

A *quantitative* trait is any well-defined aspect of an organism's morphology, physiology, biochemistry, or behavior that can be characterized on a quantitative scale of measurement (e.g. inches, kilograms, degrees, seconds, micromoles, number of offspring, and so on), and that tends to exhibit a continuous distribution of such measurements, rather than occurring in just a few discrete states. Such traits tend to be *polygenic* (under the control of alleles at many different genetic loci) and they tend to be *influenced by the environment*. The distribution of character states (measurements) within a population is usually *normal* (gaussian or "bell-shaped"), at least after suitable (e.g. logarithmic) transformation of the raw data. Why should this be so?

The reason turns out to be simple and all but inevitable, given mendelian inheritance and random mating. If there are many loci affecting the trait, each with alleles that tend to either increase (+) or decrease (-) the trait, and if these loci tend to segregate independently at meiosis, then it is much more likely that an individual will receive a roughly equal number of + and - alleles from its parents (over the entire set of such loci), than it is that the individual will receive predominantly + or predominantly - alleles. Since the individual's *genotypic value* (i.e. its expected phenotype given its genotype) amounts to a *sum* of individually small contributions (+ or -) from each of many loci, this sum will obey the statistical "law of large numbers" which states that the distribution of a sum of independently sampled terms tends to become more and more normal as the number of terms in the sum increases. Thus the distribution of genotypic values will tend to be normal. Environmental influences on the development of the trait further "smooth" the resulting overall distribution of phenotypes, since more individuals experience average environments than extreme ones.

How can we study the evolution of such traits? It might seem hopeless, since we can't establish any simple, one-to-one relationships between genotypes and phenotypes. (Indeed, the reason why most genotypic values fall near the mean is that there are so *many* different combinations of + and - alleles that give such sums, but relatively few that give extremely high or low sums.) Fortunately, this terrifying complexity evaporates as soon as we realize that we can ignore the messy details and work at a "macroscopic" level where we directly analyze the genetic and phenotypic *variation* of the population. In this approach the *phenotypic variance* ( $V_P$ ) is "partitioned" (decomposed, subdivided) into *components* that *add up* to the total (observed, phenotypic) variance. The first partition divides the *genetic* ( $V_G$ ) and *environmental* ( $V_E$ ) components; the genetic variance is then subdivided into *additive* ( $V_A$ ), *dominance* ( $V_D$ ), and *interaction* ( $V_I$ ) components.

$$V_P = V_G + V_E = (V_A + V_D + V_I) + V_E .$$

Some of these components can be further subdivided, but these four are usually the ones of greatest interest. They can be estimated by fitting appropriate genetic models to the results of controlled breeding experiments.

"Broad-sense" *heritability* is defined as  $H^2 = V_G/V_P$ , which is the proportion of the total phenotypic variance accounted for by all of the genetic variance components. The non-additive components  $V_D$  and  $V_I$  contribute to the overall phenotypic variation, but they do *not* contribute to the evolutionary *response* to selection. Only  $V_A$ , the *additive* part of the genetic variance, provides a basis for evolution.

"Narrow-sense" heritability is this additive component divided by the total variance:  $h^2 = V_A/V_P$ . It can be understood most easily as the *slope* of the regression line describing the relationship between the phenotypes of offspring and their parents. If the slope of this regression line is  $1/2$ , then a parent one unit above the mean will produce, on average, an offspring one *half* unit above the mean. If the slope is 0, then a parent's phenotype gives no information about the likely phenotype of the offspring; our best guess as to the offspring's phenotype is simply the population mean.  $V_A$  and  $V_P$  can be estimated from measurements taken directly from a population of parents and offspring, *if* it can be assumed that the *environments* in which offspring develop are *uncorrelated* with those in which their parents developed.

Given this geometric interpretation of  $h^2$ , it follows at once that  $R = h^2S$ , where  $R$  is the *response to selection* (= mean phenotype of the offspring generation - mean phenotype of the parental generation), and  $S$  is the *selection differential* (= mean phenotype of reproductively successful or "selected" parents - mean phenotype of the entire parental generation). If there is no narrow-sense heritability for a trait, then selection can have no effect on the population's mean phenotype.

Selection tends to *deplete* the genetic variance components (in particular the additive component), by pulling some alleles to very high frequencies and thus pushing others to very low frequencies, and this *decreases* the selected trait's heritability. This implies that at an evolutionary equilibrium, there should be little or no *additive* genetic variance for *fitness effects* associated with the trait, although there might be some dominance, interaction, and environmental variance that affects fitness, and additive variation for aspects of the phenotype with little effect on fitness. *Fitness* itself is a quantitative trait, so we expect its narrow-sense heritability to be low, most of the time, since selection is constantly and strongly favoring increased fitness!

