THE HERITABILITY OF ALL-OR-NONE TRAITS:
VIABILITY OF POULTRY

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O NE of the fundamental steps in any analysis of the inheritance of quantitative characters is the partitioning of the total phenotypic variance of the trait under study into its genetic and environmental portions.

The various techniques for this purpose for characters showing continuous or graded types of variation are well known (see WHATLEY 1942). They are based on methods of variance and correlation analysis derived in the main from the studies of WRIGHT (1921), although many phases of the general problem were attacked earlier by WRIGHT (1917) and several other investigators (among them WEINBERG 1909; FISHER 1918 *et al.*). In the case of characters whose phenotype is expressed in an all-or-none manner these methods do not directly apply. However, special techniques based on the inverse probability transformation have been used in this connection by WRIGHT (1934a, 1934b, 1943), while more recently LUSH, LAMOREUX and HAZEL (1948) considered the problem in a more direct relationship to applied animal breeding practice. Reference to the methodology and results of this investigation will be made in the course of discussion. It may also be noted that at least one other attempt at what may be interpreted as the estimation of heritability of an all-or-none trait (multiple births in cattle) is on record, an attempt which, however, did not consider fully the difficulties of application of the analysis of variance to binomial data (KORKMAN 1948).

It is actually possible to derive a simple technique for dealing with all-or-none data by considering certain properties of the "degree of heritability," which for quantitative characters may be defined as the proportion of the phenotypic variance due to additively genetic differences between individuals. The purpose of this communication is to describe a method of approximate determination of the degree of heritability of traits expressed in an all-or-none manner, and to discuss briefly the significance of the results obtained. The derivation presented is applied to raw percentage data as contrasted with similar formulas developed by WRIGHT (*loc. cit.*) and applied to transformed data.

The problem was suggested by the junior author who also supplied the material, the general solution and actual analysis having been carried out by the senior author, while the responsibility for the presentation and discussion is shared by both.

MATERIAL

The data used in the present study consist of the 18-months mortality records for 12 years of a flock of Single Comb White Leghorns, maintained at the UNIVERSITY OF CALIFORNIA in Berkeley. Detailed descriptions of the selection procedures used in breeding for egg production and other pertinent information regarding the flock have been presented elsewhere (TAYLOR and LERNER 1938; LERNER and HAZEL 1947; DEMPSTER and LERNER 1947). It may be sufficient to say here that the main efforts in the breeding program were directed towards increasing the first-year egg *production index* (average hen-housed egg production), which represents a combination of egg production, viability, and the interaction between them.

The only other point in connection with the data which requires comment is the fact that all of the birds which died in the first laying year were subjected to post-mortem examination. The birds which were not kept after the end of the first laying year (approximately 18 months of age) were killed and autopsied. Any animals in this category which exhibited significant pathological lesions were included in the group of dead rather than surviving birds. The autopsies were performed by the Department of Veterinary Science of the UNIVERSITY OF CALIFORNIA in all of the years, except for a period during the war, when some were carried out by members of the Division of Poultry Husbandry. In addition to the study of the heritability of total first year mortality, two specific types of pathological disturbance are also discussed. The first is lymphomatosis, a complex of neoplastic diseases of particular importance in the commercial field (see TAYLOR *et al.* 1943, for a report on breeding investigations on resistance to lymphomatosis in this flock), while the second may be termed *reproductive* disorders, consisting of various types of breakdown of the genital system. This type of pathological involvement represented the major single cause of loss in the flock under study (1083 from a total of 5064 birds involved) while lymphomatosis accounted for 411 deaths from a total of 2107 deaths recorded.

THE DETERMINATION OF HERITABILITY

The change in the average genotype for a given character in successive generations under selection is a function of the heritability and the selection differential. Hence, if this change in successive generations and the selection differential are known, it is possible to estimate the heritability. In other terms the heritability is the regression of genotype on phenotype and can be determined with equal accuracy, whether or not the distribution of the independent variable (in this case, the phenotype) is normal.

With respect to mortality in a population in which no artificial selection is applied, there exists a natural selection differential for viability since birds with a genotype for low viability will have a lower chance of surviving to become parents of the next generation than the birds with a genotype for high viability. The improvement in the average genotype by natural selection may easily be calculated and allows a ready determination of the degree of heritability of mortality.

