mathrm{G} + \mathrm{E}. \tag{1}$$

The distributions of breeding values and environmental effects are each assumed to be multivariate normal, as expected for polygenic characters with a Gaussian phenotype distribution. Any system of mating consistent with multivariate normality is allowed, including random or assortative mating by phenotype (Fisher 1918, Lande 1977, 1980*a*) or age.

It is sometimes convenient to analyze life history variation and evolution by considering homologous

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traits at different ages as separate characters, such as age-specific body sizes. Organisms with complex life cycles, as in many arthropods and amphibians, naturally lend themselves to this approach. But if several characters are measured at many ages, the analysis may become impractical. A powerful method of reducing the number of characters is through the analysis of parameters of individual growth curves. In many species of plants, reptiles, birds, and mammals, the observable ontogeny of an individual can be described by a few constant parameters such as allometric coefficients, growth rates, and initial and final size and shape. Life history variation and evolution can then be analyzed in terms of these individual growth curve parameters (Kidwell et al. 1952, 1979, Cock 1966, Atchley and Rutledge 1981). For species with determinate growth, the models also indicate conditions under which the evolution of the adult phenotype can be analyzed independently of preadult characters (see Discussion), further reducing the number of characters.

Population demography

The basic demographic model of age-structured populations in continuous time is used (Lotka 1956, Keyfitz 1968). However, the age-specific survival and fecundity rates are allowed to depend on the quantitative characters which compose the phenotype of an individual during its lifetime. Letting individuals with life history phenotype $\mathbf{z}$ have fertility and mortality rates at age a denoted as $m_a(\mathbf{z})$ and $\mu_a(\mathbf{z})$, the probability of survival from conception (age 0) to age a is (Keyfitz 1968:Chap. 1)

$$l_a(\mathbf{z}) = \exp\left[-\int_0^a \mu_y(\mathbf{z})\, dy\right], \tag{2}$$

and the fitness at age a of individuals with life history $\mathbf{z}$ is defined as

$$w_a(\mathbf{z}) = l_a(\mathbf{z})m_a(\mathbf{z}). \tag{3}$$

The expected fitness at age a of a cohort of newly conceived individuals is written as $\bar{w}_a$.

In most biologically feasible situations where the lifetime reproduction of individuals is bounded, as when there is a maximum age for reproduction or survival, the intrinsic rate of increase of a population at any time can be defined as the unique real root r of the equation (Feller 1941, Keyfitz 1968:Chap. 5)

$$\int_0^\infty e^{-ra}\bar{w}_a\, da = 1. \tag{4}$$

The intrinsic rate of increase of a population with a given phenotypic composition is the exponential rate of increase of total population size that would eventually be achieved in the absence of evolutionary changes (Charlesworth 1974, 1980:136), as when all of the phenotypic variation is nonheritable or when the population is at an evolutionary equilibrium.

The stable age distribution of such a population is

$$c_a = be^{-ra}\bar{l}_a, \tag{5}$$

where

$$b^{-1} = \int_0^\infty /e^{-ra}\bar{l}_a\, da, \tag{6}$$

and $\bar{l}_a$ is the expected probability of survival to age a. From Eq. 4 it can be shown that b is the per capita birth rate for the population at the stable age distribution. The per capita death rate d can then be obtained from $r = b - d$ (Lotka 1956, Keyfitz 1968).

The stable distribution of age at reproduction in a population with a constant phenotypic composition is, using Eq. 4,

$$q_a = e^{-ra}\bar{w}_a. \tag{7}$$

Also, q_a is the proportion of newly conceived individuals contributed by parents aged a, and will be referred to as the present productivity of age-class a. The generation time, or mean age of reproducing parents, is

$$T = \int_0^\infty aq_a\, da. \tag{8}$$

The reproductive value of individuals aged a is their eventual demographic contribution to future generations relative to individuals at conception, defined following Fisher (1958) as

$$v_a/v_0 = e^{ra}\bar{l}_a^{-1}\int_a^\infty q_y\, dy \tag{9}$$

with $v_0 = 1$. The average reproductive value in a population with a stable age distribution equals the per capita birth rate per generation (Keyfitz 1968: Chap. 5.2),

$$\int_0^\infty c_a v_a\, da = bT.$$

Eqs. 4–9 are defined in terms of mean demographic statistics for a population, $\bar{l}_a$ and $\bar{w}_a$, and are the same as would be calculated from standard procedures ignoring individual variation.