Changes in the environment can alter *both* the environmental ( $V_E$ ) and genetic ( $V_G$ ) variance components, thereby increasing or decreasing the narrow-sense heritability. Thus "heritability" is a property of a *particular population* in a *particular environment*. It is *not* a property of the "trait" itself! This very important fact is often ignored in discussions of human heredity.

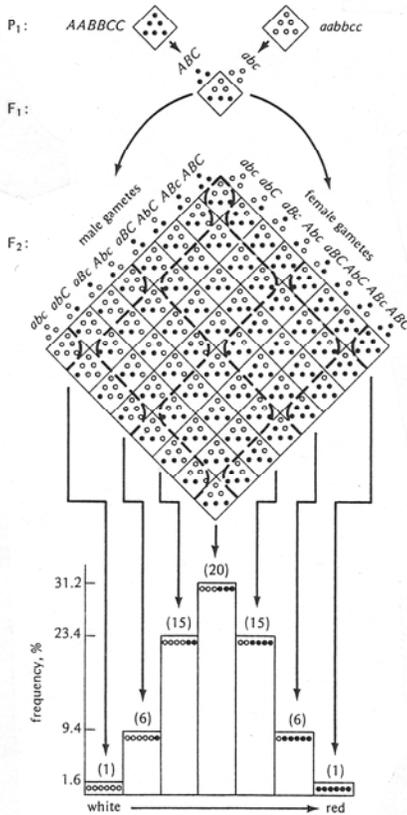


Figure 14-2

Results of crosses between two strains differing in three gene pairs that determine grain color in wheat. The F<sub>2</sub> distribution of color frequencies is shown in the histogram (each black dot represents a red color gene).

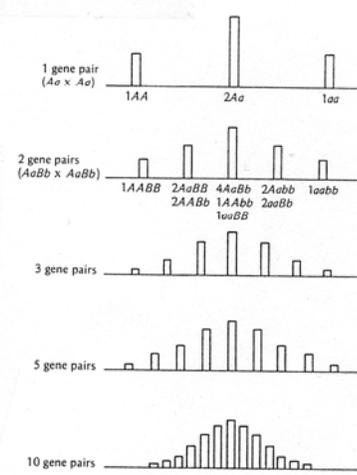


Figure 14-3

Relative frequencies (length of columns) of genotypes produced from crosses between individuals heterozygous for various numbers of independently segregating gene pairs. Illustrations using genes Aa and Bb are given for the top two conditions.

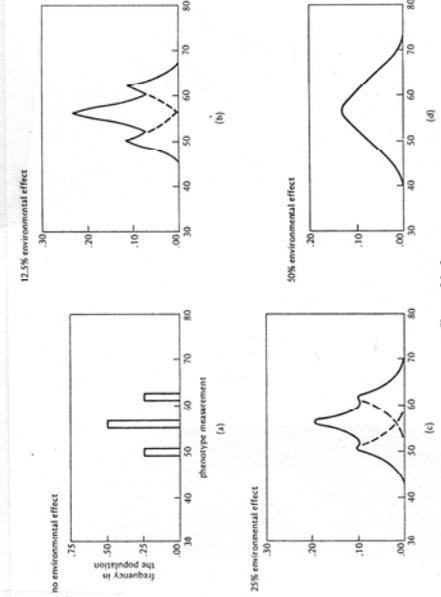
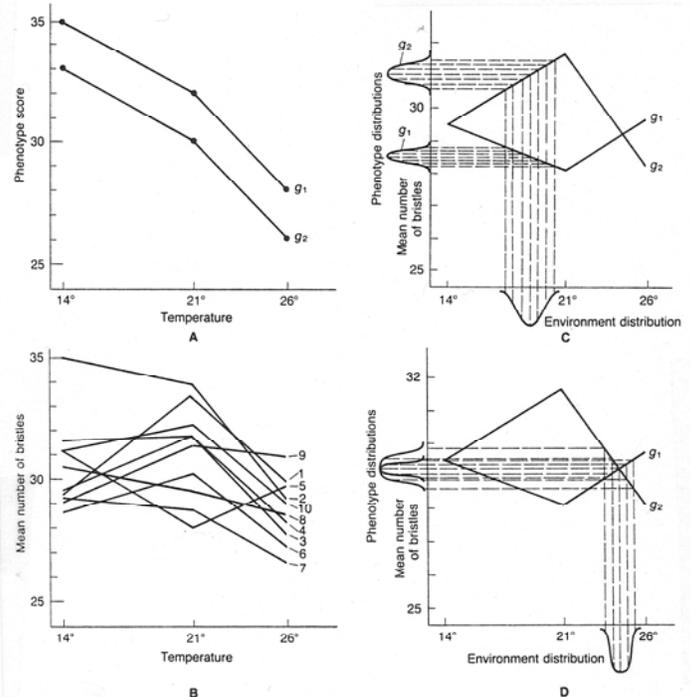