Let the genotypic values for viability of members of one generation be $p_1, p_2, \cdots, p_n$, with the mean $\bar{p}$ and the variance σ_p^2. That is to say that p_1, p_2, etc. are the probabilities of survival of the corresponding genotype under the postulated given array of environmental conditions to which the birds are exposed. The phenotype for survival of the m-th bird will then be $p_m + e_m$ where e_m is the environmental component, and includes all factors determining the phenotypic value with the exception of the genetic one. This phenotype will necessarily take the value of either 0 or 1. The mean genotype of survivors, which may be designated as $_1\bar{p}$ to distinguish it from the mean genotype of all birds ($\bar{p}$), is then

$$_1\bar{p} = \frac{\displaystyle\sum_{m=1}^{n} p_m(p_m + e_m)}{\displaystyle\sum_{m=1}^{n} (p_m + e_m)} .$$

Since the expected values of

$$\sum_{m=1}^{n} e_m \quad \text{and} \quad \sum_{m=1}^{n} p_m e_m$$

are equal to zero, if birds are kept under conditions in which there is no correlation between genotype and environment, we have

$$E(_1\bar{p}) = \frac{E\left(\displaystyle\sum_{m=1}^{n} p_m^2\right)}{E\left(\displaystyle\sum_{m=1}^{n} p_m\right)}$$

where E denotes the "expected value" for the expression in parentheses.

The expected gain over the previous generation is[1]

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[1] It may be helpful to give a broader form of derivation in addition to the above. If g_m is taken as the total genotypic value of the m-th bird, p_m as its additively genotypic value, and e_m as the environmental contribution, the additive genotype of the survivors is $\dfrac{\sum p_m(g_m + e_m)}{\sum (g_m + e_m)}$. The expected values of $\sum p_m e_m$ and of $\sum e_m$ will by definition be zero. Hence, the additive genotype of the survivors and the mean additive genotype of the next generation is $\dfrac{\sum p_m g_m}{\sum g_m}$. The improvement in the mean genotype then is $\dfrac{\sum p_m g_m}{\sum g_m} - \bar{p}$. Since $\bar{g}$ here equals $\bar{p}$, the expectation for the gain is $\dfrac{E[g_m(p_m - \bar{p})]}{\bar{p}} = \dfrac{\sigma_p^2}{\bar{p}}$, as given in the text.

$$E(_1\bar{p} - \bar{p}) = \frac{E\left(\sum_{m=1}^{n} p_m{}^2\right)}{E\left(\sum_{m=1}^{n} p_m\right)} - \bar{p}$$

$$= \frac{n(\bar{p}^2 + \sigma_p{}^2)}{n\bar{p}} - \bar{p}$$

$$= \frac{\sigma_p{}^2}{\bar{p}} .$$

The phenotypic selection differential applied in this case is then $1 - \bar{p}$, being the difference between the phenotype of the survivors (taken as unity) and the mean phenotype of the population. Therefore,

$$\text{heritability} = \frac{\text{genetic improvement}}{\text{phenotypic selection differential}}$$

$$= \frac{\sigma_p{}^2}{\bar{p}(1 - \bar{p})} .$$

This gives an expression for the heritability in terms of the genotypic variance for viability and the mean viability, an expression which also can be derived more directly (compare WRIGHT 1943).

The determination of the genotypic variance is possible by a consideration of the application of the analysis of variance to binomial data. The direct analysis of the data, in which a death or a survival is considered as a separate observation with possible values of zero or of unity, has theoretical objections, since the variance is not independent of the mean, thus violating one of the fundamental requirements of variance analysis. This difficulty can be overcome by the arc-sine transformation. However, the genotypic variance in our data proved to be so small that the direct method could be used on the raw data.

There is, of course, the general question as to the significance of the degree of heritability derived from the raw data as compared with that obtained by using one or another type of transformation. We shall not enter the various aspects of this problem here except for the discussion of the probit transformation in connection with the variation in heritability related to mortality level (see below). The reason why this discussion is made in specific reference to the "probit" rather than to the "inverse probability" transformation of WRIGHT (1926), which is essentially the same, lies in the fact that LUSH, LAMOREUX and HAZEL (1948), whose results are being referred to, have couched their analysis in probit terms.

In data on poultry, the estimation of genotypic variance is greatly simplified as compared to the case of mammals by the relatively high numbers of offspring each bird may leave, especially in the case of sires. In the data here considered, each dam had an average of about eight offspring and each male an average of

about 40 (after elimination of smaller classes as noted below). It then becomes possible to carry out an analysis of variance between the offspring of males within years, and between the offspring of females within males within years, and thus obtain an estimate of the component of variance due to the differences between classes, much as has been done previously in the case of continuously distributed characters (for instance by WHATLEY 1942).

Consider, for instance, the analysis between the offspring of males in a given year. The table of data will then read as follows:

Offspring of male	Total	Surviving
I	n_1	a_1
II	n_2	a_2

etc. with a total of N males.

The total of survivors in the offspring of male I can then be considered as the total sum of n_1 observations (that is, a_1 taken as unity and $n_1 - a_1$ taken as zero). By analogy with the case of continuous variation, the sum of squares between classes is

$$\sum_1^N \frac{a_1^2}{n_1} - \frac{(\sum a_1)^2}{\sum n_1}$$

with $N-1$ degrees of freedom.

