CHAPTER 11

Quantitative Genetics

ANITA M. COLLINS

I. INTRODUCTION

Quantitative genetics of honey bees has received less attention than other areas of bee genetics. The majority of work has dealt with geographic variation and classification (Ruttner, Chapter 2), population genetics, especially in relation to sex alleles (Cornuet, Chapter 9), and, of course, visible mutations (Tucker, Chapter 3). The study of visible mutants has been a major topic because bee biologists, like Mendel with his peas, used mutants to test the fundamentals of inheritance in honey bees. Crosses between individuals differing in discrete characters produced offspring which had uniform phenotypes. Relationships between offspring and parents were easily seen and described. Results of crosses where the trait examined was not easily divided into clear groups were difficult to interpret. The methodology for analyzing such variation and describing the inheritance of the characters involved was developed in part because of this difficulty. Provine (1971) presented an interesting history of a conflict between the Mendelians and the biometricians early in this century which stimulated the development of statistics as a science. This conflict parented the study of continuously variable characters, called quantitative genetics, which now complements Mendelian genetics.

There is no longer any argument that the biological mechanics of the inheritance of quantitative traits differ in any fundamental way from those that determine discrete traits. The major difference between them is the

number of genes having significant control on their expression. A discrete trait has distinct visible differences that are attributable to a single locus (gene) with several possible variants called alleles. A quantitative trait is controlled by a number of genetic loci, each of which may have many alleles, and possibly significant modifiers at other loci. This multifactorial nature of the inheritance of traits such as tongue length, wing length, weight, honey production, and defensive behavior requires the study of populations of individuals and descriptions of the properties of these traits at the population level. Important measures include the population mean value, the variation, and the relationships between different continuous characters that may be genetically or phenotypically interactive. Such descriptions are more complex than the simple classical Mendelian descriptions of the inheritance of discrete traits.

Quantitative genetic theory describes the biological bases for the changes observed in animal and plant breeding programs. This theory deals with many aspects of populations and their quantitative traits including the structure of hypothetical randomly breeding populations, results of crosses between inbred lines, and mechanisms of selection. For more extensive explanations of this basic theoretical work, the reader is referred to Falconer (1981), Hill (1984a,b), Lush (1945), and Mather (1949). This chapter will present the basic ideas of theoretical quantitative genetics and their application to honey bees.

II. APPROACHING QUANTITATIVE CHARACTERS

A. Measurement

Quantitative traits usually involve many genes, each contributing a small effect. In some cases there are modifier genes at other loci which have indirect, or pleiotropic, effects on the genes directly affecting the character. There is no difference in basic chromosomal mechanics for these two types of genes, although in some cases the genes involved in controlling a continuous character may be rather closely associated on a chromosome (Falconer, 1981). Such groups of many genes affecting a single character are called polygenes.

Many of the characters of honey bees that have economic importance are quantitative. To aid in more clearly defining the underlying genetic complexity, the visible expression of characters, the phenotype, must be clearly described. Some traits, such as morphological ones, are easily measured. Physiological traits, such as hormone or pheromone levels and disease resistance, may require more complex assessment. Behavioral traits, such as

pollen collection and honey production, may require the development of a measurement system that divides the complex behavior into smaller more easily studied parts.

Measurement of quantitative traits has three major requirements. First, the measurement techniques must be appropriate for the characteristic. Second, the scale must be appropriate to show existing variation between individuals. Third, the distribution of the measurements must be normal, falling symmetrically around the population mean, to meet assumptions made in the basic theory. Sometimes this is achieved by measuring in one scale and then recasting the values using a logarithmic or other transformation.

Some traits may be measurable only on certain individuals or at certain times. For example, egg-laying capacity is expressed only by reproductive females, and levels of chemicals such as alarm pheromones vary with age and must be measured at specific times in the development of an individual. Other traits may require an organism's death and dissection to measure (ovariole development in queens or laying workers) or may be evaluated by measurement of close relatives—progeny or siblings. Also, the behavior of social insects is often group behavior rather than individual behavior. Here, measurements must be made on a group of related individuals, either a subsample or the entire colony.

B. Objectives

One of the basic objectives of quantitative genetics is predicting the outcome of a selection or breeding program based on observations of existing populations. Measurements are made on groups of relatives which are used to predict how future offspring will express a character. This process uses estimates of population parameters such as means, variances, and covariances.

A second objective of quantitative genetics is to discover how an organism's phenotype is influenced by its genotype and the environment in which it develops and exists. This can be done by comparing individuals or groups having the same or similar genotypes (genetic constitution) in different environmental conditions. Alternately, individuals or groups with different genotypes can be compared in similar environmental conditions.

The basic relationship underlying the value of such comparisons is expressed in Eq. (1). The phenotype (P) equals the sum of the effect of the genotype (G) and the environment (E).

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

If we could reduce variation due to environmental influences to zero, then

the phenotype would directly reflect the underlying genotype. For some discrete traits this may be true, but it is rarely true for quantitative characters. Theoretically, it is possible to measure a population having a single genotype over all ranges of its normal environment and, by calculating the mean value, have a measure of the actual genotypic value. However, this is not practical, and rough estimates of genotypic value are based on phenotypes expressed in one or only a few environments.

III. RESEMBLANCE—WHY SISTERS ARE ALIKE

A. Average Effect and Breeding Value

There are a number of concepts that are important to understanding genotypic value in the context of a population. First, the *average effect* of a gene is the mean deviation from the population mean of individuals which receive that gene from one parent. Thus it is the mean deviation caused by replacing a single gene in an array of genotypes with one allele of the gene in question. The inclusion of this single allele would change the phenotypes in several specific ways. The average of these changes is the average effect of this gene. Expressed another way, it is the average effect a gene will have against the whole background of possible genotypes in the population.

Not only are we interested in the average effect of a single gene, but we are interested in the complex of genes that an individual can pass on to its offspring. The value associated with this collection of genes carried by an individual and potentially transmitted to its offspring is referred to as the individual's *breeding value.* This is frequently estimated by making measurements on the progeny and assigning the mean value of these measurements as the breeding value of the parent. This is the sum of the average effects of all the genes that this parent carries.

