Evolution, 45(4), 1991, pp. 853–861

ON THE LOW HERITABILITY OF LIFE-HISTORY TRAITS

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Abstract.—Life-history traits such as longevity and fecundity often show low heritability. This is usually interpreted in terms of Fisher's fundamental theorem to mean that populations are near evolutionary equilibrium and genetic variance in total fitness is low. We develop the causal relationship between metric traits and life-history traits to show that a life-history trait is expected to have a low heritability whether or not the population is at equilibrium. This is because it is subject to all the environmental variation in the metric traits that affect it plus additional environmental variation. There is no simple prediction regarding levels of additive genetic variance in life-history traits, which may be high at equilibrium. Several other patterns in the inheritance of life-history traits are readily predicted from the causal model. These include the strength of genetic correlations between life-history traits, levels of nonadditive genetic variance, and the inevitability of genotype-environment interaction.

Key words.—Components of fitness, genetic correlation, genotype-by-environment interaction, heritability, life-history traits, path analysis.

Received October 4, 1989. Accepted December 4, 1990.

Life-history traits—such as longevity and fecundity—tend to have lower heritabilities than morphological, physiological, and behavioral traits (metric traits; Gustafsson, 1986; Charlesworth, 1987; Mousseau and Roff, 1987; Roff and Mousseau, 1987; Hartl and Clark, 1989 Ch. 8; Falconer, 1989 Ch. 10). This finding has been interpreted widely in terms of Fisher's fundamental theorem, which predicts that in a population at evolutionary equilibrium no additive genetic variance in total fitness is present (Fisher, 1930; Wright, 1930; Price, 1972; Charlesworth, 1987). Because life-history traits are more closely connected to fitness than metric traits, the reasoning has been that they should have less additive genetic variance and therefore lower heritability. The empirical results have consequently been viewed as support for the theorem and the proposition that populations are near equilibrium.

In this paper we show that low heritabilities in life-history traits are theoretically expected to result from high levels of environmental variance, whether or not the population is at equilibrium. Our conclusions stem from the observation that variation in metric traits underlies variation in life-history traits (Arnold, 1983; Crespi and Bookstein, 1989; Falconer, 1989 Ch. 20). For example, survival of a Darwin's finch is influenced by its body size (Boag and Grant, 1981; Price et al., 1984), lifespan of a female *Drosophila* is affected by her fat and glycogen content (Service, 1987; Luckinbill et al., 1989) and fecundity of a neotropical herb is partly determined by its corolla length (Schemske and Horvitz, 1989). Consequently, the genetic and environmental components that make up the underlying traits also indirectly make up the life-history traits. Any additional environmental variation affecting a life-history trait enlarges only its total environmental component, and reduces its heritability. By contrast, the traditional explanation for the low heritability of life-history traits based on low additive genetic variance does not appear to have theoretical justification.

Before developing the theory we illustrate the reasoning with an explicit example, taken from studies on a population of Darwin's Medium Ground finches living on a small island in the Galápagos (Boag and Grant, 1981; Price et al., 1984; Schluter et al., 1985). In this population larger adults often live longer than smaller individuals because they

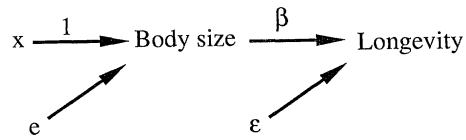

FIG. 1. Causal relationship between a single metric trait, body size, and a single life-history trait, longevity. Arrows connect dependent (arrowhead) with independent variables. x refers to the additive genetic component of the metric trait and e to the environmental component. The life-history trait is subject to additional environmental noise (ϵ) that does not influence the metric trait, and consequently the life-history trait has lower heritability. Numbers or letters along arrows are the regression coefficients.

