

18

QUANTITATIVE INHERITANCE

STUDY OBJECTIVES

1. To understand the patterns of inheritance of phenotypic traits controlled by many loci 531
2. To investigate the way that geneticists and statisticians describe and analyze normal distributions of phenotypes 535
3. To define and measure heritability, the unit of inheritance of variation in traits controlled by many loci 542

STUDY OUTLINE

Traits Controlled by Many Loci 531

- Two-Locus Control 531
- Three-Locus Control 532
- Multilocus Control 532
- Location of Polygenes 533
- Significance of Polygenic Inheritance 534

Population Statistics 535

- Mean, Variance, and Standard Deviation 536
- Covariance, Correlation, and Regression 539

Polygenic Inheritance in Beans 541

Selection Experiments 541

Heritability 542

- Realized Heritability 542
- Partitioning of the Variance 543
- Measurement of Heritability 544

Quantitative Inheritance in Human Beings 545

- Skin Color 545
- IQ and Other Traits 546

Summary 547

Solved Problems 548

Exercises and Problems 549

Critical Thinking Questions 551

Box 18.1 Mapping Quantitative Trait Loci 537

Box 18.2 Human Behavioral Genetics 547



Much human variation is quantitative.

(© Jim Cummins/FPG International.)

When we talked previously of genetic traits, we were usually discussing traits in which variation is controlled by single genes whose inheritance patterns led to simple ratios. However, many traits, including some of economic importance—such as yields of milk, corn, and beef—exhibit what is called *continuous variation*.

Although some variation occurred in height in Mendel's pea plants, all of them could be scored as either tall or dwarf; there was no overlap. Using the same methods that Mendel used, we can look at ear length in corn (fig. 18.1). With Mendel's peas, all of the F_1 were tall. In a cross between corn plants with long and short ears, all of the F_1 plants have ears intermediate in length between the parents. When both pea and corn F_1 plants are self-fertilized, the results are again different. In the F_2 generation, Mendel obtained exactly the same height categories (tall and dwarf) as in the parental generation. Only the ratio was different—3:1.

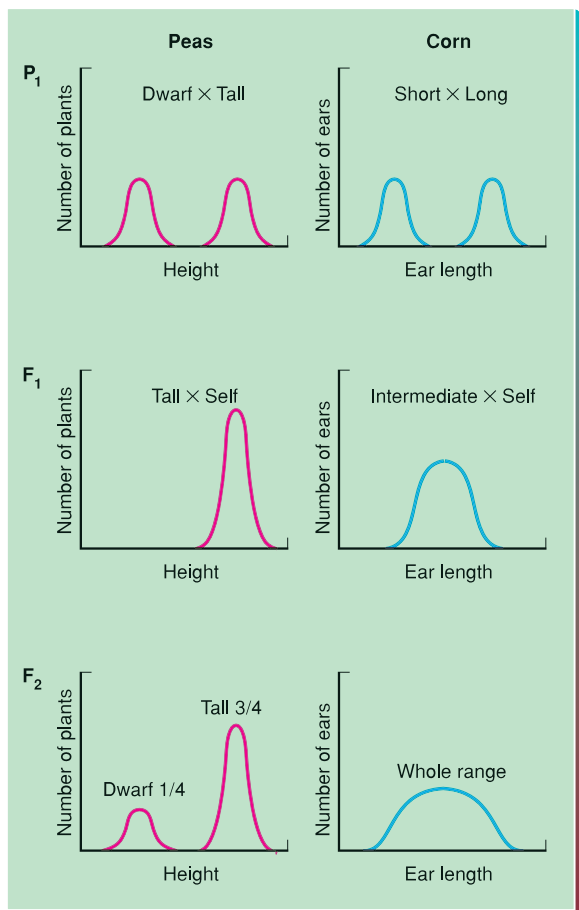


Figure 18.1 Comparison of continuous variation (ear length in corn) with discontinuous variation (height in peas).

In corn, however, ears of every length, from the shortest to the longest, are found in the F_2 ; there are no discrete categories. A genetically controlled trait exhibiting this type of variation is usually controlled by many loci. In this chapter, we study this type of variation by looking at traits controlled by progressively more loci. We then turn to the concept of heritability, which is used as a statistical tool to evaluate the genetic control of traits determined by many loci.

TRAITS CONTROLLED BY MANY LOCI

Let us begin by considering grain color in wheat. When a particular strain of wheat having red grain is crossed with another strain having white grain, all the F_1 plants have kernels intermediate in color. When these plants are self-fertilized, the ratio of kernels in the F_2 is 1 red:2 intermediate:1 white (fig. 18.2). This is inheritance involving one locus with two alleles. The white allele, a , produces no pigment (which results in the background color, white); the red allele, A , produces red pigment. The F_1 heterozygote, Aa , is intermediate (incomplete dominance). When this monohybrid is self-fertilized, the typical 1:2:1 ratio results. (For simplicity, we use dominant-recessive allele designations, A and a . Keep in mind, however, that the heterozygote is intermediate in color.)

Two-Locus Control

Now let us examine the same kind of cross using two other stocks of wheat with red and white kernels. Here, when the resulting intermediate (medium-red) F_1 are self-fertilized, five color classes of kernels emerge in a ratio of 1 dark red:4 medium dark red:6 medium red:4 light red:1 white (fig. 18.3). The offspring ratio, in sixteenths, comes from the self-fertilization of a dihybrid in which the two loci are unlinked. In this case, both loci affect the same trait in the same way. In figure 18.3, each capital

P_1	Red	×	White
	AA		aa
F_1	Intermediate color		
	Aa		
F_2	Red	:	Intermediate
	AA	:	Aa
		:	White
			aa
	1 : 2 : 1		

Figure 18.2 Cross involving the grain color of wheat in which one locus is segregating.

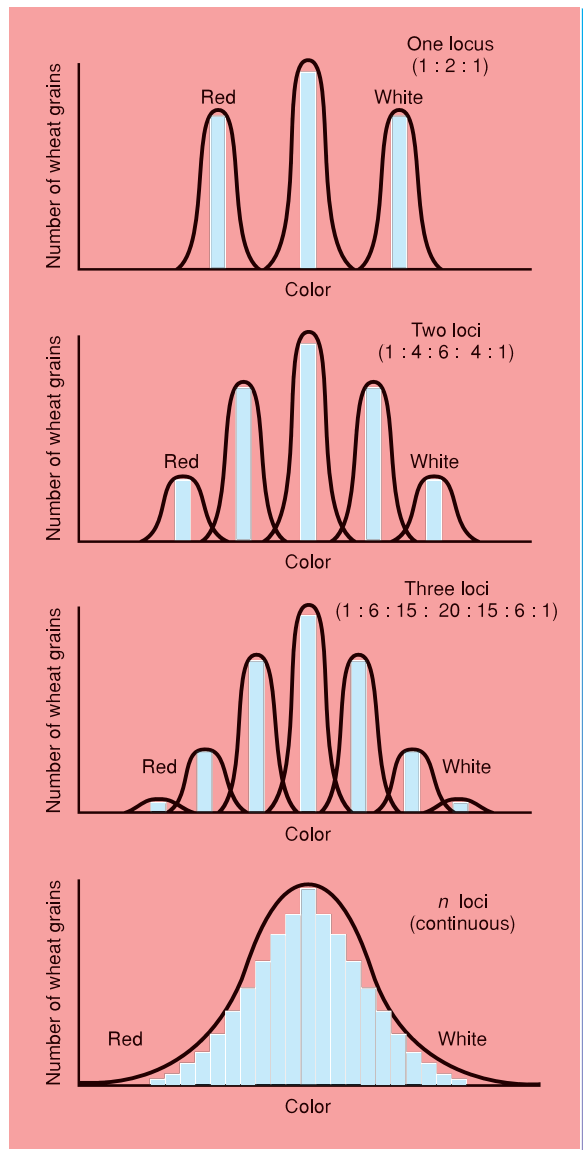


Figure 18.5 The change in shape of the distribution as increasing numbers of independent loci control grain color in wheat. If each locus is segregating two alleles, with each allele affecting the same trait, eventually a continuous distribution will be generated in the F_2 generation.

different color classes down to the level of the eye's ability to perceive differences in wavelengths of light. In fact, we rapidly lose the ability to assign unique color classes to genotypes because the variation within each genotype soon causes the phenotypes to overlap. For example, with three loci, a color somewhat lighter than medium dark red may belong to the medium-dark-red class with

three color alleles, or it may belong to the medium-red class with only two color alleles (fig. 18.5).

The variation within each genotype is due to the environment—that is, two organisms with the same genotype may not necessarily be identical in color because nutrition, physiological state, and many other variables influence the phenotype. Figure 18.6 shows that it is possible for the environment to obscure genotypes even in a one-locus, two-allele system. That is, a height of 17 cm could result in the F_2 from either the aa or Aa genotype in the figure when there is excessive variation (fig. 18.6, column 3). In the other two cases in figure 18.6, there would be virtually no organisms 17 cm tall. Systems such as those we are considering, in which each allele contributes a small unit to the phenotype, are easily influenced by the environment, with the result that the distribution of phenotypes approaches the bell-shaped curve seen at the bottom of figure 18.5.

Thus, phenotypes determined by multiple loci with alleles that contribute dosages to the phenotype will approach a continuous distribution. This type of trait is said to exhibit **continuous, quantitative, or metrical variation**. The inheritance pattern is **polygenic** or **quantitative**. The system is termed an **additive model** because each allele adds a certain amount to the phenotype.

From the three wheat examples just discussed, we can generalize to systems with more than three polygenic loci, each segregating two alleles. From table 18.1, we can predict the distribution of genotypes and phenotypes expected from an additive model with any number of unlinked loci segregating two alleles each. This table is useful when we seek to estimate how many loci are producing a quantitative trait, assuming it is possible to distinguish the various phenotypic classes. For example, when a strain of heavy mice was crossed with a lighter strain, the F_1 were of intermediate weight. When these F_1 were interbred, a continuous distribution of adult weights appeared in the F_2 generation. Since only about one mouse in 250 was as heavy as the heavy parent stock, we could guess that if an additive model holds, then four loci are segregating. This is because we expect $1/(4)^n$ to be as extreme as either parent; one in 250 is roughly $1/(4)^4 = 1/256$.