This concept considers only the additive effects of genes. It does not include any interaction between alleles of the same locus or between genes at different loci. In the development of a genetic theory, Mendel looked at single genes or loci occuring in diploid organisms having two alleles, although a locus might have many possible alleles or forms. Quantitative genetics looks at many genes, each of which may have two alleles present at any one time in the standard diploid organism. In honey bees, we must remember that while queens and workers are diploid (arising from fertilized eggs) and will have two alleles for each locus, drones are haploid (arising from unfertilized eggs) and have only one allele present per locus. This must

be taken into account when applying normal diploid quantiative genetics to honey bees.

B. Dominance and Epistasis

There are two possible types of interaction between loci that also influence expression of the phenotype. The interaction between alleles of the same gene when it occurs in a diploid organism is referred to as *dominance.* Dominance can only be expressed in diploid organisms. The parameter "degree of dominance" is the deviation of the heterozygote from the midpoint value for the homozygous parents. If we assign a value of $-A$ to a parent who is homozygous for an allele and the value of $+A$ to the other parent who is homozygous for the other allele, then the value of the F_1 heterozygote will indicate the degree of dominance. If there is no dominance, the value of the heterozygote will be the zero point midway between the two homozygotes. However, if there is dominance, the value of the heterozygote will deviate to one side or the other of the midpoint. With incomplete dominance, the heterozygote value will fall somewhere between the midpoint and one of the parents. With complete dominance, the value of the heterozygote will coincide with the value of one of the homozygotes. Overdominance occurs when the heterozygote value lies outside the range of either of the two parents.

If dominance is a factor in the expression of the genotype of a diploid individual, it will have a bearing on the average effect of the gene. The average effect of a gene will be dependent on its dominance relationship to its allelic pair. For example, an allele that is recessive and paired with a dominant allele will have no effect on the phenotype of the heterozygote. However, if that same allele is inserted against the background of an equivalent recessive allele, the phenotype does change. Quantitative genetics assesses the average affect of a gene across a population of genotypic backgrounds. Therefore, the average effect of a gene having dominance will be considerably influenced by the genetic composition of the population. The relative allelic frequencies in the population will change the average effect.

The other interaction occurring in polygenic systems is the interaction between genes at different loci. This is also referred to as the epistatic deviation, or *epistasis.* These interactions may be very complex and are not readily amenable to dissection.

The G in Eq. (1) has now been separated into its component parts. Genotypic value is equal to the additive effects (A), or breeding value, plus the effect of dominance (D) and interactions between multiple genes (I):

$$G = A + D + I \tag{2}$$

IV. VARIATION—WHY COLONIES DIFFER

A phenotype is the product of the genotype and the environment in which it is expressed. But something more is necessary before a genetic study of a metric character is possible. That is the variation present in a population. As the phenotype of an individual is the sum of effects of both genotype and environment, a population's variation can also be partitioned as

$$\begin{aligned} V_P &= V_G + V_E \\ &= V_A + V_D + V_I + V_E \end{aligned} \tag{3}$$

The phenotypic variation, V_P, is equal to the genotypic variation, V_G, plus the environmental variation, V_E. The genotypic variation can be further subdivided into an additive portion (V_A), a deviation due to dominance interactions (V_D), and an interaction (epistatic) deviation (V_I).

In reality, when measurements are made on individuals and average values are calculated for populations of individuals, they are estimates of these component parts. Earlier it was proposed that if one could eliminate the effects of environment, then the phenotype would directly reflect the value of the genotype. While the variation due to environment cannot be completely removed, it can be reduced in controlled experiments. If the variation due to the environment is reduced, then the variation due to genotype can more easily be seen. Environmental influences that are controlled during an experiment might include those due to nutrition, climate, errors of measurement, or maternal effects. For the honey bee, the effect of a common colony environment might be thought of as equivalent to maternal effects in mammals. Also, there are a number of intangible, and possibly uncontrollable, influences in both developmental and immediate environments.

It is possible to estimate the effects due to environment by controlling all the variation due to genotype. This can be done by using a group of identical genotypes (i.e., highly inbred individuals or F_1 hybrids of inbred lines). This is routinely done with a number of mammals where highly inbred lines have been maintained for many generations. However, this is difficult with honey bees since inbred lines very quickly lose their fitness value and often cannot be maintained (Chapters 9 and 13).

The seemingly straightforward relationship of the underlying influences of environment and genotype and their variance components can be complicated by several natural phenomena. The amount of variation due to environment could be different for different genotypes within the same environment. It has been shown that inbred lines show more variation due

to environmental causes than do outbred lines of the same species. It is hypothesized that the inbreds are less well buffered against the environment because of their much greater frequency of homozygosity at many loci.

The genotype and the environment may also interact, which may influence the partitioning of the variation. The honey bee represents a very clear example of this correlation. A bee spends a good portion of its life within a colony. This colony environment influences the development of the bee and is in part determined by the genotypes of the related bees who are maintaining the colony. These kinds of correlations may be somewhat controlled in experiments through techniques such as cross-fostering of individuals between colonies or sibling groups. However, generally the influence of correlation between genotype and environment is accepted as being part of the genotypic variation.

Generally, an assumption is made that the environment has the same effect or magnitude of effect on different genotypes, but this is not always the case. Such variation also is generally accepted as part of the variation due to environment and not considered further. With certain experimental designs, it is possible to estimate the influence of an environment on different genotypes by use of a two-way analysis of variance of various genotypes across various environments or treatments. With the statistical analysis, a value can be computed for the magnitude of the interaction between differing environment and differing genotype.

