

versely, a certain change in environment may make a large change in individuals of some genotypes but only a small change in other individuals whose genotypes make them less labile.

Positive correlation between heredity and environment makes the whole population more variable by preventing the plus effects of variations in heredity from being canceled in individual animals by the minus effects of variations in environment, or vice versa, as often as would be the case if the two were uncorrelated. Such a correlation is an ever-present possibility in data which are collected from a variety of farms, since it often happens that the man who tries hardest to give his animals the best environment also tries hardest to select the animals with the best heredity and has some degree of success in both efforts. Correlation between heredity and environment is also likely to exist in data concerning the mental and social traits of man, since inherited aptitudes on the part of the parents tend to cause them to create in their own homes environments which favor the development of those same special abilities in their children. These children will also inherit some of the same genes which made those parents have those aptitudes in the first place.

It seems likely that the nonadditive combination effects of heredity and environment are generally small in amount,² but some interactions of this kind do occur.

MODES OF GENE EXPRESSION

The simplest way in which the effects of various genes are combined is that the substitution of a gene for its allele produces a certain plus or minus shift in the measurement of the characteristic affected and that this change—the “effect” of that gene substitution—is the same, regardless of what other genes are present. As a physical example, consider how adding or subtracting one more brick makes exactly the same increase or decrease in the weight of a brick pile, regardless of the number or kind of bricks the pile already contains. Some genes may combine their effects exactly in this simple way and many seem to do so to some extent, yet many genes are known to interact with each other so that the outward result of substituting a gene for its allele is larger in some genotypes and smaller or zero or even reversed in other genotypes. Thus the actual effect of the gene substitution in each separate individual may depend partly on what other genes are present.

A simple example is dominance. If dominance exists, the outward effect of substituting gene *A* for *a* is larger when the substitution is

²For some extreme examples, consult Chapter 5 of Hogben's *Nature and Nurture*.

made in an individual which is *aa* than when it is made in one which is *Aa*, although the effect on breeding value of the individual is the same; namely, that it now transmits *A* to one-half of its offspring which would otherwise have received *a* from it. Dominance is nonadditive combination of the effects of genes which are in the same allelic series. When the effect of making two such gene substitutions in an *aa* individual is not simply twice as large as the effect of one, some degree of dominance exists.

Genes which are not allelic may also modify the magnitude or even the direction of each other's effects. A classic example is Bateson's case of white and purple flowers in sweet peas. He found that two different pairs of genes, both showing dominance, were necessary for the production of the purple color. Plants which were *cc* had white flowers, no matter whether they were *RR*, *Rr*, or *rr*; and plants which were *rr* had white flowers, no matter whether they were *CC*, *Cc*, or *cc*. But plants which were either *CC* or *Cc* and were also *RR* or *Rr* had purple flowers. It is as if *R* produced an enzyme necessary for developing color and *C* produced the substrate on which the enzyme could work. Whether the substitution of *C* for *c* will produce a change from white to purple depends on whether *R* is also present, as well as on whether the substitution is made in a *cc* or in a *Cc* individual. The difference between purple and white is a *joint effect* the credit or blame for which cannot wholly be divided fairly, part to one gene and the rest to the other. An example in which the direction of the effect depends on other genes is the case of the *E* gene in guinea pigs, which darkens certain colors in the presence of the *P* gene but lightens them in *pp* individuals. Many other kinds of nonadditive combinations of the effects of genes are known. Some common examples are: inhibiting genes, threshold effects, and the general class of cases in which the outward extreme is genetically an intermediate. The latter may be very common among physiologically complex characteristics where the degree of expression of the characteristic depends on the harmonious interplay of a number of different organs and processes.

SUBDIVISION OF HEREDITARY VARIATION

In an actual population there will be genes acting in all these ways, and the number of genes and possible kinds of interactions between them is so enormous that there is no possibility of learning exactly what each gene does in every combination. The simplest way to think of this tangled situation is to imagine that one could average the effects (some of them large, some small, some positive, some negative, etc.)

which a gene substitution actually does have in that particular population and then proceed as if this *average effect* were the *actual effect* of that gene substitution in all genotypes and under all environmental circumstances which occur in that population. In effect this is what we do when we speak of a gene as "good" or "bad," or as "a gene for high production."

By adding these average effects of all the genes which an animal has we can obtain an "expected" value, or measurement of the appearance or individual performance of this animal. The expected and the actual characteristics may not be exactly the same, if the genes interact in non-additive ways. The expected value of an individual corresponds more closely to its breeding value than its own appearance or performance does.

The variation of the expected values from each other is the additively genetic portion of the actual variation. Differences between the expected and the actual values are deviations from the simple additive scheme. It is convenient to divide these nonadditive deviations into two groups, the first being the deviations caused by dominance and the second being the nonadditive interactions of genes which are not allelic to each other. For brevity these latter are called "epistatic" deviations in this book, although this is a broader use of epistatic than Bateson intended when he introduced the word.

To understand the principles of what we do when we separate the additively genetic variation from that due to dominance deviations, consider the two cases shown in Figure 6. Polled is considered a simple dominant over horns in cattle.³ The frequencies of the three genotypes in this example were assumed to be: PP , .01; Pp , .18; and pp , .81. These are not far from the present frequencies of the three types in the Hereford breed as a whole in the United States. Probably there actually are slightly more PP and fewer Pp individuals than this.

The actual or phenotypic values are indicated by Y's and the expected or genetic or breeding values are indicated by G's. The G values come nearer to agreeing with the Y values than could any other three values which lie on a straight line.⁴ In technical statistical terms,

³This fits most of the facts as far as yet known, but there are a few sets of data in which the situation seems more complicated. Also dominance is not always complete, scurs being some indication of heterozygosis.

⁴The G values must lie on a straight line since they are made to conform to the assumption of no dominance; i.e., the phenotypic change expected from substituting P for p is to be the same when the substitution is made in Pp individuals as when it is made in pp individuals. The G values are completely determined by the requirement that the sum of the squares of their deviations from the Y values shall be the least possible for any set of three values which are on a straight line. This is the "least squares" method for fitting a straight line as closely as possible to observations which do not actually lie on a straight line.

the line connecting the G values shows the regression of phenotypic values on breeding values. If there were no dominance Y_{Pp} would lie on a straight line connecting Y_{pp} and Y_{PP} and the G values would fit the Y values exactly. Hence the discrepancies between each G and the corresponding Y (on the vertical scale) are called dominance deviations. Vertical differences between the G values are the additively genetic deviations. If we let the difference between horned and polled be one unit on the vertical scale, we can compare on a quantitative basis the additively genetic variation and the dominance deviations and find how important both are. We get the following values:

Genotype	pp	Pp	PP
Frequency81	.18	.01
Actual phenotype, Y	y	$y + 1.00$	$y + 1.00$
G value	$y + .01$	$y + .91$	$y + 1.81$
$Y - G$	—.01	.09	—.81

When we summarize the variation in terms of "variance" (See page 79) we find that 18/19 of the actual variation is included in the variation of the G values, while only 1/19 of it has to be charged against the domi-

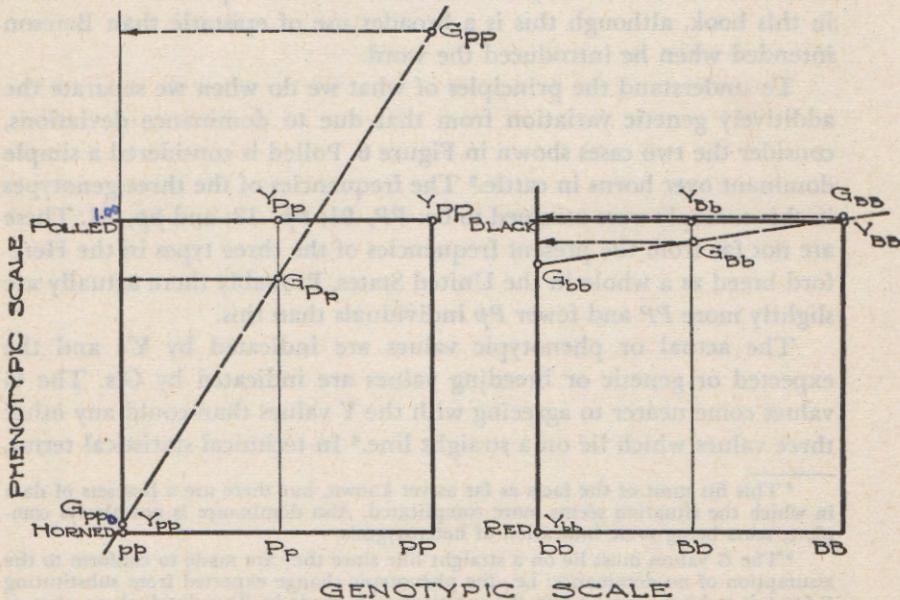


FIG. 6. Diagram of regression of phenotype on genotype where only additively genetic variance and dominance deviations are involved. Left: A case where the dominant is rare, the G-values are far apart, and most of the variance is additive. Right: A case where the recessive is rare and the dominance deviations cause much more variance than the differences between G-values.

nance deviations. In this case the additive scheme comes near to telling all the truth, even though dominance is complete. The G values are far apart and the variation between them is large. The discrepancy between G and Y is very small for the pp genotype which includes most of the individuals. Nearly all the rest of the individuals are in the Pp group where the dominance deviation is also rather small. The dominance deviation is large for the PP group, but the PP animals are rare and hence contribute only a few deviations to the total and do not have much influence.

