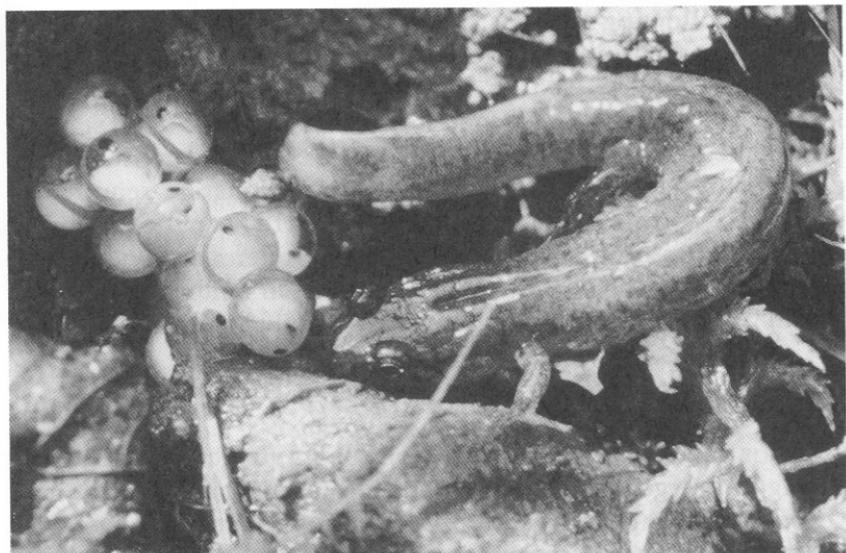


Multivariate Inheritance and Evolution: A Review of Concepts

Stevan J. Arnold



A female dusky salamander (*Desmognathus fuscus*), uncovered while she was brooding her eggs. The mass of yolk provided by the female affects the size of the hatchling larvae. Female behaviors that affect yolk size (e.g., feeding behavior) are said to exert maternal effects on hatchling size. Females also protect their developing young by attacking small predators (Forester 1978). Such antipredator behaviors are said to experience maternal selection (Kirkpatrick and Lande 1989).

The aim of this chapter is to review basic concepts in quantitative genetics. Most readers have heard of heritability and the axiom that genetic response to selection is a function of heritability and selection intensity. That axiom is now at least fifty years old, and the discipline of quantitative genetics has matured considerably

in the intervening years. One important recent development has been the ability to predict genetic responses to selection acting simultaneously on multiple traits. The development of a useful multivariate theory has attracted the attention of evolutionary biologists, because natural selection inevitably acts on multiple traits. Curiously, heritability no longer holds center stage in the new multivariate theory of inheritance and response to selection. Instead, the most general theory of quantitative genetics is cast in terms of genetic variances and covariances, or in terms of the more basic concept of covariance between additive genetic value and phenotypic value. Heritability's fall from grace and the emergence of a new pantheon of concepts is easiest to understand from a historical perspective.

In the following sections I give a simplified account of conceptual developments in evolutionary quantitative genetics. (For more advanced overviews, see Lande 1988; Barton and Turelli 1989; and Arnold 1992b). Heritability and its antecedents are a good starting point, but my main goal is to outline the spirit and content of recent theoretical developments. Some equations are required to succinctly outline these results; I have tried to keep the number of equations at a minimum. Derivations are referenced and are not produced here. Matrix equations are given in boldface, and some useful rules for interpreting them are given in appendix 2.1.

SHORT-TERM RESPONSE TO SELECTION

A major goal in quantitative genetics is to predict how selection acting in one generation will produce genetic change in the next generation. A series of response to selection equations has evolved in the discipline. These equations always have the same form: response to selection is a function of selection and inheritance. *Selection* is a phenotypic concept that describes statistical change in the means of traits in the parental generation. *Inheritance* is a genetic concept that describes how the effects of selection are transmitted from the parental generation to the offspring generation. In the sections below I outline a series of progressively more general equations. The whole series can be written in either of two ways: (1) with inheritance described as a covariance and selection described as a regression or (2) with inheritance described as a regression and selection as a covariance. Both formats have their merits, but I have adopted the first format because it makes the continuity between the various models somewhat clearer.

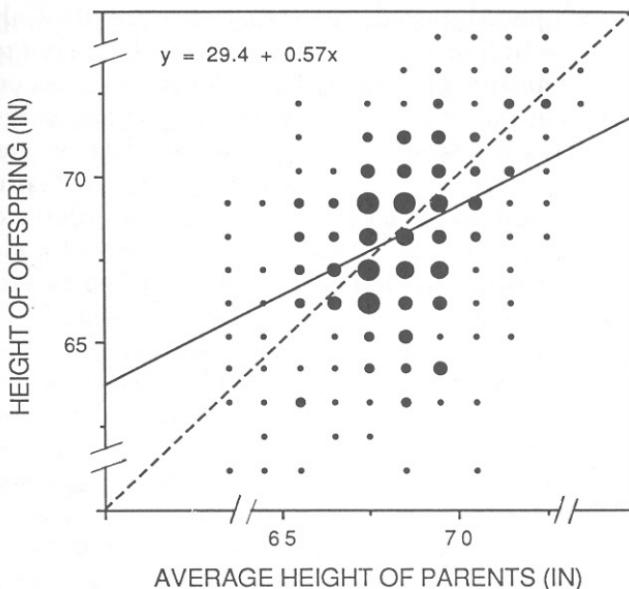


Fig. 2.1 Galton's (1889) data showing the height of adult human offspring ($N = 928$) as a function of the average height of their parents ($N = 205$ sets) in a British population. The dashed line describes perfect inheritance. The calculation of the regression slope (solid line) excluded 36 offspring for which the exact heights of offspring and/or parents were not reported. These 36 extreme points are for parents with average heights less than 63.5 inches or greater than 73.5 inches and for offspring less than 61.2 or greater than 74.2 inches in height.

Response to Selection on a Single Trait

Resemblance between offspring and their parents is the fundamental concept in quantitative genetics because it enables us to predict responses to selection. Galton (1889) introduced a graphical portrayal of offspring-parent resemblance that persists to the present day. An example of a Galton plot is shown in figure 2.1, which shows for a human population the height of adult offspring plotted against the average height of their parents. Such data plots convinced Galton's contemporaries and succeeding generations that there were regularities in the inheritance of traits with continuous distributions. Aside from an enduring graphical format, Galton's contribution was to show that a least squares line fitted to his data deviated from the line representing perfect inheritance (fig. 2.1). Offspring of exceptionally tall parents regressed downward toward the offspring mean, whereas offspring of exceptionally short par-

ents regressed upward toward the mean. Consequently, Galton referred to his best fit line as a regression, a term which later took on a more general meaning in the field of statistics. Regression in its original incarnation as a descriptor of offspring-parent resemblance is still the fundamental concept in quantitative genetics.

The problem facing the generations that succeeded Galton was to reconcile the regression of offspring on parents with the emerging laws of Mendelian inheritance. Mendelian roots for Galton's plots and regressions were independently unearthed by Weinberg (1908), Fisher (1918), and Wright (1921). The basic idea is that many genes affect a trait such as human height, but each gene has only a small effect. If we allow the genes to display dominance in their effects on height and work through an algebraic model, we obtain a disconcerting result: it is not simply the genotypic properties of parents that produce resemblance with their offspring. Instead, we need to define a particular genetic property of parents that encodes the transmission of height characteristics to offspring. At the level of a single genetic locus, this property is known as the *average effect* of the gene, which, perhaps not surprisingly, is defined by a regression of phenotypic value on number of gene copies (zero, one, or two). Falconer (1989) gives a lucid account of the algebra. We can now define the elusive genetic property of parents that produces resemblance with offspring. That genetic property is simply the sum across gene loci of the average effects of alleles on height. Because of the summation, the key genetic property of parents has come to be known as *additive genetic value*. In other words, the recurrent adjective "additive" in the field of quantitative genetics heralds the early twentieth-century triumph in grafting Mendelian roots onto Galton's observations, while accounting for dominance in gene action.

Armed with the concept of additive genetic value, we return to Galton's plot. We can now show that Galton's best fit line is the regression of additive genetic values (observed as the average heights of offspring) on the phenotypic values of parents (observed as their measured heights). The regression slope is known as *heritability*. The slope turns out to be equivalent to the ratio of variance in additive genetic values (*additive genetic variance*) to variance in phenotypic values (*phenotypic variance*). We have now assembled the basic vocabulary used in textbook accounts of quantitative inheritance. Our goal, however, is to understand contemporary extensions of quantitative genetics to evolutionary biology.

To achieve those goals it will be helpful to have an explicit model of the concepts we have reviewed.