The component of variation having most interest in quantitative genetics is the additive variance or the breeding value (V_A). This factor is the chief cause for resemblance between related individuals and therefore is closely connected to observed genetic properties of a population and responses of that population to selection. It is also the most readily estimated component of a population's variation. Equation (3) might more realistically be expressed as

$$V_P = V_A + V_N + V_E \tag{4}$$

where the variation of the phenotype (V_P) is equal to the variation due to additive genetic causes (V_A) plus variation due to nonadditive genetic causes (V_N) plus variation due to environmental causes (V_E). The value of additive genetic variation is not usually presented in this form but is expressed as the proportion of variation due to additive genetic causes as a part of the total phenotypic variation. This ratio of additive genetic to total phenotypic variation is referred to as *heritability.* This will be further discussed later.

The presence of additive variation in the variation of a quantitative char-

acter does not imply anything about the mode of action of the underlying polygenes. These polygenes may be additive in nature, they may have dominance interactions, and they may have epistatic interactions. Only if all the genetic variation is additive is the mode of action implied by the presence of additive variation. Perhaps this can be looked at as the degree of resemblance between individuals with genes having common ancestry as opposed to the level of dissimilarities between relatives. The dominance variation is due to the association of alleles that show some form of dominance relationship. With multiple loci the epistatic interaction generally is included as part of the genetic variance. However, this is usually very small. Probably it could be further subdivided if one knew the number of loci involved in the polygenes. For further discussion of the epistatic deviation, see Falconer (1981). The additive variance component of the genetic variance does take into account multiple alleles at a single locus through the average effect of all the alleles across the population being studied.

The total phenotypic or observed variation in a population can be partitioned into its several components. Using resemblances between relatives, estimates of the additive genetic variation, or breeding value, are possible, and by using inbred lines the variation of a character due to environmental causes can be estimated. Estimations of the breeding value of an individual are relatively easy to make and are important for the estimation of heritability. Heritability is important for the major objective of predicting success from proposed breeding schemes. This leaves the nonadditive genetic variation, which is included in effects due to dominance and to epistasis. With very large numbers of individuals in a population and more elaborate statistical techniques it is also possible to estimate these two components as separate entities. This is probably not practical for honey-bee quantitative genetics.

V. MORE ON RESEMBLANCE

The previous discussion has dealt with partitioning the variation in a theoretical way. Practically, partitioning of the observable phenotypic variation is begun by grouping individuals into families—that is, groups of individuals with genes that are common by descent. Statistical techniques are then used to compare the similarity of individuals within a group with differences between groups. For groups of siblings, either full sibs (in the case of honey bees, supersibs) or half sibs, the intraclass correlation coefficient t is used, and for the special case of offspring with a parent, or average of two parents (mid-parent), the regression coefficient b is used. These coefficients are estimates of the covariance of related individuals. They will

estimate the value of the components of the observed phenotypic variance, with different proportions of these components being estimated based on the type of relationship. For more detailed derivations of these relationships, see Lush (1945), Falconer (1981),and Kempthorne (1955).

Offspring can be grouped by commonality of one parent or an average of both parents. By definition, the average value of offspring is equal to half of the breeding value (V_A) of one of the parents. If mid-parent is used, the relationship will still be true because mid-parent represents the mean of two parents and an estimate of the mean breeding value of the population. Therefore the covariation of offspring on parents is equal to $\frac{1}{2}V_A$. When a regression coefficient b is calculated, it represents the ratio of covariance to total phenotypic variation. Thus, the regression coefficient will estimate $\frac{1}{2}V_A/V_P$, which is one-half the heritability.

For half sibs, those individuals with only one parent in common, the probability is that they will have $\frac{1}{4}$ of their genes by common descent from that one parent, and therefore their covariance is $\frac{1}{4}$ of the breeding value of that parent, $\frac{1}{4}V_A$. For full sibs, the situation is more complicated. They have a probability of having $\frac{1}{2}$ of their genes in common from two parents plus a $\frac{1}{4}$ chance of having the same alleles for a single locus. This probability adds to the covariance, in addition to $\frac{1}{2}$ the breeding value, V_A, a quantity $\frac{1}{4}$ the variation due to dominance, V_D. For honey-bee supersibs, all the genes from the haploid male parent are identical, so the probability is $\frac{3}{4}$ for genes in common and a $\frac{1}{2}$ chance of the same alleles. For all instances using sibs, the intraclass correlation coefficient is calculated and represents the ratio of the covariation to the total variation observed. Therefore, t will be equal to $\frac{1}{4}$ of the heritability for half sibs, a minimum value of $\frac{1}{2}$ of the heritability for full sibs, and a minimum of $\frac{3}{4}$ for supersibs. In the case of full sibs and supersibs, if dominance is not a factor in the variation, the correlation coefficient will be equal to the heritability. If dominance is a factor, the correlation coefficient will overestimate the heritability by $\frac{1}{2}$ the factor of dominance variation.

One other consideration is that relatives frequently occur in a common environment. This common environment increases the similarity between relatives and increases the difference between family groups. This would therefore increase the estimates of heritability where common environment was important. Generally it is ignored except for instances of full sibs where maternal environment plays a part. Probably for honey bees the equivalent common environment could be viewed as the colony. In this case it would influence both calculations with super- sibs and half sibs. It would be up to the individual investigator to judge how important this common environment is, based on a biological understanding of the character under study.

There are a number of assumptions that are made about the basic popula-

tion from which these calculations have been drawn. We assume that the population is a randomly breeding one having no changes of gene frequency from generation to generation. We also assume no selection and no inbreeding are affecting the gene frequencies in the population. Finally, we assume that there is no differential fitness of genotypes; heterozygotes have the same chance of surviving as either of the two homozygous types. If these assumptions are not true, then the estimates will be correspondingly inaccurate.

VI. HERITABILITY

The term heritability, has two major definitions. The first, a more general definition, is the state or quality of being heritable, inherited, or common by descent. In other words, it is the biological phenomenon of the mode of inheritance of a particular trait. Quantitative geneticists, however, use the term in a much more restrictive sense, as a measure of the degree to which a phenotype is genetically influenced and can be modified by selection (King, 1968). Care must be taken in using this word correctly. There are a number of papers in honey-bee genetic literature that use the word heritability in the title, but are not quantitative in subject.