Location of Polygenes

The fact that traits with continuous variation can be controlled by genes dispersed over the whole genome was shown by James Crow, who studied DDT resistance in *Drosophila*. A DDT-resistant strain of flies was created by growing them on increasing concentrations of the insecticide. Crow then systematically tested each chromosome for the amount of resistance it conferred. Susceptible flies were mated with resistant flies, and the sons from this cross were backcrossed. Offspring were

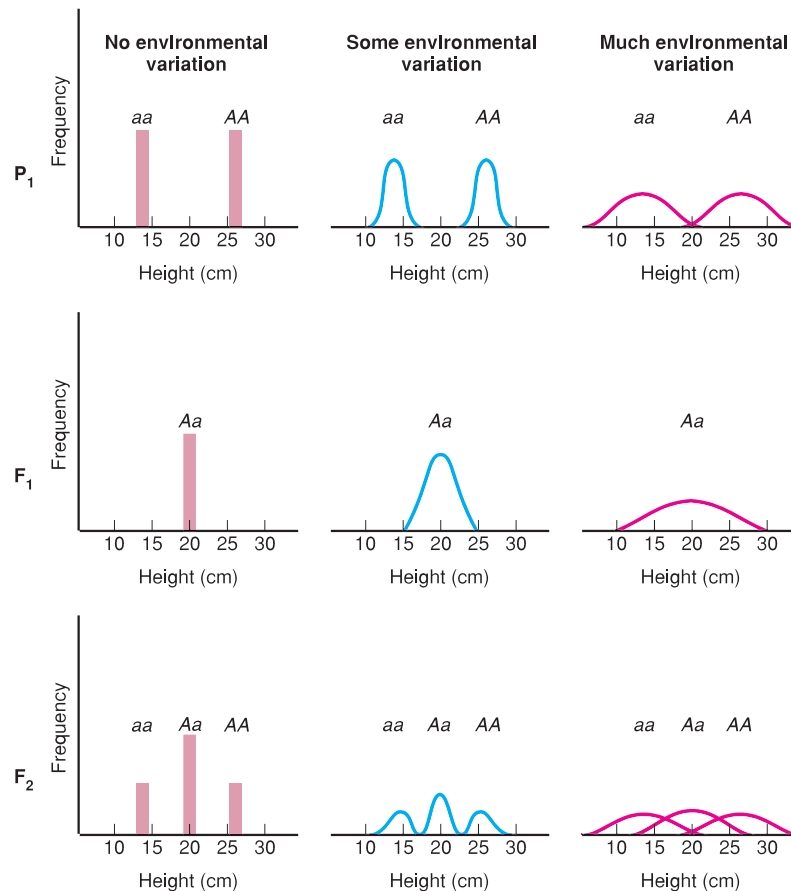


Figure 18.6 Influence of environment on phenotypic distributions.



James F. Crow (1916–).
(Courtesy of Dr. James F. Crow.)

then scored for the particular resistant chromosomes they contained (each chromosome had a visible marker) and were tested for their resistance to DDT. Sons were used in the backcross because there is no crossing over in males. Therefore, the sons would pass resistant and susceptible chromosomes on intact. Crow's results are shown in figure 18.7. As you can see, each chromosome has the potential to increase the fly's resistance to DDT. Thus, each chromosome contains

loci (polygenes) that contribute to the phenotype of this additive trait (box 18.1).

Significance of Polygenic Inheritance

The concept of additive traits is of great importance to genetic theory because it demonstrates that Mendelian rules of inheritance can explain traits that have a continuous distribution—that is, Mendel's rules for discrete characteristics also hold for quantitative traits. Additive traits are also of practical interest. Many agricultural products, both plant and animal, exhibit polygenic inheritance, including milk production and fruit and vegetable yield. In addition, many human traits, such as height and IQ, appear to be polygenic, although with substantial environmental components.

Historically, the study of quantitative traits began before the rediscovery of Mendel's work at the turn of the century. In fact, biologists in the early part of this century debated as to whether the "Mendelians" were correct or whether the "biometricians" were correct in regard to

Table 18.1 Generalities from an Additive Model of Polygenic Inheritance

	One Locus	Two Loci	Three Loci	<i>n</i> Loci
Number of gamete types produced by an F ₁ multihybrid	2 (<i>A, a</i>)	4 (<i>AB, Ab, aB, ab</i>)	8 (<i>ABC, ABc, AbC, Abc, aBC, aBc, abC, abc</i>)	2 ^{<i>n</i>}
Number of different F ₂ genotypes	3 (<i>AA, Aa, aa</i>)	9 (<i>AABB, AABb, AAbb, AaBB, AaBb, Aabb, aaBB, aaBb, aabb</i>)	27 (<i>AABBCC, AABBCCc, AABBcc, AABbCC, AABbCc, AABbcc, AAbbCC, AAbbCc, AAbbcc, AaBBCC, AaBBCCc, AaBBcc, AaBbCC, AaBbCc, AaBbcc, AabbCC, AabbCc, Aabbcc, aaBBCC, aaBBCCc, aaBBcc, aaBbCC, aaBbCc, aaBbcc, aabbCC, aabbCc, aabbcc</i>)	3 ^{<i>n</i>}
Number of different F ₂ phenotypes	3	5	7	2 <i>n</i> + 1
Number of F ₂ as extreme as one parent or the other	1/4 (<i>AA</i> or <i>aa</i>)	1/16 (<i>AABB</i> or <i>aabb</i>)	1/64 (<i>AABBCC</i> or <i>aabbcc</i>)	1/4 ^{<i>n</i>}
Distribution pattern of F ₂ phenotypes	1:2:1	1:4:6:4:1	1:6:15:20:15:6:1	(<i>A + a</i>) ^{2<i>n</i>}

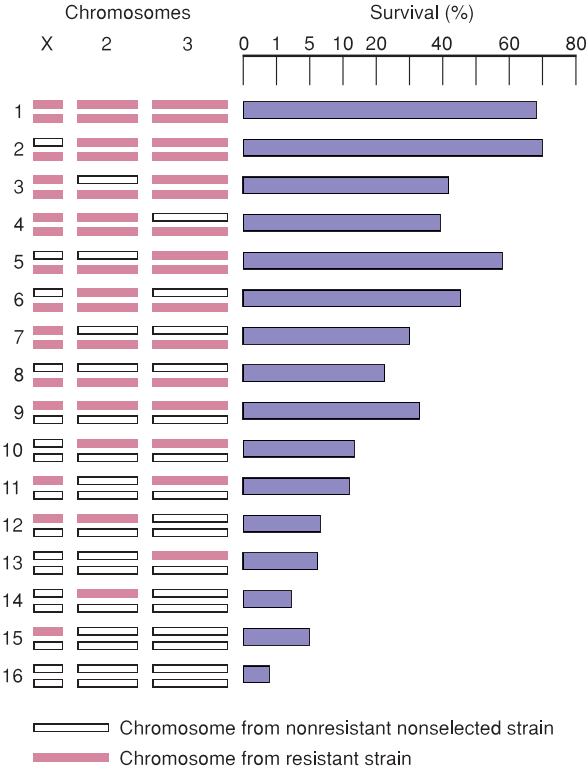


Figure 18.7 Survival of *Drosophila* in the presence of DDT. Numbers and arrangements of DDT-resistant and susceptible chromosomes vary. (Reproduced with permission from the *Annual Review of Entomology*, Volume 2, © 1957 by Annual Reviews, Inc.)

the rules of inheritance. Biometricians used statistical techniques to study traits characterized by continuous variation and claimed that single discrete genes were not responsible for the observed inheritance patterns. They were interested in evolutionarily important facets of the phenotype—traits that can change slowly over time. Mendelians claimed that the phenotype was controlled by discrete “genes.” Eventually the Mendelians were proven correct, but the biometricians’ tools were the only ones suitable for studying quantitative traits.

The biometric school was founded by F. Galton and K. Pearson, who showed that many quantitative traits, such as height, were inherited. They invented the statistical tools of correlation and regression analysis in order to study the inheritance of traits that fall into smooth distributions.

POPULATION STATISTICS

A distribution (see fig. 18.5, bottom) can be described in several ways. One is the formula for the shape of the curve formed by the frequencies within the distribution. A more functional description of a distribution starts by defining its center, or **mean** (fig. 18.8). As we can see from the figure, the mean is not itself enough to describe the distribution. Variation about this mean determines the actual shape of the curve. (We confine our discussion to symmetrical, bell-shaped curves called **normal distributions**. Many distributions approach a normal distribution.)

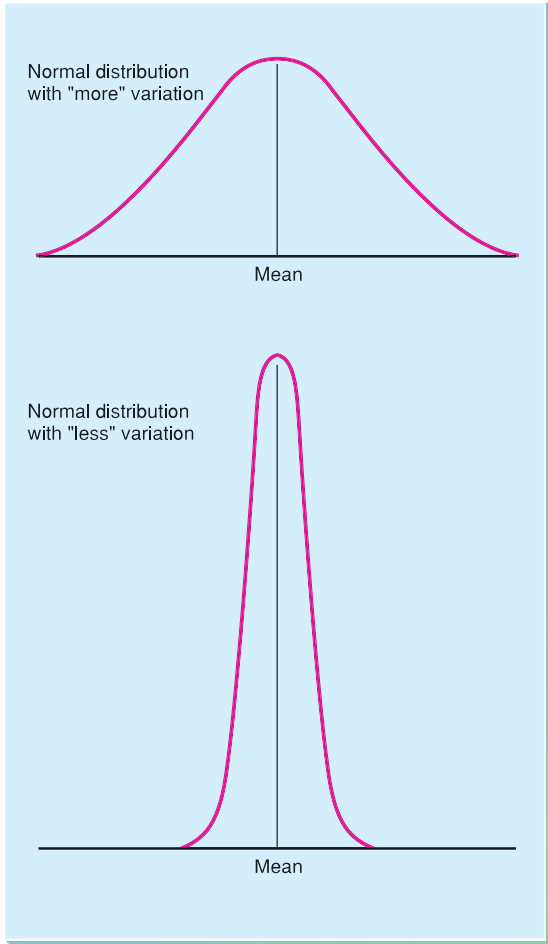


Figure 18.8 Two normal distributions (bell-shaped curves) with the same mean.