more efficiently feed on the large seeds that at times predominate on the island. Although the relationship between body size and adult longevity is highly significant, body size explains only a small proportion of the variability in survival (Price et al., 1984) and there are many causes of death not directly related to size. For example, finches are eaten by egrets and owls. They suffer from accidents such as getting stuck between *Opuntia* cactus pads, getting caught in the sticky exudates of *Boerhavia erecta,* getting the husk of an *Opuntia* seed caught round the beak, and being used as a plaything by a young Blue-footed booby (pers. obs.). Many of the reasons a finch dies are clearly not heritable (i.e., parents and offspring die from dissimilar causes), and the only identified heritable character affecting probability of mortality is body size. Thus genetic and environmental differences among individuals in adult longevity are attributable to genetic and environmental differences in body size, but additional environmental noise affects longevity that does not affect body size. Hence, longevity must have lower heritability than body size (Fig. 1). Data are not yet available to directly test this proposition. Other heritable characters beside body size presumably also affect survival, but the basic conclusion that additional environmental variation affects life-history traits that does not affect metric traits remains unaltered.

In the following sections we show how the heritability of a life-history trait can be computed from the inheritance of the underlying metric variables and additional random factors. We use the results to consider the relationships among metric traits, life-history traits, and total fitness in levels of environmental and additive genetic variance. There are no simple predictions about levels of genetic variance in life-history traits even when the population is at equilibrium. Finally, we show how the causal relationship predicts other genetic properties of life-history traits, including levels of nonadditive genetic variance, genetic correlations between life-history traits, and the ubiquity of genotype-environment interaction.

THEORY

Heritability of a Life-History Trait.—Let L be the value of the life-history trait and $\mathbf{z}$ be a vector whose elements $z_1, z_2, \ldots z_n$ are the phenotypic values of n underlying metric traits. We assume that L is causally and linearly related to $\mathbf{z}$ as

$$L = \alpha + \beta^{\mathrm{T}}\mathbf{z} + \epsilon, \qquad (1)$$

where α is a constant, ϵ is a random error term, and T denotes transpose. The relationships are diagrammed in Figure 2. The vector β is the directional selection gradient with respect to the life-history trait (Lande and Arnold, 1983), and its elements are the partial regression coefficients of L on each metric trait. β is commonly used to indicate the strength of directional selection acting directly on metric traits over episodes of the life history (Price et al., 1984; Kalisz, 1986; Schluter and Smith, 1986). In reality, the relationship between metric traits and a life-history trait may be partly nonlinear (Lande and Arnold, 1983; Schluter, 1988), in which case equation (1) should include higher order terms (such as z_1^2, z_1z_2, etc., with their corresponding partial regression coefficients). We assume for now that nonlinear terms are absent from the model, and discuss the consequences of including them in a later section.

Equation (1) can also be used to model total fitness (Charnov, 1989), in which case β represents selection over the whole life history (Lande, 1979). However, our goal is to model the genetic properties of fitness components (life-history traits) and we will show that these properties are largely dissociated from those of total fitness. Thus, to avoid confusion we use β to represent selection only in association with individual

fitness components (e.g., longevity and fecundity).

We assume that each metric trait is itself determined as the sum of two independent components:

$$\mathbf{z} = \mathbf{x} + \mathbf{e}. \quad (2)$$

The elements of the vector **x** are the additive genetic values for the n metric traits and the elements of **e** are corresponding environmental values, including nonadditive genetic effects. Genotype-environment interactions or correlations involving the metric traits are assumed to be absent.

We define the heritability of the life-history trait as the ratio of the variance in the additive genetic values to the total phenotypic variance (see Charlesworth, 1987). Using (1) and (2) the heritability of the life-history trait is derived from the underlying variables to yield

$$h_L^2 = \frac{\beta^T \mathbf{G} \beta}{\beta^T \mathbf{P} \beta + \sigma_\epsilon^2}. \quad (3)$$