Our model for the inheritance of a behavior, or any other attribute affected by many genes, is that an individual's phenotypic value is composed of an additive genetic value (which is transmitted to offspring) and a part which is not transmitted to offspring. The nontransmitted part is conveniently referred to as the individual's environmental value, but it is composed of nonadditive genetic effects (due to dominance and epistasis) as well as purely environmental effects. If we denote the phenotypic value as z , the additive genetic value as a , and the environmental value as e , then our model is

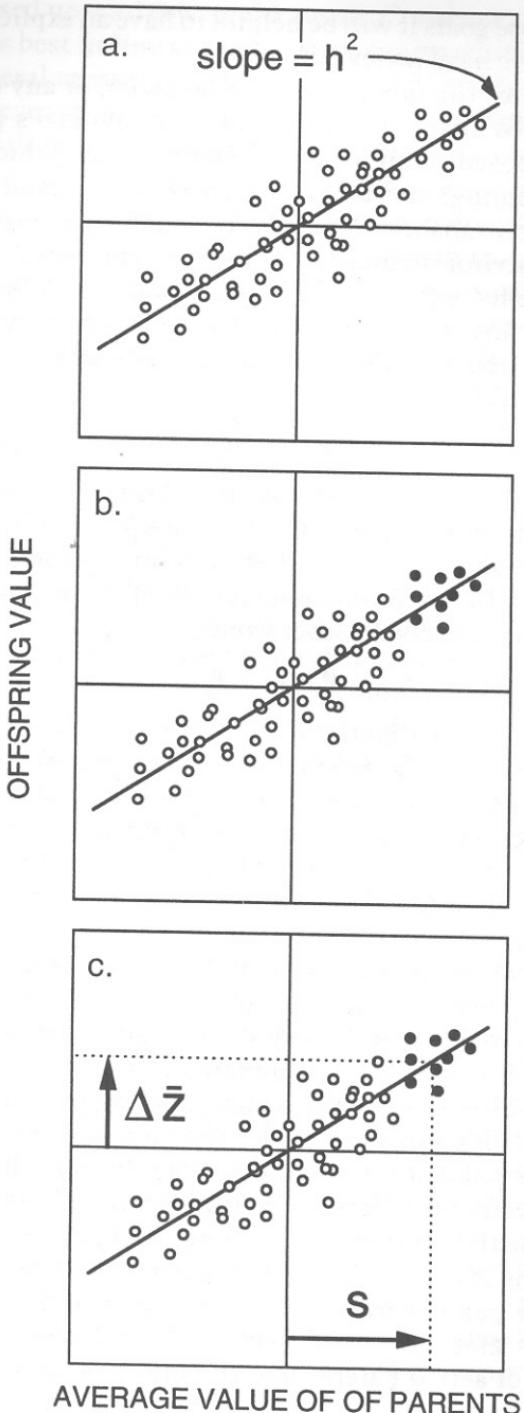
$$z = a + e \quad (2.1)$$

(see appendix 2.1). The mean phenotypic value in the population is similarly composed of two parts, and consequently the population variance in phenotypic values, P , is the sum of an additive genetic variance, G , and a environmental variance, E , which includes non-additive genetic effects. In other words,

$$P = G + E, \quad (2.2)$$

with the assumption that there is no correlation between the additive genetic and environmental values of individuals (see appendix 2.1). We shall return to this assumption later. If we also assume that there is no correlation between the environmental values of parents and their offspring, the slope of the regression line relating offspring values to parental values is $h^2 = G/P$, (h^2 is the standard symbol for heritability).

The primary importance of heritability is that it enables us to predict the genetic consequences of selection that acts on a single trait (e.g., a behavioral attribute). We have discussed the concept of heritability. How shall we conceptualize selection? A useful way to think about selection is to focus on its phenotypic manifestations. Consider the idealized Galton plot shown in figure 2.2a. The projection of the points onto the x -axis represents the distribution of phenotypic values for all the potential parents of the next generation. Suppose that only a subset of potential parents actually produce offspring (fig. 2.2b). The actual parents are a biased subset of all potential parents by virtue of selection acting on the phenotypic trait graphed on the x -axis. The difference between the mean value of actual parents and the mean value of all potential



parents is a useful measure of the strength of selection. This difference in means is known as the *selection differential*. By graphing the selection differential on Galton's plot, we see that the slope of the regression line enables us to predict the genetic consequences of selection (fig. 2.2c). In making this prediction we implicitly assumed that the regression for actual parents is the same as the regression for all potential parents. Pearson (1903) showed that this is the case. As Lush (1945) put it, heritability is the fraction of the selection differential that is transmitted to the next generation. Symbolically, we have the following result: the change in mean from one generation to the next when selection acts on a single trait is

$$\Delta\bar{z} = h^2 s, \quad (2.3)$$

where s is the selection differential. Equation 2.3 is a powerful tool for an experimentalist or a plant or animal breeder who imposes selection on a single trait and wants to know how much change (improvement) to expect. In the natural world, however, selection undoubtedly acts simultaneously on many traits, so we will need an even more powerful tool.

Response to Selection on Multiple Traits

To predict the evolutionary response to selection acting on multiple traits it is convenient to use a new concept of selection, the *selection gradient* (Lande 1979). The selection gradient measures the direct force of selection on a trait. It is equivalent to the partial regression of relative fitness on the trait, holding other traits constant (Lande and Arnold 1983). In contrast, the selection differential measures both the direct force of selection on a trait and the

Fig 2.2 A graphical portrayal of response to selection on a single trait. (a) Resemblance between offspring and their parents. The set of all potential parents and their offspring is shown. The slope of the regression of offspring values on parental values estimates heritability (h^2). The vertical line indicates the mean of parental values (before selection) and the horizontal line indicates the mean offspring value when there is no selection in the parental generation. (b) Same conventions as a but with the set of actual parents and their offspring values indicated with solid symbols. (c) The selection differential, s , is the difference between the mean values of actual parents and all potential parents. The response to selection, $\Delta\bar{z}$, is the difference between the mean offspring values of actual parents and all potential parents. The response to selection is proportional to the selection differential and heritability. (Adapted from Lush 1945 and Falconer 1989.)

indirect effects of selection acting on other traits. The selection differential is equivalent to a covariance between a trait and relative fitness (Robertson 1966).

The only other new concept we need in order to deal with selecting acting on multiple traits is the idea of *additive genetic covariance*. The idea here is that traits may be genetically coupled. Consequently, when selection acts directly on one trait, it may produce reverberations in traits that are genetically coupled to it. Genetic coupling will be revealed in a Galton plot in which we plot offspring values for one trait on the y-axis and parental values for some other trait on the x-axis. A nonzero regression in such a plot is due to covariance between additive genetic values for the two traits. This covariance is known as additive genetic covariance. Such covariance or coupling arises from pleiotropy (individual genes exerting effects on both traits) or linkage disequilibrium. By linkage disequilibrium we do not mean the physical linkage of genes on chromosomes, but rather statistical associations between alleles at different loci (which may or may not be on the same chromosome). Now, the additive genetic covariance between the two traits may be positive, zero, or negative. In the last case, there might be a preponderance of genes with positive pleiotropic effects on one trait, but negative effects on the other trait. Standardized additive genetic covariances are known as *genetic correlations* and can take values between +1 and -1. When dealing with multiple traits it is convenient to arrange the additive genetic covariances in a table known as the *additive genetic variance-covariance matrix*, \mathbf{G} . In this table the first row and column refer to the first trait, the second row and column refer to the second trait, and so on. For p traits, the \mathbf{G} -matrix looks like this

$$\begin{bmatrix} G_{11} & G_{12} & \dots & G_{1p} \\ G_{21} & G_{22} & \dots & G_{2p} \\ \vdots & \vdots & \ddots & \vdots \\ \vdots & \vdots & \ddots & \vdots \\ \vdots & \vdots & \ddots & \vdots \\ G_{p1} & G_{p2} & \dots & G_{pp} \end{bmatrix}$$

where G_{11} is the additive genetic variance for the first trait, G_{12} ($= G_{21}$) is the additive genetic covariance between the first and the second trait, and so forth.