The average effect of substituting P for p may be computed as follows: For every 100 individuals in the population there will be 180 p genes and 20 P genes. Of the p genes 18 are in Pp individuals where changing them to P would produce no phenotypic effect. The other 162 are in pp individuals and for any one of these a change to P would change the phenotype of its possessor one full unit. Hence the average effect of substituting P for p in this particular population would be $\frac{162}{180} = .9$ unit.⁵

Obviously, the size of the average effect and the comparative importance of additive and dominance variations will depend on the frequency of the genotypes, as well as on the degree of dominance. These are part of the description of this particular population.

The right side of Figure 6 shows for comparison how different the situation is when the recessive is very rare. The Bb pair of genes determines the contrast between black and red in cattle, black being completely dominant. The frequencies assumed for the genotypes are about what they would be in black breeds of cattle in which one calf in each 200 is born red. The actual variation is small and the variation between

the G values accounts for only $\frac{14}{107}$ of it. Dominance deviations

account for the rest, $\frac{93}{107}$. The contrast between the left and the right sides of Figure 6 illustrates the general fact (of which more in Chapter 11) that dominance is an *important* source of confusion or hindrance to progress by selection only when the undesired recessive is already rare. Even when dominance is complete, the variance among the G values

will account for $\frac{2(1-q)}{2-q}$ of the actual variance in random breeding

⁵This simple arithmetical way of computing the average effect illustrates its meaning and will be correct when the heterozygotes are present in the same proportion as they would be under random mating. When they are more abundant or less abundant than that, the average effect should be computed by the more technical procedure which is called the least squares method of fitting a straight line.

populations, q being the frequency of the dominant gene.

For a numerical example of epistatic variance, let us take again the case of purple and white flowers in sweet peas. Its genetic basis is definite and well-known, and it is generally considered to be a rather extreme case of epistasis—although that idea may need revision when we learn more about the usual results of making several gene substitutions at one time. If the sweet peas were breeding at random,⁶ and if the frequencies of the C gene and of the R gene were each .5, then the various genotypes would occur in the proportions shown in column 1 of Table 5.

If we let the difference between purple and white be one unit on the scale on which we measure color, then in this population the average effect of substituting C for c is $3/8$ of a unit. That may be computed as

TABLE 5
ILLUSTRATION OF THE BASIS FOR SEPARATING ADDITIVE GENETIC VARIATIONS, FROM DEVIATIONS CAUSED BY DOMINANCE AND EPISTASIS, USING BATESON'S CASE OF PURPLE AND WHITE COLOR IN SWEET PEAS

Frequency With Which the Various Genotypes Would Occur	Fraction of the c Genes of the Whole Population Which Are in This Genotype	Values on a Scale on Which White = 0 and Purple = 1		
		Actual	"Expected"	Deviations Due to Dominance and Epistasis
1 $ccrr$	1/8	0	- 3/16	+3/16
2 $Ccrr$	1/8	0	+ 3/16	-3/16
2 $ccRr$	2/8	0	+ 3/16	-3/16
1 $CCrr$	none	0	+ 9/16	-9/16
1 $ccRR$	1/8	0	+ 9/16	-9/16
4 $CcRr$	2/8	1	+ 9/16	+7/16
2 $CcRR$	1/8	1	+15/16	+1/16
2 $CCRr$	none	1	+15/16	+1/16
1 $CCRR$	none	1	+21/16	-5/16

follows: One-half of all the c genes are in Cc individuals (the second, sixth, and seventh lines in Table 5), where, on account of dominance, the substitution would make no change in the color. One-eighth of the c genes are in $ccrr$ individuals (the first line in Table 5), where the substitution would produce no outward effect because the R gene, which is also necessary for the production of purple, is not present. The remaining three-eighths of the c genes are in $ccRR$ or $ccRr$ individuals, where the substitution of C for c would produce the full change from white to purple. One unit of change in three-eighths of the cases plus no change in five-eighths of the cases makes an average effect of three-eighths of a

*The sweet pea does not really fulfill this condition, since it is largely self-fertilizing.

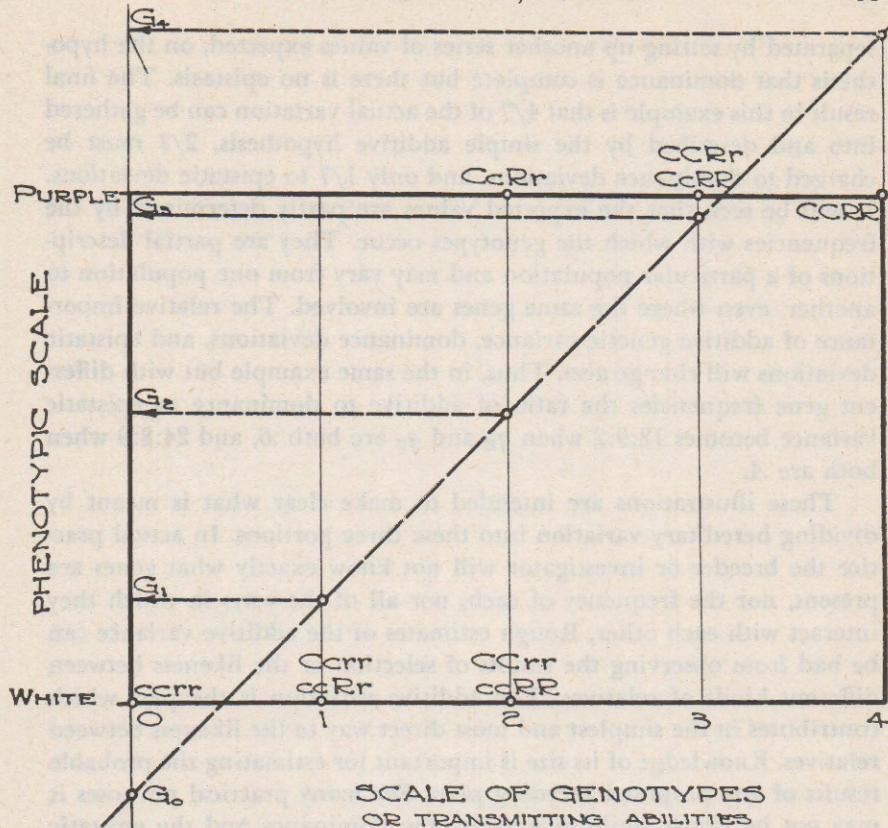


FIG. 7. Regression of phenotypes on transmitting ability in a case involving epistatic deviations in addition to dominance deviations and additive differences. G_0 — G_4 are the expected values or transmitting abilities of individuals which have 0, 1, -- 4; respectively, of the C and R genes. Differences (on the vertical scale) between the G values are the additive variation. Differences (on the vertical scale) between each G value and the corresponding actual value are deviations caused by dominance and epistasis together.

unit for substituting C for c in that population, although the actual change would not be exactly three-eighths of a unit in any one plant.⁷ The expected values shown in Table 5 were found by the additional requirement that the average of the expected values must be the same as the average of the actual values. Figure 7 shows graphically how near the actual and the "expected" values are to each other. The differences between them (the last column in Table 5) are the deviations caused jointly by dominance and epistasis.