G and **P** are, respectively, the additive genetic covariance matrix and the phenotypic covariance matrix for the n metric traits, and σ_ϵ^2 is the additional random error variance. The numerator in equation (3) is a weighted sum of the genetic variances and covariances in the metric traits, where the weightings are measurements of intensities of selection on the traits. Similarly, the first term in the denominator is a weighted sum of the phenotypic variances and covariances of the metric traits. It can be shown that in the absence of the other extraneous influences on the life history trait (i.e., $\sigma_\epsilon^2 = 0$), h_L^2 may exceed or fall below the mean heritabilities of the n metric traits. It may be larger, for example, if the metric traits with the strongest effects on the life history have relatively low heritability, or if there are negative environmental covariances among the underlying metric traits. Thus, the strict result from the case with one metric trait (Fig. 1), that the heritability of the life-history trait is always less than or equal to the heritability of the underlying metric trait, does not hold when many metric traits are influencing the life-history trait. Nevertheless, additional environmental variation is inevitable ($\sigma_\epsilon^2 > 0$) and its effect is exactly as before: to inflate the overall environmental component of the life-history trait without altering its genetic component (eq. 3). The magnitude of this extra environmental variance is unknown. It may often be large in nature because many factors unrelated to an individual's phenotype influence fitness components.

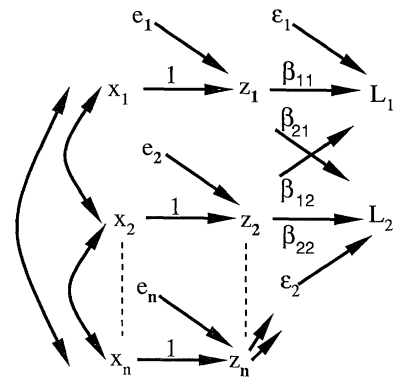

FIG. 2. Causal relationships between n metric traits and two life-history traits L_1 and L_2. Single headed arrows connect dependent with independent variables. x_i, e_i, and z_i refer respectively to the additive genetic, environmental, and phenotypic values of metric trait i. ϵ_1 and ϵ_2 are the additional environmental components of variation in the two life-history traits. Double headed arrows indicate possible genetic correlations between metric traits. For clarity environmental correlations are omitted.

Genetic Variation in Life-History Traits.— Our conclusions regarding relative levels of environmental variance in metric and life-history traits are applicable whether or not the population is at evolutionary equilibrium. The alternative explanation for low heritability of life-history traits is an explanation based on the additive genetic component, and assumes equilibrium. Here we show that, in contrast to levels of environmental variance, there is no simple prediction about expected levels of genetic variance. Indeed, previous theoretical work has shown that high levels of additive genetic variance in life-history traits can be maintained at equilibrium (Lande, 1982; Rose, 1982). Here we explore the levels of additive genetic variance expected for a life-history trait in equilibrium and nonequilibrium situations. To do this we return to the simplified case in which life-history variation depends on a single metric trait, such as body size (Fig. 1).

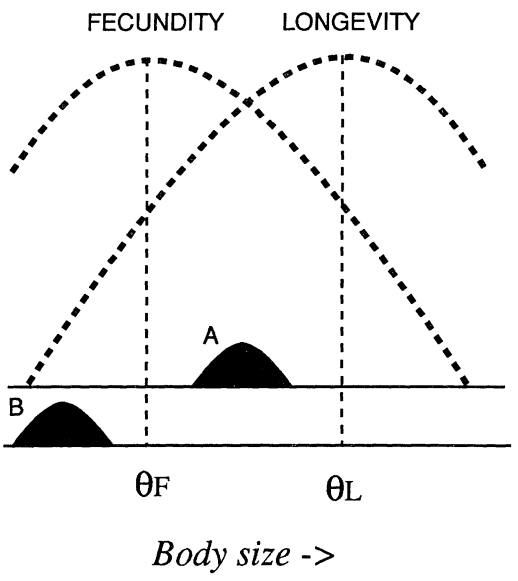

FIG. 3. Fecundity and longevity as functions of body size, with the respective optima, θ_L and θ_F, indicated. Solid curves indicate phenotypic distributions for body size in two populations (A and B). Body size is assumed to be heritable. Case A: the population is at an evolutionary equilibrium and the additive genetic variance in fitness is near zero. Case B: the population is in a nonequilibrium state and additive genetic variance in fitness is present.