Dynamics of life history evolution

A further assumption necessary to render the analysis tractable is that selective forces on a population are weak and change only slowly with time, so that life history evolution is slow in comparison with normal demographic processes. Except possibly for a few generations after its initial establishment, a population then closely tracks a slowly evolving "stable" age distribution, and the total population size N grows approximately at the exponential rate r,

$$dN/dt = rN. \tag{10}$$

Allowing for the maintenance of additive genetic variation by mutation and recombination under the

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joint action of weak directional selection and stabilizing selection (Lande 1975, 1980*a*), the genetic and phenotypic variance-covariance matrices G and P, as well as the age-specific fecundity and mortality rates for each phenotype, are assumed to remain nearly constant during intervals of at least a few generations. With these assumptions the rate of evolution of the mean life history phenotype in unselected (newly conceived) individuals is derived in the Appendix as approximately

$$d\bar{\mathbf{z}}/dt = G\nabla r \tag{11a}$$

where

$$\nabla r = (\partial r/\partial \bar{z}_1, \ldots, \partial r/\partial \bar{z}_n)^T.$$

∇r is the gradient vector of the intrinsic rate of increase of the population with respect to changes in the mean life history, and will be referred to as the selection gradient. The rate of evolution of the mean life history in unselected individuals equals approximately the additive genetic variance-covariance matrix of the characters times the selection gradient. The ith character thus evolves at the approximate rate

$$d\bar{z}_i/dt = \sum_{j=1}^{n} G_{ij}\partial r/\partial \bar{z}_j, \tag{11b}$$

where G_{ij} is the additive genetic covariance between characters i and j.

Although the phenotypic variance and covariance of age-specific characters can be partitioned into additive genetic and environmental (plus nonadditive genetic) parts using correlations among relatives or artificial selection experiments, there are no comparable techniques for estimating selective forces acting on characters of age-structured populations. For this purpose, two formulas for the selection gradient are presented here. Robertson (1966) and Price (1970, 1972) noted that the observed selection differential on a quantitative character (the difference in the mean phenotypes of selected and unselected individuals) is equal to the covariance of the character with relative fitness, and proportional to the slope of the linear regression of relative fitness on the character. This approach can be generalized to multiple characters, as suggested by S. J. Arnold (*personal communication*).

The Appendix shows that the selection gradient can be written as

$$\nabla r = P^{-1}\mathrm{Cov}[w(\mathbf{z}), \mathbf{z}] \tag{12a}$$

where the relative fitness per unit time of individuals with life history phenotype $\mathbf{z}$ is approximately

$$w(\mathbf{z}) = T^{-1}\int_0^\infty e^{-ra}w_a(\mathbf{z})\,da. \tag{12b}$$

From Eq. 4 it can be seen that $\bar{w} = T^{-1}$. Equivalent expressions for relative fitness of individuals in an age-structured population have been obtained by Haldane (1927), who assumed $r = 0$, and by Charlesworth (1974, 1980:158).

The right hand side of Eq. 12a is a vector of partial regression coefficients of relative fitness on the characters of individuals in a population (Kendall and Stuart 1973:Eq. 27.42). In other words, in Eq. 11b, $\partial r/\partial \bar{z}_j$ equals the slope of the linear regression of relative fitness on z_j, holding all other characters constant. Thus the selection gradient ∇r represents the actual forces of directional selection, while the observed selection differential $\mathrm{Cov}[w(\mathbf{z}), \mathbf{z}] = P\nabla r$ includes changes caused by phenotypic correlations among traits.