Variance due to dominance and variance due to epistasis can be

⁷For a more detailed discussion of this conception of the average effect of a gene substitution, see the first few pages of Wright's article beginning on p. 243 of volume 30 of the *Jour. of Genetics*, or pages 53-56 in volume 11 of *Annals of Eugenics*, 1941.

separated by setting up another series of values expected, on the hypothesis that dominance is complete but there is no epistasis. The final result in this example is that 4/7 of the actual variation can be gathered into and described by the simple additive hypothesis, 2/7 must be charged to dominance deviations, and only 1/7 to epistatic deviations. It will be seen that the expected values are partly determined by the frequencies with which the genotypes occur. They are partial descriptions of a particular population and may vary from one population to another, even where the same genes are involved. The relative importance of additive genetic variance, dominance deviations, and epistatic deviations will change also. Thus, in the same example but with different gene frequencies the ratio of additive to dominance to epistatic variance becomes 12:9:2 when q_R and q_C are both .6, and 24:8:9 when both are .4.

These illustrations are intended to make clear what is meant by dividing hereditary variation into these three portions. In actual practice the breeder or investigator will not know exactly what genes are present, nor the frequency of each, nor all of the ways in which they interact with each other. Rough estimates of the additive variance can be had from observing the results of selection or the likeness between different kinds of relatives. The additive variation is the part which contributes in the simplest and most direct way to the likeness between relatives. Knowledge of its size is important for estimating the probable results of any proposed breeding plan. For many practical purposes it may not be worth while to separate the dominance and the epistatic portions from each other.

The additive genetic variation caused by a gene can become zero only when the average effect of that gene is zero; that is, when the sum of all the plus changes which it causes is exactly equal to the sum of all the minus changes which it causes in other genotypes in that same population. Most of the variation which a gene causes will be included in the additive portion if its average effect is large. For most of its variation to be epistatic requires that its average effect be near zero but that it produce large plus effects in some genotypes and correspondingly large minus effects in other genotypes. As yet there are only a few actual data to indicate whether epistatic variations are abundant and important, or so rare and small that ignoring them in practice would not cause many errors.

THE MEASUREMENT OF VARIATION

The methods of measuring variation are inconveniently technical for those not trained in statistical methods. Moreover, there are several

of them, and each has advantages for certain purposes. For reasons which do not concern us here, the importance of various causes in producing the variability of a population is most conveniently expressed in terms of the "variance" (σ^2) of that population. The variance may be defined as the average of the squared deviations of the individuals from the population average.⁸ An equivalent definition is that the variance is one-half of the average squared difference between pairs of individuals chosen at random. The square root of the variance is called the "standard deviation" (σ), which, since it is expressed in the same terms as the original measurements, is often more convenient for expressing the variations of individual items than is the variance, which is expressed in squares of the original measurements. In a "normally distributed" population about two-thirds of the individuals differ from the average by less than the standard deviation, while about one-sixth of the individuals will be more than one standard deviation above the average. The remaining sixth will be below the average by more than one standard deviation. Only about one-fortieth of the individuals will be more than twice the standard deviation above the average and another fortieth will be more than twice the standard deviation below the average. In small populations the standard deviation is usually about one-fourth to one-sixth of the difference between the largest and the smallest individuals (the "range"); but that rule is not very accurate, since the range depends on only two individuals and is subject to large sampling errors. The arithmetic average of the deviations, neglecting signs, is about .8 as large as σ , but for various reasons is not as dependable and is almost never used.

Not all populations are "normally" distributed, although most of those encountered in breeding practice are nearly enough so that the statistics of the normal curve may be used with little error for practical purposes. The normal curve is frequently called the "Gaussian curve" after the mathematician who first studied it in detail, or the "curve of error" because its first application, which is still its principal application in some sciences, was in making allowance for unavoidable but random errors of observation. It is symmetrical and bell-shaped, as will be seen in Figure 8.

The statistical cornerstone for the genetics of populations is the "binomial distribution," which is obtained by expanding the expression $(a + b)^n$. This is just the mathematical description of what results

⁸ Since the known average of the n individuals in the sample studied may not be exactly the same as the unknown average of the much larger population from which the sample comes, the sum of the n squared deviations of individuals from the average of the sample is divided by $n - 1$ instead of n to obtain the best estimate of the variance of the population.

naturally from the duplicateness of inheritance and the "one-or-otherness" of gene transmission; that is, of Mendel's laws of segregation and recombination. The Mendelian mechanism guarantees that in a random-mating population the zygotes will be distributed according to the square of the gametic ratio. The genotypes for characteristics determined by n pairs of genes with equal frequencies and equal effects will have the binomial distribution corresponding to $[qA + (1 - q)a]^{2n}$.

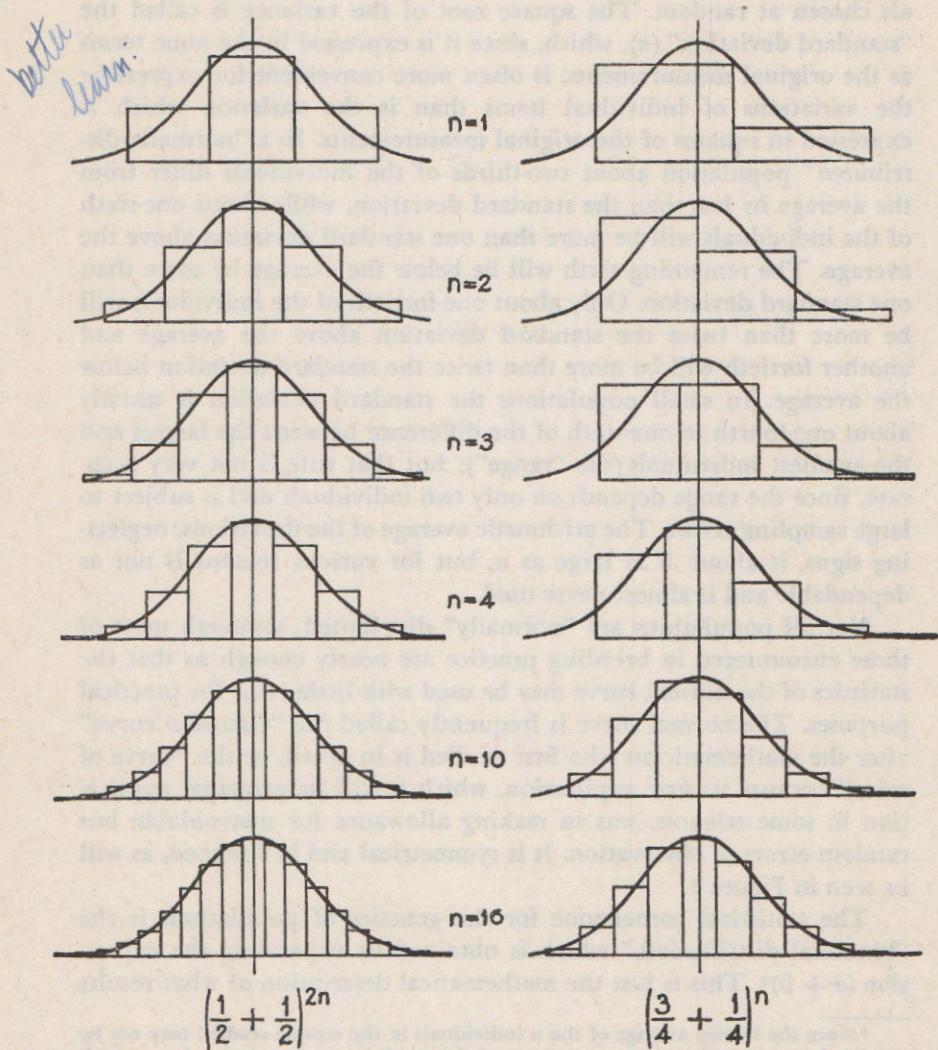
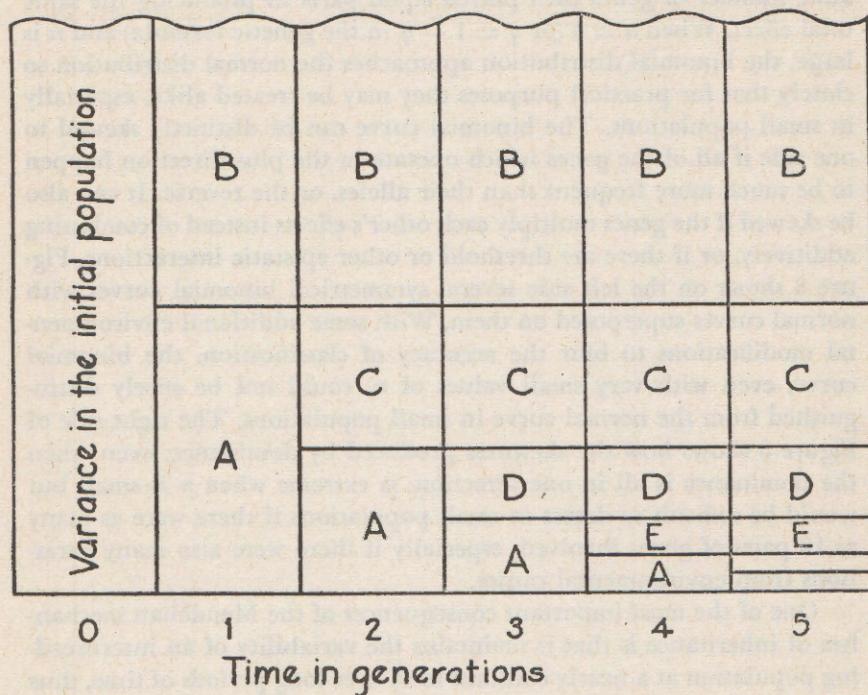