FIG. 4. A numerical example based on Figure 3, showing how the heritabilities of fecundity and total fitness change as a function of body size. The quadratic fitness functions of Figure 3 are approximated by Gaussian curves (e.g., see Lande, 1981) with width, $\omega^2 = 100$. The small quantity of genetic variance in the life-history traits, which arises due to curvature in the fitness functions (see text), is ignored. Fecundity selection is assumed to precede viability selection. Calculated using formulae in Lande (1981) and equation (3) of this paper. Parameters: $\theta_F = 20$, $\theta_L = 40$, $\sigma_z^2 = 10$, $h_z^2 = 0.5$. The extra environmental variance (σ_ϵ^2) in association with fecundity is 5, and in association with total fitness is 10.

We assume that body size affects two life-history traits, for example, fecundity and longevity, and that fecundity is maximized at a different body size than longevity (Fig. 3). This appears to be the case in the Darwin's finch example discussed earlier. Although larger females had higher survival over the nonbreeding season, smaller females came into breeding condition at an earlier age (Price, 1984). For simplicity, we also assume that all variation in fitness is accounted for by variation in fecundity and in longevity. Equilibrium and nonequilibrium conditions are contrasted in Figure 3. In the first case (A), the survival advantage of a larger size is counterbalanced by the fecundity advantage of a smaller size, and mean size in the population is stabilized at an intermediate value. In case B, all individuals are below both the fecundity and longevity optima, and the population is in the process of evolving to a larger size.

Additive genetic variance in a life-history trait is given by the numerator in equation (3). In the case of a single metric trait (body size) affecting a life-history trait (e.g., longevity), the additive genetic variance of the life-history trait (g_L^2) is obtained from the numerator of equation (3) as $g_L^2 = \beta^2 g_z^2$, where g_z^2 is the additive genetic variance of the metric trait. Additive genetic variance in a life-history trait does not simply decline as a population evolves toward equilibrium. For example, as body size evolves from a small mean value (case B in Fig. 3) to the equilibrium size (case A), the slope of the function relating fecundity to body size in the population changes from positive to zero to negative. Correspondingly, the additive genetic variance (and the heritability) of fecundity first declines and then rises again as the population nears equilibrium (Fig. 4).

Levels of additive genetic variance in life-history traits will often be a poor reflection of the amount of additive genetic variation in total fitness. Total fitness would have low levels of additive genetic variance at equilibrium (Fig. 4) but additive genetic variance in the life-history traits may neverthe-

less be substantial [see Lande (1982) and Rose (1982) for further discussion]. Conversely, a population occurring far away from equilibrium with its mean value for the metric trait close to the value that maximizes fecundity would exhibit positive amounts of additive genetic variance in total fitness, but only low amounts of additive genetic variance in fecundity (Fig. 4). Hence, the notion that genetic variance in life-history traits at equilibrium should be low is not predicted by theory.

How should levels of additive genetic variance in life-history traits compare with those of metric traits? It is difficult to directly compare the two because they are measured in different units. Transformations such as the use of logarithms or coefficients of additive genetic variation have been suggested (Charlesworth, 1987), but there is no obvious reason why these measures appropriately correct for scale (Bryant, 1986). However, if such transformations are used it is easy to show that the additive genetic variance of the life-history trait can be higher (or lower) than that of the underlying metric traits. For the example of Figure 3, additive genetic variance of fecundity exceeds that of body size whenever the slope of the fecundity function (β) is steep across the range of sizes spanned by the population (specifically, whenever $\beta^2 > 1$) and this may occur even at equilibrium. The same principle can be demonstrated using coefficients of additive genetic variance.

Non-linear Selection.—So far we have assumed that selection on the metric characters in association with a particular life-history trait is linear across the range of phenotypes in the population. Empirical studies have often found directional selection, but this is generally accompanied by some curvature in the fitness function (Schluter, 1988). The curvature can be accommodated by including higher order terms in the regression equation (1). For example, the complete regression model for the relationship of a single character subject to quadratic selection over a life-history episode, such as that depicted in Figure 3, is

$$L = \alpha + \beta(z - \bar{z}) + \frac{\gamma}{2}(z - \bar{z})^2 + \epsilon \quad (4)$$

(Lande and Arnold, 1983). From equations (3) and (4) it can be seen that the genetic variation in L depends on genetic variation in both the linear and quadratic terms, and on the regression coefficients of directional selection, β and quadratic selection, γ.