Heritability, h^2, is one of the most important properties of a quantitative trait. It represents the ratio of the additive genetic variance to the total, or phenotypic, variance:

$$h^2 = V_A/V_P \tag{5}$$

This is the proportion of total variance that is attributable to additive effects, the average effects of all genes affecting a character. The size of h^2 indicates the similarity of related organisms. The most important function of h^2 in the study of quantitative traits is that it can predict how reliable the phenotypic value is as a guide to the individual's actual breeding value. This is so because heritability estimates the proportion of the phenotypic variation that is attributable to genetic causes amenable to selection. This is why it is so important to accurately measure the desired trait. If the environmental variance, V_E, is high due to poor measuring techniques, the estimate of the proportion resulting from additive genetic causes, and thus heritability, would be reduced. A poor measure of the phenotype will result in poor success in a selection program.

The value of heritability ranges from 0 (no genetic influence on the trait) to 1 (all variation of the trait is genetically produced). Traits that are closely related to reproductive fitness, such as egg-laying capacity [$h^2 = 0.16$: Ave-

tisyan and Grankin (1976)], generally have low heritabilities. Higher h^2 values are expected in characters less important to reproductive fitness such as body color, or body-part size [$h^2 = 0.53-0.92$: Avetisyan and Grankin (1976)]. A variety of estimates of heritability for the honey bee are presented in Table 1.

Importantly, heritability is a property not only of a specific character but also of the population and of the environmental circumstances. Thus, heritability value estimates made on one population may be different for that same population in another environment, or for a different population. Environmental variance depends on the conditions of culture or management of the organisms being studied — more variable conditions reduce the heritability, and more uniform conditions increase it. The genetic components of heritability are influenced by the gene frequencies in the population, and these may differ between populations of the same organism because of their different biological histories. Small populations maintained for a long time may become more genetically uniform than do large, randomly mating populations, and they may therefore show lower heritabilities.

A. Special Considerations in Honey Bees

The standard approaches for measuring heritability require comparing the merits of related individuals and estimating heritability from the covariance between them or from a regression or correlation. However, the theoretical and applied work that has been done has largely concentrated on diploid domesticated animals, such as sheep and cows, and the laboratory standby, the fruit fly. The honey bee, a haplo–diploid organism living in colonial aggregations and showing caste differences as well as sex differences, requires slightly modified theoretical bases. Rinderer (1977) and Oldroyd and Moran (1983) have addressed the biological differences for honey bees and the necessary modifications to be made in systems for estimating heritability.

First of all, the haplo–diploid condition changes the relatedness of relatives. Rinderer (1977) discusses the changes in the coefficient of relatedness between sister-workers under three different mating conditions. For a queen mated to a single drone, all the sperm are identical, and the daughter-workers are related at a level of 0.75. When the queen has been mated by many drones, all of them from the same drone-mother, the relatedness between two workers can be either 0.75 or 0.50. Under the more natural situation of a queen multiply mated with drones from many sources, the relatedness is either 0.75 or 0.25 for workers in the same colony. This is contrary to the normal situation in diploid organisms where full sibs (having

TABLE 1. Some Heritability (h^2) Estimates for Honey-Bee Traits

Source	Trait		h^2	Method
Soller and Bar-Cohen (1967)	Winter honey weight		0.57	Analysis of variance
	Spring honey weight		0.60	
	Total honey		0.58	
	Winter brood		0.76	
	Spring brood		0.33	
	Final brood area			
el Banby (1967)	Brood rearing:			Regression: offspring on dams
	winter		0.95	
	citrus season		0.51	
	clover season		0.34	
	cotton season		0.28	
	yearly average		0.90	
	Honey production:			
	clover		1.00	
	cotton		0.75	
Pirchner *et al.* (1962)	Honey production		0.23	Analysis of variance
	Brood		0.35	
Vesely and Siler (1963)	Honey yield		0.16–0.19	Regression: offspring on midparent
	Brood 6 weeks before flow		0.30–0.41	
Gonçalves and Stort (1978)	Number of hamuli		0.76	Regression: offspring on parents
Collins (1979)	Time to react to isopentyl acetate (IPA)		0.68	Regression: offspring on midparent
Rinderer *et al.* (1983)	Time to react to IPA		0.03 ± 0.006	Analysis of variance
	Longevity		0.32 ± 0.27	
Oldroyd and Moran (1983)	Number of hamuli		0.68 ± 0.183	Intraclass correlation/ average relatedness
Collins *et al.* (1984)	Hoarding	E[a]	0.92 ± 0.44	Analysis of variance
	(three day average)	A	0.66 ± 0.69	
	Time to react	E	1.28 ± 0.04	
	to IPA	A	0.31 ± 0.01	
	Time to react to	E	0.31 ± 0.20	
	moving target	A	0.69 ± 0.31	
	Number of stings	E	0.57 ± 0.24	
	in target	A	NE[b]	
	Number of bees	E	0.93 ± 0.03	
	responding	A	0.17 ± 0.01	
	Comb cell size	E	NE	
		A	1.15 ± 0.11	
Milne and Friars (1984)	Pupal weight		0.645 ± 0.065	Analysis of variance
Milne (1985a)	Corbicular area		1.014 ± 0.195	Analysis of variance
Milne (1985b)	Hoarding		0.187 ± 0.029	Analysis of variance
Milne (1985c)	Worker longevity		0.196 ± 0.024	Analysis of variance
Moritz (1985)	Postcapping stage		0.68 ± 0.001	Intraclass correlation/ average relatedness
			0.97 ± 0.06	Regression: offspring on midparent

[a] E, European bees; A, Africanized bees

[b] NE, not estimable.

both parents the same) are related at a probability of 0.50 and half sibs (one parent in common) are related at a level of 0.25. Crow and Roberts (1950) and Laidlaw and Page (Chapter 13) discussed the calculation of the coefficient of relationship for a number of mating systems. A somewhat different approach was taken by Polhemus *et al.* (1950) and Laidlaw and Eckert (1950) for application of quantitative genetic theory to honey bees. They were able to look at relatedness in the same manner as a diploid system by considering that a mating took place between a dam-queen and a sire-queen.