FIG. 8. Binomial distributions for n pairs of genes with equal effects and superposed normal curves with equal mean, equal area, and equal variance. Left: No dominance. Right: Complete dominance in the same direction in all pairs of genes.

If the gene frequencies are not the same for all pairs of genes, the distribution will be somewhat less variable than if they were all equal but had the same average. If the different pairs of genes do not have equal effects, the distribution of the genotypes is more variable than if the same number of genes each played equal parts in producing the same total effect. When $a = b$ (or $q = 1 - q$ in the genetic formula) and n is large, the binomial distribution approaches the normal distribution so closely that for practical purposes they may be treated alike, especially in small populations. The binomial curve can be distinctly skewed to one side if all of the genes which operate in the plus direction happen to be much more frequent than their alleles, or the reverse. It can also be skewed if the genes multiply each other's effects instead of combining additively, or if there are threshold or other epistatic interactions. Figure 8 shows on the left side several symmetrical binomial curves with normal curves superposed on them. With some additional environmental modifications to blur the accuracy of classification, the binomial curve, even with very small values of n , could not be surely distinguished from the normal curve in small populations. The right side of Figure 8 shows how the skewness produced by dominance, even when the dominance is all in one direction, is extreme when n is small but would be difficult to detect in small populations if there were as many as 10 pairs of genes involved, especially if there were also many variations from environmental causes.

One of the most important consequences of the Mendelian mechanism of inheritance is that it maintains the variability of an interbreeding population at a nearly constant level over long periods of time, thus maintaining a supply of variability available for selection or other breeding practices. The importance of this may be shown most clearly by contrasting it with what would be expected under the blending theory of inheritance. Under that theory, if the sire deviated x and the dam deviated y from the mean of the race, *every* offspring from that mating would be expected to deviate $\frac{x+y}{2}$. The average squared deviation (the variance) of the parents would be $\frac{x^2 + y^2}{2}$. The average squared deviation of the offspring would be $\frac{x^2 + 2xy + y^2}{4}$. If the parents mated at random, sires with positive values of x having no especial tendency to mate with dams which had positive values of y , the term $2xy$ would be zero (since the negative terms would cancel the positive ones); and the average squared deviation of each generation would be *only half as large as that of the preceding one*. The group would thus

approach perfect uniformity at a tremendous rate. Even a pronounced tendency for like to mate with like would delay this approach only a little (by causing $2xy$ to have a positive value) unless the tendency of like to mate with like were perfect. Figure 9 shows how rapidly the



- [A] Remaining initial variance
- [B] Variance from mutations in the preceding generation
- [C] " 2nd "
- [D] " 3rd "
- [E] " 4th "

FIG. 9. Rate at which initial hereditary variance was supposed to be lost and the supposed recent origin of the hereditary variations existing at any one time, according to the former theory that inheritance really blended.

hereditary variability existing at any one time would be "swamped" if inheritance really were blending, and how much of the hereditary variability existing at any one moment must have come from mutations which had just occurred in the last few generations, if the variability of

a population were to remain the same from generation to generation. Much of the skepticism about Darwin's theory of evolution by natural selection had its roots in the tacit assumption that inheritance is "blending" and the consequent belief that selection would have to be almost instant and perfect in its seizure of new variations if these were to be incorporated into the species before they were lost. In the older view, heredity was a conserving force and variation was contradictory to it. That is now obsolete as it is seen that the mechanism of heredity conserves individual variation also. Knowledge of Mendelism and of the hereditary variation to be expected between full brothers has freed us from the supposed necessity of thinking that mutations are frequent or important in practical breeding problems. It has also relieved us from the necessity of believing that selection must act almost at once if it is to utilize variations before they are "swamped" or lost. In many respects Mendelism has rounded out the Darwinian theory of the power of natural selection by showing that some of its most serious supposed weaknesses do not exist.⁹

Mendelism gives us a picture of a stable population composed of changing individuals, or of endless individual variations which added together result in an almost constant population. A Mendelian population may be compared to a group of bees around a hive. Almost every bee is constantly in motion and yet the average position of the swarm may remain almost the same hour after hour. The individuals are dynamic—the population is almost static. Changing a population by selection may be compared roughly to driving a herd of many hundred steers. At any one time there are steers moving in all possible directions, yet the drivers, by constantly discouraging those which attempt to move toward the rear and leaving the road open to those which go in the desired direction, can succeed in moving the herd a few miles each day.

The *correlation coefficient* is a measure of how closely two things tend to vary in the same direction. Examples are the tendency in human data for father and son both to be tall or both to be short and the tendency for tall men to weigh more than short men. In both cases the tendency is pronounced enough that it is a matter of common knowledge, even to those who have never heard it expressed quantitatively. In both cases there are frequent and striking exceptions. In the former case the measurements are the same kind (i.e., stature), and they are paired together on account of the genetic relationship of their possessors. In the latter case the measurements (i.e., stature and weight) are different in kind and are paired together because they apply to the same

⁹See pp. 1-12 of Fisher's *The Genetical Theory of Natural Selection*.

individual. The general idea of correlation is simple and universally understood, but the coefficient for measuring degrees of correlation is technical enough that considerable practice in computing it on various kinds of data is usually necessary for proficiency in understanding it. The coefficient of correlation is expressed on a scale running from + 1.0, where two characteristics vary in perfect step with each other, through zero, where there is no correspondence at all, to — 1.0, where there is a perfect tendency to vary in exactly opposite directions from each other.

Regression is the general statistical term for expressing how much one variable may be expected to change per unit change in some other variable. As a concrete example, in dairy cattle regression of daughter's fat yield on dam's fat yield is the average amount of increase in the fat production of the daughter which we may expect for each extra unit of fat the dam produced. Historically regression gets its name from a certain aspect of correlation wherein it was observed that the offspring of extreme parents are usually nearer to the average of the population than their parents were; i.e., they regress toward the mean of the race. This idea of regression of the offspring from the parents toward the mean of the race was later extended to all kinds of equations for predicting one variable from another, where the relation between the two is not perfect. Regression is now used in many cases which do not involve questions of heredity at all and even in cases where the idea of the correlation coefficient would be artificial, as in time trends. The customary symbol for the correlation coefficient, r , was originally chosen because it was the first letter of the word regression. Thus, it is a reminder of the once intimate relation between the two ideas!

SUMMARY

Variation is the raw material with which the breeder works. For purposes of subdivision into its constituent parts, variation is best measured in terms of "variance," which is the average squared deviation of the individuals from the population average. According to its causes, variance may be divided into three main parts: that due to variations in environment (σ_E^2), that due to differences in heredity (σ_H^2), and that due to joint effects of variations in heredity and environment (σ_{HE}^2) which are nonadditive or otherwise intertwined so that they cannot fairly be ascribed to heredity or to environment alone. The hereditary variance may be further subdivided into three portions: the additive genetic variance, which includes all that can be described by assuming that the effects of the whole combination of genes in the individual equal the sum of the average effects of those genes (σ_G^2); the variance

caused by dominance deviations from the additive scheme (σ_D^2); and the variance caused by epistatic deviations from the additive scheme (σ_I^2).

For expressing the variation of individuals, or for expressing differences between expected and actual values, variation is most conveniently expressed in the form of the standard deviation (σ), which is the square root of the variance. In most populations about two-thirds of the individuals differ from the population average by less than the standard deviation.