The amount of genetic variance expected in $(z - \bar{z})^2$ has been determined for a variety of explicit genetic models for z (Wright, 1935*a*; Tachida and Cockerham, 1988). Even if all the genetic variance in z is additive, substantial nonadditive genetic variance (epistatic and dominance variance) is expected in $(z - \bar{z})^2$ (Wright, 1935*a*; Tachida and Cockerham, 1988). Hence, nonlinear selection on metric traits gives rise to nonadditive genetic variance in life-history traits. Tachida and Cockerham (1988) show that the amounts of nonadditive variance depend on the details of the genetic model used.

The proportion of nonadditive genetic variance in life-history traits is often found to be small (Charlesworth, 1987; Tachida and Cockerham, 1988) although there are some exceptions (Falconer, 1989 Ch. 8). This suggests that the relationship between metric traits and life-history traits is often approximately linear [an alternative explanation based on nonlinear selection and a particular model for the determination of the underlying traits has been discussed by Tachida and Cockerham (1988)]. The effect of cubic and higher order terms on levels of additive and nonadditive genetic variance is unknown and needs to be investigated.

Genetic Correlations between Life-History Traits.—Our emphasis has been on levels of environmental and genetic variance in life-history traits, but at least two other results follow from the causal model (Figs. 1, 2). The first is that the additive genetic correlation between any two life-history traits is derived from the genetics of the metric traits that jointly affect them, and from the selection intensities (Fig. 2). From equations (1) and (2) we calculate the genetic correlation (r_L) as:

$$r_L = \frac{\beta_1^{\mathrm{T}}\,\mathbf{G}\beta_2}{\sqrt{\beta_1^{\mathrm{T}}\mathbf{G}\beta_1\beta_2^{\mathrm{T}}\mathbf{G}\beta_2}} \quad (5)$$

(see also Schluter and Smith, 1986). β_1 and β_2 are the selection gradients associated with the first and second of the two life-history

traits. That is, when calculating the genetic correlation between longevity and fecundity the selection gradients would be obtained from separate multiple regressions of longevity and fecundity on the complete suite of metric characters (Fig. 2). The derivation assumes that survival and reproduction are simultaneous, whereas in reality the life-history episodes are separate to some extent, such that the genetic correlation itself is changing within each generation. An estimate of the genetic correlation before any selection in the generation may be best obtained by weighting the second selection gradient to account for the changing covariances and variances. The appropriate weighting procedure has been developed by Wade and Kalisz (1989).

Many authors have discussed the theoretical significance of negative genetic correlations between life-history traits (Lande, 1982; Rose, 1982; Charlesworth, 1990). Equation (5) shows that negative genetic correlations between life-history traits arise in two ways. First, they can result from negative genetic correlations between the underlying metric traits, even if all the selection coefficients are positive. In general, this would represent a nonequilibrium situation unless the negative genetic correlations between the metric traits are sufficiently strong (Charnov, 1989; Charlesworth, 1990).

Second, negative genetic correlations between life-history traits may be a consequence of opposing selection pressures on underlying metric traits. This can be illustrated by the simplified example of Figure 3, in which a single metric trait determines variation in the life-history traits. Consider the situation where mean body size in the population falls between the two optima, as it would at equilibrium (case A). Selection favors decreased body size in association with fecundity and increased body size in association with longevity. The selection gradients for fecundity and longevity are of opposite sign, yielding a genetic correlation of -1.0 between fecundity and longevity. The negative correlation results because the evolution of increased longevity requires an increase in body size, and fecundity would consequently decline. A population with body size falling outside the range of both optima (e.g., case B) would experience a genetic correlation of $+1.0$. In reality correlations will often be between -1.0 and $+1.0$ because more than one metric trait influences the life-history traits.