Figure 14-4  
Effect of different degrees of environmental influence on the phenotypic expression of F<sub>2</sub> genotypes produced by segregation of a single gene difference. The two homozygous genotypes in (a) have respective measurements of 50 and 62, and the heterozygotes are exactly intermediate at 56. Environmental difference (or "environmental variance," see Chapter 15) will modify the measurements of a genotype so that the greater this effect, the more varied will be the measurements. For example, homozygous genotypes whose measurements were all at 50 in the absence of an environmental effect (a) will range in measurements from about 43 to 57 when the environmental effect or variance is 25 percent (c). (After Allard.)

FIGURE 3

Genotype × environment interaction. (A) An idealized, hypothetical case in which there is no interaction between genotype and environment. At higher temperatures, each genotype has a lower phenotypic score (e.g., number of bristles), but the two genotypes, although different in phenotype, respond similarly to the environment. (B) The mean number of bristles on sternites 4 and 5 of the abdomen of male *Drosophila pseudoobscura* of 10 genotypes, reared at three temperatures. The genotypes respond differently to temperature, so there is an interaction between genotype and environment. (C) The curves from genotypes 5 and 10 in (B) are shown to illustrate the distribution of phenotypes that might be observed in a population of these two genotypes if the flies developed under a range of temperatures from about 17°–20°. Most of the phenotypic variation would be genetic, because at these temperatures the genotypes differ greatly in phenotype. (D) As for (C), but the environment varies from about 23°–25°. The population is genetically the same as in (C), and the amount of variation in the environment is similar, but most of the phenotypic variation would be environmental because the genotypes have similar responses at these temperatures. (From Gupta and Lewontin 1982)



**Supplement to Quantitative Characters I**

**The simplest polygenic model: one locus, two alleles, no environment!** Let the quantitative phenotype ( $x$ ) be +1, 0, and -1 in individuals of genotypes **AA**, **Aa**, and **aa**, respectively. Then the *mean* phenotype is

$$p^2(1) + 2pq(0) + q^2(-1) = p^2 - q^2 = 2p - 1$$

and the *variance* is

$$p^2(1 - (2p-1))^2 + 2pq(0 - (2p-1))^2 + q^2(-1 - (2p-1))^2 = 2p(1-p)$$

**The next simplest model: trait controlled by two such loci.** Now add a second locus that also affects  $x$  and in just the same way: **BB**, **Bb**, and **bb** contribute +1, 0 or -1 to  $x$ , on top of whatever is contributed by the A locus. So here are the resulting phenotypes, in the form of a Punnett square.

	<b>BB</b>	<b>Bb</b>	<b>bb</b>
<b>AA</b>	2	1	0
<b>Ab</b>	1	0	-1
<b>aa</b>	0	-1	-2

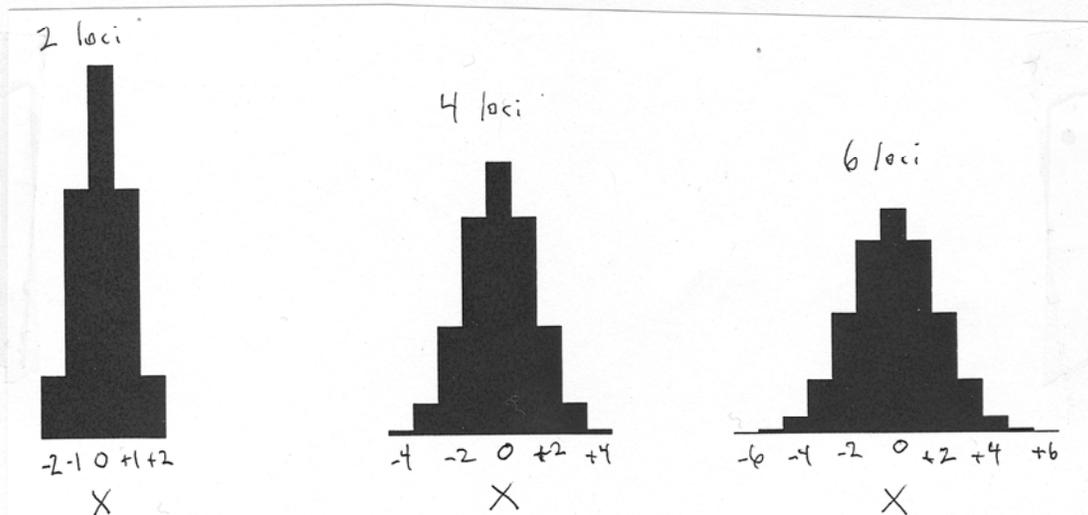
Now what's the mean? If we let  $r$  be the frequency of **B**, and assume that genotypes at the A and B loci are independent of each other (so that the frequency of **AA, BB** individuals is  $p^2r^2$ , and so on), then the mean is just the sum of the average contribution from each locus,