The Appendix also demonstrates that the relative fitness of life history phenotype $\mathbf{z}$ can be approximated as a weighted average of age-specific fecundity and mortality rates,

$$w(\mathbf{z}) = T^{-1}\int_0^\infty [q_a\bar{m}_a{}^{-1}m_a(\mathbf{z}) - b^{-1}c_a v_a \mu_a(\mathbf{z})]\,da. \tag{12c}$$

The relative fecundity rates $\bar{m}_a{}^{-1}m_a(\mathbf{z})$ and mortality rates $\mu_a(\mathbf{z})$ are weighted, respectively, by the present productivity and the future reproductive value of each age-class as a whole. These weighting coefficients give the sensitivities of the intrinsic rate of increase of a population to a small change in the age-specific fecundity or mortality rate of all phenotypes (using Eq. 4),

$$\partial r/\partial m_a = T^{-1}q_a \tag{13a}$$

$$\partial r/\partial \mu_a = -(bT)^{-1}c_a v_a. \tag{13b}$$

Reproductive value weighting of age-specific selective forces was alluded to by Fisher (1930, 1958:38), who seems to have assumed no fecundity selection. Equivalent expressions for the sensitivities (Eq. 13a, b) were first calculated for a monomorphic population by Hamilton (1966) in a heuristic study of selection on mortality rates. Hill (1974) derived reproductive-value weighting in a model of artificial selection on a single character, assuming no fecundity selection and $r = 0$.

An important conclusion apparent from Eqs. 12a, b, c is that changes in the age-specific fecundity and mortality rates which are independent of phenotype can nevertheless alter the relative fitnesses of individuals with different life-histories. Early reproduction is advantageous in expanding populations, while delayed reproduction is selected in declining populations (Mertz 1971, Charlesworth 1980:Chaps. 4, 5).

The mean absolute fitness per unit time, derived from Eq. 10, is $\bar{W} = e^r$. This permits the selection gradient in the dynamic Eq. 11a, b to be written as $\nabla r = \nabla \ln \bar{W}$, which is consistent with the discrete generation model of Lande (1979). The absolute fitnesses of individuals per unit time can then be written using Eq. 12b or 12c as $W(\mathbf{z}) = e^r T w(\mathbf{z})$.

The dynamic Eq. 11a, b implies a fundamental principle of adaptation for phenotypic characters in age-

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structured populations. If the phenotype- and age-specific fecundity and mortality rates remain constant in time, the intrinsic rate of increase of a population evolves at the rate

$$\begin{aligned} dr/dt &= (\nabla r)^T d\bar{\mathbf{z}}/dt \\ &= (\nabla r)^T \mathbf{G} \nabla r \geq 0 \end{aligned} \quad (14)$$

which is always non-negative since G is a variance-covariance matrix. Thus in a constant selective regime, life history evolution continually increases the intrinsic rate of increase of a population, until an equilibrium is reached. A stable equilibrium of the mean life history occurs at a local maximum of r, provided the additive genetic variance-covariance matrix is not singular. A stable equilibrium can also exist when selection acts to change a combination of characters for which there is no additive genetic variance. The intrinsic rate of increase of a population, r, can therefore be viewed as an adaptive topography for life history characters, similar to Wright's (1932, 1969) adaptive topography for gene frequencies. The population is represented as a point on a multidimensional landscape with height r and other dimensions $\bar{z}_1, \ldots, \bar{z}_n$. For a population at any position on the adaptive landscape, the selection gradient ∇r is a vector of directional selection pressures pointing in the steepest uphill direction. The population responds by moving uphill, but with rate and direction modified by G, the additive genetic variance-covariance matrix of the characters. This model gives quantitative expression to an intuitive concept of an adaptive topography for phenotypic evolution that has been widely used by evolutionary biologists (e.g., Dobzhansky 1951, 1971:Chap. 1, Simpson 1953). It is important to recall, however, that various factors such as changing environments, frequency-dependent selection, and random genetic drift in small populations can decrease the level of adaptation of a population (Wright 1932, 1949, 1969, 1977:Chap. 13). Fisher (1930, 1958:Chap. 2) concluded that natural selection in a constant environment increases the growth rate of a population, but that changes in physical and biotic factors, including increased population density, tend to produce a deterioration of the environment that results in decreased population growth rate, so that r usually fluctuates around zero.

While the joint evolution of all life history characters under constant phenotype- and age-specific fecundity and mortality rates increases the intrinsic rate of increase of a population, no such principle applies to any subset of characters that are genetically correlated with other selected traits. The rate of evolution of a particular trait is a sum of the response to direct selection on that trait and the correlated responses to selection on genetically correlated traits (Eq. 11b). A character may evolve in opposition to the actual force of selection on it due to antagonistic selection on genetically correlated characters. The adaptive response to selection on the entire life history may thus involve compromises or trade-offs between characters that are genetically correlated.