Mean, Variance, and Standard Deviation

The mean of a set of numbers is the arithmetic average of the numbers and is defined as

$$\bar{x} = \sum x / n \tag{18.1}$$

in which

- \bar{x} = the mean
- $\sum x$ = the summation of all values
- n = the number of values summed

In table 18.2, the mean is calculated for the distribution shown in figure 18.9. The variation about the mean is calculated as the average squared deviation from the mean:

$$s^2 = V = \frac{\sum(x - \bar{x})^2}{n - 1} \tag{18.2}$$

Table 18.2 Hypothetical Data Set of Ear Lengths (x) Obtained When Corn Is Grown from an Ear of Length 11 cm

x	$(x - \bar{x})$	$(x - \bar{x})^2$
7	-4.12	16.97
8	-3.12	9.73
9	-2.12	4.49
9	-2.12	4.49
10	-1.12	1.25
10	-1.12	1.25
10	-1.12	1.25
10	-1.12	1.25
10	-1.12	1.25
10	-1.12	1.25
10	-1.12	1.25
11	-0.12	0.01
11	-0.12	0.01
11	-0.12	0.01
11	-0.12	0.01
11	-0.12	0.01
11	-0.12	0.01
11	-0.12	0.01
11	-0.12	0.01
12	0.88	0.77
12	0.88	0.77
12	0.88	0.77
13	1.88	3.53
13	1.88	3.53
13	1.88	3.53
14	2.88	8.29
14	2.88	8.29
16	4.88	23.81
$\sum x = 278$		$\sum(x - \bar{x})^2 = 96.53$
$n = 25$		
$\bar{x} = \frac{\sum x}{n} = \frac{278}{25} = 11.12$		
$s^2 = V = \frac{\sum(x - \bar{x})^2}{n - 1} = \frac{96.53}{24} = 4.02$		
$s = \sqrt{s^2} = \sqrt{4.02} = 2.0$		

This value (V or s^2) is called the **variance**. Observe that the flatter the distribution is, the greater the variance will be.

The variance is one of the simplest measures we can calculate of variation about the mean. You might wonder why we simply don't calculate an average deviation from the mean rather than an average squared deviation. For example, we could calculate a measure of variation as

$$\frac{\sum(x - \bar{x})}{n - 1}$$

BOX 18.1

Experimental Methods

Mapping Quantitative Trait Loci

Mapping the location of a standard locus is conceptually relatively easy, as we saw in the mapping of the fruit fly genome. We look for associations of phenotypes that don't segregate with simple Mendelian ratios and then map the distance between loci by the proportion of recombinant offspring. However, with quantitative loci we have a problem: We can't do simple mapping because genes contributing to the phenotype are often located across the genome. Thus, a particular continuous phenotype will be controlled by loci linked to numerous other loci, many unlinked to each other. However, with the advent of molecular techniques, it has become feasible to map polygenes.

In chapter 13, we showed how a locus can be discovered and mapped in the human genome (and other genomes) by association with molecular markers. That is, as the Human Genome Project has progressed, we have discovered restriction fragment length polymorphisms (RFLPs) that mark every region of all the chromosomes. Conceptually, there is not

much difference between finding the gene for cystic fibrosis and finding the gene that contributes to a quantitative trait.

In theory, we look at a population of organisms and note various RFLPs or other molecular markers. We then look for the association of a marker and a quantitative trait. If an association exists, we can gain confidence that one or more of the polygenes controlling the trait is located in the chromosomal region near the marker. The closer the polygenes are to the markers, the more reliable our estimates are, because they depend on few crossovers taking place in that population. With many crossovers, the association between a particular marker and a particular effect diminishes. Since we don't know immediately from this method whether the region of interest has one or more polygenic loci, a new term has been coined to indicate that ambiguity. Instead of talking about polygenic loci directly, we talk of **quantitative trait loci**.

For example, consider the search for polygenes associated with geotactic behavior in fruit flies (see fig. 18.13). As selection proceeds, flies in the high and low lines diverge in their geotactic scores. The lines are also becoming homozygous for many loci since only a few parents are chosen to begin each new generation (see chapter 19). Thus, quantitative trait loci can become associated with different molecular markers in each line (fig. 1). If flies from each line are crossed, heterozygotes will be produced of both the markers and the quantitative trait loci. If there is very little crossing over between the two, three classes of F₂ offspring will be produced. These offspring can be grouped according to their RFLPs and then tested for their geotactic scores. If, as figure 1 suggests, a relationship exists between a locus influencing geotactic score and an RFLP,

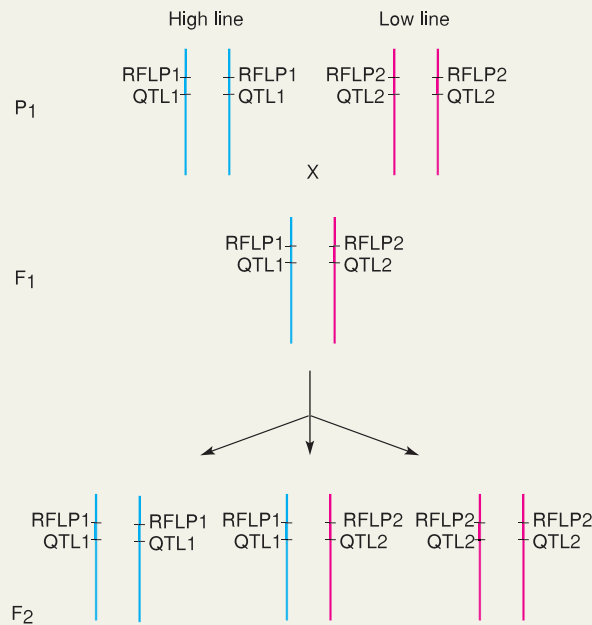


Figure 1 Mapping a quantitative trait locus (QTL) to a particular chromosomal region using a restriction fragment length polymorphism (RFLP) marker. A hypothetical chromosome pair in the fruit fly is shown. The flies have been selected for a geotactic score; QTL1 is the locus in the high line, and QTL2 is the locus in the low line. RFLP1 is homozygous in the high line and RFLP2 is homozygous in the low line.

continued

BOX 18.1 CONTINUED

then the three groups will have different geotactic scores. We can then conclude that the region of the chromosome that contains the RFLP also contains a quantitative trait locus. Finding the right RFLP is, of course, a tedious and time-consuming task.

In a recent summary of the literature, Steven Tanksley reported that numerous quantitative trait loci have been mapped in tomatoes, corn, and other organisms. For example, five quantitative trait loci have been mapped in tomatoes for fruit growth, and eleven quantitative trait loci have been mapped in corn for plant height. Enough data seem to be present to recognize an interesting gener-

ality. That is, our definition of an additive model may need to be rethought because it appears that in almost every case studied so far, one or more of the quantitative trait loci account for a major portion of the phenotype, whereas most of the loci had very small effects. Thus, the additive model that assumes that all polygenes contribute equally to the phenotype may be wrong. However, additive models that allow different loci to contribute different degrees to the phenotype are still supported.

Also of value from locating quantitative trait loci is a new ability to estimate the number of loci affecting a quantitative trait. In this chapter, we

use an estimate of extreme F_2 offspring to estimate the number of polygenes. There are other methods, including sophisticated statistical methods, that we will not develop here. Mapping quantitative trait loci gives us a third method, that is, simply counting the number of quantitative trait loci mapped.

As the methods of mapping quantitative trait loci have been developed, they have also been refined. High-resolution techniques under development will help us determine whether quantitative trait loci are, in fact, individual polygenes or clusters of polygenes.

(We will get to why we use $n - 1$ rather than n in the denominator in a moment.) Note, however, that the above measure is zero. By the definition of the mean, the absolute value of the sum of deviations above it is equal to the absolute value of the sum of deviations below it—one is negative and the other is positive. However, by squaring each deviation, as in equation 18.2, we create a relatively simple index—the variance—which is not zero and has useful properties related to the normal distribution.

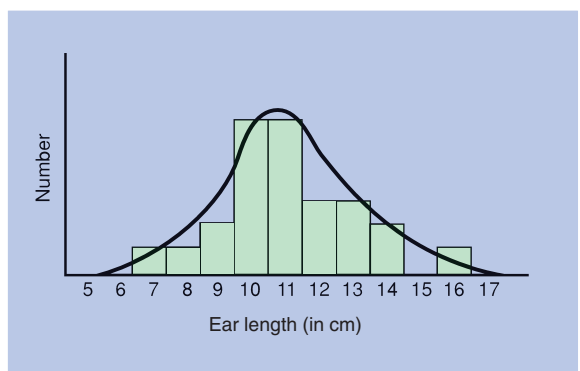


Figure 18.9 Normal distribution of ear lengths in corn. Data are given in table 18.2.

The ear lengths measured in table 18.2 are a sample of all ear lengths in the theoretically infinite population of ears in that variety of corn. Statisticians call sample values **statistics** (and use letters from the Roman alphabet to represent them), whereas they call population values **parameters** (and use Greek letters for them). The sample value is an estimate of the true value for the population. Thus, in the variance formula (equation 18.2), the sample value, V or s^2 , is an estimate of the population variance, σ^2 . When sample values are used to estimate parameters, one degree of freedom is lost for each parameter estimated. To determine the sample variance, we divide not by the sample size, but by the degrees of freedom ($n - 1$ in this case, as defined in chapter 4). The variance for the entire population (assuming we know the population mean, μ , and all the data values) would be calculated by dividing by n . The sample variance is calculated in table 18.2.

The variance has several interesting properties, not the least of which is the fact that it is additive. That is, if we can determine how much a given variable contributes to the total variance, we can subtract that amount of variance from the total, and the remainder is caused by whatever other variables (and their interactions) affect the trait. This property makes the variance extremely important in quantitative genetic theory.