$$(2p - 1) + (2r - 1) = 2p + 2r - 2$$

and the variance is also just the sum of the variances contributed by each locus,

$$2p(1-p) + 2r(1-r) .$$

**Any number of loci: variance components add, and distribution becomes normal.** More generally, this principle of additivity applies to *any number of loci*, as long as the contributions to  $x$  are independent of each other and the genotypes assort randomly. Also, the distribution of *genotypic values* becomes more and more Gaussian or "normal", because there are many ways to have intermediate genotypes but relatively few ways to have extreme genotypes. You can see this even for our simple model with just two loci. Suppose  $p = r = 1/2$ . Then the distribution of genotypic values (and phenotypes) will be as shown on the left. This is already looking rather "normal", and it rapidly becomes more so as the number of loci increases. The middle and right-hand distributions show the same simple model with four and six loci, respectively.



## Some quantitative genetics problems

1 Pig farmers in Illinois would like to reduce the average number of offspring per litter, because pigs from larger litters tend to be of lesser quality. One way to do this is to select for smaller litter sizes. The distribution of litter sizes in a parental population is shown at the right (above). All individuals with litter sizes of 10 or fewer were mated at random among themselves; the resulting distribution of litter sizes among their offspring is shown below the parental distribution. Calculate the mean and variance of litter size for the parental and offspring populations. Determine the selection differential (S), the response to selection (R), and the heritability. Is this *broad-sense* or *narrow-sense* heritability? Which *variance components* can we estimate from these data, and what are their values?

Parents					
		x	x	x	
	x	x	x	x	x
	<hr/>				
	9	10	11	12	13
Offspring					
		x	x		
	x	x	x	x	
	x	x	x	x	
	<hr/>				
	9	10	11	12	

2 A population of *asexually* reproducing slugs shows genetic variance for fecundity (egg number, which translates directly into fitness) in a certain back yard in Seattle. There are, at the moment, three *equally frequent* genotypes in this population, with distributions of fecundity (lifetime egg number) as given in the table. These samples of four individuals are representative of the population as a whole. What is the mean fecundity in *this* generation? What will it be *next* generation? (*Hints*: You must find the heritability and the selection gradient or differential; there are *several* ways to find the heritability; for example, you could figure out the parent-offspring regression by noting that these slugs are *asexual*; what other approaches could you use?) What will the *equilibrium* fecundity be in this population? (Assume that there is no significant immigration or mutation for many generations.) At equilibrium, what is the additive genetic variance? The phenotypic variance?

Genotype	Fecundities
1	150, 170, 190, 210
2	170, 190, 210, 230
3	190, 210, 230, 250

3 Five generations of selection for decreased plasma cholesterol in mice changed the mean from 2.15 mg/100 ml to 2.01 mg/100 ml. The selection differential was -0.07 mg/100 ml. What is the realized heritability?

4 Two inbred varieties of tobacco are crossed and the variance of leaf number among  $F_1$  plants is 1.5. In the  $F_2$  generation the variance is 6.0. What are the genotypic and environmental variance components and the broad-sense heritability in the  $F_2$  generation?

5 In a population of the flour beetle *Tribolium* the mean weight of pupae is 2 mg (2000  $\mu\text{g}$ ). The phenotypic variance is 400  $\mu\text{g}^2$  and the additive genetic variance is 100  $\mu\text{g}^2$ . If individuals with a *mean* pupal weight that is *two phenotypic standard deviations* above the mean are selected as parents, what is the expected average pupal weight among the progeny?