Discussion

A constraint of special importance in life history evolution is encountered when natural selection acts to increase simultaneously a set of characters with negative genetic correlations. Then any progress in one character results in correlated decreases in the others, which if sufficiently severe can prevent any net increase in adaptation. This situation may often arise for characters that are major components of fitness, as illustrated by artificial selection experiments. If population size is not too small to maintain substantial genetic variability, strong directional selection on a character (such as fitness itself) tends rapidly to fix pleiotropic or linked combinations of genes with predominantly positive effects on the components of the selected character, and to leave segregating those genes with opposing effects on the component traits. This decreases the additive genetic correlation among the component traits, and depletes the additive genetic variance in the selected character without necessarily exhausting that in any of its components (Dickerson 1955, Falconer 1960:Chap. 19). To give several examples, Dickerson (1955) observed that in a flock of chickens at a selection limit for a combination of characters including egg size and egg number, these two traits had a large negative genetic correlation and a phenotypic correlation near zero, indicating a positive environmental correlation. In a wild-derived population of *Drosophila* flies, Rose and Charlesworth (1981*a*, *b*) found large negative genetic correlations of early fecundity with both late fecundity and longevity in females, with phenotypic correlations near zero or positive. Within populations of ryegrass that could not be selected for increased yield, Cooper and Edwards (1961) detected large negative genetic correlations and positive environmental correlations between leaf size and rate of leaf production, and between tiller number and dry mass per tiller. Adams (1967) demonstrated that three components of crop yield in navy beans: pods per plant, seeds per pod, and seed size, had phenotypic correlations near zero or slightly positive in low density plantings but negative in high density plantings. In an extensive study of the genus *Plantago*, Primak (1978) found that within populations phenotypic correlations among four components of seed yield were near zero or positive, but that the same correlations among species included some large negative values.

These papers, and others cited in them, support the contention that in large populations near an evolutionary equilibrium, major components of fitness tend to have negative genetic correlations, with the corresponding environmental correlations tending to be positive in good conditions and negative in harsh or

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limiting conditions. Thus the genetic correlations between major components of fitness tend to be more negative than the phenotypic correlations. The evolutionary constraints on major components of fitness imposed by genetic correlations therefore may often not be accurately reflected in the phenotypic correlations within populations. It is worth noting, however, that a population with no phenotypic variance for a certain combination of characters must also lack both additive genetic and environmental variance for that combination.

Morphological, physiological, and behavioral characters of natural populations are often under stabilizing selection toward a joint intermediate optimum; the genetic variance and covariance of such characters within populations usually are largely additive, and resemble the environmental variance-covariance pattern (Bailey 1956, Falconer 1960:Chaps. 8, 19, 20, Hashiguchi and Morishima 1969, Hegmann and DeFries 1970, Leamy 1977). In contrast, stabilizing selection on quantitative characters creates substantial genotype-environment interaction and nonadditive genetic variance in fitness itself. As the primary phenotypic characters evolve toward a joint optimum the additive genetic correlations between major components of fitness are expected to decrease as described above, depleting the additive genetic variance in fitness and increasing the proportion of nonadditive genetic variance in fitness (Wright 1935, Falconer 1960:Chaps. 8, 10, 20, Leng 1963).

For characters other than fitness or its major components, the assumption that the additive genetic and phenotypic variance-covariance matrices remain constant during the evolution of the mean phenotypes often yields a good approximation for several generations even after the onset of intense artificial selection (Falconer 1960, Bell and Burris 1973, Cheung and Parker 1974). In natural populations where genetic variation is maintained by a continual flux of pleiotropic mutations, and where the pattern of stabilizing selection around a (moving) optimum phenotype may be very conservative, the additive genetic and phenotypic variance-covariance matrices may remain nearly constant for long periods of time (Lande 1980*a*). The temporal and taxonomic limits within which this assumption is valid can be ascertained empirically for a given set of characters by comparing genetic and phenotypic variation patterns in related populations. For instance, Lande (1979) inferred substantial constancy of genetic variation parameters of adult brain and body masses in species of insectivores and rodents, but inferred rather different values in primate species, and Arnold (1981) estimated similar patterns of genetic variation in feeding preferences in divergent races of garter snakes.