The tendency for two different characteristics of the same individual, or for the same characteristic in pairs of individuals related in a certain way, to vary in the same direction is measured by the "coefficient of correlation." The equation for predicting the value of one characteristic which will most probably correspond with a given value of another characteristic is called a "regression" equation.

Regression can also indicate that more extensive effects can be anticipated in some directions than in others. Thus, for example, if two traits are correlated in such a way that "height" tends to "increase" while "width" decreases, then the regression coefficient will be negative, since height tends to increase and width to decrease. If, however, the regression coefficient is positive, then height tends to increase and width to decrease. If the regression coefficient is zero, then height and width are uncorrelated.

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CHAPTER 8

Heredity and Environment

In the strictest sense of the word, the question of whether a characteristic is hereditary or environmental has no meaning. Every characteristic is both hereditary *and* environmental, since it is the end result of a long chain of interactions of the genes with each other, with the environment and with the intermediate products at each stage of development. The genes cannot develop the characteristic unless they have the proper environment, and no amount of attention to the environment will cause the characteristic to develop unless the necessary genes are present. If either the genes or the environment are changed, the characteristic which results from their interactions may be changed.

Nevertheless, it is often convenient to speak of a characteristic as "hereditary" or "highly hereditary" when we wish to emphasize that most of the differences we usually see between individuals in that characteristic are caused by differences in the genes they have, and only a few of the differences between individuals are caused by differences in the environments under which they developed. The difference between black and red coat color in cattle is such an example of a highly hereditary characteristic. Environmental circumstances, such as exposure to sunshine, may cause the black to vary from a jet black to a rusty or brownish black: but that is a tiny variation compared with the large difference between black and red which is caused by differences in genes.

With equal logic it is often convenient to call a characteristic "environmental" or "only slightly hereditary" when most of the differences ordinarily found between individuals in that population are caused by differences in the environments under which they developed and only a small part of those differences between individuals are caused by differences in the genes they have. Examples of such largely environmental characteristics are degrees of fatness and of lameness. In most populations variations in those are much more apt to have resulted from previous management, feeding, accidents, or condition of general health than from differences in heredity. Yet it is certain that some indi-

viduals have genes which make them fatten more readily than others, or have genes causing structural weaknesses which predispose them to lameness.

The whole matter of whether a characteristic is hereditary or environmental, if we find it convenient to state it in that way, is a question of how much of the variation in that characteristic in that population is caused by differences in heredity and how much is caused by differences in environment.

The question of whether heredity or environment is the more important can be phrased precisely for a particular trait in a particular population and, if the data are available, can be answered. It does not have a single answer true for all traits in one population nor for the same trait in all populations. Let σ_o^2 = the actually observed variance, σ_h^2 = that part of the variance caused by differences in the heredity which different individuals have, and σ_e^2 = that part of the variance caused by differences in the environments under which different individuals developed. Then¹ $\sigma_h^2 + \sigma_e^2 = \sigma_o^2$ and $\frac{\sigma_h^2}{\sigma_h^2 + \sigma_e^2} = \frac{\sigma_h^2}{\sigma_o^2}$ = the portion of the observed variance for which differences in heredity are responsible. When this fraction is large, we say that the characteristic is highly hereditary; when this fraction is small, we call the characteristic slightly hereditary or largely environmental.

The value of $\frac{\sigma_h^2}{\sigma_h^2 + \sigma_e^2}$ can be altered by changing either σ_h^2 or σ_e^2 .

If we try to make the environment exactly the same for all individuals, as is usually attempted in genetic experiments, we may go far in that direction although we can hardly hope to control the environment perfectly. So far as we succeed in making σ_e^2 smaller than it is in the general population, we make the variations in that characteristic more highly hereditary in our material than they were in the general population. If we also enlarge σ_h^2 in our material by mating like to like while selecting for opposite extremes or by inbreeding in several separate lines, we increase σ_h^2 and make variations in that characteristic in our material still more highly hereditary. Unless we are quite aware of what we have done in partially controlling the environment (and thus making σ_e^2 tend toward zero) and in selecting or breeding to increase the genetic diversity of our laboratory material, we are apt to get an exag-

¹ In order not to confuse the argument, the nonadditive interactions of heredity and environment are neglected here. There is reason to think that those joint effects will generally be small. This definition includes as "hereditary" the dominance and epistatic deviations since they result from differences between whole genotypes, although they will not contribute so much to the likeness between relatives as the additive differences do.

gerated idea of the importance of heredity in causing variations in that characteristic in the general population. On the other hand, if we are experimenting with the effects of some procedure in nutrition or in management, we will probably try to make σ_H^2 as small as possible by using a uniform stock, discarding at the beginning of the experiment any which appear to deviate much in either direction from the population average, and minimizing the hereditary differences between lots by putting litter mates in different lots, etc. Then we will also make our experimental (environmental) treatments so contrasting that we will be reasonably sure to find differences in their results. Again, unless we are quite aware of what we did in neutralizing hereditary differences and in magnifying environmental differences in our laboratory material, we are apt to get an exaggerated idea of how important our environmental differences are in causing the variations in the general population. If this were clearly understood, a considerable amount of fruitless controversy would be avoided.

A quantitative statement of the relative importance of heredity and environment is a partial description of the causes of the variation in a particular characteristic in the specified population. It is useful in estimating the probable results of certain breeding systems in the next generation or two, but it tells nothing about the ultimate limits of the changes which might be made in that population either by breeding or by altering its environment.

METHODS OF ESTIMATING HERITABILITY

All methods of estimating heritability rest on measuring how much more closely animals with similar genotypes resemble each other than less closely related animals do. The techniques suitable for doing this vary with the material and according to whether environmental correlations between relatives and the peculiarities of the mating system, if it was other than random, can be measured and discounted by other means.

Variation within isogenic lines is wholly environmental. Comparing this with the variation in an otherwise similar random breeding population may give an estimate of heritability. This method is of little use in farm animals because among them are no isogenic lines except occasional pairs of identical twins. These are difficult to identify but if sought diligently and studied intensively might finally give reliable information. Identical twins are to be compared with ordinary twins, rather than with pairs of individuals unrelated to each other, lest the similarities in the environments of twins might lead to errors in the interpretation. The method of isogenic lines is the only method likely

to measure all of the epistatic and dominance variations as well as the additive ones but, because of the rarity of identical twins in farm animals, is not promising as a source of information fairly free from sampling errors.

In selection experiments, if we can measure the amount by which those selected to be the parents exceeded the average of their generation, we can divide that into the amount by which the average of the offspring exceeded the average of the generation in which their parents were born. This gives a measure of the additive portion of the variance plus a portion (somewhat less than half) of the epistatic variance. In order not to be misled by unnoticed environmental changes, it is usually necessary that selection be practiced in opposite directions at the same time, so that the interpretation will be based on differences between the high and the low lines, rather than on the absolute values of the averages. Sometimes this method can be followed in experiments especially designed for this purpose at some research institution, but it is not often available to the breeder, since he can rarely afford to select in the undesired direction just to get information on heritability.

The resemblance between parent and offspring is the most widely useful method, but is likely to include some environmental correlation between parent and offspring. Also it will include something from the resemblance of the offspring to the other parent, if mating were not random. In using this method the procedure is as follows: (1) observe the correlation between parent and offspring, (2) subtract from that the environmental contribution, (3) double the remainder, and (4) divide by one plus the correlation between mates.² The second step is always likely to be difficult, and the fourth will be unless the deviations from random mating are known more exactly than is usually the case.

A useful dodge which makes steps two and four unnecessary is to divide the mates of each sire into a high and a low half on their own performance, combine the data for all sires, divide the difference between the daughters of the high and the low halves by the difference between the two groups of dams and double the results. This measures the additive portion and a bit of the epistatic portion of the differences between such dams as were mated to the same sire. It leaves unanalyzed the differences between the groups of cows which get mated to different sires. A similar division of the offspring of the high and low sires mated to the same dam would answer as well in principle but, because of the

² This should be the genetic correlation (coefficient of relationship) if the departures from random mating were of the inbreeding kind, but should be the actually observed correlation if the mating choices were based on each animal's own individuality for the characteristic being studied. One will rarely be certain about this.

usually small number of offspring per dam, is rarely possible with farm animals.