6 How many generations of selection with a selection differential of 20 would be required to increase the average number of eggs laid per hen per year from 180 to 220, given a heritability of 0.20?

7 After a lengthy study of flower-size variation in a population of wild poppies growing in southwestern Utah, you find that flower diameters (equivalent to a measure of petal lengths) are distributed *bimodally*, as shown below. The gap between the two main peaks is real: you measured several flowers on each of hundreds of individual plants. The flowers on any given plant tend to be similar in size, relative to the variation among plants. The bimodality does not result from a sex difference, because all individuals in this species are hermaphrodites. Nor is it some strange effect of the environment, because you get similar distributions when you harvest seeds from adults and grow them up to mature plants in the greenhouse. What's going on here? There are several possibilities. Describe as many as you can think of, and explain how you would *test* them.



8 "In general, we expect the heritabilities of quantitative traits to be *lower* in *small* populations than in large ones." Critically evaluate this assertion. Whether you agree or disagree, *explain your reasoning*.

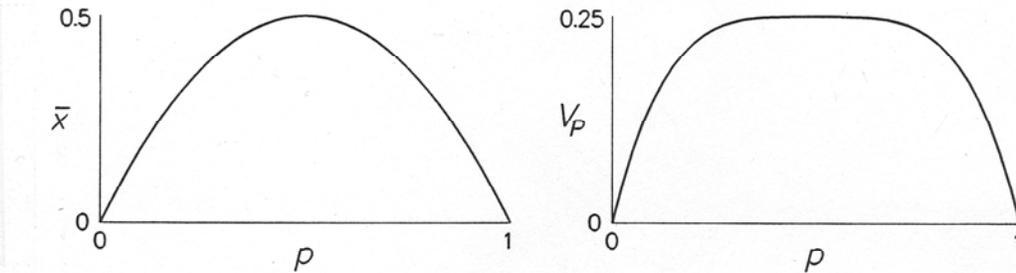
## Quantitative characters II: understanding the variance components

The total phenotypic variance of a quantitative trait ( $V_P$ ) is the sum of two major components, the *variance of genetic effects* on the trait ( $V_G$ ) and the *variance of environmental effects* ( $V_E$ ). These two components are fairly easy to understand intuitively, because we can easily imagine (and even carry out) experiments that distinguish them. For example, we can estimate the environmental variance directly as the phenotypic variance seen in a population of clones or very highly inbred (hence nearly identical) family members. But it's not so easy to grasp the meaning of the variance of *additive* genetic effects ( $V_A$ ), which is the component of  $V_G$  that accounts for the resemblance among relatives and the response to selection. (Recall that  $V_G = V_A + V_D + V_I$ , and  $h^2 = V_A/V_P$ .)

To see that not all genetic variance is additive, consider a trait that is controlled in a symmetrically *overdominant* manner by two alleles at one locus, and assume that there is no environmental variance.

genotype	phenotype ( $x$ )	frequency
AA	0	$p^2$
Aa	1	$2p(1-p)$
aa	0	$(1-p)^2$

Thus  $\bar{x} = 2p(1-p)$ , and  $\text{Var}(x) = V_P = V_G = E(x^2) - [E(x)]^2 = 2p(1-p)[1 - 2p(1-p)]$ .



Now, consider what would happen if we selected for high values of  $x$ . The heterozygotes (Aa) would have the highest fitness, and the gene frequency would quickly approach  $p=1/2$ , where  $\bar{x}$  is maximized and (as it happens), so is  $V_G (=V_P)$ . Selection may continue, but there will be no further response. The "realized" heritability is zero ( $R=h^2S$ ), despite the *abundance* of phenotypic variation caused by genes! Individuals with low values of  $x$  have low values because of their genotypes, but neither of the two *alleles* (A and a) is specifically *associated* with low values of  $x$ , when  $p=1/2$ . Individuals with  $x=0$  have an average gene frequency of  $1/2$ , and so do individuals with  $x=1$ . Thus there is no *additive* effect of these genes on the value of the trait ( $V_A=0$ ). Although the genetic variance is large, it consists entirely of *dominance* variance ( $V_D=V_G=V_P=\text{Var}(x)=0.25$ ). It turns out that  $V_A$  and  $V_D$  can easily be calculated, as shown in detail in many textbooks such as those by Falconer and by Hartl & Clark. We begin by representing the genotypic values as follows.