The second major difference seen in honey bees is the production of castes in individuals that are derived from the same combinations of genetic material (a fertilized egg) but reared under different environmental conditions. In some cases, a trait may be expressed differently, or not at all, by queens and workers. This could be considered an example of variation due to epistasis, where genes controlling caste differentiation have an effect on the expression of genes controlling other characters. In many cases, especially those related to an economic character, a phenotypic value for an individual may be measured on a sibling of a different sex or caste. In these instances the phenotypic value of the reproductive (queens or drones) is based on the phenotype of sisters (workers) with coefficients of relatedness of 0.75, 0.50, or 0.25 in the case of queens, or a coefficient of relatedness equal to 0.50 for drones. This inaccuracy in actually measuring the phenotype of the reproductive individual has consequences for the accuracy of the estimate of heritability.

A third important situation present in honey bees is that they live as a colony of more or less related individuals. In diploid organisms one must be alert to increased covariance due to a common environment. This is most clearly seen in animals that have litters or clutches where there is a strong effect of the common maternal environment. The individual honey-bee investigator must assess the effect of the common colony environment on the trait he is investigating.

Another aspect of the colonial sociality of honey bees is that some of the characteristics that are of most interest are the result of behavior of the individuals within the colony and cannot be readily measured on single individuals. A good example of this is honey production, commonly expressed in terms of weight gain by a colony during a honey production season. A technique proposed by Rothenbuhler (1960) attempted to deal with this problem. He proposed that inbred queens be inseminated by single drones. All the semen from the single drone will be identical, and most of the loci of the inbred queen can be considered to be homozygous. From such a mating a colony of workers of almost identical genotype can be produced. A measurement of the colony behavior, therefore, is the average expression

of almost all of these similar genotypes, and a good estimate of their phenotype.

B. Estimation

The simplest way to estimate heritability is to measure a population of mixed genotypes and one of identical genotype in several environments. The first population would provide an estimation of total phenotypic variance. The second would measure only environmental variance, because all genotypes would be identical. The difference between these two phenotypic variances would be the additive genetic value. Heritability could then be directly calculated from the ratio of additive genetic variance to total phenotypic variance.

A common straightforward method for estimating heritability is from the regression of offspring on parents. The data, measurements of parents and the mean values of their offspring, are used to calculate a regression coefficient b. If this is the regression of offspring on one parent, b_{op}, it is a valid measure of $\frac{1}{2}h^2$; if the regression is offspring on midparent, b_{mp}, it actually measures heritability. Some examples of honey bee h^2 values calculated using this method are presented in Table 1. Both Rinderer (1977) and Oldroyd and Moran (1983) caution that this approach is inappropriate for measurements made on different castes. However, it has been used in a number of instances where the queen's phenotypic value was based on measurements made on sister workers (Collins, 1979; Milne, 1985a,b,c; Milne and Friars, 1984; and Moritz, 1985). For characters, such as brood patterns or egg-laying abilities, that are measured only on queens, the situation mimics a diploid one. The midparent then is the average of the phenotypic values of the dam-queen and the sire-queen. Another possibility is to use pooled mixed semen from more than 20 drones, where the resulting segregation of gametes from the sire-queen can be interpreted as a diploid system.

For a trait that can be measured on both queens and workers, an appropriate collection of matings can be used to compare queens with the parental queens, and workers with those same parental queens. Differences in these two regression values will show the magnitude of environmental, dominance, and epistatic effects. Heritability values from the regression of queens on queens would be higher than the values for workers on the same queens. The only limitation on this system is that the phenotypic variance must be the same for both castes for the statistical procedures to be appropriate.

Historically, heritability is most often estimated by sib analysis. Each of several males (sires) is mated to several females (dams), and some offspring

TABLE 2. Calculation of Phenotypic Variance from Analysis of Variance Mean Squares (MS) for a Sampling of Sib/Half-Sib Families[a]

Source of variance	Variance	Calculation[b]
Between sires	σ^2_{sire}	$(MS_{sire} - MS_{dam})/dk$
Between dams	σ^2_{dam}	$(MS_{dam} - MS_{within})/k$
Within offspring	σ^2_{within}	MS_{within}
Total population	σ^2_{total}	$\sigma^2_{sire} + \sigma^2_{dam} + \sigma^2_{within}$

[a] For more detail see Falconer (1981) and Henderson (1953).

[b] d, Number of dams; k, number of offspring per dam.

from each female are measured. The individuals measured form a population of half-sib and full-sib families. An analysis of variance divides the phenotypic variance into components attributable to differences in sires, dams mated to the same sires, and among offspring of the same female. The variance components from sires, dams, and the total may be calculated from the mean square values (Table 2). The total variance, or phenotypic variance, is calculated because it is not necessarily equal to the observed variance as estimated from the total sum of squares, though the two seldom differ by much. With these values, estimates of heritability can be made from the sire component, the dam component, or a combination of the two (Table 3).

The use of sib analysis has been carefully examined by Rinderer (1977). In order to really be comparing half-sib and supersib families, the character must be measured on the appropriate caste in both cases, that is, queens or workers but not both. This is because the relationship of workers between colonies of sister queens is that of cousins but not sibs. He proposed a scheme using randomly selected dam-queens mated to randomly selected sire-queens. This is a useful sibling system for estimating heritability. If the

TABLE 3. Calculation of Heritability from Phenotypic Variances Based on Sib and Half-Sib Families[a]

Estimate	Calculation
h^2_{sire}	$4\sigma^2_{sire}/\sigma^2_{total}$
h^2_{dam}	$4\sigma^2_{dam}/\sigma^2_{total}$
$h^2_{combined}$	$2(\sigma^2_{sire} + \sigma^2_{dam})/\sigma^2_{total}$

[a] For more detail see Falconer (1981).

matings are single drone inseminations, then the sire component from the analyses of variance is the best source for estimates of h^2. If mixed pooled semen for more than 20 drones from one sire-queen is used, then both the sire and dam component are acceptable for estimates. If the behavior under study is a colony behavior, then only the sire component can be used regardless of the number of drones in each mating.