The expected value of this sum of squares is

$$(N - 1)\bar{p}(1 - \bar{p}) + n_0 r \sigma_p^2$$

where r is the difference in genetic relationship between and within classes, and

$$n_0 = \sum n_1 - \frac{\sum n_1^2}{\sum n_1} - (N - 1)$$

[see SNEDECOR 1946, p. 234, and KENDALL 1945, p. 123, eq. (5.29)]. The within classes component is $\bar{p}(1-\bar{p})$, which is the normal expression for the variance of a binomial population. The genetic variance then is

$$\sigma_p^2 = \frac{\left[\sum \frac{a_1^2}{n_1} - \frac{(\sum a_1)^2}{\sum n_1} \right] - (N - 1)\bar{p}(1 - \bar{p})}{r n_0},$$

and heritability equals

$$\frac{\sigma_p^2}{\bar{p}(1 - \bar{p})} = \left[\frac{\sum \frac{a_1^2}{n_1} - \frac{(\sum a_1)^2}{\sum n_1}}{\bar{p}(1 - \bar{p})} - (N - 1) \right] \Big/ r n_0.$$

However, the first term in the numerator is the heterogeneity χ^2 in the $2 \times N$ table which the data form, so that heritability may be expressed as

$$\frac{\chi^2 - (N - 1)}{r n_0}$$

The mean value of χ^2 for $N-1$ degrees of freedom is $N-1$. Hence the numerator of the above expression is the excess of the observed χ^2 above its expected value.

The relation of the heritability to the χ^2 statistic raises a further point. It is well-known that the χ^2 test is unreliable if the expected number in any subclass is small, the minimum per subclass being usually taken as five. COCHRAN (1936) has considered the distribution of the heterogeneity χ^2 in the case of the $2 \times N$ table with a constant number n of observations in each of the N classes. He has shown that the mean value of χ^2 is slightly greater than $N-1$, being given by

$$\chi^2_{exp} = N - 1 + \frac{1}{n}\left[1 - \frac{1}{N}\left(1 - \frac{1}{n}\right)\right].$$

The excess depends largely on n and is independent of the expected numbers in subclasses. In the cases here considered, we have approximately,

(a) in the analysis between sires within years,

$$N = 10, \quad n = 40,$$

(b) in the analysis between dams within sires within years,

$$N = 5, \quad n = 8.$$

For the present analysis, the samples were made fairly homogeneous as regards n by elimination of some of the observations. In case (a), all sires with n less than 12 and in case (b) all dams with n less than five were excluded. It is obvious by reference to the above formula that in the analysis between sires, n is so large that no correction need be made even for causes of death with low incidence. In the analysis between dams, however, a correction was made in all cases. It was of the order of 0.10 for each χ^2 calculated and, in the case of total mortality, for instance, reduced the estimate of heritability by about one in eight.

It is possible to give a rough estimate of the standard error of the estimates of heritability, since the method used above is similar to the determination of the intra-class correlation in the case of continuous variation. FISHER (1941) has shown that in a case similar to that discussed above and using the same symbols,

$$\sigma_t = [1 + (n - 1)t](1 - t) \cdot \sqrt{\frac{2}{n(n - 1)(N - 2)}}$$

where t denotes the intra-class correlation. Since heritability is then equal to t/r, the standard error of the estimate of heritability is given by σ_t/r.

In the data here discussed, the average size of the flock was 400–500 birds. Data for about eight breeding males, each mated to an average of five females, were used for each of the years analyzed. Owing to the considerable variation in mortality from year to year, the analyses were performed within years and the values of χ^2 and n_0 for the separate years pooled to give the final estimate.

With this flock structure, the value of r was 0.285 in the analysis between sires (each group being a mixture of full and half sisters) and 0.250 in the analysis between dams within sires (that is, for full sisters in a population of half sisters). Table 1 shows the results.

TABLE 1

Estimates of heritability

| TYPE OF MORTALITY | MEAN INCIDENCE | BETWEEN SIRES | | BETWEEN DAMS WITHIN SIRES HERITA-BILITY | JOINT ESTIMATE OF HERITABILITY |
		POOLED χ^2	HERITA-BILITY		
1. Total	0.416	206.39	0.0832	0.1123	0.0893 ± 0.028
2. Lymphomatosis	0.081	165.86	0.0506	0.0315	0.0475 ± 0.017
3. Reproductive disorders	0.234	130.68	0.0223	0.0553	0.0264 ± 0.015
4. Other than lymphoma- tosis	0.335	185.25	0.0662	0.0633	0.0656 ± 0.020

In the analysis between sires, the pooled values of n_0 and the degrees of freedom were 4358.7 and 103 in all cases. This gave a standard error of the final estimates of around 0.02–0.03. In the analysis between dams, the pooled values were 2752.3 and 414 respectively, n_0 being less than in the former case owing to the exclusion of dams with less than five progeny. This gave a standard error of the estimates of heritability of 0.04–0.05. There is no significant difference in the heritability of the different causes of death as derived from an analysis between the different sexes. In the joint estimate, the heritabilities from the two sexes were weighted according to their standard error, which resulted, on the average, in that between sires having a weight five times that between dams. An independent measure of the standard error of the joint heritability can be obtained from a consideration of the variation of the heritability between the different years. In the case of total mortality, the heritability was estimated to have a standard error of ±0.028 from the above formula and ±0.024 from the variation between years.