Note that if we consider a suite of life-history traits, then some pairs can be positively genetically correlated even at equilibrium (Charlesworth, 1990). For example, imagine a revised version of Figure 3 in which the fecundity curve is replaced by two curves nearly superimposed, one for fecundity early in life and another for fecundity later in life. At equilibrium the metric trait will lie above the optima for the two fecundity curves, and early and late fecundity will be positively genetically correlated.

Genotype-Environment Interaction. — One other feature of life-history inheritance is readily predicted from the causal model. In nature selection pressures vary spatially and temporally (Endler, 1986; Kalisz, 1986; Schluter and Smith, 1986; Gibbs and Grant, 1987; Schemske and Horvitz, 1989) and it follows from equations (3) and (5) that the heritabilities, additive genetic variances, and genetic correlations of life-history traits will also fluctuate (i.e., life-history traits will exhibit genotype-environment interaction). This will be so even when the genetic parameters for the underlying metric traits are constant across environments. The effect can be illustrated using Figure 3. In this example, if the positions of the body size optima for longevity and fecundity were to fluctuate temporally, then the genetic correlation between longevity and fecundity would also fluctuate, alternating between the values of -1.0 and $+1.0$.

Genetic parameters of life-history traits are known to be highly dependent on the environment in which they were measured (Dingle and Hegmann, 1982; Via, 1984; Service and Rose, 1985; Mitchell-Olds and Rutledge, 1986; Dingle et al., 1988). Our analysis shows that genotype-environment interaction in life-history traits will be present virtually whenever selection pressures vary with the environment.

Discussion

In this paper we model variation in life-history traits from the perspective of directional selection on the underlying metric traits. Our chief conclusion is that life-his-

tory traits have lower heritability than metric traits because they are one step further down the causal pathway from genes to phenotype; additional random factors come into play at each step. Low heritability of life-history traits is expected whether or not populations are at evolutionary equilibrium. We suggest that the low heritability of life-history traits has little to do with Fisher's fundamental theorem, and that the heritabilities of single life-history traits cannot be used to indicate whether or not populations are close to equilibrium. The only way to assess the heritability of total fitness seems to be to directly measure it.

There is no simple prediction regarding the amount of additive genetic variance expected in a life-history trait even at equilibrium. A substantial amount of additive genetic variance in a life-history trait can be maintained at equilibrium because the life-history trait is affected by underlying metric traits that are themselves heritable. We present a realistic example in which the amount of additive genetic variance (and the heritability) of a life-history trait rises as a population approaches equilibrium.

The empirical question remains: do life-history traits have lower heritability than metric traits because they possess lower levels of additive genetic variance or because they have higher levels of environmental variance? This question is difficult to answer because metric and life-history characters are measured in different units. A preliminary test may be gained by comparing coefficients of additive genetic variance and also coefficients of environmental variance between metric and life-history traits (although the coefficient of variation may not fully correct for differences in scale; Bryant, 1986). For example, Gustafsson (1986) showed that the average heritability of three life-history traits in female Collared flycatchers (lifespan, number of fledged young, and clutch size) was lower than that for five metric traits (length of tarsus, tail, wing, beak and first primary) (setting negative values equal to zero, $\bar{h}^2 = 0.11$ and 0.57, respectively, L. Gustafsson, pers. comm.). The difference seems to be primarily due to differing levels of environmental variance (coefficients of environmental variation = 0.38 and 0.01, respectively) rather than to differing levels of genetic variation (coefficients of additive genetic variation = 0.01 and 0.02, respectively).

For simplicity, we have developed a model in which we assume that heritable variation in the underlying metric characters is constant. In fact, the variation is expected to be affected by selection, mutation, and other evolutionary processes (e.g., Houle, 1989; Turelli and Barton, 1989). This does not affect computations of the heritability of the life-history traits within a single generation. However, a more complete model describing the evolution of the variances of life-history traits should ultimately incorporate changes in the variances and covariances among the metric traits in addition to the evolution of their mean values.