We can use the \mathbf{G} -matrix and the set of selection gradients for

the traits to predict responses to selection acting simultaneously on many traits. In tabular (matrix) form our equation looks like this,

$$\begin{bmatrix} \Delta\bar{z}_1 \\ \Delta\bar{z}_2 \\ \vdots \\ \vdots \\ \Delta\bar{z}_p \end{bmatrix} = \begin{bmatrix} G_{11} & G_{12} & \dots & G_{1p} \\ G_{21} & G_{22} & \dots & G_{2p} \\ \vdots & \vdots & \ddots & \vdots \\ \vdots & \vdots & \dots & \vdots \\ G_{p1} & G_{p2} & \dots & G_{pp} \end{bmatrix} \begin{bmatrix} \beta_1 \\ \beta_2 \\ \vdots \\ \vdots \\ \beta_p \end{bmatrix}$$

in which $\Delta\bar{z}_1$ is the change across generations in the mean of the first trait, β_1 is the selection gradient for the first trait, and so forth. The set of changes in means and the set of selection gradients are known as column vectors. The whole set of equations is conveniently represented as

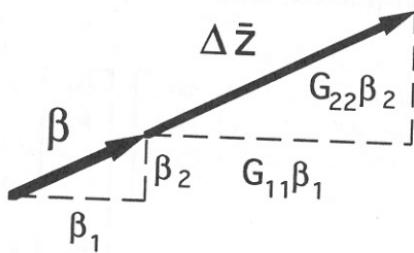
$$\Delta\bar{z} = G\beta, \quad (2.4)$$

in which $\Delta\bar{z}$ and β are the column vectors given above. We can now let the equations do some work for us. Using the rules of matrix algebra (see appendix 2.1), we can write out the equation for the change in mean of the first trait, which is

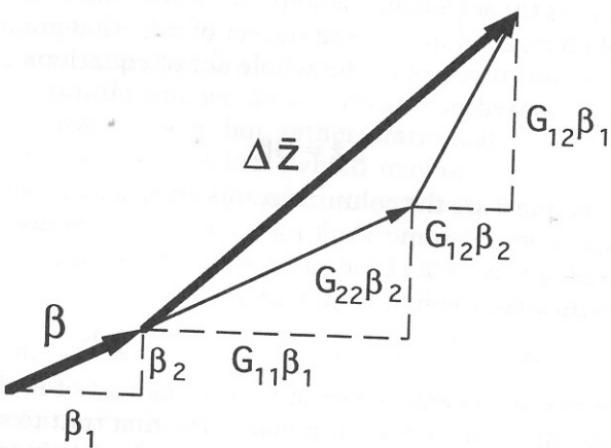
$$\Delta\bar{z}_1 = G_{11} \beta_1 + G_{12} \beta_2 + \dots + G_{1p} \beta_p$$

We see before us several important consequences of multivariate selection. The evolutionary response of the first trait to selection, i.e., $\Delta\bar{z}_1$, is composed of several possible contributions. One of these is the direct response to selection, $G_{11} \beta_1$, representing the change in the mean of trait 1 due to selection acting directly on trait 1. The other contributions are correlated responses to selection. For example, $G_{12} \beta_2$ represents change in the mean of trait 1 due to selection acting directly on trait 2 and inducing change in trait 1 via the additive genetic covariance G_{12} (fig. 2.3). It is possible that the change in the mean of trait 1 is wholly or largely due to direct response to selection on trait 1. But, it is also possible that the many possible correlated responses to selection will dominate or swamp the direct response. In the extreme case, selection might favor high values of the first trait (positive β_1), yet the trait will evolve toward a lower mean value because of negative additive genetic covariances (fig. 2.3c) or because of selection for low values of other traits. In general, if we think of selection as a pool cue (β) and

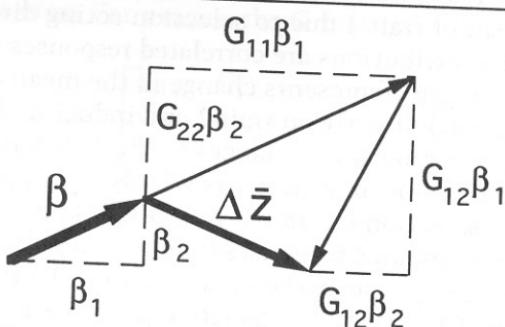
a.



b.



c.



response to selection as the path of the ball ($\Delta\bar{z}$), the effect of genetic covariance is to throw the ball's path out of alignment with the cue (fig. 2.3).

Estimation of Genetic Variances and Covariances

Defining behavioral traits. How to define a behavior is an important decision that needs to be made before launching a program of genetic parameter estimation. Two important considerations to keep in mind are relevance to ecological circumstances or evolutionary history and simplicity of scoring. If a study of inheritance is conducted with traits that have straightforward ecological or evolutionary significance, the results can add a genetic dimension to ecological or evolutionary issues. Simplicity of scoring is crucial because the behaviors must be scored on many individuals (and sometimes many times on each individual) to estimate genetic parameters. The study of behavioral inheritance is especially challenging because the expression of a particular behavior by an individual may fluctuate over time, show maturational change, or be influenced by experience or condition.

1. Fluctuation without a temporal trend can be handled by making repeated scores on each individual. The issue of how many scores to make can be decided in a pilot experiment in which the behavior of each individual is scored two or more times. The among-individual component of variance, expressed as a fraction

Fig. 2.3 A graphical portrayal of response to selection on two traits. Our response to selection equation for the first trait is, $\Delta\bar{z}_1 = G_{11}\beta_1 + G_{12}\beta_2$, and for the second trait, $\Delta\bar{z}_2 = G_{22}\beta_2 + G_{12}\beta_1$. In each of the three cases shown, direct selection on the first trait, β_1 , is positive and twice as strong as direct selection on the second trait, β_2 , and the additive genetic variances for the two traits are equal, $G_{11} = G_{22} = 1$. The vector representing direct selection, β , is shown as a heavy arrow. The vector representing response to selection, $\Delta\bar{z}$, is shown as a medium arrow. The vectors representing direct and correlated responses are shown as light arrows.
 (a) No genetic covariance between the two traits, $G_{12} = 0$. The response to selection is composed only of direct responses to selection, $G_{11}\beta_1$ and $G_{22}\beta_2$. Because the two genetic variances are equal and genetic covariance is zero, the response is aligned with the direction of selection. (b) Positive genetic covariance between the two traits, $G_{12} = 0.75$. The two correlated responses to selection, $G_{12}\beta_1$ and $G_{12}\beta_2$, throw the total response out of alignment with the direction of selection. (c) Negative genetic covariance between the two traits, $G_{12} = -0.75$. The genetic covariance, acting through the correlated responses, now produces an extreme departure from alignment. Notice that trait z_2 evolves in the opposite direction to the selection acting directly on it.

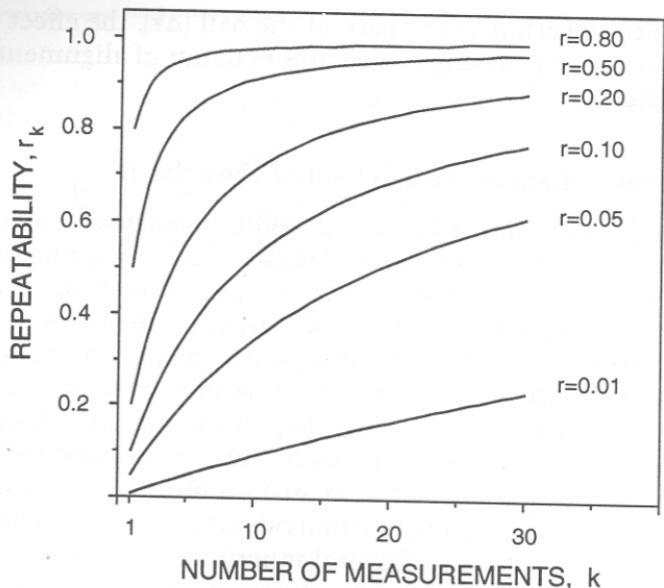


Fig. 2.4 The repeatability of k measurements or trials, r_k , as a function of single-trial repeatability, r , and the number of measurements or trials, k . Using formulas given in Winer (1971) and Falconer (1989), $r_k = kr/[kr - r + 1]$. In Falconer's (1989) notation, r_k is also equal to $(V_G + V_{Eg})/V_{P(k)}$, where $V_G + V_{Eg}$ is the among-individual component of variance and $V_{P(k)}$ is the total phenotypic variance for the average of k trials.

of total phenotypic variance, is known as the intraclass correlation, reliability, or *repeatability* (Winer 1971; Falconer 1989). Such a repeatability refers to individual consistency of single-trial scores, but we can also compute the repeatability of the average of two or more trials or measurements (fig. 2.4). We see from figure 2.4 that repeatability increases with the number of measurements, k . Thus, if a pilot experiment indicates that single-trial repeatability is 50%, by making four measurements on each individual and using the four-trial averages, we can increase repeatability to 80%. Because repeatability places an upper bound on heritability (Boake 1989b; Falconer 1989), we are more likely to be able to detect statistically significant heritability if our behavioral score is a four-trial average rather than a single-trial score. The use of multiple-trial averages can be justified on biological as well as statistical grounds. An animal has only one left eye, so measuring the repeatability of the width of the left eye is an exercise in assessing measurement error. But an animal may perform a courtship display

hundreds or even thousands of times during its life. Consequently, the repeatability of a behavior, such as a courtship display, depends on the time scale over which behavioral averages are assessed. The proper time scale in turn depends on the question being asked. If the issue is response to lifetime selection, then life span is the proper time scale, and k is the average number of times the behavior is expressed in a lifetime. If we are interested in female discrimination of male behavior, then k might be the average number of male displays given to an attending female. Houck, Arnold, and Thisted (1985) give an example of time scale for repeatability as an ecological issue. An important technical point is to keep estimates of repeatability and heritability in register. If repeatability is computed using the sum or average of k trials, then the heritability for k trials should be computed, rather than the single-trial heritability. Arnold and Bennett (1984) give an example.