The **standard deviation** is also a measure of variation of a distribution. It is the square root of the variance:

$$s = \sqrt{V} \quad (18.3)$$

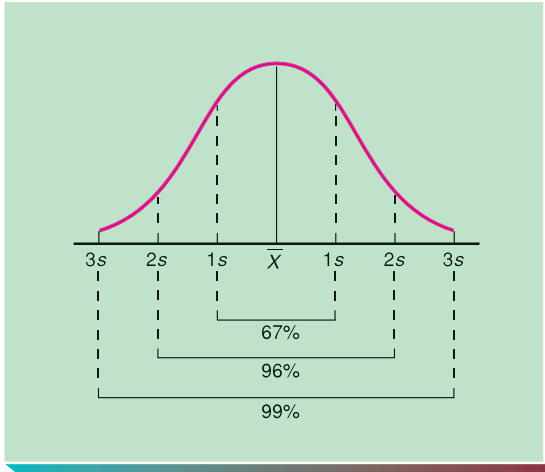


Figure 18.10 Area under the bell-shaped curve. The abscissa is in units of standard deviation (s) around the mean (\bar{x}).

In a normal distribution, approximately 67% of the area of the curve lies within one standard deviation on either side of the mean, 96% lies within two standard deviations, and 99% lies within three standard deviations (fig. 18.10). Thus, for the data in table 18.2, about two-thirds of the population would have ear lengths between 9.12 and 13.12 cm (mean \pm standard deviation).

One final measure of variation about the mean is the **standard error of the mean (SE)**:

$$SE = s/\sqrt{n}$$

The standard error (of the mean) is the standard deviation about the mean of a distribution of sample means. In other words, if we repeated the experiment many times, each time we would generate a mean value. We could then use these mean values as our data points. We would expect the variation among a population of means to be less than among individual values, and it is. Data are often summarized as “the mean \pm SE.” In our example of table 18.2, $SE = 2.0/\sqrt{25} = 2.0/5.0 = 0.4$. We can summarize the data set of table 18.2 as 11.1 ± 0.4 (mean \pm SE).

Covariance, Correlation, and Regression

It is often desirable in genetic studies to know whether a relationship exists between two given characteristics in a series of individuals. For example, is there a relationship between height of a plant and its weight, or between scholastic aptitude and grades, or between a phenotypic measure in parents and their offspring? If one increases, does the other also? An example appears in table 18.3; the same data set is graphed in figure 18.11, in what is referred to as a scatter plot. A relation does appear between

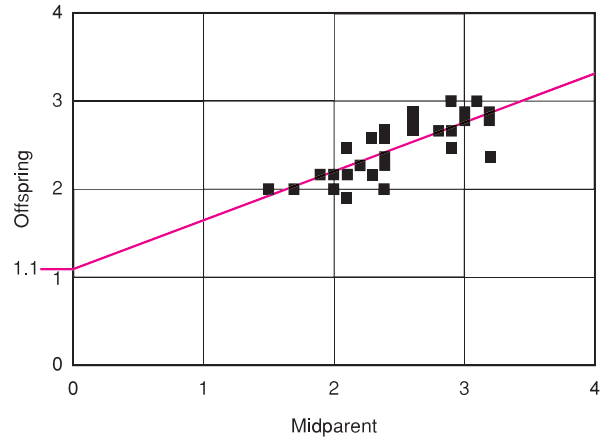


Figure 18.11 The relationship between two variables, parental and offspring wing length in fruit flies, measured in millimeters. *Midparent* refers to the average wing length of the two parents. The line is the statistical regression line. (Source: Data from D. S. Falconer, *Introduction to Quantitative Genetics*, 2d ed. [London: Longman, 1981].)

the two variables. With increasing wing length in midparent (the average of the two parents: x-axis), there is an increase in offspring wing length (y-axis). We can determine how closely the two variables are related by calculating a **correlation coefficient**—an index that goes from -1.0 to $+1.0$, depending on the degree of relationship between the variables. If there is no relation (if the variables are independent), then the correlation coefficient will be zero. If there is perfect correlation, where an increase in one variable is associated with a proportional increase in the other, the coefficient will be $+1.0$. If an increase in one is associated with a proportional decrease in the other, the coefficient will be -1.0 (fig. 18.12). The formula for the correlation coefficient (r) is

$$r = \frac{\text{covariance of } x \text{ and } y}{s_x \cdot s_y} \quad (18.4)$$

where s_x and s_y are the standard deviations of x and y , respectively.

To calculate the correlation coefficient, we need to define and calculate the **covariance** of the two variables, $\text{cov}(x, y)$. The covariance is analogous to the variance, but it involves the simultaneous deviations from the means of both the x and y variables:

$$\text{cov}(x, y) = \frac{\sum(x - \bar{x})(y - \bar{y})}{n - 1} \quad (18.5)$$

The analogy between variance and covariance can be seen by comparing equations 18.5 and 18.2. The variances, standard deviations, and covariance are calculated

in table 18.3, in which the correlation coefficient, r , is 0.78. (There are computational formulas available that substantially cut down on the difficulty of calculating these statistics. If a computer or calculator is used, only the individual data points need to be entered—most computers and many calculators can be programmed to do all the computations.)

Many experiments deal with a situation in which we assume that one variable is dependent on the other (in a cause-and-effect relationship). For example, we may ask, what is the relationship of DDT resistance in *Drosophila* to an increased number of DDT-resistant alleles? With more of these alleles (see fig. 18.7), the DDT resistance of the flies should increase. Number of DDT-resistant alleles is the independent variable, and resistance of the flies is the dependent variable. That is, a fly's resistance is dependent on the number of DDT-resistant alleles it has,

Table 18.3 The Relationship Between Two Variables, x and y (x = the midparent—average of the two parents—in wing length in fruit flies in millimeters; y = the offspring measurement)

x	y	x	y	x	y	x	y
1.5	2	2.2	2.3	2.4	2.7	2.9	2.7
1.7	2	2.3	2.2	2.4	2.7	2.9	2.7
1.9	2.2	2.3	2.6	2.6	2.7	2.9	3
2	2	2.4	2	2.6	2.7	3	2.8
2	2.2	2.4	2.3	2.6	2.8	3	2.8
2	2.2	2.4	2.4	2.6	2.9	3	2.9
2.1	1.9	2.4	2.6	2.8	2.7	3.1	3
2.1	2.2	2.4	2.6	2.8	2.7	3.2	2.4
2.1	2.5	2.4	2.6	2.9	2.5	3.2	2.8
						3.2	2.9
$\Sigma x = 92.7$		$n = 37$		$\Sigma y = 93.2$			
$\bar{x} = \frac{\Sigma x}{n} = 2.51$				$\bar{y} = \frac{\Sigma y}{n} = 2.52$			
$s_x^2 = \frac{\Sigma(x - \bar{x})^2}{n - 1} = 0.19$				$s_y^2 = \frac{\Sigma(y - \bar{y})^2}{n - 1} = 0.10$			
$s_x = \sqrt{s_x^2} = 0.44$				$s_y = \sqrt{s_y^2} = 0.32$			
$\text{cov}(x, y) = \frac{\Sigma(x - \bar{x})(y - \bar{y})}{n - 1} = 0.11$							
$r = \frac{\text{cov}(x, y)}{s_x s_y} = \frac{0.11}{(0.44)(0.32)} = 0.78$							

Source: Data from D. S. Falconer, *Introduction to Quantitative Genetics*, 2d ed. (London: Longman, 1981).

Note: Data are graphed in figure 18.11.

not the other way around. Going back to figure 18.11, we could make the assumption that offspring wing length is dependent on parental wing length. If this were so, a technique called *regression analysis* could be used. This analysis allows us to predict an offspring's wing length (y variable) given a particular midparental wing length (x variable). (It is important to note that regression analysis assumes a cause-and-effect relationship, whereas correlation analysis does not.)

The formula for the straight-line relationship (regression line) between the two variables is $y = a + bx$, where b is the slope of the line (change in y divided by change in x , or $\Delta y/\Delta x$) and a is the y -intercept of the line (see fig. 18.11). To define any line, we need only to calculate the slope, b , and the y intercept, a :

$$b = \text{cov}(x, y)/s_x^2 \tag{18.6}$$

$$a = \bar{y} - b\bar{x} \tag{18.7}$$

Thus equipped, if a cause-and-effect relationship does exist between the two variables, we can predict a y value given any x value. We can either use the formula $y = a + bx$ or graph the regression line and directly determine the y value for any x value. We now continue our examination of the genetics of quantitative traits.

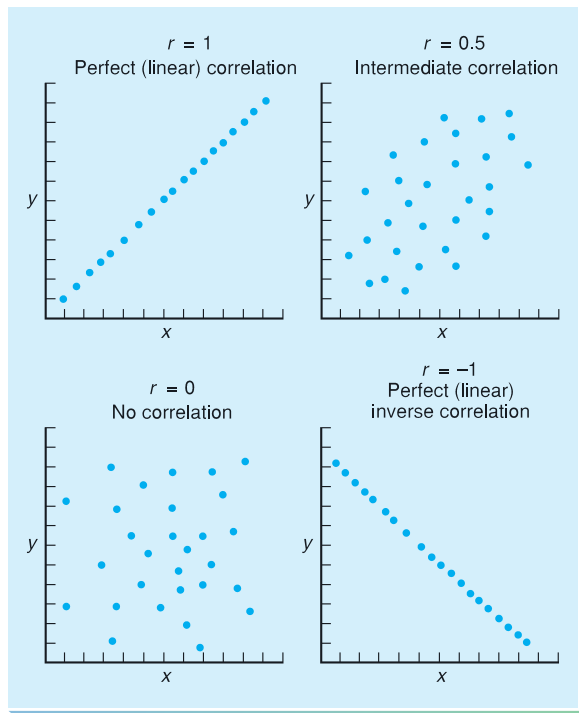


Figure 18.12 Plots showing varying degrees of correlation within data sets.