Oldroyd and Moran (1983) proposed a somewhat different system that has the advantage of not requiring artificial insemination. They use the relationship of intraclass (sib group) correlations (t) for a population and the relatedness (r) of workers in a colony (sib groups). This is expressed as

$$h^2 = t/r \tag{6}$$

For naturally mated queens having a large number of effective matings (eight or more), the value of r approaches $\frac{1}{4}$, the situation in diploid half-sibs. The intraclass correlation value of t is calculated from measurements made on worker offspring. If the trait is one expressed by the colony, such as defense and honey production, the correlation of repeated or replicate measures on groups of the worker offspring would be higher than that from measures from individuals. This would give an upward bias to the heritability estimates. This bias could be reduced by utilizing many replicates or taking measurements across a period of time.

VII. EFFECTS OF SELECTION

Earlier, a number of assumptions were made about the population. One was that the population was not undergoing selection. In many cases, however, we are using quantitative genetics to predict the effect of planned selection programs. Once selection has begun the effects from generation to generation can be monitored. By observing the changes in the population means, variance, covariances, etc., the effects of the underlying changes in gene frequencies brought about by selection can be described. There are three ways that gene frequencies change: (1) by artificial selection of parents for each generation, (2) through differential fertility in parents within a generation, and (3) through differential viability in the offspring. Ways (2) and (3) are major aspects of natural section and are generally always present. Usually these are not correlated with the characters in question. However, their function in the selection system should not be forgotten.

There are two parameters that we can measure in a selection program. One is the response to selection, R. This is the mean deviation of the offspring from the original, or parental, population before selection. This is

identical to the breeding value of the selected parents. The other is the selection differential S, which is defined as the deviation of the mean parental value from the mean of the whole parental generation before selection. The relationship of R to S is expressed as

$$R = h^2 S \tag{7}$$

and, where b_{op} is the regression of offspring on the midparent, as

$$R = b_{op} S \tag{8}$$

Assumptions are that there are no nongenetic causes of resemblance between the offspring and parents, such as common environment, and that there is no natural selection.

The heritability value was used to predict the response to the selection. This value of heritability is, in theory, only valid for prediction for one generation because it is estimated from the resemblance between relatives in the parental generation and the genetic make-up of this population changes with selection. In practice, however, the original estimate of h^2 is useful for several generations (five to 10). It can be seen from Eqs. (7) and (8) that it is possible to calculate an estimate of h^2 from the relationship between response to selection and selection differential. This information is obtained from later generations in a selection program. This h^2 is referred to as *realized heritability.* Estimates of realized h^2 are valid in the absence of inbreeding, with reduced environmental effects, a weighting of selection differential to account for natural selection, and no maternal effects. Realized heritability is the most useful way of comparing the effectiveness of selection in different experiments. Because of the many external effects that cannot be totally eliminated from any experiment, the best estimates of realized heritability are those that are predicted over a number of generations of selection. The actual calculation involves plotting the generation means against the cumulative differential and calculating the slope of the regression line. This value, essentially the regression of offspring on the midparent, is the realized heritability. An example of such a plot is presented in Fig. 1 based on data from Rothenbuhler *et al.* (1979), with calculations by Collins. Several other estimates of realized heritability are presented in Table 4.

The selection differential S depends on the proportion of the population selected as parents (a number limited by the minimum number of parents necessary to maintain a viable population and by the variability of the character). Intensity of selection i is a standardized form of S expressed as

$$S/\sigma_p = i \tag{9}$$

where i is dependent only on the proportion selected, and can be deter-

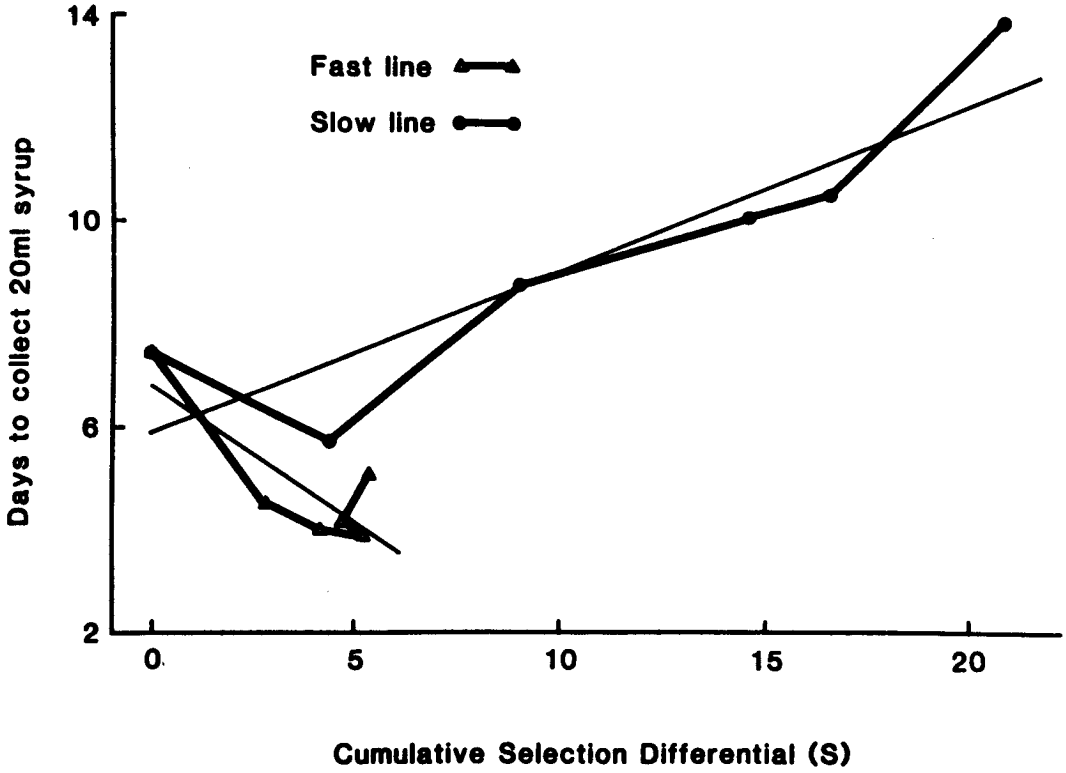

Fig. 1. Two-way selection for hoarding of sugar syrup in honey bees. Response (generation means) plotted against cumulative selection differential (S). The slopes are not significantly different ($p = 0.408$). Data taken from Rothenbuhler *et al.* (1979). Compare with Rinderer and Collins, Chapter 6, Fig. 4.