The dynamic equations for life history evolution can be combined with information on the additive genetic variances and covariances among characters to analyze the cumulative selective forces during an interval of time in which G remains approximately constant. Although the selective forces may be slowly fluctuating, the net selection gradient required to produce an observed change in the mean life history of a population can then be calculated by summing Eq. 11a over the appropriate span of time and inverting G, after eliminating linear combinations of characters for which there is no additive genetic variance so that G is not singular,

$$\int_0^t \nabla r \, dt' = G^{-1}[\bar{z}(t) - \bar{z}(0)]. \tag{15}$$

This method can also be extended to deduce the direction of past selective forces which acted to differentiate contemporary populations descended from a common ancestral population (Lande 1979). The use of this formula requires some assurance that the phenotypic changes being analyzed result from genetic evolution and are not caused by immediate effects of different environments. In the absence of genotype-environment interaction, immediate effects of the environment can be accounted for by raising samples from the populations under common conditions. For species with determinate growth, in which individuals achieve a final adult size and shape, evolution of the adult phenotype can be analyzed as an autonomous system independent of preadult characters if the former are genetically uncorrelated with the latter, or if selection on preadult characters is negligible, as indicated by Eqs. 11a, b and 15.

Differences in the phenotypic (or genetic) variance-covariance structures of homologous sets of characters can often be interpreted as resulting from different patterns of multivariate selection operating on similar genetic systems. Additive genetic variance and covariance of quantitative characters may change rapidly following a sudden alteration of the multivariate pattern of stabilizing and/or disruptive selection (Falconer 1960, Lande 1980*a*). The environmental portion of phenotypic variances and covariances between characters is also subject to genetic modification, and evolves in response to changing patterns of selection (Schmalhausen 1949, Waddington 1960, Prout 1962, Scharloo 1964). Thus Berg (1960) found that the dimensions of flower parts are more highly correlated phenotypically in plant species with specialized insect pollinators than in selfing or wind-pollinated species. Wu (1981) showed that emigration rate is genetically correlated with egg size, body size, and fecundity in *Tribolium castaneum* (which disperses by flight under some conditions) but not in *T. confusum* (which has never been observed to fly). Several studies of the mammalian skeleton have demonstrated that high phenotypic and genetic correlations tend to occur between characters that are developmentally and/or functionally related (Kurtén 1953, Bailey 1956, Olson and Miller 1958, Leamy 1977, Cheverud 1982).

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APPENDIX

The distribution of life history characters in unselected individuals is assumed to be Gaussian with mean $\bar{\mathbf{z}}$ and variance-covariance matrix P,

$$p(\mathbf{z}) = \sqrt{(2\pi)^{-n}\left|\mathrm{P}\right|^{-1}}\, e^{\frac{1}{2}(\mathbf{z}-\bar{\mathbf{z}})^{\mathrm{T}}\mathrm{P}^{-1}(\mathbf{z}-\bar{\mathbf{z}})}. \tag{A1}$$

The distributions of additive genetic and environmental effects $g(\mathbf{x})$ and $\xi(\mathbf{e})$ are also assumed to be Gaussian with the respective variance-covariance matrices G and E summing to P. For a constant distribution of environmental effects, let $\bar{\mathbf{e}} = \mathbf{0}$ so that $\bar{\mathbf{z}} = \bar{\mathbf{x}}$. In a population under weak selection and near a slowly evolving stable age distribution, the matrices G and P and the age-specific forces of selection are assumed to remain nearly constant for periods of at least a few generations. The mean life history phenotype expressed by unselected individuals is taken to be that of new conceptions (aged 0), $\bar{\mathbf{z}} = \bar{\mathbf{z}}_0 = \bar{\mathbf{x}}_0$, which can be approximated as the average breeding value of parents reproducing at time t,

$$\bar{\mathbf{z}}(t) = \int_0^\infty q_a \bar{\mathbf{x}}_a(t)\, \mathrm{d}a. \tag{A2}$$

$\bar{\mathbf{x}}_a(t)$ is the mean breeding value of individuals aged a at time t, which is approximately equal to the mean life history of unselected individuals conceived at time $t - a$, $\bar{\mathbf{z}}(t-a)$, plus the cumulative genetic selection differential due to viability selection from conception to age a and fertility selection at that age, $\boldsymbol{\delta}_a$,

$$\bar{\mathbf{x}}_a(t) \simeq \bar{\mathbf{z}}(t-a) + \boldsymbol{\delta}_a. \tag{A3}$$