Table 18.4 Johanssen’s Findings of Relationship Between Bean Weights of Parents and Their Progeny

Weight of Parent Beans	Weight of Progeny Beans (centigrams)																n	Mean ± SE
	15	20	25	30	35	40	45	50	55	60	65	70	75	80	85	90		
65-75				2	3	16	37	71	104	105	75	45	19	12	3	2	494	58.47 ± 0.43
55-65			1	9	14	51	79	103	127	102	66	34	12	6	5		609	54.37 ± 0.41
45-55			4	20	37	101	204	287	234	120	76	34	17	3	1		1,138	51.45 ± 0.27
35-45	5	6	11	36	139	278	498	584	372	213	69	20	4	3			2,238	48.62 ± 0.18
25-35		2	13	37	58	133	189	195	115	71	20	2					835	46.83 ± 0.30
15-25			1	3	12	29	61	38	25	11							180	46.53 ± 0.52
Totals	5	8	30	107	263	608	1,068	1,278	977	622	306	135	52	24	9	2	5,491	50.39 ± 0.13

POLYGENIC INHERITANCE IN BEANS

In 1909, W. Johanssen, who studied seed weight in the dwarf bean plant (*Phaseolus vulgaris*), demonstrated that polygenic traits are controlled by many genes. The parent population was made up of seeds (beans) with a continuous distribution of weights. Johanssen divided this parental group into classes according to weight, planted them, self-fertilized the plants that grew, and weighed the F₁ beans. He found that the parents with the heaviest beans produced the progeny with the heaviest beans, and the parents with the lightest beans produced the progeny with the lightest beans (table 18.4). There was a significant correlation coefficient between parent and progeny bean weight ($r = 0.34 \pm 0.01$). He continued this work by beginning nineteen lines (populations) with beans from various points on the original distribution and selfing each successive generation for the next several years. After a few generations, the means and variances stabilized within each line. That is, when Johanssen chose, within each line, parent plants with heavier-than-average or lighter-than-average seeds, the offspring had the parental mean with the parental variance for seed size. For example, in one line, plants with both the lightest average bean weights (24 centigrams) and plants with the heaviest average bean weights (47 cg) produced offspring with average bean weights of 37 cg. By selfing the plants each generation, Johanssen had made them more and more homozygous, thus lowering the number of segregating polygenes. Therefore, the lines became homozygous for certain of the polygenes (different in each line), and any variation in bean weight was then caused only by the environment. Johanssen thus showed that quantitative traits were under the control of many segregating loci.

SELECTION EXPERIMENTS

Selection experiments are done for several reasons. Plant and animal breeders select the most desirable individuals as parents in order to improve their stock. Population geneticists select specific characteristics for study in order to understand the nature of quantitative genetic control.

For example, *Drosophila* were tested in a fifteen-choice maze for geotactic response (fig. 18.13). The maze was on its side, so at every intersection, a fly had to make a choice between going up or going down. The flies with the highest scores were chosen as parents for the “high” line (positive geotaxis; favored downward direction), and the flies with the lowest score were chosen as parents for the “low” line (negative geotaxis; favored upward direction). The same selection was made for each generation. As time progressed, the two lines diverged quite significantly. This tells us that there is a large genetic component to the response; the experimenters are successfully amassing more of the “downward” alleles in the high line and more of the “upward” alleles in the low line. Several other points emerge from this graph. First, the high and low responses are slightly different, or asymmetrical. The high line responded more quickly, leveled out more quickly, and tended toward the original state more slowly after selection was relaxed. (The relaxation of selection occurred when the parents were a random sample of the adults rather than the extremes for geotactic scores.) The low line responded more slowly and erratically. In addition, the low line returned toward the original state more quickly when selection was relaxed.

The nature of these responses (fig. 18.13) indicates that the high line became more homozygous than the low line. This is shown by the former’s response when selection is relaxed: It has exhausted a good deal of its variability for the polygenes responsible for geotaxis. The low line,

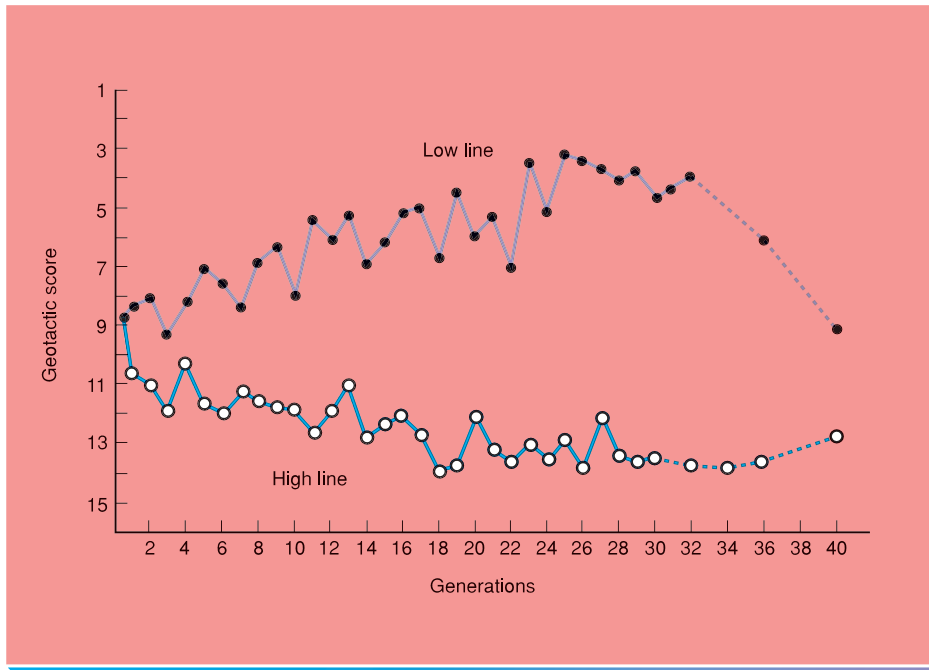


Figure 18.13 Selection for geotaxis. The *dotted lines* represent relaxed selection. (Source: Data from T. Dobzhansky and B. Spassky, "Artificial and natural selection for two behavioral traits in *Drosophila pseudoobscura*," *Proceedings of the National Academy of Sciences, USA*, 62:75–80, 1969.)

however, seems to have much of its original genetic variability, because the relaxation of selection caused the mean score of this line to increase rapidly. It still had enough genetic variability to head back to the original population mean. The response to a selection experiment is one way that plant and animal breeders can predict future response.

HERITABILITY

Plant and animal breeders want to improve the yields of their crops to the greatest degree they can. They must choose the parents of the next generation on the basis of this generation's yields; thus, they are continually performing selection experiments. Breeders run into two economic problems. They cannot pick only the very best to be the next generation's parents because (1) they cannot afford to decrease the size of a crop by using only a very few select parents and (2) they must avoid **inbreeding depression**, which occurs when plants are self-fertilized or animals are bred with close relatives for many generations. After frequent inbreeding, too much homozygosity occurs, and many genes that are slightly or partially deleterious begin to show themselves, depressing vigor and yield. (Chapter 19 presents more on in-

breeding.) Thus, breeders need some index of the potential response to selection so that they can then get the greatest amount of selection with the lowest risk of inbreeding depression.

Realized Heritability

Breeders often calculate a **heritability** estimate, a value that predicts to what extent their selection will be successful. Heritability is defined in the following equation:

$$H = \frac{Y_O - \bar{Y}}{Y_P - \bar{Y}} = \frac{\text{gain}}{\text{selection differential}} \quad (18.8)$$

in which

H = heritability

Y_O = offspring yield

\bar{Y} = mean yield of the population

Y_P = parental yield

From this equation, we can see that heritability is the gain in yield divided by the amount of selection practiced (fig. 18.14). $Y_O - \bar{Y}$ is the improvement over the population average due to $Y_P - \bar{Y}$, which is the amount of difference between the parents and the population average. If there is no gain ($Y_O = \bar{Y}$), then the heritability

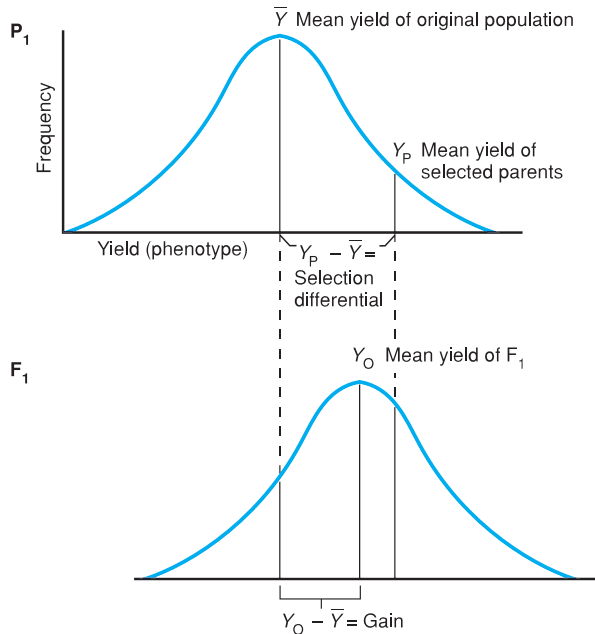


Figure 18.14 Realized heritability is the gain in yield divided by the selection differential when offspring are produced by parents with a mean yield that differs from that of the general population.

will be zero, and breeders will know that no matter how much selection they practice, they will not improve their crops and might as well not waste their time. Since this value is calculated after the breeding has been done, it is referred to as **realized heritability**. Some typical values for realized heritabilities are shown in table 18.5.

The following example may help to clarify the calculation of realized heritability. The number of bristles on the sternopleurite, a thoracic plate in *Drosophila*, is under polygenic control. In a population of flies, the mean bristle number was 6.4. Three pairs of flies served as parents; they had a mean of 7.2 bristles. Their offspring had a mean of 6.6 bristles. Hence, $Y_o = 6.6$, $\bar{Y} = 6.4$, and $Y_p = 7.2$. Dividing the gain by the selection differential—that is, substituting in equation 18.8—gives us

$$H = \frac{6.6 - 6.4}{7.2 - 6.4} = \frac{0.2}{0.8} = 0.25$$

If both a low line and a high line were begun, and if both were carried over several generations, the heritability would be measured by the final difference in means of the high and low lines (gain) divided by the cumulative selection differentials summed for both the high and low lines.