mined by the ratio of the value of the trait at truncation to the proportion of individuals selected as parents. It is given in standard deviation units, and if one knows the standard deviation of the trait, S can be predicted. If we then calculate S following selection, weighted by the number of offspring each parent produces, we can calculate an effective value of S. The relative magnitudes of the predicted S and the effective S give us a measure of the importance of artificial and natural selection as it is functioning in our

TABLE 4. Estimates of Realized Heritability (h^2)

Source	Trait	Realized h^2
Soller and Bar-Cohen (1967)	Citrus honey production	0.36
Rothenbuhler *et al.* (1979)	Hoarding of syrup by caged bees	Fast line, 0.553[a] Slow line, 0.324
Hellmich *et al.* (1985)	Pollen hoarding	High line, 0.556 ± 0.161 Low line, 0.063 ± 0.190

[a] Calculation by A. M. Collins.

selection scheme. The effective *S* reflects both, and the predicted *S* reflects only artificial selection.

There are a number of other phenomena associated with selection that are of interest in a discussion of quantitative genetics of the honey bee. The most important of these, inbreeding depression, has a serious effect on the fitness of selected lines. A more complete discussion of this problem is presented by Cornuet (Chapter 9) and Laidlaw and Page (Chapter 13). Its converse, heterosis, has been used to advantage in a number of honey-bee breeding programs discussed by Kulinčević (Chapter 16). These made use of the greater fitness expressed in certain heterozygotes compared to the respective homozygous conditions. In some cases combinations between certain homozygotes are more fit than other heterozygote combinations, a phenomenon known as combining ability. Both inbreeding depression and heterosis occur because of the dominance interactions within loci, and the amount of heterosis one sees following a cross between two lines or populations is dependent on differences in gene frequencies between the two populations.

VIII. CORRELATIONS

Correlations sometimes occur between characters. These correlations can arise because of commonality, or similarity, in the underlying genotypes. The genetic cause of correlation between characters is largely due to pleiotropy, although linkage of genes on the same chromosome is a transient cause of correlation. Pleiotropy, as discussed earlier, is a property of a gene whereby it effects more than one character. Thus, the degree of correlation between two characters is the extent to which they are controlled by the same genes. Some of these pleiotropic effects may produce negative correlations and others may produce positive correlations.

The correlations that we actually measure are the phenotypic correlations. As with variance, this correlation, or covariance, can be partitioned into its component parts. These are genetic, the correlation of the breeding values for the two traits, and environmental, which includes both environmental and nonadditive genetic correlations. The correlation is calculated by the appropriate covariance divided by the product of the two standard deviations of the characters. The genetic and environmental correlations may be quite different in magnitude and sign. If they are different in sign it means that the genotype and the environment affect the character through different physiological mechanisms.

Given the same kinds of experimental designs for super sib and half-sib families or for regressions of offspring on parents, appropriate calculations

TABLE 5. Significant Correlations between Traits of the Honey Bee

Source	Trait A	Trait B		Correlations: Phenotypic	Correlations: Genetic
Soller and Bar-Cohen (1967)	Spring honey weight	Total honey		0.92	1.02
	Winter brood	Final brood area		0.91	1.15
	Total honey	Winter brood		0.51	1.12
	Winter honey weight	All others		Small and negative	
	Spring honey weight	Winter brood		0.45	1.06
	Spring honey weight	Final brood area		0.37	1.30
	Total honey	Final brood area		0.45	1.32
el Banby (1967)	Honey production on cotton	Brood in cotton season		0.86	1.06
	Honey production for whole year	Whole year brood		0.82	0.77
Kerr *et al.* (1974)	Amount of 2-heptanone	Time to first sting		−0.75	Not calculated
	Amount of 2-heptanone	Time bees were irritated		−0.821	Not calculated
	Amount of 2-heptanone	Number of stings in ball		0.741	Not calculated
	Time bees were irritated	Number of stings in ball		−0.759	Not calculated
Rinderer *et al.* (1983)	Time to react to IPA (cage)	Initial activity level (cage)		−0.61	NE[a]
	Time to react to IPA (cage)	Longevity		−0.06	0.72 ± 0.87
	Response to *Nosema apis*	Longevity		0.76	NE
Collins *et al.* (1984)	Time to react to IPA (cage)	Initial activity level (cage)	E[b]	−0.57	1.0
			A	−0.26	1.0
	Time to react to IPA (cage)	Time to react to moving target	E	−0.01	−0.82 ± 0.03
			A	−0.17	−0.36 ± 0.08
	Time to react to IPA (cage)	Number of stings in moving target	E	0.09	1.0
			A	−0.43	NE
	Hoarding (13-day average)	Initial activity level (cage)	E	0.05	1.0
			A	0.54	1.0
	Hoarding (13-day average)	Time to react to IPA (cage)	E	0.11	1.0
			A	−0.70	−0.28 ± 0.10
	Hoarding (13-day average)	Time to react to moving target	E	0.15	0.80 ± 0.12
			A	−0.32	−1.0
	Hoarding (13-day average)	Number of stings in target	E	−0.05	−1.0
			A	0.61	NE
	Time to react to moving target	Initial activity level (cage)	E	−0.19	1.0
			A	0.12	1.0
	Time to react to moving target	Number of stings in target	E	−0.58	−1.0
			A	−0.16	NE

[a] NE, not estimable.