With weak selective forces and rates of evolution nearly constant during the reproductive lifespan of most individuals,

$$\bar{\mathbf{z}}(t-a) \simeq \bar{\mathbf{z}}(t) - a\mathrm{d}\bar{\mathbf{z}}/\mathrm{d}t. \tag{A4}$$

Substitution of Eqs. A3 and A4 into Eq. A2, using Eqs. 7 and 8, gives approximately

$$\mathrm{d}\bar{\mathbf{z}}/\mathrm{d}t = T^{-1}\int_0^\infty q_a \boldsymbol{\delta}_a\, \mathrm{d}a. \tag{A5}$$

Thus the rate of evolution of the mean life history phenotype in unselected individuals is approximately equal to the average genetic selection differential of reproducing individuals. Haldane (1927) used an approximation similar to Eq. A4 to obtain a simplified dynamic equation for gene-frequen-

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cy evolution in an age-structured population under weak selection (Charlesworth 1980:Chap. 4). Expressions of the form of Eq. A5 have previously been derived under similar assumptions for a single character that does not change with age (Dickerson and Hazel 1944, Rendel and Robertson 1950, Hill 1974).

The genetic selection differential at age a resulting from mortality selection from conception to age a, and fecundity selection at that age, can be derived following the method of Lande (1979). The phenotype distribution (Eq. A1) can also be written as the convolution of the additive genetic and environmental distributions,

$$p(\mathbf{z}) = \int_{-\infty}^{+\infty} g(\mathbf{x})\xi(\mathbf{z} - \mathbf{x})\, d\mathbf{x} \tag{A6}$$

where

$$\int_{-\infty}^{+\infty} d\mathbf{x} \equiv \int_{-\infty}^{+\infty} \cdots \int_{-\infty}^{+\infty} dx_1 \cdots dx_n.$$

From Eqs. 3 and A6 the mean fitness at age a can be written as an average over either phenotypes or breeding values,

$$\bar{w}_a = \int_{-\infty}^{+\infty} p(\mathbf{z})w_a(\mathbf{z})\, d\mathbf{z} = \int_{-\infty}^{+\infty} g(\mathbf{x})\bar{w}_a(\mathbf{x})\, d\mathbf{x} \tag{A7}$$

where

$$\bar{w}_a(\mathbf{x}) = \int_{-\infty}^{+\infty} \xi(\mathbf{z} - \mathbf{x})w_a(\mathbf{z})\, d\mathbf{z}$$

equals the mean fitness at age a of individuals with breeding value $\mathbf{x}$. The phenotypic selection differential at age a is the difference in mean life history phenotype between unselected individuals and those undergoing mortality selection from conception to age a, and fertility selection at that age,

$$\mathbf{s}_a = \bar{w}_a^{-1} \int_{-\infty}^{+\infty} (\mathbf{z} - \bar{\mathbf{z}})p(\mathbf{z})w_a(\mathbf{z})\, d\mathbf{z}. \tag{A8}$$

The genetic selection differential at age a is the difference in mean breeding values of selected and unselected individuals at that age,

$$\boldsymbol{\delta}_a = \bar{w}_a^{-1} \int_{-\infty}^{+\infty} (\mathbf{x} - \bar{\mathbf{x}})g(\mathbf{x})\bar{w}_a(\mathbf{x})\, d\mathbf{x}. \tag{A9}$$

Applying to Eq. A7 the gradient operator ∇ defined in Eq. 11a, recalling $\bar{\mathbf{z}} = \bar{\mathbf{x}}$ and assuming $w_a(\mathbf{z})$ is constant in time, yields

$$\bar{w}_a^{-1}\nabla\bar{w}_a = \mathbf{P}^{-1}\mathbf{s}_a = \mathbf{G}^{-1}\boldsymbol{\delta}_a. \tag{A10}$$

Substituting Eq. A10 into Eq. A5 and using Eq. 7 gives

$$d\bar{\mathbf{z}}/dt = \mathbf{G}\mathbf{P}^{-1}T^{-1} \int_0^{\infty} q_a\mathbf{s}_a\, da = \mathbf{G}T^{-1} \int_0^{\infty} e^{-ra}\nabla\bar{w}_a\, da. \tag{A11}$$

But the gradient operator applied to Eq. 4 shows that

$$\int_0^{\infty} e^{-ra}\nabla\bar{w}_a\, da = T\nabla r. \tag{A12}$$

Eqs. A11 and A12 reveal the simple dynamic law in Eq. 11a.