Note from figure 18.13 that the response to selection declines with time as the selected population becomes homozygous for various alleles controlling the trait. As the response declines, the calculated heritability value it-

Table 18.5 Some Realized Heritabilities

Animal	Trait	Heritability
Cattle	Birth weight	0.49
	Milk yield	0.30
Poultry	Body weight	0.31
	Egg production	0.30
	Egg weight	0.60
Swine	Birth weight	0.06
	Growth rate	0.30
	Litter size	0.15
Sheep	Wool length	0.55
	Fleece weight	0.40

self declines. After intense or prolonged selection, heritability may be zero. It does not mean that the trait is not controlled by genes, only that there is no longer a response to selection. Hence, heritability is specific for a particular population at a particular time. Intense selection exhausts the genetic variability, rendering the response to selection, and thus the heritability itself, zero.

Quantitative geneticists treat the realized heritability as an estimate of **true heritability**. True heritability is actually viewed in two different ways: as *heritability in the narrow sense* and *heritability in the broad sense*. We define these on the basis of partitioning of the variance of the quantitative character under study.

Partitioning of the Variance

Given that the variance of a distribution has genetic and environmental causes, and given that the variance is additive, we can construct the following formula:

$$V_{Ph} = V_G + V_E \quad (18.9)$$

in which

V_{Ph} = total phenotypic variance

V_G = variance due to genotype

V_E = variance due to environment

Throughout the rest of this discussion, we will stay with this model. We could construct a more complex variance model if there are interactions between variables. For example, if one genotype responded better in one soil condition than in another soil condition, this environment-genotype interaction would require a separate variance term (V_{GE}).

The variance due to the genotype (V_G) can be further broken down according to the effects of additive polygenes (V_A), dominance (V_D), and epistasis (V_I) to give us a final formula:

$$V_{Ph} = V_A + V_D + V_I + V_E \quad (18.10)$$

We can now define the two commonly used—and often confused—measures of heritability. *Heritability in the narrow sense* is

$$H_N = V_A/V_{Ph} \quad (18.11)$$

This heritability is the proportion of the total phenotypic variance caused by additive genetic effects. It is the heritability of most interest to plant and animal breeders because it predicts the magnitude of the response under selection.

Heritability in the broad sense is

$$H_B = V_G/V_{Ph} \quad (18.12)$$

This heritability is the proportion of the total phenotypic variance caused by all genetic factors, not just additive factors. It measures the extent to which individual differences in a population are caused by genetic differences. This measure is the one most often used by psychologists. We are concerned primarily with H_N , heritability in the narrow sense.

Measurement of Heritability

Three general methods are used to estimate heritability. First, as discussed earlier, we can measure heritability by the response of a population to selection. Second, we can directly estimate the components of variance by minimizing one component; the remaining variance can then be attributed to other causes. For example, by minimizing environmental causes of variance, we can estimate the genetic component directly. Or, by eliminating the genetic causes of variance, we can estimate the environmental component directly. Third, we can measure the similarity between relatives. We look now at the latter two methods.

Variance components can be minimized in several different ways. If we use genetically identical organisms, then the additive, dominance, and epistatic variances are zero, and all that is left is the environmental variance. For example, F. Robertson determined the variance components for the length of the thorax in *Drosophila*. The total variance (V_{Ph}) in a genetically heterogeneous population was 0.366 (measured directly from the distribution of the trait, as in tables 18.2 and 18.3). He then looked at the variance in flies that were genetically homogeneous. These were from isolated lines inbred in the laboratory over many generations to become virtually homozygous. Robertson studied the F_1 in several different matings of inbred lines and found the variance in thorax length to be 0.186 (V_E). By subtraction ($0.366 - 0.186$), we know that the total genetic variance (V_G) was 0.180. From this, we can calculate heritability in the broad sense as

$$H_B = V_G/V_{Ph} = 0.180/0.366 = 0.49$$

To calculate a heritability in the narrow sense, it is necessary to extract the components of the genetic variance, V_G ,

Genetic variance can be measured directly by minimizing the influence of the environment. This is most easily done with plants grown in a greenhouse. Under that circumstance, environmental variables, such as soil quality, water, and sunlight, can be controlled to a very high degree. Hence, the variance among individuals grown under these circumstances is almost all genetic variance. The total phenotypic variance can be obtained from the plants grown under natural circumstances. This allows us to calculate heritability in the broad sense.

Several methods exist to sort out the additive from the dominant and epistatic portions of the genetic variance. The methods rely mostly on correlations between relatives. That is, the expected amount of genetic similarity between certain relatives can be compared with the actual similarity. The expected amount of genetic similarity is the proportion of genes shared; this is a known quantity for any form of relatedness. For example, parents and offspring have half their genes in common. The relation of observed and expected correlations between relatives is a direct measure of heritability in the narrow sense. We can thus define

$$H_N = r_{obs}/r_{exp} \quad (18.13)$$

in which r_{obs} is the observed correlation between the relatives, and r_{exp} is the expected correlation. The expected correlation is simply the proportion of the genes in common.

We must point out that the observed correlation between relatives can be artificially inflated if the environments are not random. Since we know that relatives frequently share similar (or correlated) environments, they may show a phenotypic similarity irrespective of genetic causes. It is important to keep that in mind, especially when we analyze human traits, where it may be almost impossible to rule out or quantify environmental similarity. Hence, r_{obs} may be inflated, which will inflate H_N .

In human beings, finger-ridge counts (fingerprints, fig. 18.15) have a very high heritability; there seems to be very little environmental interference in the embryonic development of the ridges (table 18.6). Monozygotic twins are from the same egg, which divides into two embryos at a very early stage. They have identical genotypes. Dizygotic twins result from the simultaneous fertilization of two eggs. They have the same genetic relationship as siblings. (However, environmental influences may be different; they may be treated differently by relatives and friends.) The data therefore suggest that human finger ridges are almost completely controlled by additive genes with a negligible input from environmental and dominance variation. Few human traits are controlled this simply (table 18.7).

This brief discussion should make it clear that the components of the total variance can be estimated. For a given quantitative trait, the total variance can be mea-

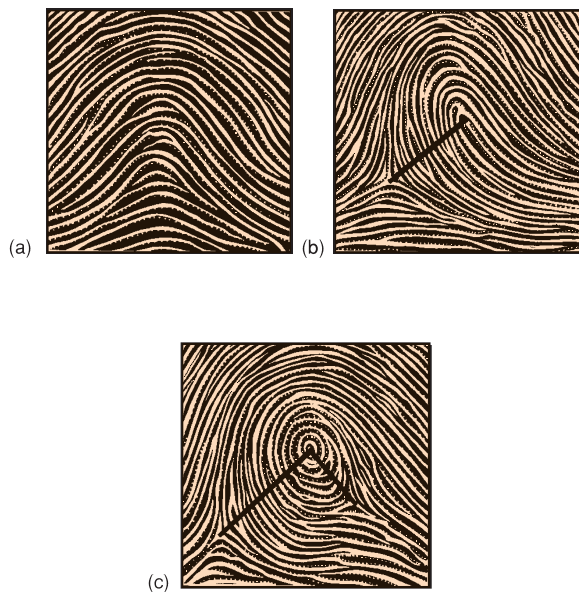


Figure 18.15 The three basic fingerprint patterns. Ridges are counted where they intersect the line connecting a triradius with a loop or whorl center. (a) An arch; there is no triradius; the ridge count is zero. (b) A loop; thirteen ridges. (c) A whorl; there are two triradii and counts of seventeen and eight (the higher one is routinely used). (From Sarah B. Holt, "Quantitative genetics of finger-print patterns," *British Medical Bulletin*, 17. Copyright © 1961 Churchill Livingstone Medical Journals, Edinburgh, Scotland. Reprinted by permission.)

Table 18.6 Correlations Between Relatives, and Heritabilities, for Finger-Ridge Counts

Relationship	r_{obs}	r_{exp}	H_N
Mother-child	0.48	0.50	0.96
Father-child	0.49	0.50	0.98
Siblings	0.50	0.50	1.00
Dizygotic twins	0.49	0.50	0.98
Monozygotic twins	0.95	1.00	0.95

Source: From Sarah B. Holt, "Quantitative genetics of finger-print patterns," *British Medical Bulletin*, 17. Copyright © 1961 Churchill Livingstone Medical Journals, Edinburgh, Scotland. Reprinted by permission.

sured directly. If identical genotypes can be used, then the environmental component of variance can be determined. By correlation of various relatives, it is possible to directly measure heritability in the narrow sense. If heritability is known, and if the total phenotypic variance is known, then all that are left, assuming no interaction, are the dominance and epistatic components. In practice, the epistatic components are usually ignored. Thus, op-

Table 18.7 Some Estimates of Heritabilities (H_N) for Human Traits and Disorders

Trait	Heritability
Schizophrenia	0.85
Diabetes Mellitus	
Early onset	0.35
Late onset	0.70
Asthma	0.80
Cleft Lip	0.76
Heart Disease, Congenital	0.35
Peptic Ulcer	0.37
Depression	0.45
Stature*	1.00+

* A heritability higher than one can be obtained when the correlation among relatives is higher than expected. This is usually the result of dominant alleles.

erationally, all that is left is the dominance variance, obtained by subtracting the additive from the total genetic variance. In addition, plant and animal breeders use sophisticated statistical techniques of covariance and variance analysis, techniques that are beyond our scope.

QUANTITATIVE INHERITANCE IN HUMAN BEINGS

As with most human studies, the measurement of heritability is limited by a lack of certain types of information. We cannot develop pure human lines, nor can we manipulate human beings into various kinds of environments or do selection experiments. However, certain kinds of information are available that allow some estimation of heritabilities.