One of the first clearly analyzed cases of the relative importance of heredity and environment was Wright's study of the amount of white spotting in a stock of guinea pigs. It will illustrate the principles and the use of nearly isogenic lines and of correlation between relatives. Besides the control stock, in which even second cousin matings had been avoided, there was a stock which came from the same foundation but which had been inbred full brother and sister for more than 10 years (probably about 20 to 25 generations in most branches of the family, nearly all of which came from a single mating in the twelfth generation), so that it must have been almost entirely homozygous and could have retained but little genetic variability. By measuring the average likeness between parents, between parents and offspring, and between litter mates, Wright was able to separate the variance into a portion due to heredity, a portion due to environment common to litter mates, and a remainder due to environment or embryological accidents which were not alike even for litter mates. Table 6 shows the findings. The

TABLE 6
PIEBALD SPOTTING IN GUINEA PIGS. PORTIONS OF THE VARIANCE ACCORDING TO CAUSES
(AFTER WRIGHT)

Causes of Variance	σ^2 in the Inbred Stock		σ^2 in the Control Stock	
	Actual Units	Per-cent age of Total	Actual Units	Per- centage of Total
Heredity.....	.010	2.8	.271	42.2
Environment common to litter mates.....	.020	5.5	.002	.3
Environment not common to litter mates.....	.334	91.7	.370	57.5
Total.....	.364	100.0	.643	100.0

most illuminating fact for our present purpose is that the variance due to environment was almost the same in actual units of measurements (.354 and .372) in both stocks but was 97.2 per cent of the variance in inbred stock and only 57.8 per cent of the variance in the control stock. Here is a case where the same characteristic in two separate stocks derived from the same colony by different breeding methods is very slightly hereditary (2.8 per cent) within the inbred stock and nearly half (42.2 per cent) hereditary within the control stock. All that really happened was that in one stock the inbreeding had caused nearly all of the initial hereditary variability to be lost and thereby had altered

greatly the proportion of hereditary to environmental variability.

The following are some examples of the kind of analysis which can be made by comparing correlations between relatives. Gowen studied Jersey Register of Merit data on milk yield and fat percentage, assuming that there was no correlation between the environments of daughter and dam. He came to the conclusion that about 50 to 70 per cent of the variance in milk production and about 75 to 85 per cent of the variance in fat percentage came from variations in the heredity of the individual cows. But if there was as much as .10 to .20 of environmental correlation between daughter and dam, as seems probable from the usually observed correlation between the records of herd mates, these figures are too high by .20 to .40. Plum's analysis of the records of cows in Iowa Cow Testing Associations led him to the figures shown in Table 7. Studies of intrasire regression of daughter on dam have gen-

Importance
TABLE 7
RELATIVE IMPORTANCE OF CAUSES OF VARIATION IN BUTTERFAT PRODUCTION

Causes of Variation	Percentage of Total Variance
Breed.....	2
Herd	
(1) Feeding policy of herd.....	12
Other causes (genetic or environmental)	21
	33
(2) Cow (mostly genetic).....	26
Residual (year to year variations)	
(3) Feeding variations within the herd.....	6
Other year to year differences.....	1
Length of dry period.....	1
Season of calving.....	3
Other factors.....	28
	39
Total.....	100

erally given values of around .15 to .30 for heritability of differences in fat production between cows in the same herd where each cow was represented by only one record.

Lush, Hetzer, and Culbertson, studying the birth weights of pigs born during 15 years at the Iowa Agricultural Experiment Station, came to the figures shown in Table 8. Part of the 29 per cent due to "other" environment common to litter mates may really have been hereditary as the result of hereditary differences among the dams, although it was environmental as far as the pigs themselves were concerned. There is much evidence that the dam's own size or other characteristics have much influence on the birth weights of her offspring.

The figures in Tables 7 and 8 illustrate how the actual data may permit subdividing the environmental variance into portions caused by certain tangible factors or groups of factors. Nearly always a considerable part of the variance will remain unidentified as to causes. These are most naturally inferred to have been individual unobserved (or at least unrecorded) variations in environment, but in some cases may really have been errors in observations or (in some methods of analysis) will also have included those portions of the epistatic or dominance variations which did not contribute to the likeness of the relatives studied. For other examples similar to Tables 6 to 8, yet each showing some special features of its own, see: *Genetics* 19:535; 21:360; 22:468; 26:217; 31:503; and *Onderstepoort Jour. Vet. Sci. and An. Husb.* 5:580.

While it is true that the animal at birth contains all the heredity it is going to have but has not yet been affected by many of the environmental circumstances which will affect it, yet the presence or absence of differences at birth is not a good criterion of whether those differences

TABLE 8
RELATIVE IMPORTANCE OF CAUSES OF VARIANCE IN BIRTH WEIGHTS OF PIGS

Causes of Variance	Percentage
Heredity of the pigs	
Breed differences.....	2
Sex.....	1
General.....	3
	6
Environment common to litter mates	
Litter size.....	7
Year.....	5
Ration.....	4
Gestation length.....	2
Other.....	29
	47
Environment not common to litter mates.....	47
Total.....	100

are hereditary. Some of the differences found at birth are the result of previous differences in intra-uterine environment, or of what for lack of a better term may be called embryological accidents. On the other hand, many genes in which individuals may differ do not produce their effects until the individual reaches a certain stage of development. Examples are the genes which affect early maturity, milk production, shape and quality of teeth, and in man such specific things as baldness, prematurely gray hair, and Huntington's chorea.

PRACTICAL APPLICATIONS

Much individual variation is left even when either the heredity or environment is perfectly controlled. For example, if half of the variance in a characteristic is hereditary and half is environmental, perfect control of heredity would still leave the standard deviation 71 per cent (the square root of one-half) as large as before. If all environmental variations in a characteristic which is 80 per cent hereditary were eliminated, the standard deviation would still be nearly 90 per cent (the square root of 80 per cent) as large as before. If the hereditary variations were entirely eliminated, the standard deviation would still be about 45 per cent as large as before. Even if a characteristic were 99 per cent hereditary, complete elimination of all hereditary variability would still leave the standard deviation 10 per cent as large as it was originally.

Only those variations which are caused by differences in heredity are themselves inherited. Variations caused by environment can be large and very important economically, but they do not change the inheritance of the animal and are not transmitted to its offspring but must be produced afresh in those offspring by repeating the environmental treatments which produced them in the parent. There is not space here to repeat the proofs for the noninheritance of environmental effects; and, as with other negative concepts, it may be impossible to prove this one rigorously. But the many experiments carefully planned to test whether the effects of environmental treatments are inherited in such a way that the offspring inherit some degree of the modifications originally produced in their parents by the environmental treatment have all given negative or doubtful results. Even one who deliberately wishes to believe that environment does affect heredity in this way must admit that the effects are so slight that they are not practically important in any one generation.³ Perhaps they do not occur at all. Both in improving the heredity and in improving the environment of his animals the breeder is likely to encounter the law of diminishing returns. Yet in improving their heredity there is the possibility that if he can achieve enough—for example, get his herd widely known as one of the three or four best sources of breeding stock in the whole breed—he may come again to a zone of increasing returns because of the high prices he will receive. The competition to get into that zone is usually very strong, however.

Improvements in heredity are permanent⁴ and each generation

³ Certain extreme environments, such as exposure to X-rays, or to barely sub-lethal temperatures, or to radium, do increase the mutation rate.

⁴ Except for gains in the epistatic effects. Those tend to disappear as the genes recombine. One must keep on selecting to hold them.

stands on the shoulders of the preceding one, whereas improvements in the environment produce almost their full effect on the animals for which they are first made. Each new generation must again receive the improved environment or the gain will be lost. Hence, in the long run it may be profitable to spend considerable effort to make small improvements in heredity, since the expense of making such improvements in one generation may yield dividends for many generations. The expense of making improvements in heredity (so far as those are additive) is a capital investment; the expense of making improvements in environment is an operating expense. Naturally the breeder will wish to do both so far as they are profitable.

Besides the economic value of its direct effects on the animals, environment needs the practical breeder's attention in two ways: First, the animals should be kept in an environment which will permit them to show readily which of them come nearest to having all the genes which have effects the breeder wishes. Second, the breeder should observe carefully the environment which applies to each animal so that he can allow for this when making his selections. It is usually impossible to disentangle the effects of environment completely from the effects of heredity in individual cases, but some effort spent in trying to do that will often do much to make selections more accurate and progress more rapid.