To derive expressions for the selection gradient ∇r in terms of individual fitnesses, note that Eqs. A11 and A12 also establish that

$$\nabla r = \mathbf{P}^{-1}T^{-1} \int_0^{\infty} q_a\mathbf{s}_a\, da. \tag{A13}$$

Substituting Eqs. 7 and 8 and changing the order of integration produces

$$\begin{aligned}\nabla r &= \mathbf{P}^{-1} \int_{-\infty}^{+\infty} (\mathbf{z} - \bar{\mathbf{z}})p(\mathbf{z})w(\mathbf{z})\, d\mathbf{z} \\ &= \mathbf{P}^{-1}\mathrm{Cov}[w(\mathbf{z}), \mathbf{z}]\end{aligned} \tag{A14}$$

where $w(\mathbf{z})$ is the relative fitness of individuals with life history phenotype $\mathbf{z}$, defined in Eq. 12b.

Alternatively, $w(\mathbf{z})$ can be written in terms of the phenotype- and age-specific fecundity and mortality rates, starting from Eqs. A13 and A8. The mean life history phenotype of individuals selected according to viability to age a is denoted as

$$\bar{\mathbf{z}}^*_a = \int_{-\infty}^{+\infty} \mathbf{z}p^*_a(\mathbf{z})\, d\mathbf{z} \tag{A15}$$

in which

$$p^*_a(\mathbf{z}) = p(\mathbf{z})l_a(\mathbf{z}) / \int_{-\infty}^{+\infty} p(\mathbf{z})l_a(\mathbf{z})\, d\mathbf{z}.$$

Separating viability and fecundity selection by subtracting and adding $\bar{\mathbf{z}}^*_a$ in Eq. A8 and rearranging gives

$$\begin{aligned}\mathbf{s}_a = \bar{m}_a^{-1} &\int_{-\infty}^{+\infty} (\mathbf{z} - \bar{\mathbf{z}}^*_a)p^*_a(\mathbf{z})m_a(\mathbf{z})\, d\mathbf{z} \\ &- (\bar{\mathbf{z}}^*_a - \bar{\mathbf{z}})\end{aligned} \tag{A16}$$

where

$$\bar{m}_a = \int_{-\infty}^{+\infty} p^*_a(\mathbf{z})m_a(\mathbf{z})\, d\mathbf{z}.$$

$\bar{m}_a$ is the mean fecundity among survivors to age a. Similarly, the last two terms in Eq. A16 can be expanded by interposing an infinite series of stages for mortality selection at each age from conception to a, substituting Eqs. A15 and 2, and differentiating,

$$\begin{aligned}\bar{\mathbf{z}}^*_a - \bar{\mathbf{z}} &= \int_0^a \frac{d}{dy}[\bar{\mathbf{z}}^*_y]\, dy \\ &= -\int_0^a \int_{-\infty}^{+\infty} (\mathbf{z} - \bar{\mathbf{z}}^*_y)p^*_y(\mathbf{z})\mu_y(\mathbf{z})\, d\mathbf{z}\, dy.\end{aligned} \tag{A17}$$

Employing Eqs. A16 and A17 to replace $\mathbf{s}_a$ in Eq. A13 and changing the order of integration, again produces Eq. A14 but with

$$w(\mathbf{z}) = T^{-1} \int_0^{\infty} q_a[\bar{m}_a^{-1}m_a(\mathbf{z}) - \int_0^a \mu_y(\mathbf{z})\, dy]\, da. \tag{A18}$$

Reversing the order of integration over the triangular region in the last term of Eq. A18, and interchanging the symbols y and a,

$$\int_0^{\infty} q_a \int_0^a \mu_y(\mathbf{z})\, dy\, da = \int_0^{\infty} \mu_a(\mathbf{z}) \int_a^{\infty} q_y\, dy\, da,$$

finally yields the aproximation given in Eq. 12c.

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