[b] E, European bees; A, Africanized bees.

to estimate genetic correlations can be made from analyses of covariance. Some examples are presented in Table 5. However, these are very seldom precise. One can estimate correlations from responses to selection in a manner similar to that for realized heritability or, a more useful process, one can predict the change in character Y following selection on character X if one knows the genetic correlation and the heritabilities for the two characters. A discussion of the calculations involved for these estimates is presented by Falconer (1981).

We can also make use of correlations between characters in a process called indirect selection. In indirect selection one selects for a character of secondary importance in order to improve a correlated character of major importance. This can be done if the secondary character has a higher h^2 than the primary character, and if the genetic correlation is high. A possible example involves sex- or caste-limited traits which are correlated to a character expressed in both sexes or castes. On the whole, however, indirect selection is not as effective as simultaneous selection for the two characters, a topic discussed more thoroughly in the next chapter.

ACKNOWLEDGMENT

The chapter was prepared in cooperation with the Louisiana Agricultural Experiment Station.

REFERENCES

Avetisyan, G. A., and Grankin, N. N. (1976). Heritability of some economically useful and some external characteristics of mid-Russian honeybees of the Orel region. *Doklady Timiryazevskoi Sel'skokhozyaĭstvennoĭ Akademii 221,* 177–180.

Banby, M. A. el. (1967). Heritability estimates and genetic correlation for brood-rearing and honey production in the honeybee. *Proc. Inter. Apic. Cong. (Apimondia) 21,* 498.

Collins, A. M. (1979). Genetics of the response of the honeybee to an alarm chemical, isopentyl acetate. *J. Apic. Res. 18,* 285–291.

Collins, A. M., Rinderer, T. E., Harbo, J. R., and Brown, M. A. (1984). Heritabilities and correlations for several characters in the honey bee. *J. Hered. 75,* 135–140.

Crow, J. F., and Roberts, W. C. (1950). Inbreeding and homozygosis in bees. *Genetics 35,* 612–621.

Falconer, D. S. (1981). "Introduction to Quantitative Genetics," 2nd ed. Ronald Press, New York.

Gonçalves, L. S., and Stort, A. C. (1978). Honey bee improvement through behavioral genetics. *Annu. Rev. Entomol. 31,* 197–213.

Hellmich, R. L., II, Kulinčević, J. M., and Rothenbuhler, W. C. (1985). Selection for high and low pollen-hoarding honey bees. *J. Hered. 76,* 155–158.

Henderson, C. R. (1953). Estimation of variance and covariance components. *Biometrics 9,* 226–252.

Hill, W. G., ed. (1984a). "Quantitative Genetics Part I: Explanation and Analysis of Continuous Variation." Van Nostrand Reinholt, New York.

Hill, W. G., ed. (1984b). "Quantitative Genetics Part II: Selection." Van Nostrand Reinholt, New York.

Kempthorne, O. (1955). The theoretical values of correlations between relatives in random mating populations. *Genetics 40,* 153–167.

Kerr, W. E., Blum, M. S., Pisani, J. F., and Stort, A. C. (1974). Correlation between amounts of 2-heptanone and iso-amyl acetate in honeybees and their aggressive behaviour. *J. Apic. Res. 13,* 173–176.

King, R. C. (1968). "A Dictionary of Genetics." Oxford University Press, London.

Laidlaw, H. H., Jr., and Eckert, J. E. (1950). "Queen Rearing." Dadant and Sons, Hamilton, Ill.

Lush, J. L. (1945). "Animal Breeding Plans," 3rd ed. Iowa State University Press, Ames, Iowa.

Mather, K. (1949). "Biometrical Genetics: The Study of Continuous Variation." Methuen and Co., London.

Milne, C. P., Jr. (1985a). An estimate of the heritability of the corbicular area of the honeybee. *J. Apic. Res. 24,* 137–139.

Milne, C. P., Jr. (1985b). A heritability estimate for honey bee hoarding behavior. *Apidologie.* (In press).

Milne, C. P., Jr. (1985c). An estimate of the heritability of worker longevity or length of life in the honeybee. *J. Apic. Res. 24,* 140–143.

Milne, C. P., Jr., and Friars, G. W. (1984). An estimate of the heritability of honeybee pupal weight. *J. Hered. 75,* 509–510.

Moritz, R. F. A. (1985). Heritability of the post capping stage in *Apis mellifera* and its relevance for varroatosis resistance. *J. Hered. 76,* 267–270.

Oldroyd, B., and Moran, C. (1983). Heritability of worker characters in the honeybee *(Apis mellifera). Aust. J. Biol. Sci. 36,* 323–332.

Pirchner, F., Ruttner, F., and Ruttner, H. (1962). Erbliche Unterschiede zwischen Ertragseigenschaften von Bienen. *Proc. Inter. Cong. Entomol. 11,* 510–516.

Polhemus, M. S., Lush, J. L., and Rothenbuhler, W. C. (1950). Mating systems in honey bees. *J. Hered. 41,* 151–155.

Provine, W. B. (1971). "The Origins of Theoretical Population Genetics." University of Chicago Press, Chicago.

Rinderer, T. E. (1977). Measuring the heritability of characters of honeybees. *J. Apic. Res. 16,* 95–98.

Rinderer, T. E., Collins, A. M., and Brown, M. A. (1983). Heritabilities and correlations of the honey bee: response to *Nosema apis,* longevity, and alarm response to isopentyl acetate. *Apidologie 14,* 79–85.

Rothenbuhler, W. C. (1960). A technique for studying genetics of colony behavior in honey bees. *Am. Bee J. 100,* 176, 198.

Rothenbuhler, W. C., Kulinčević, J. M., and Thompson, V. C. (1979). Successful selection of honeybees for fast and slow hoarding of sugar syrup in the laboratory. *J. Apic. Res. 18,* 272–278.

Soller, M., and Bar-Cohen, R. (1967). Some observations on the heritability and genetic correlation between honey production and brood area in the honeybee. *J. Apic. Res. 6,* 37–43.

Vesely, V., and Siler, R. (1963). Possibilities of the application of quantitative and population genetics in bee breeding. *Proc. Inter. Apic. Cong. (Apimondia) 19,* 120–121.