2. Behaviors that change with age can be treated as age-specific attributes. Examples of genetic analysis of behavioral ontogenies can be found in Hahn et al. (1990).

3. Behaviors that can be affected by experience call for special experimental procedures. One simple approach is to score behavior in neonates that are either completely naive or all have identical, limited experience (e.g., Arnold 1981a). If the inheritance of learning is the issue of interest, the difference in behavioral scores before and after experience can be treated as a trait. Repeated measures or profile analysis of variance are useful tools for data analysis (Winer 1971; Bock 1975; Harris 1975; Simms and Burdick 1988).

4. Techniques for handling behaviors that vary with nutritional condition or body size are discussed by Travis (chap. 8).

Reference population. The choice of a reference population is also a critical decision. Genetic variances and covariances may vary from one population to the next, even within a species. Therefore, if we draw sets of relatives from more than one population, the genetic parameters we estimate may not be characteristic of any natural population. Likewise, genetic parameters may change over several generations of captive propagation (e.g., due to inbreeding and/or drift), so that estimation based on a long-maintained laboratory line may not be characteristic of any natural population. If the aim of genetic parameter estimation is to make inferences about inheritance and responses to selection in nature, the best procedure generally is to sample directly from a single natural

population. A second reason for having a reference population is that the genetic results will apply to a particular population with definable ecology and phylogenetic history. The ecology of the reference population can be studied along with its behavioral inheritance with synergistic results. Grant and Grant's (1989) study of Galápagos finches, for example, illustrates the power of focusing both genetic and ecological studies on the same reference population.

Overview of parameter estimation. Genetic variances and covariances (or their standardized analogues, heritability and genetic correlation) can be estimated in two ways: by conducting selection experiments or by assembling replicated sets of relatives. Falconer (1989) gives an elementary account of selection experiments; Hill (1980) discusses design considerations. I will focus on the use of sets of relatives. The classic approach in using sets of relatives is to assemble replicated sets of first-degree relatives (e.g., sets of parents and offspring, full sibs, or paternal half sibs) or to produce them in a breeding design. The basic tools of data analysis are regression and analysis of variance (Cockerham 1963; Becker 1984; Falconer 1989). A more recent approach is to use maximum likelihood to estimate the genetic parameters. This approach can be used with sets of first-degree relatives (Thompson 1977; Shaw 1987) or sets of individuals related by complex pedigrees (Lange, Westlake, and Spence 1976; Hopper and Matthews 1982; Lange, Weeks, and Boehnke 1988).

The basic idea in using sets of relatives to estimate genetic parameters is that observable phenotypic resemblance among different sets of relatives can be used to isolate and estimate the additive genetic and other interesting parts of the total phenotypic variance in behavior. Think of a stick representing the total variance, with marks indicating the additive genetic variance and other parts of the total. By observing the phenotypic resemblance among parents and offspring, among full sibs, among paternal half sibs, etc., we can break the stick at well-defined points and so isolate the segment that interests us most—the segment representing additive genetic variance. The same analogy applies to a stick representing the total phenotypic covariance between a pair of traits. Some complicated breeding designs have the goal of breaking the stock into as many interesting pieces as possible. I will focus on some simple sets of relatives and their use in estimating additive genetic variances and covariances.

Parents and offspring. We have already discussed the correspondence between the regression of parental on offspring scores and heritability. Two important assumptions were made in equating the regression slope with heritability. One assumption was that there is no correlation between the environmental values of parents and their offspring. This assumption can be violated in many ways. For example, if territories are passed from parents to their offspring, then the properties of territories, rather than genes, may promote resemblance between parents and their offspring. In a laboratory setting, care can be taken to standardize environments, but in sets of free-ranging parents and offspring the possibility of cross-generational correlation in environmental effects should be considered in the interpretation of resemblance between parents and offspring. If parents directly affect the behavior of their offspring (e.g., by tutoring or by nutritional effects), a more complicated model of phenotypic value should be considered (see below). A second critical assumption was that additive genetic values are not correlated with environmental values. Falconer (1989) gives the example of larger individuals, with higher additive genetic values for body size, also getting more to eat and so inducing a positive correlation between genetic and environmental values for size. In many instances such covariances between environmental values of parents and offspring or between genetic and environmental values within a generation may be implausible, but they should be considered.

In situations where the complications just discussed can be put aside, regression analysis can be used to estimate genetic variances and covariances from parent-offspring data. Estimates can be made using data on both parents or data on just one parent (Falconer 1989, chap. 9). When *family size* (i.e., the number of offspring in a brood) varies, the technical problem of how to weight families arises. Unfortunately the best weight is itself a function of the genetic parameters being estimated. Bulmer (1985) discusses an iterative solution to the problem. A shortcut is to compute two regressions, weighting families by their sizes and weighting them equally. The correct weighting is somewhere in between these extremes. If the regression slopes found using the two extreme weightings do not differ, it is probably not worth resorting to the more exact iterative approach.

Full sibs. Sets of offspring each with a different mother and father (full sibs) produce more ambiguous estimates of genetic parame-

ters than any of the other sets of relatives. The procedure with sets of full sibs is to estimate the within- and among-sibship components of variance using a random effects analysis of variance (Sokal and Rohlf 1981). Unfortunately, in this case the stick representing total phenotypic variance is broken in a rather jagged way, so we do not get a clean estimate of additive genetic variance. The among-sibship variance component estimates additive genetic variance plus part of the genetic variance due to dominance and environmental variance arising from the common conditions experienced by sibs. One can minimize the common family environmental variance by standardizing as much as possible the environment during pregnancy and/or rearing. Even so, one needs to qualify the conclusions made from full sib data because the estimates of additive genetic parameters are inflated (or depressed) by some unknown amount.

Paternal half sibs. In contrast to using sets of full sibs, using sets of paternal half sibs enables us to break the stick of variation in a very clean way. Paternal half sibs are usually produced using a deliberate breeding design in which each of a series of males (sires) is mated to a different set of females (dams). Consider first the analysis when phenotypes are scored on only the offspring. The data structure is a nested one, with dams nested within sires. The data are analyzed using a two-level analysis of variance, with the goal of estimating the among-sire component of variance (Falconer 1989, chap. 10). Unless there are paternal effects (see below), the among-sire variance component estimates one-fourth of the additive genetic variance. Likewise, a two-level analysis of covariance is conducted to estimate the among-sire components of covariance and hence the additive genetic covariances between traits. If phenotypic scores are available for parents as well as for offspring (and if both generations were reared under equivalent conditions), all of the data can be used to estimate genetic parameters. The best approach is probably to use maximum likelihood estimation (Shaw 1987).

Maternal half sibs. Another way to cleanly break the stick of variation is to produce both maternal and paternal half sibs. The primary gain from the extra work is an estimate of the contribution of maternal effects. The production of maternal half sibs is usually possible only in animals with external fertilization (e.g., most anurans, some fishes). The most feasible design is to randomly assign breeding animals to blocks, with d dams and s sires in each block.

The ova from each dam in a block are divided into s groups and each group is fertilized with sperm from a different sire in that block. The offspring are reared individually and their phenotypes are scored. The data in a block have a factorial structure, sires crossed with dams. The data can be analyzed with a two-way analysis of variance (e.g., Simms and Rausher 1989). The among-sire component of variance (or covariance) can be used to estimate additive genetic variance (or covariance). The contrast between the among-sire and the among-dam components of variance can be used to isolate the contribution of maternal effects (Willham 1963, 1972).

Sample size. When heritability is high (>70%), a statistically significant effect may be detected with only a dozen or two parent-offspring sets or sibships. But when heritability is low (<30%), many dozens or scores of parent-offspring sets or sibships will be needed to show a statistically significant effect. Mousseau and Roff's (1987) survey suggests that behavioral heritabilities are usually in the low range. Under these circumstances, investigators often adopt one of two postures: "genetics without tears," or the more ambitious parameter estimation approach. The goal of the "genetics without tears" approach is to see whether a genetic effect can be detected with a modest sample of families or sibships (Antonovics 1982). This approach is one of exploration and can be justified if the phenotype or behavior in question has never been scrutinized from the heritability point of view. The gamble is that statistically significant genetic effects might be detected. If they are not, the investigator is in no position to argue that heritability is nonexistent, because the experimental design lacks power. An advantage of "genetics without tears" is that family structure can be incorporated into an experimental design and permit qualitative tests for genetic differences as part of a larger set of issues (e.g., Newman 1988; Trexler and Travis 1990; Arnold 1992a). In contrast, the estimation of parameters approach focuses on making standard errors around estimates of genetic parameters as small as possible. More effort is expended and more precision is the reward. A disadvantage of parameter estimation is that the thorough pursuit of one or a few genetic issues may cause the investigator to sacrifice other pursuits.