Breeding animals should be kept under environment like that for which their offspring are being bred. If animals are being bred for resistance to unfavorable conditions, they should be kept under unfavorable conditions so that the breeder will have a chance to learn which ones have the genes that will make them most nearly what he wants.⁵ If cows are being bred for specialized and intensive dairying, they should be well fed and milked three times a day, provided those are the conditions under which commercial cows are to be kept in the specialized dairying of the future. To feed them poorly or milk them only twice a day would prevent some of the genes, useful under the more specialized

⁵This reaches its extreme form in breeding for disease resistance, a practice to which is now devoted a large portion of the efforts in plant breeding, but which is still in the laboratory stage in animal breeding except where (as, for example, in the tropics or in the breeds of sheep native to marshy regions) some effective natural selection has been practiced automatically. For fewest mistakes when breeding for disease resistance, the exposure or inoculation dose should be severe enough that somewhere between 30 and 70 per cent of the animals would contract the disease. That may not be practical for diseases which have a high mortality. Those which cause only a low mortality may not be important enough to warrant much effort in breeding for disease resistance. Animal breeders now look first of all for prevention, vaccines, or medicaments as a more economical way out. But if those fail and we are driven to breed for disease resistance, the above considerations show how far we will have to depart from the (at present) more orthodox practices of sanitation and efforts to prevent exposure.

conditions, from showing their presence. On the other hand, to force the cows by extravagant feeding and by such extreme practices as milking four times a day would magnify the differences between their production records and to that extent would be a help in selecting animals adapted to these conditions; but for most practical purposes this gain would probably be more than offset by the fact that some of the cows would respond more than others to those forced conditions, without there being a corresponding increase in what they would produce under the usual conditions for which they are being bred. The breeder of dual-purpose cattle will make fewer mistakes in his selections if his breeding cows are tested under twice-a-day milking and other management like that under which he expects their descendants to be used.

The question of testing under forced conditions or under ordinary conditions can perhaps be clarified by an analogy. If an athletic coach were to examine all the men in a college to find the best runners for his track team and were to test them in a race of only one length, such as a two-mile race, it is true that most of those who did well in this long race would also do better than average in the short races; yet there would certainly be some who had not the endurance for the long race but could do well in the 100-yard dash. There would be others who could win in the long race but have not the bursts of speed necessary to make them good performers in the short races. In brief, each man has a certain amount of general ability to run, and that will manifest itself in races of all lengths; but each individual also has certain special abilities or disabilities which will help him or hinder him in certain lengths of races but not in others. In order to find the very best runners for each kind of race the coach will need to test them in that kind of race. Yet if the correlation between their abilities at one length of race and at others is high, perhaps he might conveniently eliminate half or more of the whole group from further consideration, by trying them on one kind of race, without much risk of losing a runner who would be really outstanding at some of the other lengths of races. Likewise with farm animals there is doubtless much correlation between an animal's ability to do well under many different environments, but that correlation is far from perfect. The genes which enable it to do well or poorly in a certain environment will manifest themselves most clearly only when the animal is kept under that environment. In the writings about race horses there is much mention of "stayers" and "sprinters," indicating that many horses are good in one of these respects but not in the other. Also they sometimes speak of "mudders" which can do well on a wet track but are outclassed by many others when the track is dry.

If a breeder can foresee that general conditions of management are

going to change in a certain way in the future, it is of course to his advantage to change his conditions of testing now, so that, when the general change comes, his animals will already have been selected for a generation or two toward adaptability to those conditions. In doing so, of course, he runs the risk that his prediction of the coming change may be wrong. If it is, his stock may be changed farther away from the real goal of the future than if he had made no attempt to foresee a change.

Also, a high record made under forced testing has considerable advertising value. Not all of the potential customers will discount this high record as much as they should for the environmental circumstances under which it was made. While this may even be a hindrance to breed improvement, yet in some cases it has a commercial value to the individual breeder which he cannot afford to ignore.

Mistaking the effects of the environment for the effects of genes dulls the keenness of selections, makes the breeder sometimes save animals he would cull if he knew what genes they had and sometimes cull animals which have better genes than most of those he saves. This source of error is very important for such things as growth rate, fertility, health, vigor in general, size of fleshy parts, ability to fatten, etc., which are economically important and physiologically complex and often much modified by environment. The most important practical consequence is a regression of the offspring toward the mean of the race. That is, the offspring of parents which are extreme in either direction will not usually average as extreme as their parents. The simplest quantitative expression for this is that for each unit which the selected parents average above the mean of their race, their offspring will most probably

average about $\frac{\sigma_H^2}{\sigma_H^2 + \sigma_E^2}$ as far above. This would be literally true if the

genes all combined their effects additively. In actual fact there will be some dominance and some nonadditive gene interactions which will produce more regression toward the mean of the race than this formula shows.⁶ Mistaking the effects of environment for the effects of genes, next to matters of health and fertility in some species, is usually the biggest obstacle to the breeder's rapid progress toward his chosen goal.

The remedy for confusing the effects of environment with those of heredity is either to control the environment physically by eliminating variations in it, testing all animals under standard conditions and thus reducing σ_E^2 toward zero, or to control the environment statistically by using correction factors to allow as best one can for unusual individual

⁶ The formula would be more nearly correct if the numerator were only the additive genetic portion of the variance; but that is a slight understatement of the case, since a portion of the epistatic variance also belongs in the numerator.

circumstances when judging what each animal would have been under standard environmental conditions or what the difference between two individuals would have been if they had been under the same environments.

Physical standardization of the environment can never be perfect. Partial control may be too expensive to carry far. Statistical control may actually introduce errors through use of the wrong correction factors. Yet so far as one makes allowances or corrections which are more often right than they are wrong, he will eliminate more of σ_{E}^2 than of σ_{H}^2 . Therefore a larger fraction of the variance in his corrected or adjusted estimates will be hereditary than was true of the variance in the original observations. Such allowances for differences in environmental conditions may vary from the vaguest kind of a mental allowance to intricate correction factors which sometimes approach the limit where the increases they make in the accuracy of selections do not pay for the labor of making the corrections. The man who sees his animals every day has an important advantage over the man who does not work with them himself and sees them only at rare intervals or knows them only through the report of his herdsman, since the former knows the environmental differences better and can make fairer allowance for them. The man who works with his animals daily is, however, more likely to make too much allowance for his favorites without being aware that he is doing so.

That the offspring of extreme parents generally are nearer to the average of the breed than their parents were (especially as concerns characteristics of low heritability), does not automatically make the breed become more uniform as time passes. The offspring from each parent will vary among themselves and environment will shove some of them far up and others far down. The extreme ones thus produced will replenish the supply of extreme individuals in the next generation.

In medical writings a distinction used sometimes to be made between "hereditary" and "familial," the former referring to cases where the offspring was obviously like one parent, and the latter to traits (such as recessives or those with low "penetrance") which "run in families" but in which the individual might not resemble either parent. Progress in genetic knowledge has now made that distinction obsolete.

"Hereditary" in the broad sense of the word has nothing to do with abundance or scarcity of a characteristic or with dominance, although some methods of estimating heritability do not gather up the variance due to dominance deviations. Black is no more and no less hereditary than the much rarer red in black breeds of cattle; rather the question of heritability concerns the cause of the contrast, black versus red.

SUMMARY

1. All characteristics are both hereditary and environmental in the strictest sense of those words.
2. Characteristics called hereditary for convenience are those for which most of the usual differences between individuals are caused by differences in the genes those individuals have.
3. Characteristics called environmental or nonhereditary are those for which most of the differences between individuals result from differences in the environments to which the individuals were exposed.
4. The effects of environment are not inherited except as extreme environments (like heavy X-ray radiation) produce mutations, and those are not *adaptively* related to the environment which produced them.
5. The breeder should keep his animals under the environments in which they and their descendants are intended to be used so that the desired genes may have a chance to express their effects and be recognized for selection.
6. The breeder will often mistake the effects of environment for those of genes and will thus make mistakes in his selections. Such mistakes are usually the most important cause of the fact that the offspring of selected extreme parents average nearer than their parents to the mean of their race.

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which the two breeds most closely related to the cow at hand. If one of these breeds is the same as the cow, then probably other relations have been omitted. In this case we know our test is just misleading.