genotype	phenotype ( $x$ )
AA	$\mu^* + a$
Aa	$\mu^* + d$
aa	$\mu^* - a$

For our particular model (above), this gives  $\mu^*=0$ ,  $a=0$ , and  $d=1$ . With this notation, the general expressions for the two genetic variance components can be shown to be

$$V_A = 2p(1-p)[a + (1-2p)d]^2, \text{ and } V_D = [2p(1-p)d]^2.$$

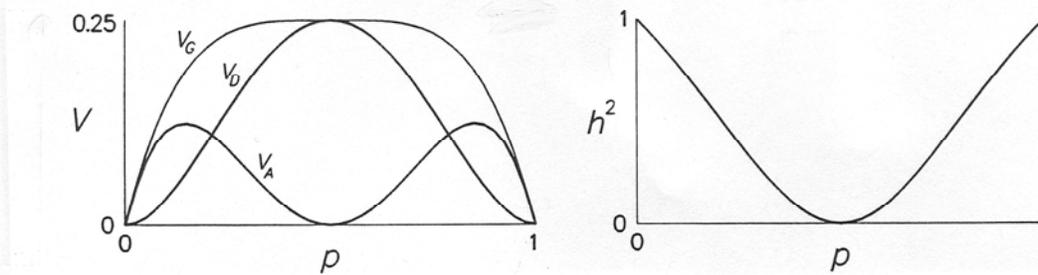
Substituting  $a=0$  and  $d=1$  gives

$$V_A = 2p(1-p)(1-2p)^2, \quad V_D = 4p^2(1-p)^2, \text{ and}$$

$$V_G = V_A + V_D = 2p(1-p)[1-2p(1-p)] [= \text{Var}(x), \text{ from the original derivation, above}].$$

Now we can calculate the heritability, which is

$$h^2 = V_A/V_P = (1-2p)^2/[1-2p(1-p)] .$$



If the *additive* variance is the part that causes (1) a resemblance among relatives and (2) an evolutionary response to selection, then the *parent-offspring regression* should be closely related to the *heritability*. In fact it is  $\frac{1}{2}h^2$ , even in this unusual model. Recall that the regression coefficient is  $b_{yx} = \text{Cov}(x,y)/\text{Var}(x)$ , where  $\text{Cov}(x,y) = E(xy) - E(x)E(y)$ . The parental and offspring variances are just  $V_P = V_G$ , and the means are  $2p(1-p)$ , as derived above. So all we need to do to find the sire-offspring regression is to calculate the covariance.

Since  $x$  and  $y$  are either 0 or 1,  $E(xy)$  is the probability that the sire and his offspring both have phenotypes of 1. (If either is 0, the product  $xy$  is 0.) Both will be 1 when both are heterozygotes. The father is  $Aa$  with probability  $2pq$ , and in that case his offspring is also  $Aa$  with probability  $\frac{1}{2}$ . (No matter which allele the mother may send to the egg, there is a  $\frac{1}{2}$  chance that the father will place the *other* allele in the sperm.) Thus  $E(xy) = p(1-p)$ , and

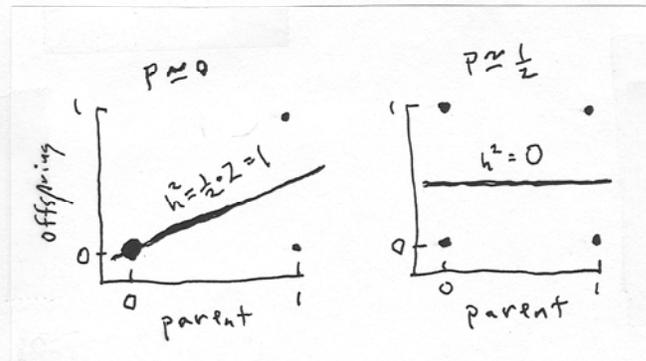
$$\text{Cov}(x,y) = p(1-p) - [2p(1-p)]^2 = p(1-p)(1-2p)^2 = \frac{1}{2}V_A .$$

It follows that

$$b_{yx} = \frac{1}{2}V_A/V_P = \frac{1}{2}h^2 .$$

This means that parents and offspring strongly resemble each other when  $p$  is near 0 or 1, but not when  $p$  is near  $\frac{1}{2}$ . How can we understand this?

Suppose  $a$  is rare. Then most individuals are  $AA$  homozygotes and the mean phenotype is near 0. A heterozygous father (phenotype = 1) will almost always mate with a homozygous  $AA$  mother (phenotype = 0). Half of their offspring will be  $AA$  and half will be  $Aa$  (like him). On average, the offspring of the rare  $Aa$  heterozygotes will have a phenotype of 0.5. But most fathers will be  $AA$  homozygotes who mate with  $AA$  homozygote females and produce exclusively  $AA$  offspring. So the regression slope is  $\frac{1}{2}$  and  $h^2 = 2b = 1$ .