Skin Color

Skin color is a quantitative human trait for which a simple analysis can be done on naturally occurring matings. Certain groups of people have black skin; other groups do not. Many of these groups breed true in the sense that skin colors stay the same generation after generation within a group; when groups intermarry and produce offspring, the F_1 are intermediate in skin color. In turn, when F_1 individuals intermarry and produce offspring, the skin color of the F_2 is, on the average, about the same as the F_1 , but with more variation (fig. 18.16). The data are consistent with a model of four loci, each segregating two alleles. At each locus, one allele adds a measure of color, whereas the other adds none.

IQ and Other Traits

In human beings, twin studies have been helpful in estimating the heritability of quantitative traits. One way of looking at quantitative traits is by the **concordance** among twins. Concordance means that if one twin has the trait, the other does also. Discordance means one has the trait and the other does not. Table 18.8 shows some concordance values. High concordance of monozygotic as compared with dizygotic twins is another indicator of the heritability of a trait. Concordance values for measles susceptibility and handedness, which are similar for both monozygotic and dizygotic twins, demonstrate the environmental influence on some traits.

Some monozygotic twins (MZ) have been reared apart. The same is true for dizygotic twins (DZ) and nontwin siblings. IQ (intelligence quotient) is a measure of intelligence highly correlated among relatives, indicating a strong genetic component. In three studies of monozygotic twins reared apart, the average correlation in IQ was 0.72. In thirty-four studies of monozygotic twins reared together, the average correlation in IQ was 0.86; dizygotic twins reared together have an average correlation of 0.60 in IQ. Thus, it is clear that there is a genetic influence on IQ. However, experts disagree strongly on the environmental role in shaping IQ and the exact meaning of IQ as a functional measure of intelligence (box 18.2).

At present, twin studies are emerging from the shadow of a scandal involving a knighted British psychologist, Cyril Burt (1883–1971), who did classical twin research on the inheritance of IQ. Burt was posthumously accused of fraud, an accusation that was almost universally accepted and that cast doubt on all of his data and conclusions. More recently, new infor-

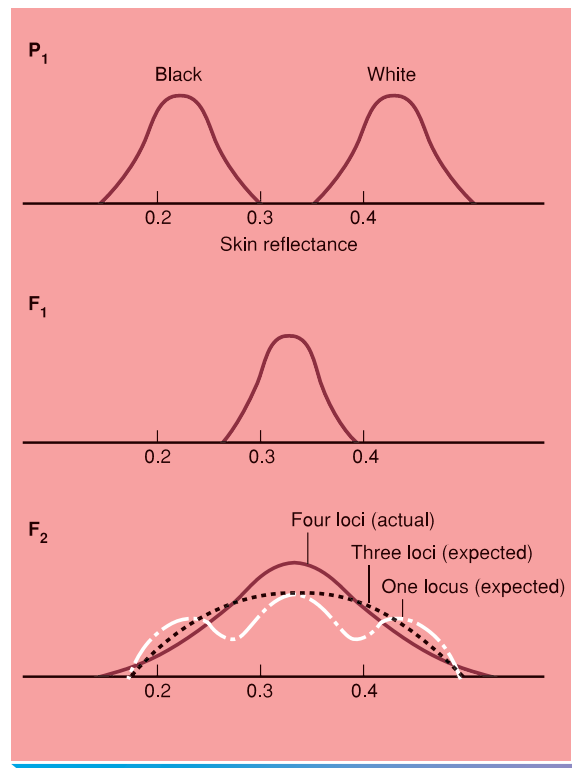


Figure 18.16 Inheritance of skin color in human beings. Four loci are probably involved.

mation seemed to cast doubt on the charges of fraud. These on-again, off-again charges have been a focus of scientific interest.

Table 18.8 Concordance of Traits Between Identical and Fraternal Twins

	Identical (MZ) Twins (%)	Fraternal (DZ) Twins (%)
Hair color	89	22
Eye color	99.6	28
Blood pressure	63	36
Handedness (left or right)	79	77
Measles	95	87
Clubfoot	23	2
Tuberculosis	53	22
Mammary cancer	6	3
Schizophrenia	80	13
Down syndrome	89	7
Spina bifida	72	33
Manic-depression	80	20

BOX 18.2

The study of human behavioral genetics was at first associated with the **eugenics** movement, founded in the late nineteenth century by Francis Galton, one of the founders of quantitative genetics. Eugenics was a movement designed to improve humanity by better breeding. This movement was tainted by bad science done by people with strong prejudices. However, although still controversial, the study of human behavioral genetics is back in vogue. The political climate has changed, and scientific methods to study human behaviors have improved. Even some of the strongest critics against these studies have changed their minds when confronted with the discovery of particular behavioral genes through the mapping of quantitative trait loci. In addition, advocates for people with many human conditions, such as homosexuality and mental illness, feel that if these traits are shown to be genetic in origin, then those who have them will be treated as people with medical conditions rather than as social outcasts.

Ethics and Genetics

Human Behavioral Genetics

Even with better methods and more objective practitioners, the study of human behavioral genetics is still difficult to achieve. Some traits are very poorly defined and may be complex mixtures of phenotypes, such as schizophrenia. Other traits are just difficult to define, such as alcoholism, criminal tendency, and aggressiveness. To make things more complicated, several recent studies that seemed to isolate genes for specific behavioral traits were not verified or were later retracted. In one case, a gene for manic-depressive behavior was isolated in an Amish population. However, when the study was expanded, new cases were discovered that were not linked to the particular marker locus. The result was that a “found” genetic locus was

lost. To their credit, the workers were quick to retract their conclusions.

Currently, there are intriguing results suggesting that divorce, aggression, and dyslexia are under genetic influence. For example, in a recent study, investigators measured a heritability of 0.52 for divorce. This doesn’t mean that “divorce genes” exist, but rather that genes for certain personality traits might predispose a person to divorce. We should make it clear that genetic control does not mean that the environment does not play a role in these traits, just that there are genes that are influential also, sometimes very significantly.

In retrospect, it should not be surprising that genes influence much of our behavior. There are numerous animal studies confirming genetic control of behaviors, indicating that the same would be found in people. As long as the research is done in a competent fashion and the results are not “politicized,” human behavior genetics should not only be a reasonable area of study, but an exciting one as we learn more about ourselves.

SUMMARY

STUDY OBJECTIVE 1: To understand the patterns of inheritance of phenotypic traits controlled by many loci 531–535

Some genetically controlled phenotypes do not fall into discrete categories. This type of variation is referred to as quantitative, continuous, or metrical variation. The genetic control of this variation is referred to as polygenic control. If the number of controlling loci is small, and offspring fall into recognizable classes, it is possible to analyze the genetic control of the phenotypes with standard methods. Polygenes controlling DDT resistance are located on all chromosomes in *Drosophila*.

STUDY OBJECTIVE 2: To investigate the way that geneticists and statisticians describe and analyze normal distributions of phenotypes 535–542

When phenotypes fall into a continuous distribution, the methods of genetic analysis change. We must describe a distribution using means, variances, and standard deviations. Then we must describe the relationship between two variables using variances and correlation coefficients.

STUDY OBJECTIVE 3: To define and measure heritability, the unit of inheritance of variation in traits controlled by many loci 542–547

Equipped with statistical tools, we analyzed the genetic control of continuous traits. The heritability estimates tell us how much of the variation in the distribution of a trait can be attributed to genetic causes. Heritability in the narrow sense is the relative amount of variance due to additive

loci. Heritability in the broad sense is the relative amount of variance due to all genetic components, including dominance and epistasis. In practice, heritability can be calculated as realized heritability—gain divided by selection differential. Estimates of human heritabilities can be constructed from correlations among relatives, concordance and discordance between twins, and studies of monozygotic twins reared apart.

S O L V E D P R O B L E M S

PROBLEM 1: In a certain stock of wheat, grain color is controlled by four loci acting according to an additive model. How many different gametes can a tetrahybrid produce? How many different genotypes will result if tetrahybrids are self-fertilized? What will be the phenotypic distribution of these genotypes?

Answer: Assume the A , B , C , and D loci with A and a , B and b , C and c , and D and d alleles, respectively. A tetrahybrid will have the genotype $Aa Bb Cc Dd$. A gamete can get either allele at each of four independently assorting loci, so there are $2^4 = 16$ different gametes. Three genotypes are possible for each locus, two homozygotes and a heterozygote. Therefore, for four independent loci, there are $3^4 = 81$ different genotypes. Phenotypes are distributed according to the binomial distribution. Thus, there will be a pattern of $(A + a)^{2n} = (A + a)^8$; a ratio of 1:8:28:56:70:56:28:8:1 of phenotypes with decreasing red color from left to right, eight red colors plus white.

PROBLEM 2: In horses, white facial markings are inherited in an additive fashion. These markings are scored on a scale that begins at zero. In a particular population, the average score is 2.2. A group of horses with an average score of 3.4 is selected to be parents of the next generation. The offspring of this group of selected parents have a mean score of 3.1. What is the realized heritability of white facial markings in this herd of horses?

Answer: This is a simple selection experiment; the data fit our equation for realized heritability (equation 18.8). In this case:

$$Y_O = \text{offspring yield} = 3.1$$

$$\bar{Y} = \text{mean yield of the population} = 2.2$$

$$Y_P = \text{parental yield} = 3.4$$

Substituting into equation 18.8:

$$H = \frac{Y_O - \bar{Y}}{Y_P - \bar{Y}} = \frac{3.1 - 2.2}{3.4 - 2.2} = \frac{0.9}{1.2} = 0.75$$

PROBLEM 3: Corn growing in a field in Indiana had a lysine (amino acid) content of 2.0%, with a variance of 0.16. When grown in the greenhouse under controlled and uniform conditions, the mean lysine content was again 2.0%, but the variance was 0.09. What measure of heritability can you calculate?

Answer: We use equation 18.9 for the calculation of heritability by partitioning of the variance ($V_{Ph} = V_G + V_E$). In this case:

$$V_{Ph} = \text{total phenotypic variance} = 0.16$$

$$V_G = \text{variance due to genotype} = 0.09$$

$$V_E = \text{variance due to environment} = ?$$

In the greenhouse, we have minimized environmental variance, meaning the total genotypic variance = 0.09. If we subtract this from the total variance, we get the original environmental variance: $0.16 - 0.09 = 0.07$. Heritability in the broad sense is the genetic variance divided by the total phenotypic variance, or $0.09/0.16 = 0.56$.