CHAPTER 9

The Nature of Differences Between Breeds (or Races or Species or Other Groups)

Averages vary less than the individual items on which they are based. This happens because the items averaged together are not all alike and their variations partly cancel each other. The average of a sample of n individuals will differ from the average of the population from which it came by one n th of the sum of the individual deviations which did not happen to be canceled. If the sample is selected at random, that difference will be small, especially if the sample is large. The variance of the averages of such random samples is one n th as large as the variance of individuals. But, if the sample is selected by some method which tended more often than not to choose individuals with plus (or with minus) deviations, those individual deviations will be prevailingly in the same direction and will not cancel each other completely. Instead, the sample average will tend to be different from the population average by an amount determined by the method of selection. In such a case the variation within the sample will be less than if it had been selected at random.

As a numerical example we may take annual butterfat production, for which the standard deviation among cows in Iowa cow testing associations is not far from 100 pounds. We are not much surprised if we choose two cows at random and find that their records differ by as much as 100 pounds. In fact, nearly half of all such pairs would differ by that much or more. But we would be surprised if the average of two groups of 10 cows, each selected entirely at random, differed by as much as 100 pounds. We would not expect that to happen oftener than once in about 20 or 25 such comparisons. If it did happen, we would wonder whether the two groups really had come from the same population or whether they had been selected in some biased way which tended to bring higher records into one group and lower ones into the other. If the one set had all been selected from one herd and the other set had all come from another herd, we would not be so surprised by that big a difference, since good or poor management or breeding in either herd

would tend to shove all the records from that herd up or all down together. In choosing only two herds at random it might easily happen that we would get one herd with poor management and another with good management; while, if each record came from a different herd, it is not at all likely that the 10 in one set would all come from well-managed or well-bred herds and the 10 in the other set would all come from poor herds, unless there was some difference in the method of choosing the records for each set.

Because averages are less variable, it is often possible to be sure that there is a real difference between two groups which have averages not very different and in which the individuals vary so widely that the two groups overlap in much of their range. This is the general situation for most breed differences, especially for differences in economically impor-

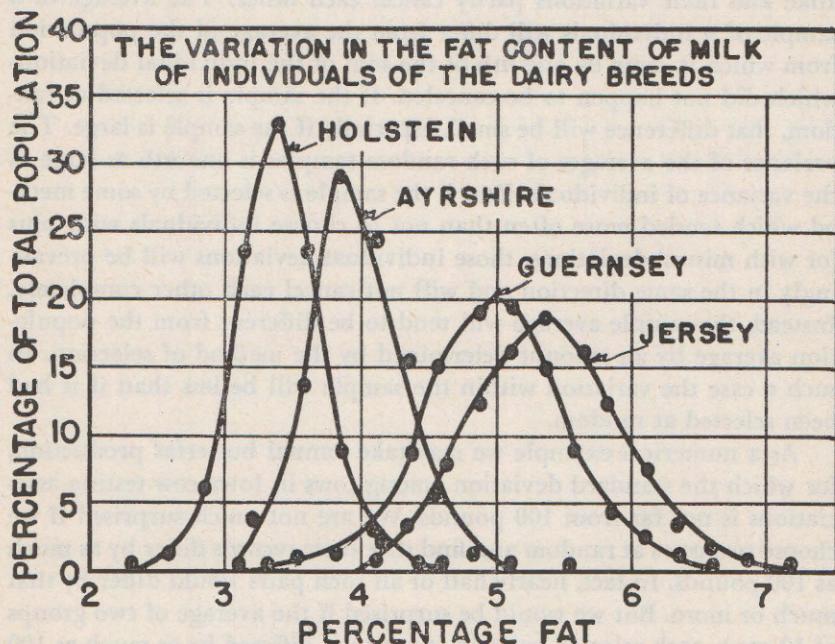


FIG. 10. Distributions of individual cows in four dairy breeds according to the percentage of butterfat in their milk. The breeds overlap, but the differences between their averages are real. (Adapted from Bul. 365 of the Mo. Agr. Exp. Station.)

tant and physiologically complex characteristics like milk production, fertility, size or shape of muscular parts, etc. Figure 10 shows this for percentage of fat in the milk of four breeds of dairy cattle. For some characteristics, notably color or details of bone dimensions or conformation, there may be no overlapping at all between breeds. Figure 11

illustrates graphically the relation between individual differences and group differences. The fairly common saying, "There is more difference within breeds than there is between breeds," is often true in the sense that the difference between the breed averages is small compared with many of the differences between individuals which belong to the same breed. This saying is quite misleading, however, if it is interpreted to mean that the differences between breeds are not real after all. Group differences of the same kind as are illustrated in Figure 11 often prevail between races and may prevail between families or other groups within a breed.

It is to be expected that breeds which have been kept separate from each other in their ancestry for many generations will usually have drifted apart in many of their characteristics on account of the sampling variations of Mendelian inheritance in small populations, or will have been drawn apart by selection which has not been equally successful in

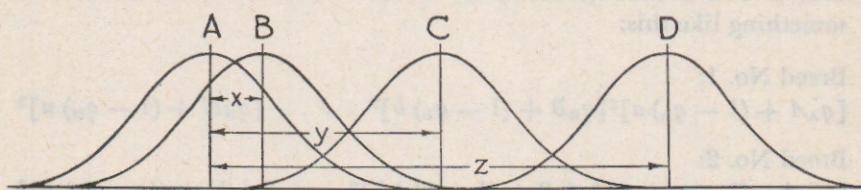


FIG. 11. Overlapping frequency curves showing the nature of differences between population averages and differences between individuals. x is the difference between the averages of populations A and B , which overlap in most of their ranges. y is the difference between the averages of populations A and C , which overlap only a little. z is the difference between the averages of populations A and D , which do not overlap at all. If two individuals are chosen at random from one population, the difference between those two will be larger than x in far more than half of such pairs and in a few pairs will be larger than y .

both breeds or has not been directed toward exactly the same ideals. Breed differences may often be so small that they are economically unimportant, especially if the breeds have been selected toward almost the same ideals; yet it would be a remarkable coincidence if the breed averages were exactly the same for any characteristic. Even where two breeds have been selected toward the same ideal and their phenotypic averages are almost the same, the sets of genes by which that phenotype is produced are likely to have become qualitatively different if the two breeds have been kept entirely from any crossing with each other for tens of generations.

The genetic basis of differences between breeds may be of two kinds. In the first place, one breed may be homozygous for one gene and another breed may be homozygous for an allele of that gene. If that were

true for all genes, we could write the Mendelian formulas of two breeds as follows:

Breed No. 1	$AABBccddEE \dots NN$
Breed No. 2	$aabbCCddEE \dots nn$

This would indicate that the two breeds are homozygous for different genes in the series for A , for B , for C and for N , but are alike in the series for d and for E . This conception of breed differences appears to have been rather widely held by those who discussed the possible practical applications of Mendelism in the early years of genetics, but it is expressed less frequently now, as the evidence accumulates that few genes are homozygous in all members of the breed. The other kind of genetic situation which may be the basis for distinct breed differences is that a pair of genes is not entirely homozygous in either breed but the proportion of one gene to its allele may be widely different in the two breeds. The Mendelian formulas for the two breeds could be written something like this:

Breed No. 1:

$$[q_A A + (1 - q_A) a]^2 [q_B B + (1 - q_B) b]^2 \dots [q_N N + (1 - q_N) n]^2$$

Breed No. 2:

$$[q'_A A + (1 - q'_A) a]^2 [q'_B B + (1 - q'_B) b]^2 \dots [q'_N N + (1 - q'_N) n]^2$$

The blood groups in the Guernsey and Holstein-Friesian breeds of cattle are a good example of this very situation (*Jour. Ani. Sci.* 3:315-321. 1944). In an extreme case of this sort, such as is illustrated in Figure 12, two breeds might be alike in the sense that every kind of gene which exists in the one also exists in the other, and yet be distinctly different outwardly and in average genotype. Genetic differences of both kinds probably exist between most breeds. Really, the first kind of difference is only an extreme limit of the second where $q = 1.0$ in one breed and is zero in the other.

A complete description of a breed involves not only a statement of the genes for which it is homozygous and different from other breeds but also a statement of the frequencies in it of genes for which it is not homozygous and which other breeds may also possess in larger or smaller frequencies. Thus, a complete description of a black breed of cattle may contain, besides the statement that the "type" (the most frequent kind) is black, the statement that in this breed the frequency of gene B is .93 and of b is .07. Perhaps in another black breed the frequency of B may be .97 or perhaps only .80, while in a breed like the Shorthorn, where solid black is unknown, the frequency of B is .00. Perhaps other