Now suppose  $p = \frac{1}{2}$ . The mean phenotype is  $\frac{1}{2}$  because half of all individuals are  $Aa$  half are homozygotes ( $AA$  or  $aa$ ). No matter who an  $Aa$  father mates, his offspring will be half heterozygotes and half homozygotes, as before, and their mean phenotype will be  $\frac{1}{2}$ . But now *this is no different from the population at large*; knowing the father's phenotype (0 or 1) tells us nothing we didn't already know about the offspring's phenotype! Our best guess is simply " $\frac{1}{2}$ ". This is very different from the situation described above, where fathers with phenotypes of 1 were much more likely to have offspring with phenotypes of 1 than were fathers with phenotypes of 0.

**Moral:** The variance components (and heritability) are functions of the allele frequency  $p$ . Thus heritability is *not* a property of the "trait" itself, but of the trait in a particular *population*. And for traits affected by the environment (as ecologically important quantitative traits always are), the variance components and the heritability are *also* properties of the particular *environment* in which that population developed. This is important because norms of reaction can vary (among genotypes) in complex ways. It sounds odd at first, but in fact a *change in the environment* (holding genotypes constant), can change the *genetic* variance components as well as the *environmental* variance! If you understand the behavior of this simple model, and the figure illustrating norms of reaction for bristle number in flies raised at different temperatures (from a previous handout), then you understand why. Congratulations!

**Quantitative characters III: response to selection in nature**

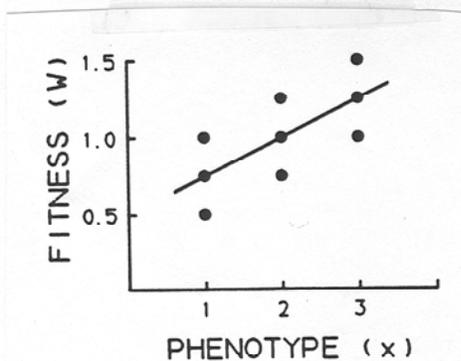
$R = h^2S$  makes obvious sense in an agricultural setting, where the parental generation is divided into two mutually exclusive categories (selected *versus* eliminated). Here we have no problem envisioning (and computing) the "mean phenotype of the selected parents". But how does this equation apply to a natural population, where fitness varies continuously from zero to many surviving offspring?

It turns out that the quantity we need is the mean parental phenotype *weighted by fitness*. That is, instead of merely calculating the mean phenotype  $\bar{x}$  as  $(1/N)\sum x_i$  (which weights each individual equally), we calculate the *weighted mean*  $(1/N)\sum (W_i/\bar{W})x_i$ . The *weight*  $(W_i/\bar{W})$  is the *individual's* fitness relative to the population *mean fitness*. If individuals with large values of  $x$  tend to have relatively high fitnesses, then the weighted mean will be greater than  $\bar{x}$  [=E(x)]. Or, if low values of  $x$  tend to confer high fitness, then the weighted mean will be less than  $\bar{x}$ . And if there is no statistical relationship between  $x$  and  $W$ , then the weighted mean will be equal to the ordinary, unweighted mean,  $\bar{x}$ . Using the language of expectation (and the fact that  $\bar{W}$ , a constant, can be taken outside the summation), we can write the selection differential as

$$S = \bar{x}_S - \bar{x}_U = E \left[ x_i \frac{W_i}{\bar{W}} \right] - \bar{x} = \frac{E(xW) - E(x)E(W)}{\bar{W}} = \frac{Cov(x,W)}{\bar{W}}$$

The direction and magnitude of selection depend on the covariance between phenotype and fitness. The following examples illustrate the weighted-mean-phenotype idea. To keep things simple we will assume that (1) there are just three different values of the phenotype, (2) each phenotype has one of three different fitnesses, (3) each of the nine different combinations of phenotype and fitness is equally frequent, and (4) the fitnesses have already been normalized to a mean of one.