These results show a fair agreement with those of LUSH, LAMOREUX and HAZEL (1948), who found a heritability of 0.083 for total mortality compared with 0.059 from lymphomatosis deaths and 0.034 for deaths from other causes. The production index referred to by LERNER and HAZEL (1947) in their paper on genetic improvement in this flock is the average production during the laying year of all pullets housed and their estimate of individual heritability for the character was 0.045. Both they and DEMPSTER and LERNER (1947) have found that this figure is sufficiently accurate to account for the changes in the production index under selection.

The production index of the flock may be considered as made up of three constituent parts:

 (i) the average production of survivors,

 (ii) the average production of birds dying before the end of the year,

 (iii) the relative proportion of birds falling into the above two classes, defining survival level or viability.

From the genetic viewpoint, there are two models of the situation available. The first model takes the production of surviving birds as its starting point and considers survival or death as affecting the environment in which the production record is made. The heritability of the survivors' production for this flock has been determined by LERNER and CRUDEN (1948) whose results agree closely with those of SHOFFNER (1946) on a totally unrelated flock in yielding the figure of 0.30–0.35. The present investigation has considered the heritability of the last component and has established it at around 0.09. The heritability of the production index is then lower than the heritability of either of the two components. There are three possible ways in which such a condition might arise:

 (i) the heritability of the second component (production of birds dying before the end of the year) may be so low as to reduce the heritability of the production index below that of the other components;

 (ii) there is a negative genetic correlation between production and viability;

 (iii) non-additive genetic effects are involved.

It is impossible to discriminate adequately between the various possibilities but considerations to be presented elsewhere would suggest that the first two probably play a significant rôle.

The second genetic model, possibly slightly more logical, is to consider total production, whether the bird lives or dies, as representing the basic genotype involved. No distinction is to be made between genes controlling viability and genes controlling egg yield. Viability then becomes important as having a high environmental correlation with production and its inclusion in a selection index is desirable. It would seem likely, however, that on this scheme, there will be considerable interaction between viability and production which will cause complications in the full application of the model.

THE GENETIC CORRELATION BETWEEN MORTALITY FROM DIFFERENT CAUSES

It should be noted that cases of double classification of mortality from both lymphomatosis and reproductive disturbances do appear in the data. If there were no complications of any sort (e.g. differences in age distribution of deaths from both causes) the environmental correlation between the two types of mortality could be readily computed. This might be done by a comparison of the number of those falling in the double classification with those falling in the separate single classes. The nature of the actual data usually available precludes the possibility of such computations.

It is possible to divide the total mortality into the deaths from lymphomatosis and those from other causes. As these are mutually exclusive classes, the environmental correlations between them can be easily calculated. The following symbols must first be defined:

 (i) the genotype for viability with reference to lymphomatosis, p_a with the

corresponding genotype for mortality q_a, equal to $1-p_a$. These will have variances of σ_a^2.

(ii) similar genotypes with reference to other causes, if lymphomatosis is not present, p_b and q_b with variances of σ_b^2.

If lymphomatosis is present, however, there will be cases of double classification which will be counted as due to lymphomatosis only. The apparent mortality from other causes is then reduced. We must then define:

(iii) the genotype for apparent viability with respect to other causes, when lymphomatosis is present in a population, p_c with corresponding q_c and σ_c^2. It is clear that q_c is simply related to q_a and q_b by the relationship

$$q_c = q_b - q_a q_b,$$

and total mortality q_T is then equal to q_a+q_c.

We can now calculate the variance of q_c and its covariance with q_a by simple algebra. We may write

$$q_a = \bar{q}_a + \delta q_a$$

where $\bar{q}_a$ is the mean of q_a and δq_a is the deviation from the mean.
Then

$$\bar{q}_c + \delta q_c = \bar{q}_b + \delta q_b - (\bar{q}_a + \delta q_a)(\bar{q}_b + \delta q_b)$$

$$= \bar{q}_b - \bar{q}_a\bar{q}_b + (1 - \bar{q}_a)\delta q_b - \bar{q}_b\delta q_a - \delta q_a\delta q_b.$$

If the variances are small compared to the mean, the last term may be ignored, giving

$$\delta q_c = \bar{p}_a\delta q_b - \bar{q}_b\delta q_a$$

and

$$\sigma_c^2 = \bar{p}_a^2\sigma_b^2 + \bar{q}_b^2\sigma_a^2 - 2\bar{p}_a\bar{q}_b r\sigma_a\sigma_b,$$

where r is the genetic correlation between p_a and p_b. Sim'larly

$$\text{cov. } (p_a, p_c) = -\bar{q}_b\sigma_a^2 + \bar{p}_a r\sigma_a\sigma_b.$$

A determination of σ_a^2, σ_c^2 and cov.(q_a, q_c) will thus allow the calculation of r.