Our conclusions concerning the low heritability of life-history traits depend only on causal relationships among variables. The conclusions are general to causal pathways, and not just applicable to the inheritance of life-history. For example, we have so far considered all metric traits as a single stage in the path model, but complex inter-relationships certainly exist. A life-history trait (e.g., longevity) may be influenced by a behavioral trait (e.g., choice of diet), which in turn has an underlying morphological basis (e.g., body size). Since additional environmental variance is added at each step along the causal chain from morphology to behavior to life history, we might expect the heritability of the behavior to be lower than that of morphology, and the life-history trait to have lower heritability than the behavioral trait. Such causal relationships may explain why behavioral traits appear to have heritabilities intermediate between morphology and life history (although we should note that this relationship has not been demonstrated at conventional levels of significance) (Mousseau and Roff, 1987).

The consequences of additional complexity are in need of further empirical and theoretical study. In some cases it is known that the environmental component of a metric trait also directly affects the life-history trait. For example, Alatalo et al. (1990) showed that nutritional state affected body size and also survival of young flycatchers, implying a correlation between the environmental component affecting body size

and the environmental component affecting survival. The simple path diagram of Figure 1 then needs to be modified to include this correlation. In this case the heritability of the life-history trait will differ from that derived using equation (3), and in special cases it may exceed that of the metric trait.

The analysis suggests that the study of selection on underlying metric traits, itself of interest for a variety of questions about adaptation, will provide a complementary approach to genetic studies of life-history evolution. This will be especially useful in nature where environments fluctuate, and genetic parameters of the life-history traits are consequently expected to have lower stability than those of the underlying metric traits. By measuring selection on the most important metric traits in the different environments it should be possible to ask how the metric traits would evolve under different circumstances, and in turn cause the life-history traits to evolve in a predictable manner (Price and Grant, 1984). This approach may be practical only when few metric characters lead to most of the heritable variation in the life-history traits. Such an approach parallels the theoretical methods of Wright (1935*a*, 1935*b*) who cast the evolution of fitness (determined by substantial nonadditive variance) in terms of nonlinear selection on an underlying additively determined character.

Acknowledgments

We thank R. Lande for much generous advice and for providing several key references, S. Frank, M. Lynch, K. Marchetti, J. B. Walsh and the reviewers for comments, and L. Gustafsson for providing unpublished data. Supported in part by a grant from NSERC Canada.

Literature Cited

Alatalo, R. V., L. Gustafsson, and A. Lundberg. 1990. Phenotypic selection on heritable size traits: Environmental variance and genetic response. Am. Nat. 135:464–471.

Arnold, S. J. 1983. Morphology, performance, and fitness. Am. Zool. 23:347–361.

Boag, P. T., and P. R. Grant. 1981. Intense natural selection in a population of Darwin's finches (Geospizinae) in the Galápagos. Science 214:82–85.

Bryant, E. H. 1986. On the use of logarithms to accommodate scale. Syst. Zool. 35:552–559.

Charlesworth, B. 1987. The heritability of fitness, pp. 21–40. *In* J. Bradbury and M. Andersson (eds.), Sexual Selection: Testing the Alternatives. Wiley, Chichester and N.Y.

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

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

Crespi, B. J., and F. L. Bookstein. 1989. A path-analytic model for the measurement of selection on morphology. Evolution 43:18–28.

Dingle, H., K. E. Evans, and J. O. Palmer. 1988. Responses to selection among life-history traits in a non-migratory population of milkweed bugs (*Oncopeltus fasciatus*). Evolution 42:79–92.

Dingle, H., and J. P. Hegmann (eds.). 1982. Evolution and Genetics of Life Histories. Springer-Verlag, N.Y.

Endler, J. A. 1986. Natural Selection in the Wild. Princeton Univ. Press, Princeton, NJ.

Falconer, D. 1989. Introduction to Quantitative Genetics, 3rd edition. Longman, N.Y.