The potential gains from the additional effort can be gauged by a study of sampling properties. Klein, DeFries, and Finkbeiner (1973), for example, give tables of expected standard errors for various values of parametric heritability and numbers of families in

the sample. Their tables, however, are constructed for students of primates and other organisms with small numbers of offspring (1–3). When family sizes are substantially larger, standard errors are considerably smaller than those given in the tables. For example, suppose one wishes to estimate heritability by regressing offspring phenotypic values on the phenotypic value of one parent. How many parent-offspring sets should be assembled to give a good heritability estimate? The answer depends on magnitude of parametric heritability and family size. A good solution is to proceed on a worst-case basis, imagining that parametric heritability might be as low as 20%. We can see from figure 2.5a that if there is only one offspring per parent we would need nearly 400 families to bound our estimate of h^2 away from zero. But, if we had ten offspring per family, we would need only about 70 families to achieve a comparably small standard error. A conscientious investigator can produce tailor-made tables of expected standard errors in less than an hour using a spreadsheet such as Lotus 1-2-3. If a full sib-half sib breeding design is being contemplated, Robertson (1959a,b) should be consulted.

The inefficiency of small family sizes can also be appreciated by considering the power of a heritability estimate. Roughly speaking, statistical power is the probability of correctly concluding that heritability is greater than zero when parametric heritability is in fact greater than zero. A number of authors and reviewers have erroneously cited Klein (1974) in reaching the conclusion that hundreds of families are needed to achieve good statistical power in estimating heritability. Klein, however, considered only the case of small family sizes ($n = 1–4$). We see from figure 2.5b that substantial statistical power can be achieved with less than a hundred families, if family size is large.

Other Models for Phenotypic Value and Variance

Genotype-environment correlation. Under some circumstances it is conceivable that environmental effects are correlated with additive genetic value. For example, in territorial species, larger individuals (which may tend to have large genetic values for body size) may obtain better territories and hence more food. The model for phenotypic values is the same as that given in equation 2.1, but the model for phenotypic variance becomes,

$$\mathbf{P} = \mathbf{G} + \mathbf{E} + 2\text{COV}(\mathbf{a}, \mathbf{e}), \quad (2.5)$$

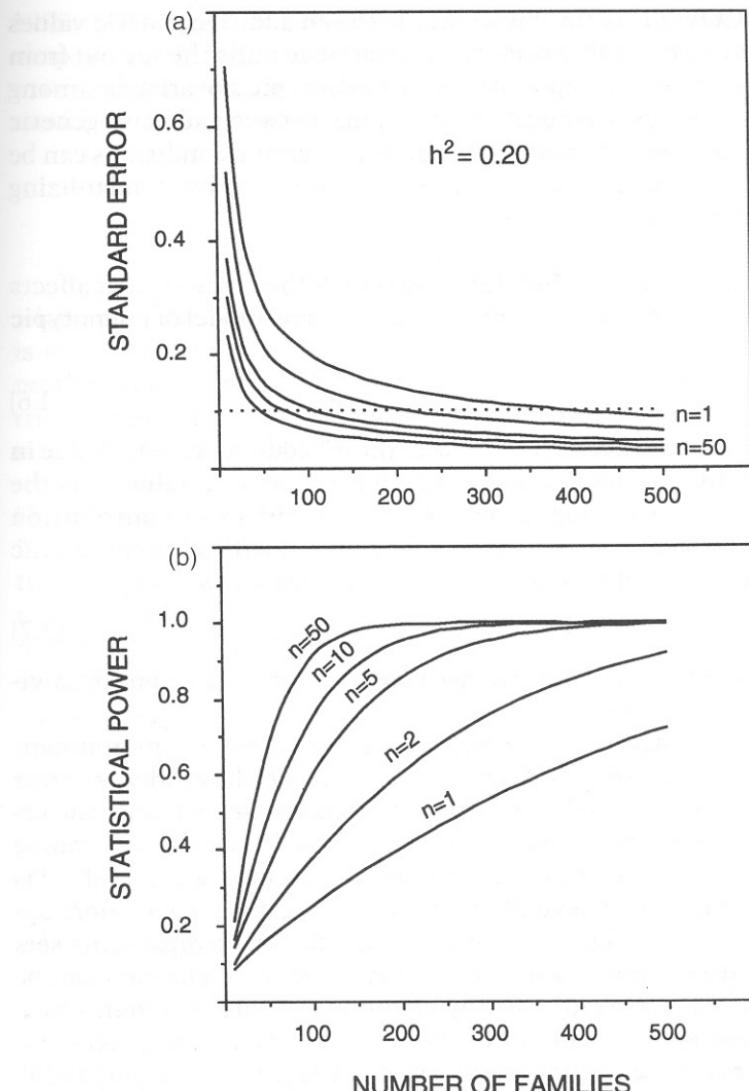


Fig. 2.5 Standard error and statistical power as a function of number of families and family size (n). A parametric heritability of 0.20 is assumed, and estimation is made by regressing offspring values on the phenotypic value of one parent.
 (a) Standard errors. The curves were drawn using formulas given in Klein, DeFries, and Finkbeiner 1973 and Falconer 1989. The horizontal dotted line shows the threshold for standard errors small enough to bound the estimate away from zero at the 0.05 level. (b) Statistical power. The curves were drawn using formulas given in Klein 1974 and Table P (areas under a normal curve) in Rohlf and Sokal 1969, with $\alpha = 0.05$ (one-tailed test).

where $\text{COV}(\mathbf{a}, \mathbf{e})$ is the covariance between additive genetic values and environmental effects. Such covariance pulls the rug out from under standard interpretations of phenotypic covariance among relatives, which assume zero covariance between additive genetic values and environmental effects. When rearing conditions can be controlled, however, $\text{COV}(\mathbf{a}, \mathbf{e})$ can be eliminated by standardizing environmental conditions.

Genotype by environment interaction. If the environment affects different genotypes differently, we have a new model of phenotypic value,

$$z_{ij} = a_i + e_j + ae_{ij}, \quad (2.6)$$

where z_{ij} is the phenotypic value of the i th additive genetic value in the j th environment, a_i is the i th additive genetic value, e_j is the effect of the j th environment, and ae_{ij} is the extra contribution from the j th environment interacting with the i th additive genetic value. Our model for phenotypic variance becomes

$$\mathbf{P} = \mathbf{G} + \mathbf{E} + \text{VAR}(\mathbf{a} \times \mathbf{e}), \quad (2.7)$$

where $\text{VAR}(\mathbf{a} \times \mathbf{e})$ is the variance contribution of genotype by environment interaction.

Most investigators routinely ignore genotype by environment interaction, but there are ways to test for it. The basic idea is to rear each genotype in a series of environments and see whether the genotypic reactions to environment are parallel. Genotypes can be replicated if inbred lines are available or if cloning is possible (as in some plants). In sexually reproducing organisms it is more appropriate to distribute sets of paternal half sibs (representing sets of additive genetic values) across environments. The data can be analyzed by testing for a sire by environment interaction in a two-way analysis of variance or by estimating the additive genetic covariance across environments (Falconer 1960; Via and Lande 1985). The choice of environmental treatments is critical. The range of environments should coincide with the range encountered under natural conditions. If the range of imposed environments greatly exceeds the natural range (e.g., Gupta and Lewontin 1982), the genotype by environment interaction detected in the laboratory may greatly overestimate the interaction in nature.