The environmental covariance can easily be calculated from the following considerations. Within a given phenotype the distribution of birds with respect to their survival or death can be arranged in a four-fold table:

		Other Causes	
		Alive	Dead
Lymphomatosis	Alive	$1-q_a-q_c$	q_c
	Dead	q_a	0

Scoring death from either lymphomatosis or other causes as zero and survivals as unity, the covariance (which here is environmental) is

$$1 - q_a - q_c - (1 - q_a)(1 - q_c), \text{ or } -q_a q_c.$$

We can then proceed as follows in the analysis of the data. The between class covariance can be obtained as in the heritability analysis from the expression,

$$\text{between class covariance} = \sum \frac{a_1 c_1}{n_1} - \frac{\sum a_1 \sum c_1}{\sum n_1}$$

where c_1 refers to birds surviving death from other causes. This, like the sum of squares between classes, consists of two terms. The first contains the environmental covariance, which can be calculated as above, and the second the genetic covariance. A comparison of the latter with σ_a^2 and σ_c^2 allows an estimation of the genetic correlation r between p_a and p_b.

The data were similarly analyzed within years but the analysis between dams was not made, since COCHRAN's correction for χ^2 made the position uncertain. The genetic correlation between p_a and p_c was estimated as $+0.096$. A further calculation on the above lines showed that the genetic correlation between p_a and p_b was 0.26. LUSH, HAZEL and LAMOREUX (1948) found a much higher correlation (.54) between the two in their data, which they interpreted as evidence of the existence of a general genetic factor controlling resistance to death. The present correlation is positive but not particularly large, suggesting that a general factor for vigor does not play a large part in this particular flock.

Since, first y, the genotype for viability from two different causes is not additive (since $p_T = p_a p_b$) and secondly, the denominator in the equation for heritability is also not additive, the heritability of the total mortality cannot be obtained simply from the heritability of its components.

Considering the case discussed above,

$$h_a^2 = \frac{\sigma_a^2}{\bar{p}_a \bar{q}_a}$$

$$h_b^2 = \frac{\sigma_b^2}{\bar{p}_b \bar{q}_b}.$$

By putting $p_a = \bar{p}_a + \delta p_a$, etc. it can easily be shown that

$$\sigma_T^2 = \bar{p}_a^2 \sigma_b^2 + \bar{p}_b^2 \sigma_a^2 + 2 \bar{p}_a \bar{p}_b r \sigma_a \sigma_b$$

and

$$h_T^2 = \frac{\sigma_T^2}{\bar{p}_a \bar{p}_b (1 - \bar{p}_a \bar{p}_b)}.$$

In the simple case, when $\bar{p}_a = \bar{p}_b$ and $\sigma_a^2 = \sigma_b^2$

$$h_T^2 = \frac{2\sigma_a^2 (1 + r)}{1 - \bar{p}_a^2}$$

$$= h_a^2 (1 + r) \frac{2\bar{p}_a}{1 + \bar{p}_a}.$$

The heritability of total mortality depends, therefore, not only on the heritability of the components but on their mean mortality levels.

THE RELATIONSHIP OF HERITABILITY TO MORTALITY LEVEL

In the course of the analysis, heritability estimates were obtained for each of the 12 years separately. There was considerable variation in the level of total mortality from year to year, as also was the case with respect to the level of the separate causes of mortality. Figures 1 and 2 show the variation of apparent
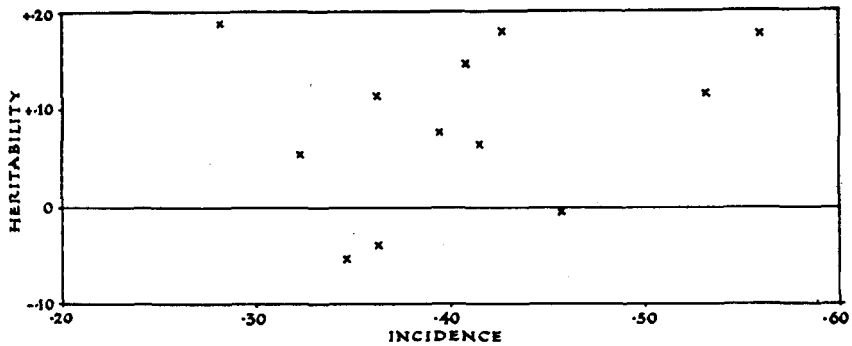
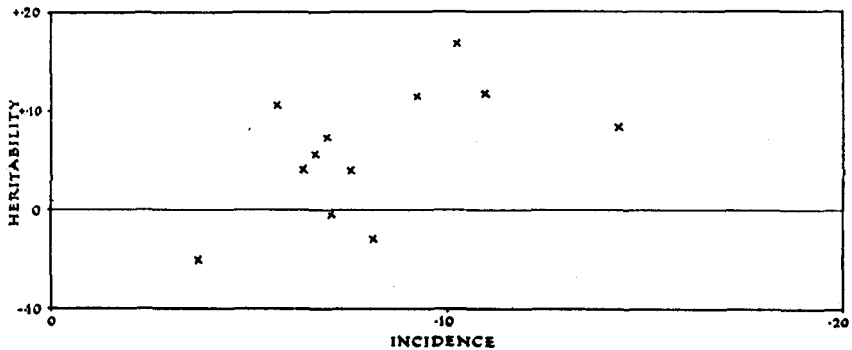FIGURE 1.—The relation between estimated heritability and incidence of total mortality.