Fisher, R. A. 1930. The Genetical Theory of Natural Selection. Oxford, N.Y.

Gibbs, H. L., and P. R. Grant. 1987. Oscillating selection on Darwin's finches. Nature 327:511–513.

Gustafsson, L. 1986. Lifetime reproductive success and heritability: Empirical support for Fisher's fundamental theorem. Am. Nat. 128:761–764.

Hartl, D. L., and A. G. Clark. 1989. Principles of Population Genetics, 2nd edition. Sinauer, Sunderland, MA.

Houle, D. 1989. The maintenance of polygenic variation in finite populations. Evolution 43:1767–1780.

Kalisz, S. 1986. Variable selection on the timing of germination in *Collinsia verna* (Scrophulariaceae). Evolution 40:479–491.

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

———. 1981. Models of speciation by sexual selection on polygenic characters. Proc. Natl. Acad. Sci. USA 78:3721–3725.

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

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

Luckinbill, L. S., T. A. Grudzien, S. Rhine, and G. Weisman. 1989. The genetic basis of selection for longevity in *Drosophila melanogaster*. Evol. Ecol. 3:31–39.

Mitchell-Olds, T., and J. J. Rutledge. 1986. Quantitative genetics in natural plant populations: A review of the theory. Am. Nat. 127:379–402.

Mousseau, T. A., and D. A. Roff. 1987. Natural selection and the heritability of fitness components. Heredity 59:181–197.

Price, G. R. 1972. Fisher's 'fundamental theorem' made clear. Ann. Hum. Genet. 36:129–140.

Price, T. D. 1984. The evolution of sexual size dimorphism in a population of Darwin's Finches. Am. Nat. 123:500–518.

Price, T. D., and P. R. Grant. 1984. Life history traits and natural selection for small body size in a

population of Darwin's finches. Evolution 38:483–494.

PRICE, T. D., P. R. GRANT, H. L. GIBBS, AND P. T. BOAG. 1984. Recurrent patterns of natural selection in a population of Darwin's finches. Nature 309:787–789.

ROFF, D. A., AND T. A. MOUSSEAU. 1987. Quantitative genetics and fitness: Lessons from *Drosophila*. Heredity 58:103–118.

ROSE, M. R. 1982. Antagonistic pleiotropy, dominance, and genetic variation. Heredity 48:63–78.

SCHEMSKE, D. W., AND C. C. HORVITZ. 1989. Temporal variation in selection on a floral character. Evolution 43:461–465.

SCHLUTER, D. 1988. Estimating the form of natural selection on a quantitative trait. Evolution 42:849–861.

SCHLUTER, D., T. D. PRICE, AND P. R. GRANT. 1985. Ecological character displacement in Darwin's finches. Science 227:1056–1059.

SCHLUTER, D., AND J. N. M. SMITH. 1986. Natural selection on beak and body size in the song sparrow. Evolution 40:221–231.

SERVICE, P. M. 1987. Physiological mechanisms of increased stress resistance in *Drosophila melanogaster* selected for postponed senescence. Physiol. Zool. 60:321–326.

SERVICE, P. M., AND M. R. ROSE. 1985. Genetic covariation among life-history components: The effect of novel environments. Evolution 39:943–945.

TACHIDA, H., AND C. C. COCKERHAM. 1988. Variance components of fitness under stabilizing selection. Genet. Res., Camb. 51:47–53.

TURELLI, M., AND N. BARTON. 1989. Dynamics of polygenic characters under selection. Theor. Pop. Biol. 38:1–57.

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

WADE, M. J., AND S. KALISZ. 1989. The additive partitioning of selection gradients. Evolution 43: 1567–1569.

WRIGHT, S. 1930. Review of *The genetical theory of natural selection* by R. A. Fisher. J. Hered. 8:349–356.

———. 1935*a*. The analysis of variance and the correlations between relatives with respect to deviations from an optimum. J. Genet. 30:243–256.

———. 1935*b*. Evolution in populations in approximate equilibrium. J. Genet. 30:257–266.

Corresponding Editor: M. R. Lynch