$x$	$W$
1	1/2
1	3/4
1	1
2	3/4
2	1
2	1 1/4
3	1
3	1 1/4
3	1 1/2



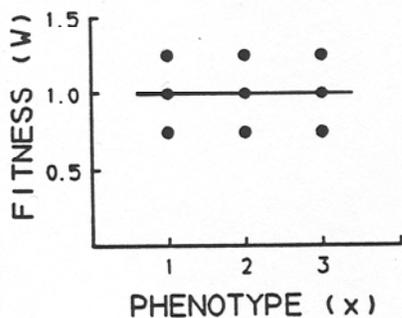
$$\bar{x} = E(x) = 2 \quad \bar{W} = E(W) = 1$$

$$\sum xW = 19.5$$

$$E(xW) = (1/9)\sum xW = 2.167$$

$$S = E(xW)/\bar{W} - E(x) = 2.167 - 2 = 0.167$$

$x$	$W$
1	3/4
1	1
1	1 1/4
2	3/4
2	1
2	1 1/4
3	3/4
3	1
3	1 1/4



$$\bar{x} = E(x) = 2 \quad \bar{W} = E(W) = 1$$

$$\sum xW = 18$$

$$E(xW) = (1/9)\sum xW = 2$$

$$S = E(xW)/\bar{W} - E(x) = 2 - 2 = 0$$

Now we can rewrite the response to selection as

$$\begin{aligned}
 \mathbf{R} &= h^2 \mathbf{S} = (V_A/V_P) \mathbf{S} = (V_A/V_P) \text{Cov}(x, W)/\bar{W} \\
 &= (\sigma_A^2/\sigma_x^2) \text{Cov}(x, W)/\bar{W} \\
 &= \sigma_A^2 [\text{Cov}(x, W)/\sigma_x^2] (1/\bar{W}) \\
 &= \sigma_A^2 b_{Wx} (1/\bar{W}) \equiv \sigma_A^2 \beta = \Delta x
 \end{aligned}$$

The parameter  $\beta$  is often referred to as the *selection gradient*. Notice that when fitnesses have been scaled so that  $\bar{W} = 1$ , then  $\beta$  is just the *regression coefficient* (slope of the line) describing the relationship between fitness and phenotype. Thus  $\Delta x$  (the change in mean phenotype) is equal to the *additive genetic variance* of the trait, times the *regression of fitness on the trait* ( $x$ ).

**Variance and the environment.** The rate of change of a quantitative trait is equal to the product of the selection gradient (which describes how phenotypes and ecology interact to affect fitness) and the additive genetic variance of the trait (which is a function of genotype frequencies, norms of reaction, and states of the environment). Thus both  $\sigma_A^2$  and  $\beta$  are affected by the environment. Selection tends to deplete the additive genetic variance, as alleles contributing to the currently favored version of the trait become fixed at loci controlling its variation.

**Genetic correlations.** If some loci contribute to variation in *two* or more traits, then those traits will be *genetically correlated*. In this case, selection acting on *one* of the traits may change the mean phenotype of the *other(s)*, even if the other trait(s) do not affect fitness and are therefore not direct targets of selection. If selection acts in *opposite* directions on two correlated traits, each will evolve more slowly than we would predict from a knowledge of its  $\sigma_A^2$  and  $\beta$ ; some traits may even evolve *against* the direction of selection. For example, tight linkage between alleles at different loci may give rise to a genetic correlation between the traits they affect, if they are not in linkage equilibrium. In this case the correlation would be expected to decay over time, as recombination restored linkage equilibrium (perhaps with some help from selection). Pleiotropic effects might also be overcome by selection, if the developmental pathways to the traits can be modified so as to uncouple them.

**Unavoidable trade-offs.** Selection in opposite directions in the two sexes can be viewed as the consequence of a genetic correlation, where "fitness of a given genotype in males" and "fitness of the same genotype in females" are the traits in question. In a similar way, the effects of a given phenotype at different times in the life cycle or in different environments can be considered to be different, genetically correlated traits. In such cases the correlation may be difficult or impossible for selection to remove, and the resulting "tradeoff" between the best state for one trait and the best state for another may persist for millions of years, as a fundamental "evolutionary dilemma" faced by many different species, each of which resolves it in a slightly different way. Evolutionary biologists often use such situations to test hypotheses about the factors that tilt the balance between conflicting demands in one direction or another, by comparing related and otherwise ecologically similar species that differ with respect to those factors.

**Problem.** The average bill depth of *Geospiza fortis* (Darwin's medium ground finch) increased by 0.5 mm in one generation (1976 to 1978) in the population studied by Peter Grant and his colleagues. The *phenotypic standard deviation* of this trait is about 1 mm, and its heritability is estimated to be  $h^2 = 0.90$ . Given these numbers, what was the *selection gradient* ( $\beta$ ) during the drought of 1977? What were the *relative fitnesses* of two otherwise equivalent birds with 9-mm and 10-mm bill depths?