FIGURE 2.—The relation between estimated heritability and incidence of lymphomatosis.

heritability of total mortality and death from lymphomatosis in relation to the mean incidence. The values of heritability are derived from those obtained from the analysis between sires and dams, with appropriate weighting, and the negative values merely reflect values of the heterogeneity χ^2 less than the number of degrees of freedom in that year. The heritability of death due to reproductive disorders was not considered, as the mean value for it is not significantly different from zero. The variation of the points is wide but not greater than that expected theoretically. In the case of deaths from lymphomatosis, the heritability seems to be related to the average incidence of disease. In the case of total mortality, there is no apparent trend of the points, although the fitting of a regression line shows a slight positive slope. However, the range

of variation of incidence is such, in this case, that heritability would not be expected to vary greatly. In the case of death from a single specific cause, the relationship between heritability and mortality can be predicted theoretically.

The question of the transformation of the data was mentioned in the presentation of the technique of estimation of heritability. Any such transformation would be expected to affect the heritability comparatively little, since the effect is to multiply both the numerator and denominator of the heritability expression by a constant factor. The arc-sine transformation makes the denominator independent of $\bar{p}$ while the genetic variance is still dependent. On the other hand, it can be shown that in the case of mortality from one single definite cause, such as a specific infection or a poison, the probit transformation makes the genetic variance independent of $\bar{p}$ while the denominator varies in a perfectly known manner, being equal to $\bar{p}\bar{q}/\bar{z}^2$ where $\bar{z}$ is the ordinate of the normal curve at the point where the intercept is equal to $\bar{p}$. The basic concept of
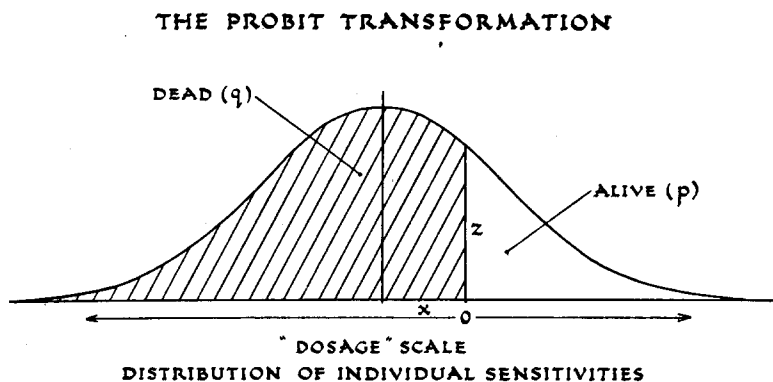

FIGURE 3.—The basic concept of the probit transformation.

the probit transformation is that the mortality is the expression of the sensitivity of the individuals in the population, the sensitivity being assumed to be normally distributed on a "dosage" scale, where the "dosage" is some measure of the level of the infection or the concentration of poison. Then all individuals whose sensitivities are below the given "dosage" will die.[2] The probit is then a measure on the dosage scale, in terms of the standard deviation within the population whose mortality is measured. Figure 3 expresses the situation pictorially.

[2] DR. EVERETT R. DEMPSTER in reviewing the manuscript has suggested a model differing from the one employed here, which may be easier to visualize but leads to the same mathematical results. He considered the genotype as the proportion of environments in which the organisms will survive. In this view the organism is seen as subjected to a distribution of environments or dosages rather than having, as in our case, its sensitivity changed by the environment and then subjected to a given dose. In our model the "probit" is derived from the dichotomy of distribution of sensitivities by the dosage; in his, from the dichotomy of the dosage distribution by the sensitivity. The theoretical implications of the two models are the same except that the concept of heritability on the dosage scale introduced by LUSH, LAMOREUX and HAZEL (see below) can not be applied to DR. DEMPSTER'S.