E X E R C I S E S A N D P R O B L E M S *

TRAITS CONTROLLED BY MANY LOCI

1. A variety of squash has fruits that weigh about 5 pounds each. In a second variety, the average weight is 2 pounds. When the two varieties are crossed, the F_1 produce fruit with an average weight of 3.5 pounds. When two of these are crossed, their offspring produce a range of fruit weights, from 2 to 5 pounds. Of two hundred offspring, three produce fruits weighing about 5 pounds and three produce fruits about 2 pounds in weight. Approximately how many allelic pairs are involved in the weight difference between the varieties, and approximately how much does each effective gene contribute to the weight?
2. In rabbit variety 1, ear length averages 4 inches. In a second variety, it is 2 inches. Hybrids between the varieties average 3 inches in ear length. When these hybrids are crossed among themselves, the offspring exhibit a much greater variation in ear length, ranging from 2 to 4 inches. Of five hundred F_2 animals, two have ears about 4 inches long, and two have ears about 2 inches long. Approximately how many allelic pairs are involved in determining ear length, and how much does each effective gene seem to contribute to the length of the ear? What do the distributions of P_1 , F_1 , and F_2 probably look like?
3. Assume that height in people depends on four pairs of alleles. How can two persons of moderate height produce children who are much taller than they are? Assume that the environment is exerting a negligible effect.
4. How do polygenes differ from traditional Mendelian genes?
5. If skin color is caused by additive genes, can matings between individuals with intermediate-colored skin produce light-skinned offspring? Can such matings produce dark-skinned offspring? Can matings be-

tween individuals with light skin produce dark-skinned offspring? (See also QUANTITATIVE INHERITANCE IN HUMAN BEINGS)

6. The tabulated data from Emerson and East ("The Inheritance of Quantitative Characters in Maize," 1913, *Univ. Nebraska Agric. Exp. Sta. Bull.*, no. 2) show the results of crosses between two varieties of corn and their F_2 offspring (see the table on ear length in corn). Provide an explanation for these data in terms of number of allelic pairs controlling ear length. Do all the genes involved affect length additively? Explain.
7. In *Drosophila*, a marker strain exists containing dominant alleles that are lethal in the homozygous condition on both chromosome 2, 3, and 4 homologues. These six lethal alleles are within inversions, so there is virtually no crossing over. The strain thus remains perpetually heterozygous for all six loci and therefore all three chromosome pairs. (Geneticists use a shorthand notation in these "balanced-lethal" systems in which only the dominant alleles on a chromosome are shown, with a slash separating the two homologous chromosomes.) The markers are: chromosome 2, Curly and Plum (Cy/Pm , shorthand for $CyPm^+/Cy^+Pm$); chromosome 3, Hairless and stubble (H/S); and chromosome 4, Cell and Minute(4) ($Ce/M[4]$). With this strain, which allows you to follow particular chromosomes by the presence or absence of phenotypic markers, construct crosses to give the strains Crow used (see fig. 18.7) to determine the location of polygenes for DDT resistance.
8. A red-flowered plant is crossed with a yellow-flowered plant to produce F_1 plants with orange flowers. The F_1 offspring are selfed, and they produce plants with flowers in a range of seven different colors. How many genes are probably involved in color production?

Ear Length in Corn (cm)

	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21
Variety P_{60}	4	21	24	8													
Variety P_{54}									3	11	12	15	26	15	10	7	2
F_1					1	12	12	14	17	9	4						
$F_2(F_1 \times F_1)$			1	10	19	26	47	73	68	68	39	25	15	9	1		

* Answers to selected exercises and problems are on page A-20.

9. A plant with a genotype of *aabb* and a height of 40 cm is crossed with a plant with a genotype of *AABB* and a height of 60 cm. If each dominant allele contributes to height additively, what is the expected height of the F_1 progeny?
10. If the F_1 generation in the cross in problem 9 is selfed, what proportion of the F_2 offspring would you expect to be 50 cm tall?
11. Two strains of wheat were compared for the time required to mature. Strain X required fourteen days, and strain Y required twenty-eight days. The strains were crossed, and the F_1 generation was selfed. One hundred F_2 progeny out of 6,200,000 matured in fourteen days or less. How many genes may be involved in maturation?

POPULATION STATISTICS

12. A geneticist wished to know if variation in the number of egg follicles produced by chickens was inherited. As a first step in his experiments, he wished to determine if the number of eggs laid could be used to predict the number of follicles. If this were true, he could then avoid killing the chickens to obtain the data he needed. He obtained the following data from fourteen chickens.

Chicken Number	Eggs Laid	Ovulated Follicles
1	39	37
2	29	34
3	46	52
4	28	26
5	31	32
6	25	25
7	49	55
8	57	65
9	51	44
10	21	25
11	42	45
12	38	26
13	34	29
14	47	30

Calculate a correlation coefficient. Graph the data, and then calculate the slope and y -intercept of the regression line. Draw the regression line on the same graph.

13. The following table (data from Ehrman and Parsons, 1976, *The Genetics of Behavior*; 121, Sunderland, Mass.: Sinauer Associates) gives heights in centimeters of eleven pairs of brothers and sisters. Calculate a correlation coefficient and a heritability. Is this realized heritability, heritability in the broad sense, or heritability in the narrow sense?

Pair	Brother	Sister	Pair	Brother	Sister
1	180	175	7	178	165
2	173	162	8	186	163
3	168	165	9	183	168
4	170	160	10	165	160
5	178	165	11	168	157
6	180	157			

How can environmental factors influence this heritability value? (See also HERITABILITY)

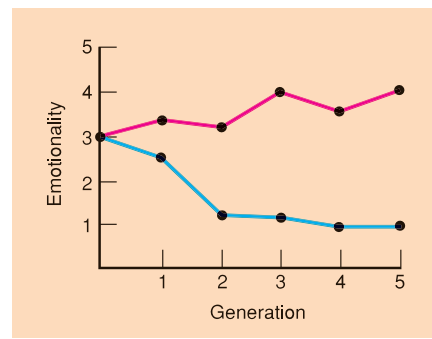
14. You determine the following variance components for leaf width in a particular species of plant:

Additive genetic variance (V_A)	4.0
Dominance genetic variance (V_D)	1.8
Epistatic variance (V_I)	0.5
Environmental variance (V_E)	2.5

Calculate the broad sense and narrow sense heritabilities. (See also HERITABILITY)

SELECTION EXPERIMENTS

15. Psychologists refer to defecation rate in rats as “emotionality.” The data shown in the accompanying figure (data modified from Broadhurst, 1960, *Experiments in Personality*, vol. 1, London: Eysenck) show mean emotionality scores during five generations in high and low selection lines. In the final generation, the parental mean was 4 for the high line and 0.9 for the low line. The cumulative selection differential is 5 for each line. Calculate realized heritability overall, and separate heritabilities for each line. Do these differ? Why? Why was the response to selection asymmetrical? (See also HERITABILITY)



16. Data were gathered during a selection experiment for six-week body weight in mice. Graph these data and calculate a realized heritability. (See also HERITABILITY)

Generation	High Line			Low Line		
	\bar{Y}	Y_P	Y_O	\bar{Y}	Y_P	Y_O
0	21			21		
1		24	22		18	20
2		24	23		18	20
3		26	23		18	20
4		26	24		16	19
5		26	23		16	18

HERITABILITY

- Outstanding athletic ability is often found in several members of a family. Devise a study to determine to what extent athletic ability is inherited. (What is “outstanding athletic ability”?)
- Variations in stature are almost entirely due to heredity. Yet average height has increased substantially since the Middle Ages, and the increase in the height of children of immigrants to the United States, as compared with the height of the immigrants themselves, is especially noteworthy. How can these observations be reconciled?
- Would you expect good nutrition to increase or decrease the heritability of height?
- Two adult plants of a particular species have extreme phenotypes for height (1 foot tall and 5 feet tall), a quantitative trait. If you had only one uniformly lighted greenhouse, how would you determine whether the variation in plant height is environmentally or genetically determined? How would you attempt to estimate the number of allelic pairs that may be involved in controlling this trait?
- The components of variance for two characters of *D. melanogaster* are shown in the following table (data from A. Robertson, “Optimum Group Size in Progeny Testing,” *Biometrics*, 13:442–50, 1957).

Estimate the dominance and epistatic components, and calculate heritabilities in the narrow and broad sense.

Variance Components	Thorax Length	Eggs Laid in Four Days
V_{Ph}	100	100
V_A	43	18
V_E	51	38
$V_D + V_I$?	?

- In a mouse population, the average tail length is 10 cm. Six mice with an average tail length of 15 cm are interbred. The mean tail length in their progeny is 13.5 cm. What is the realized heritability?
- The narrow sense heritability of egg weight in chickens in one coop is 0.5. A farmer selects for heavier eggs by breeding a few chickens with heavier eggs. He finds a difference of 9 g in the mean egg weights of selected and unselected chickens. By how much can he expect egg weight to increase in the selected chickens?
- If, in a population of swine, the narrow sense heritability of maturation weight is 0.15, the phenotypic variance is 100 lb², the total genetic variance is 50 lb², and the epistatic variance is 0, calculate the dominance genetic variance and the environmental variance.
- A group of four-month-old hogs has an average weight of 170 pounds. The average weight of selected breeders is 185 pounds. If the heritability of weight is 40%, what is the expected average weight of the first generation progeny?

QUANTITATIVE INHERITANCE IN HUMAN BEINGS

- Does schizophrenia seem to have a strong genetic component (see table 18.8)? Explain.

C R I T I C A L T H I N K I N G Q U E S T I O N S

- Several cases mentioned in the text reported and then retracted the discovery of human genes controlling specific traits. Barring fraud, what might cause a scientist to retract a study of this type?
- Monozygotic twins share identical genes. Under what conditions could they show discordance of traits?