Maternal effects. The phenotype of a mother may directly affect the phenotypes of her offspring (Cowley 1990; Mousseau and Dingle

1990, 1991; Reznick 1990; Bernardo 1991; Cheverud and Moore, chap. 4). Such maternal effects complicate the interpretation of maternal-offspring resemblance. Consider the following linear model for the offspring's phenotypic value for the i th trait,

$$z_{o_i} = a_{o_i} + e_{o_i} + \sum_{j=1}^n M_{ij} z_{m_j}^*, \quad (2.8)$$

where a_{o_i} is the offspring's additive genetic value for the i th trait, e_{o_i} is the environmental value for the offspring's i th trait, $z_{m_j}^*$ is the mother's phenotypic value for her j th trait (with the asterisk denoting that her phenotypic value is evaluated after selection), and M_{ij} is a maternal effect coefficient describing the effect of the mother's j th trait on the offspring's i th trait (Kirkpatrick and Lande 1989). Our earlier model for the composition of phenotypic variance (eq. 2.2) no longer holds. Furthermore, when selection acts on a single trait, response to selection is no longer a simple function of heritability and selection differential (eq. 2.3) (see Cheverud and Moore, chap. 4). And when selection acts on multiple traits, response to selection is no longer a simple function of the **G**-matrix and the selection gradients (eq. 2.4). Instead, we have a new response to selection equation which involves a new matrix, \mathbf{C}_{az} , representing the covariances between additive genetic values and phenotypic values. Our new equation for response to multivariate selection in generation t is

$$\Delta\bar{\mathbf{z}}(t) = \mathbf{C}_{az} \beta(t) + \mathbf{M}\Delta\bar{\mathbf{z}}_m^*(t-1), \quad (2.9)$$

where $\mathbf{M}\Delta\bar{\mathbf{z}}_m^*(t-1)$ is a sum of terms representing change in the mean phenotypic values of mothers after selection in the preceding generation (i.e., from generation $t-2$ to generation $t-1$) (Kirkpatrick and Lande 1992). Thus, the response to selection in the current generation is partly due to selection acting in that generation and partly due to selection acting in the mothers' generation and then affecting offspring values via maternal effects. The important message of equation 2.9 is that in the presence of maternal effects the key genetic parameters are no longer heritabilities or even the **G**-matrix. Instead, the main targets for estimation are two matrices, \mathbf{C}_{az} and \mathbf{M} .

The estimation of genetic parameters when there are maternal effects, but no paternal effects (eq. 2.8), is discussed by Lande and Price (1989). The basic idea is that maternal effects contribute to

the phenotypic resemblance between mothers and offspring but they do not contribute to the resemblance between fathers and offspring. Consequently, if we have data on the phenotypic values of mothers (including the attributes that exert maternal effects), fathers, and offspring, we can estimate the matrix of maternal effects, \mathbf{M} , as well as the matrix \mathbf{C}_{az} . Alternatively, one can use a breeding design that generates a large set of different kinds of relatives and/or cross-fostering (Eisen 1967; Hanrahan and Eisen 1973; Cheverud et al. 1983; Riska, Rutledge, and Atchley 1985). (See Cheverud and Moore, chap. 4, for more details.)

Maternal and paternal effects. In some fishes, birds, and mammals both sexes care for the offspring (Clutton-Brock 1991). In such species, paternal as well as maternal effects are possible. Our model for phenotypic value becomes

$$z_o = a_o + e_o + \mathbf{M}z_m^* + \mathbf{F}z_f^* \quad (2.10)$$

where $\mathbf{M}z_m^*$ is algebraic shorthand for the sum of terms on the right side of equation 2.8, z_f^* represents the phenotypic values of fathers (after selection), and \mathbf{F} represents a matrix of paternal effect coefficients that translate values of fathers' phenotypes into effects on offspring traits. The response to selection equation is analogous to equation 2.9 but involves an additional term representing the change in mean values of phenotypes of fathers in the preceding generation. The key target for genetic parameter estimation is again the matrix \mathbf{C}_{az} , but there are now two matrices of parental effect coefficients that need to be estimated, \mathbf{M} and \mathbf{F} .

The appearance of a new inheritance matrix, \mathbf{C}_{az} , in our response to selection equations is at first a disconcerting result. The reason we need \mathbf{C}_{az} instead of \mathbf{G} becomes clear, however, when we consider our underlying model. To predict offspring values from parental values we need the covariance between additive genetic values (revealed as the average values of offspring) and phenotypic values of parents (Dickerson 1947). This covariance is \mathbf{C}_{az} . We see from our model (applying eq. 2.10 to the parental generation) that the phenotypic value of a parent is composed of its additive genetic value, a value transmitted directly from its mother, a value transmitted directly from its father, and an environmental deviation (The latter deviation or value will not concern us, because we have assumed that it is uncorrelated with additive genetic value). Because phenotypic value is composed of three relevant parts, when we consider its covariance with additive genetic value, we find that the covariance, \mathbf{C}_{az} , is composed of three parts: \mathbf{G} , representing the

covariance between additive genetic values; $1/2 \mathbf{C}_{az} \mathbf{M}^T$, representing the covariance between additive genetic value and maternally transmitted value; and $1/2 \mathbf{C}_{az} \mathbf{F}^T$, representing the covariance between additive genetic value and paternally transmitted value. In other words,

$$\mathbf{C}_{az} = \mathbf{G} + \frac{1}{2} \mathbf{C}_{az} \mathbf{M}^T + \frac{1}{2} \mathbf{C}_{az} \mathbf{F}^T \quad (2.11)$$

The superscript T denotes matrix transposition (see appendix 2.1). With maternal effects but no paternal effects, we drop the last term on the right in equation 2.11 (Kirkpatrick and Lande 1989). In the absence of both maternal and paternal effects, $\mathbf{C}_{az} = \mathbf{G}$, and we could have substituted \mathbf{C}_{az} for \mathbf{G} in our response to selection equation (eq. 2.4). Thus, \mathbf{C}_{az} is the fundamental inheritance concept that runs through all of our response to selection equations (eqs. 2.3, 2.4, 2.9, 2.12, 2.13).

Estimation of genetic parameters when both sexes exert parental effects has been little explored, and there is virtually no empirical work. One possible experimental route is to rear broods allowing only mothers or fathers to exert care, but cross-fostering is probably the most efficient approach.

Selection Exerted by Relatives

Maternal selection. In species with maternal care, the attributes of the mother may directly affect the fitness of offspring. Such maternal selection is different from the maternal effects we considered in the preceding section. A maternal attribute exerts a maternal effect by affecting the expression of a trait in the offspring. A maternal attribute exerts maternal selection by directly affecting the fitness of the offspring (Kirkpatrick and Lande 1989). In many cases, the maternal traits exerting maternal selection will be behaviors. When a mother bird performs a display that distracts predators from her nest, the distraction display directly affects her offspring's fitness. In other words, the display exerts maternal selection. Such maternal selection constitutes an additional force promoting the evolution of distraction displays in the offspring generation. In addition, maternal selection on the distraction display can evoke correlated responses in traits that are genetically coupled to the distraction display.

The nature of response to selection when maternal selection prevails depends on the system of inheritance. In the absence of

maternal effects (i.e., eq. 2.1), the evolutionary response to multivariate selection is relatively simple,

$$\Delta \bar{z} = G\beta_o + \frac{1}{2} G\beta_m \quad (2.12)$$

where β_o denotes the offspring selection gradient and β_m denotes the maternal selection gradient (Kirkpatrick and Lande 1992). In the presence of maternal effects (i.e., eq. 2.8), the evolutionary response to multivariate selection is somewhat more complicated,

$$\Delta \bar{z}(t) = C_{az}\beta_o(t) + \frac{1}{2} C_{az}\beta_m(t) + M\Delta \bar{z}_m^*(t-1) \quad (2.13)$$

(using equations 2 and 3 of Kirkpatrick and Lande 1992). (See Cheverud and Moore, chap. 4, for more discussion of the first two terms on the right side of equation 2.13 for the case in which selection acts on a single trait.)

Maternal and paternal selection. When both sexes perform parental care, both maternal and paternal selection are likely to prevail. The multivariate response to selection equations are analogous to equations 2.12 and 2.13 but with additional terms representing contributions from paternal selection, β_f , and in the case of equation 2.13, an additional term representing a contribution from selection acting on fathers in the preceding generation.

Nonlinear maternal and paternal selection. In the preceding sections we considered directional parental selection in which the attributes of parents have linear effects on the fitness of their offspring. The attributes of parents can also have nonlinear effects on offspring fitness. The simplest nonlinear effects are quadratic effects, which involve the squared phenotypic values of maternal or paternal attributes and the products of phenotypic values of mothers, fathers, and offspring. Such nonlinear parental selection does not directly contribute to the evolutionary response to selection (the change in trait means across generations), but it does affect the maintenance and evolution of genetic variances and covariances. As in the case of directional parental selection, nonlinear parental selection will often involve behavioral attributes that shelter or protect the offspring from hazards.

Overview of Short-term Selection Response

The genetic concept that runs through these various cases of response to selection is the covariance between additive genetic value and phenotypic value, C_{az} . In the breeder's response equation (eq. 2.3), this covariance cryptically resides in the numerator of heritability. In the equation for response to multivariate selection (eq. 2.4), C_{az} masquerades as the G -matrix. When we consider parental effects, we are forced to make a distinction between C_{az} and G (eq. 2.11). In this revealing case, we find that it is C_{az} (rather than heritability or additive genetic variance/covariance) that plays a central role in predicting response to selection.