The probit transform of q, the mortality, is then $5+x$, the 5 being introduced to avoid negative values, and x is measured in terms of the standard deviation of the group whose probit it is. In the case of the probit of a given genotype, it would be expressed in terms of the environmental variation of that genotype on the dosage scale. The whole population is then made up of the sum of all the genotypic distributions. If it be assumed that the environmental distribution on the dosage scale is independent of genotype, then on the movement of the point 0 along the scale (thereby changing the mean mortality) the mean of all the probits will be altered by a constant amount, and the variance will remain constant. That is to say, the genetic variance on the probit scale is independent of mean mortality. We can thus predict the variation of heritability with $\bar{p}$, since

$$\text{heritability} = \frac{\sigma_x{}^2 \cdot \bar{z}^2}{\bar{p}(1 - \bar{p})}$$

and $\sigma_x{}^2$ is constant (see figure 4 below). This point has essentially been made by FISHER (1947, p. 217) in noting that if the dosage-mortality relationship (corresponding to the standard deviation of the distribution mentioned above) has been determined, the toxicity of any material (and correspondingly the toxicity of a given material to an unknown genotype) can be established with an accuracy proportional to z^2/pq. The analysis presented will, however, not be applicable if $\sigma_x{}^2$ is large. Because of the interaction introduced by the change of scale, $\bar{p}(1-\bar{p})/\bar{z}^2$ is no longer an accurate measure of the total variance. There is, for instance, no a priori limit to $\sigma_x{}^2$ and therefore the heritability
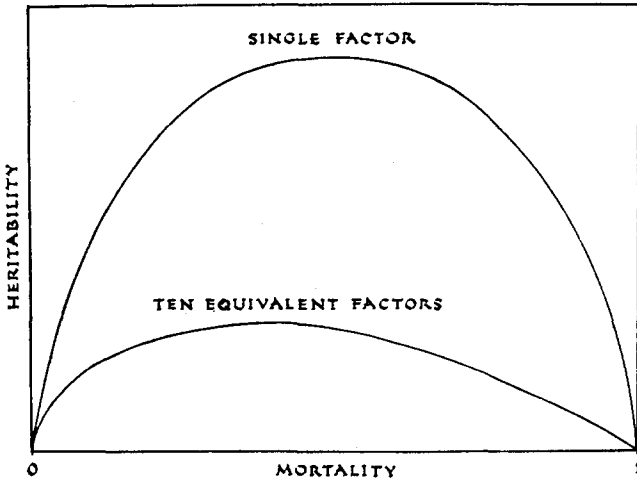

FIGURE 4.—The relationship between heritability and mortality level, in the case a) where mortality is due to a single cause to which the concept of sensitivity is applicable, where

$$h^2 = \frac{K\bar{z}^2}{\bar{p}(1-\bar{p})} ,$$

and in the case b) where the total mortality is compounded from that due to ten such independent equivalent causes, where $h_T{}^2$ is obtained as in the text expression.

could be greater than one. There seems to be some confusion on this point in the report of LUSH, LAMOREUX and HAZEL (1948). They have introduced the concept of "heritability on the probit scale," which, however, is not the real heritability of sensitivity but the genetic variance. The assumption implicit in their reasoning is that the probit units for the whole population and for the individual genotypes are the same, whereas in reality they are different. The probit for individual genotypes takes as its unit of measurement the environmental standard deviation on the sensitivity scale. That for the whole population takes as its unit the total phenotypic standard deviation. In terms of variance the latter is $(1 + \sigma_x^2)$ times the former. Thus, the heritability on the sensitivity scale is equal to

$$\frac{\sigma_x^2}{1 + \sigma_x^2},$$

rather than to σ_x^2 as considered by LUSH, LAMOREUX and HAZEL (compare WRIGHT 1934a).

The above discussion applies only to mortality from a definite single cause whose "dosage" scale has a definite biological meaning. In omnibus classifications such as "total mortality" and "other mortality" considered by LUSH, LAMOREUX and HAZEL from a similar viewpoint, the dosage scale ceases to have any definite meaning. It can only be defined in terms of the mortality level itself, which is made up of any number of different factors with different heritabilities, and may be so made up in an infinite number of possible ways. The situation is considerably removed from the case of the single disease where the change in mortality can be described as a point moving along a sensitivity scale. We can then only speak of the average heritability at a given level, the average being taken over all possible components of the factors which combine to give that total level. It is obvious that if we have two separate factors with very different heritabilities, the heritability of the joint mortality at the level of 50 percent will differ greatly depending whether, for instance, the highly heritable factor has a level of 40 percent and the other a level of 17 percent, or *vice versa*.