EVOLUTIONARY MODELS (LONG-TERM RESPONSE TO SELECTION)

For many years response to selection equations were used to predict only one or a few generations of genetic change. Lande (1976a, 1980a, 1984, 1988) broadened the scope of applications by arguing that genetic variances and covariances might be maintained in equilibrium by a balance between input from mutation/recombination and loss to selection. Or, as Kurtén (1959) put it, "the replenishing of genetic material keeps pace with the appetite of selection." In this view, relative constancy of the G -matrix (eq. 2.4) might be maintained while the phenotypic mean undergoes a substantial evolution over many generations. Lande's argument for constancy of the G -matrix has been challenged by Turelli (1984, 1985, 1986, 1988a), who argued that the amount of genetic variation maintained at equilibrium depends on untested assumptions about the statistical distribution of allelic effects on phenotypic traits. Empirical tests of the constancy issue have been made by comparing G -matrices after varying intervals of phylogenetic separation. (Methodologies for comparing matrices are discussed by Flury 1988; Shaw 1991, 1992; and Cowley and Atchley 1992.) These tests have yielded results ranging from rough structural similarity of G -matrices (Arnold 1981a; Atchley, Rutledge, and Cowley 1981; Lofsvold 1986; Kohn and Atchley 1988) to striking constancy (Wilkinson, Fowler, and Partridge 1990; Arnold 1992b). Thus, the jury is still out on the general issue of G -matrix constancy as well as on the related issue of how much G -matrix similarity is required to model evolution with quantitative genetics. Meanwhile, a series of evolutionary models has been pursued, predicated on the constancy of the G -matrix (reviewed by Lande 1988).

The range of evolutionary problems that can be treated using equation 2.4, under the assumption of **G**-matrix constancy, is very broad and includes the evolution of allometric relationships (Lande 1979), behavioral ontogenies (Arnold 1990), plasticity (Via and Lande 1985), sexual dimorphism (Lande 1980b), life history (Lande 1982a), and evolution by sexual selection (reviewed by Arnold 1987b; Pomiankowski 1988). In all these models, selection is described by a surface, as will be discussed below. One solves for the phenotypic composition of the population at equilibrium and for the trajectories of populations evolving toward that equilibrium. A selling point for these models is that they do not assume that populations are in equilibrium. Instead they give a picture of how populations might evolve toward equilibria, and they make predictions about how long the transit to equilibrium might take. A further attractive feature of the models is that they are cast in terms of observable properties of continuously varying traits (genetic variances and covariances, selection gradients). The models provide an agenda of issues to be tackled in natural populations, as well as insights about evolutionary dynamics. Heisler (chap. 5) provides more discussion of quantitative genetic models of evolution as they are applied to the evolution of mating behavior. Partridge (chap. 6) and Roff (chap. 3) discuss how other approaches can be used to complement a quantitative genetic study or model.

The concept of selection as a surface is a fundamental feature of quantitative genetic models of long-term evolution. The basic ideas can be grasped in a two-dimensional phenotypic space. Imagine phenotypic values for one trait plotted on the *x*-axis and phenotypic values for a second trait plotted on the *y*-axis. The bivariate values for the distribution in a population might take the form of a bivariate normal distribution. At each point in this distribution we plot the expected lifetime fitness of individuals in a third dimension. Or we might represent expected individual fitness as contours on the *y*- by *x*-space. The selection gradient in equation 2.4 is the direction of steepest uphill slope on this *surface of expected individual fitness*, averaged over the bivariate phenotypic distribution. In other words, at each point on the surface of individual fitness we can evaluate the slope or gradient of the surface. If we weight each of these slopes by the proportion of individuals in the population with the appropriate combination of phenotypic values, the weighted average of the slopes turns out to be the selection gradient. The selection gradient in an actual population can be estimated as the slope of a partial regression of individual fit-

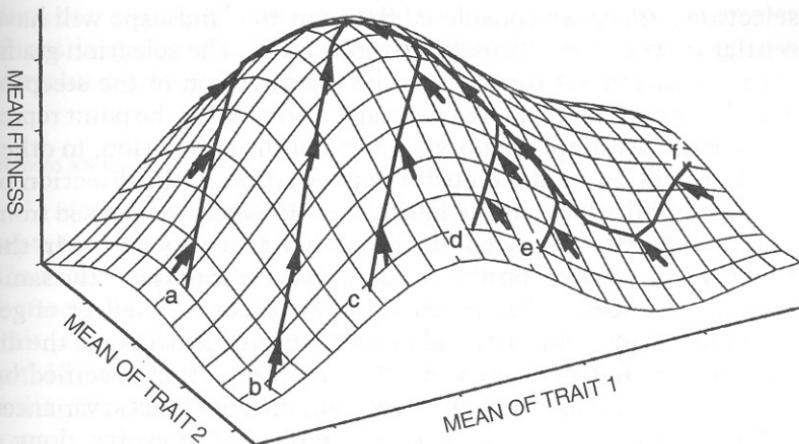


Fig. 2.6 Evolutionary trajectories of populations evolving on a hill-shaped adaptive landscape. The two horizontal axes represent the mean value of two quantitative traits, and the vertical axis represents mean fitness in the population. The genetic correlation between the two traits is strongly positive and is assumed to be constant. The additive genetic variances of the two traits are equal and constant. Heavy lines show evolutionary trajectories from six different starting points (a–f). Arrows show selection gradients (directions of steepest uphill slope) at representative points along the trajectories. Because of the genetic correlation between the traits, generally the evolutionary trajectories are not aligned with the selection gradients. However, selection gradients are aligned with evolutionary paths for the trajectory beginning at *b* and for the common trajectory of paths beginning at *e* and *f*. Those exceptional trajectories are known as the eigenvectors of the dynamical system. (Adapted from Lande 1980b.)

ness on a trait, holding the other traits constant (Lande and Arnold 1983).

The evolutionary movement of the population on an *adaptive landscape* is a useful way of summarizing the results of a quantitative genetic model of long-term phenotypic evolution. The adaptive landscape is closely related to the selection surface we just considered. Instead of plotting the phenotypic values of individuals, we plot the average phenotypic values for the two traits. The bivariate mean of the population is now represented as a point in the *x*- and *y*-space. The evolution of the population can be represented as the movement of the point in the *x*- and *y*-space. Instead of plotting expected individual fitness in the third dimension, we plot average fitness in the population. The resulting plot is called an adaptive landscape (fig. 2.6). It will generally be similar to the

selection surface we considered first, but the landscape will have gentler curvature (Phillips and Arnold 1989). The selection gradient in equation 2.4 turns out to be the direction of the steepest uphill slope on the adaptive landscape, evaluated at the point representing the bivariate phenotypic mean of the population. In other words, selection tends to push the population in the direction of steepest uphill slope on the landscape. However, we can see from equation 2.4 that the population will not generally evolve in the direction of steepest uphill slope unless all the traits have the same genetic variances and zero genetic covariances. The effect of genetic constraints, summarized in the G -matrix, is to distort the direction of evolution away from the uphill direction specified by selection. In general, the population (with unequal genetic variances and/or nonzero genetic covariances) will tend to evolve along a curved trajectory (fig. 2.6), upward on the landscape but not straight toward the highest point of the landscape (Lande 1979). If the adaptive landscape does not change from generation to generation (e.g., if selection is not frequency dependent), a large population will eventually evolve to the highest local point on the landscape, but its trajectory will be more or less circuitous, depending on the constraints embodied in the G -matrix (Lande 1979, 1980b). Another message from equation 2.4 is that the rate of evolution in each generation will be proportional to the strength of selection. In other words, the population will evolve fastest when it is located on the steep flanks of a landscape. The evolutionary rate will gradually decelerate as the population climbs toward a gentle summit, until finally the population stops at the summit. The population is now said to be in evolutionary equilibrium. If the population drifts away from the summit, selection will tend to push it back toward the summit. (See Heisler, chap. 5, especially fig. 5.3, for more discussion of bivariate evolutionary trajectories.)

Thus, models for long-term evolution on an adaptive landscape allow us to visualize the joint operation of many themes in evolutionary biology: selection, inheritance, genetic constraints, maximization of fitness, evolutionary equilibrium, rates and paths of evolutionary change. The effects of small population size (e.g., drift in phenotypic mean) and alternative evolutionary outcomes can be incorporated as well. Because such dynamic models integrate so many themes, they provide a useful framework for quantitative genetic studies of behavior. Dynamic models can be used as a vehicle to connect an inheritance study with a wide variety of evolutionary issues. The possibilities of these multiple connec-

tions can also provide the motivation for mastering the elements of matrix algebra that are required to use the most powerful version of evolutionary genetic theory.

SUMMARY

1. Heritability and the selection differential are the key concepts for predicting genetic response to selection on a single trait. Thus, heritability and the selection differential can be used to predict responses when deliberate selection is imposed on a single trait (e.g., in selection experiments or in plant and animal breeding). In nature, however, selection acts simultaneously on multiple traits, so other parameters of inheritance and selection are needed to predict response to selection.

2. Additive genetic variances and covariances and the selection gradient are the crucial concepts for predicting genetic responses to multivariate selection. This formulation of selection response assumes no covariance between additive genetic and environmental values and no maternal (or paternal) effects.

3. Additive genetic variances and covariances can be estimated by conducting selection experiments or by analyzing variation and covariation in replicated sets of relatives. Offspring-parent sets or sets of paternal half sibs give less ambiguous estimates of genetic variances and covariances than sets of full sibs.

4. Covariance between additive genetic and phenotypic values and the selection gradient are the critical concepts for predicting response to multivariate selection in the presence of parental (maternal and/or paternal) effects. By parental effects we mean the direct effects of parental phenotypes on offspring phenotypes (e.g., parental care that affects offspring size). The basic tools for estimating genetic parameters when parental effects prevail are measurement of traits in parents and offspring, the production of paternal and maternal half sibs, and cross-fostering.

5. When the attributes of parents directly affect the fitness of their offspring, we need an additional concept, parental selection, to predict genetic responses to selection. In addition to the partial regression of offspring fitness on offspring traits (the ordinary or offspring selection gradient), we need to consider the partial regression of offspring fitness on parental traits (the parental selection gradient).

6. Quantitative genetic models of long-term evolutionary response to selection are predicated on the somewhat controversial

assumption that genetic parameters equilibrate in a mutation-selection balance and remain constant. A wide variety of evolutionary processes have been modeled on this equilibration assumption (e.g., sexual selection, allometric evolution, plasticity, and life history evolution). An evolutionary trajectory on an adaptative landscape is the fundamental concept in these models.

7. These quantitative genetic models can also be used as bridges between behavioral inheritance and a wide variety of evolutionary issues.

ACKNOWLEDGMENTS

I am grateful to P. Gowaty and L. Konigsberg for comments on the manuscript, to J. Gladstone for help with graphics, and to M. Kirkpatrick and R. Lande for consultations regarding parental effects and selection. The preparation of this manuscript was supported by NSF Grants BSR 89-06703, BSR 89-18581, and BSR 91-19588.

APPENDIX 2.1 SOME RULES OF MATRIX ALGEBRA

Many texts cover the basic elements of matrix (or linear) algebra (e.g., Campbell 1965; Finkbeiner 1966; Bradley 1975; Searle 1982). Only a few simple rules for matrix operations are reviewed here.

Addition of two column vectors. The addition of two column vectors is similar to the addition of ordinary numbers. Consider the two-trait case of equation 2.1, $\mathbf{z} = \mathbf{a} + \mathbf{e}$, which represents

$$\begin{bmatrix} z_1 \\ z_2 \end{bmatrix} = \begin{bmatrix} a_1 \\ a_2 \end{bmatrix} + \begin{bmatrix} e_1 \\ e_2 \end{bmatrix}.$$

The vector sum is

$$\begin{bmatrix} z_1 \\ z_2 \end{bmatrix} = \begin{bmatrix} a_1 + e_1 \\ a_2 + e_2 \end{bmatrix}.$$

or $z_1 = a_1 + e_1$ and $z_2 = a_2 + e_2$.

Addition of two matrices. Matrix addition is a simple extension of the rule for adding two vectors. Thus, the two-trait case of equation 2.2, $\mathbf{P} = \mathbf{G} + \mathbf{E}$, is

$$\begin{bmatrix} P_{11} & P_{12} \\ P_{12} & P_{22} \end{bmatrix} = \begin{bmatrix} G_{11} & G_{12} \\ G_{12} & G_{22} \end{bmatrix} + \begin{bmatrix} E_{11} & E_{12} \\ E_{12} & E_{22} \end{bmatrix}$$

or

$$\begin{bmatrix} P_{11} & P_{12} \\ P_{12} & P_{22} \end{bmatrix} = \begin{bmatrix} G_{11} + E_{11} & G_{12} + E_{12} \\ G_{12} + E_{12} & G_{22} + E_{22} \end{bmatrix}$$

Thus, the phenotypic variance for trait 1 (P_{11}) is the sum of the trait's additive genetic variance (G_{11}) and its environmental variance (E_{11}).

In general, the lower left-hand element in a 2×2 matrix would not be equal to the upper right-hand element and would be denoted P_{21} , G_{21} , or E_{21} . However, our **P**-, **G**- and **E**-matrices are *symmetric*, which means that $P_{12} = P_{21}$, $P_{13} = P_{31}$, etc.

Multiplying a matrix and a column vector. The basic rule to remember in multiplying a matrix and a vector or two matrices is "rows times columns." For example, consider the two-trait case of equation 2.4, $\Delta\bar{\mathbf{z}} = \mathbf{G}\beta$, which represents

$$\begin{bmatrix} \Delta\bar{z}_1 \\ \Delta\bar{z}_2 \end{bmatrix} = \begin{bmatrix} G_{11} & G_{12} \\ G_{12} & G_{22} \end{bmatrix} \begin{bmatrix} \beta_1 \\ \beta_2 \end{bmatrix}$$

To evaluate the product $\mathbf{G}\beta$, we first multiply the first row of **G** by the column β . The product is the first element in the first row of **G**, G_{11} , times the first element of β , β_1 , plus the second element of the first row of **G**, G_{12} , times the second element of β , β_2 . In other words,

$$\Delta\bar{z}_1 = G_{11}\beta_1 + G_{12}\beta_2.$$

Likewise, the second element of the product $\mathbf{G}\beta$ is

$$\Delta\bar{z}_2 = G_{12}\beta_1 + G_{22}\beta_2.$$

The rule is easily extended to the many-trait case.

Multiplying two matrices. Again, the rule is "rows times columns," but now there is more than one column. Consider the second term in equation 2.11, $\frac{1}{2}\mathbf{C}_{az}\mathbf{M}^T$, in the two-trait case. Before we take the product $\mathbf{C}_{az}\mathbf{M}^T$, we note that \mathbf{M}^T is the so-called *transpose* of the matrix **M**; the rows of **M** are the columns of \mathbf{M}^T . In other words, if

$$\mathbf{M} = \begin{bmatrix} M_{11} & M_{12} \\ M_{21} & M_{22} \end{bmatrix},$$

then

$$\mathbf{M}^T = \begin{bmatrix} M_{11} & M_{21} \\ M_{12} & M_{22} \end{bmatrix},$$

The product $\mathbf{C}_{az}\mathbf{M}^T$ is then

$$\begin{bmatrix} C_{az_{11}} & C_{az_{12}} \\ C_{az_{21}} & C_{az_{22}} \end{bmatrix} \begin{bmatrix} M_{11} & M_{21} \\ M_{12} & M_{22} \end{bmatrix}$$

The upper left-hand element (with subscript 11) of the product is the *first* row of \mathbf{C}_{az} times the *first* column of \mathbf{M}^T ; the upper right-hand element of the product (subscript 12) is the *first* row of \mathbf{C}_{az} times the *second* column of \mathbf{M}^T ; the lower left-hand element (subscript 21) of the product is the *second* row of \mathbf{C}_{az} times the *first* row of \mathbf{M}^T , and finally, the lower right-hand element (22) of the product is the *second* row of \mathbf{C}_{az} times the *second* row of \mathbf{M}^T . Our matrix product is

$$\begin{bmatrix} C_{az_{11}}M_{11} + C_{az_{12}}M_{12} & C_{az_{11}}M_{21} + C_{az_{12}}M_{22} \\ C_{az_{21}}M_{11} + C_{az_{22}}M_{12} & C_{az_{21}}M_{21} + C_{az_{22}}M_{22} \end{bmatrix}$$

The symbol $\frac{1}{2}$ in front of $\mathbf{C}_{az}\mathbf{M}^T$ in equation 2.11 denotes an ordinary number or scalar. Multiplying the scalar $\frac{1}{2}$ times the product $\mathbf{C}_{az}\mathbf{M}^T$ means that each element in $\mathbf{C}_{az}\mathbf{M}^T$ is to be multiplied by $\frac{1}{2}$. Thus,

$$\frac{1}{2}\mathbf{C}_{az}\mathbf{M}^T = \begin{bmatrix} \frac{1}{2}C_{az_{11}}M_{11} + \frac{1}{2}C_{az_{12}}M_{12} & \frac{1}{2}C_{az_{11}}M_{21} + \frac{1}{2}C_{az_{12}}M_{22} \\ \frac{1}{2}C_{az_{21}}M_{11} + \frac{1}{2}C_{az_{22}}M_{12} & \frac{1}{2}C_{az_{21}}M_{21} + \frac{1}{2}C_{az_{22}}M_{22} \end{bmatrix}$$

Matrix multiplication is unlike ordinary or scalar multiplication in that the order of multiplication matters. Notice that $\mathbf{C}_{az}\mathbf{M}^T \neq \mathbf{M}^T\mathbf{C}_{az}$!