The problem may be considered by use of the method presented in the last section for calculating the total heritability of two factors. The method can be extended to any number of factors (A, B, C, etc.) to give the general result

$$h_T^2 = \frac{p_T}{1 - p_T} \left(\sum \frac{K_a z_a^2}{p_a^2} + 2 \sum r_{ab} \frac{\sqrt{K_a K_b}\, z_a z_b}{p_a p_b} \right)$$

where K_a is the genetic variance of mortality due to A on the probit scale, and r_{ab} the genetic correlation of mortality due to A and B, or A and C, and so forth.

This formula has been applied to two cases as follows:

(i) the total mortality is made up of several equivalent uncorrelated factors. Figure 4 shows the heritability of total mortality made up of ten factors, the upper curve being the heritability of one such factor alone. It will be

seen that the heritability of the compound mortality is always less than that of the single factor at the same level, the relative difference being greater at high mortalities. At 50 percent mortality it seems to be roughly true that the heritability of mortality due to m equivalent factors is $1/\sqrt{m}$ times the heritability of one such factor at the same level. In addition, the curve is no longer symmetrical but is pushed over towards the lower mortality levels. It is obvious that the variation with mortality level is quite different from that of the single factor.

(ii) the total mortality is made up of two uncorrelated factors, the variation from season to season being due mainly to one factor only. Figure 5 shows the variations of heritability with total mortality when two factors, A and B, are involved, such that:

(a) A has a heritability of 0.10 at its mean level of 10 percent with a standard deviation of mean level from season to season of 0.04.

(b) B has a heritability of 0.02 at its mean level of 33.3 percent with a standard deviation of mean level from season to season of 0.13.
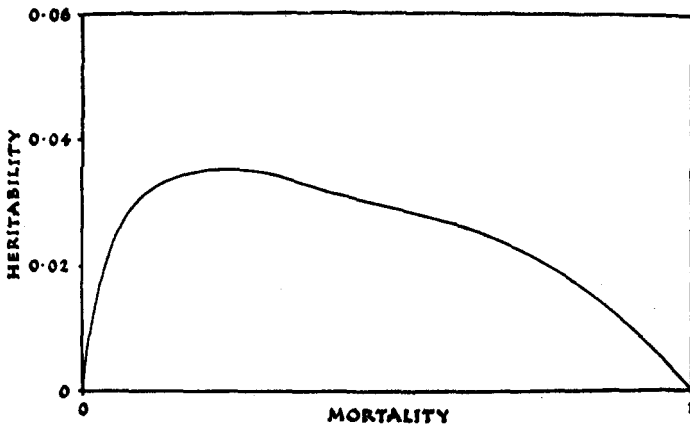

FIGURE 5.—The relationship between heritability and compound mortality from two causes for the conditions specified in the text.

Here the curve is very skew with a maximum around 20 percent mortality. In fact, the heritability varies little over the range 0.10–0.50. By varying the different constants controlling A and B, the maximum could be shifted to any desired level. This case, in which most of the yearly variation is contributed by a component, whose heritability is relatively low, will be the one departing most from the theoretical curve.

It would seem likely from these two instances that in the majority of practical cases, although it will be impossible to predict the exact variation of heritability of total mortality with incidence, the maximum will probably be in the region between 25 percent and 60 percent mortality.

The genetic variance on the probit scale of any two independent factors would not be expected to be additive, due to the high interaction between the

TABLE 2

Probit values in relation to the level of mortality from two causes

MORTALITY DUE TO	LEVEL	PROBIT	ADDITIONAL DUE TO A
B	10%	3.72	
A+B	19%	4.12	0.40
B	50%	5.00	
A+B	55%	5.12	0.12

two probits as is shown by table 2, which illustrates the effect of adding factor A at 10 percent mortality to factor B at 10 percent and 50 percent. The difficulties of the use of the probit transformation in the case of deaths from independent causes has been fully discussed by FINNEY (1947 p. 136).

SUMMARY

1. A method is presented for the determination of the heritability of all-or-none characters, with special reference to mortality. It can be extended to cover the genetic correlation between mortality from two different causes.

2. Analysis of the mortality records of the production flock of the UNIVERSITY OF CALIFORNIA gave a heritability of 0.089 for total mortality, 0.026 for deaths from reproductive disorders, 0.048 for deaths from lymphomatosis and 0.066 for deaths from other causes than lymphomatosis. The genetic correlation between mortality from lymphomatosis and from other causes was found to be +0.26.

3. The heritability varied considerably with the mean level of mortality. A theoretical expression for this variation is derived for the case of mortality from a single specific cause.

4. The relationship of the heritability of total mortality to incidence is, however, more complex and formulae are presented giving the heritability of total mortality in terms of the levels and heritabilities of its components. These are applied to two general cases, showing that the simple laws pertaining to deaths from single causes do not hold for aggregates of causes. The deviations are likely to be greatest when the variation in mean level from season to season is mainly due to a factor with relatively low heritability.

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