BIOMETRICAL GENETICS

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PREFACE

GENETICS is a young subject in the history of science but is already pervading the whole of biological science in a similar way to the pervasion of physics through physical science. The reasons for this are many, but probably one should pick out two: on the one hand, it is a beautiful subject from a purely aesthetic viewpoint and on the other hand it has impact on the whole range of human activity from the study of what is life itself to the utilization of economic plants and animals. It is a branch of science which must be consulted whenever any problem of living matter is under investigation.

The rapid growth of genetics is undoubtedly in part attributable to the growing use of stochastic representations of phenomena and to the development in the past 60 years of procedures for drawing uncertain inferences. No doubt is possible concerning the role of the growth of statistical inference on the growth of genetics, and this is particularly so because the basic genetic mechanism is probabilistic.

It would be rash to attempt a definitive partition of the subject matter of genetics, but it has become apparent in the last 30 or 40 years that there are complementary areas each of which has status in its own right, even though all stem back to classical Mendelian genetics. Perhaps a good way of representing what is happening is to distinguish two approaches requiring somewhat different talents. One approach is the biological one including chemical and physical aspects, and the other is mathematical, including ordinary mathematics, probability and statistics.

One can also make a partition of the subject matter of genetics on the basis of whether it is directed to the identification and understanding of genotypes or whether it is directed to the situation in which genotypes are not identifiable. Text books of genetics abound in examples of the former and text books on animal or plant breeding abound in examples of the latter. It is the latter which is the subject of this volume.

If we consider attributes like milk production of a cow, growth rates of swine, yield of grain of a cereal, or endocrine activity of mice, we can certainly envisage the possibility of identifying genetic factors which are influential and should always be aware of this possibility, but we should also be aware of the fact that there are undoubtedly many different genotypes which give essentially the same value of the attribute. Also we must take account of the fact that such attributes are affected to varying degrees by variations in the environment, in contrast to (say) wrinkled peas which are wrinkled providing one has any reasonable environment.

These two facts, non-identifiability of genotypes and dependence on environment even within a restricted class of environments such as the soil and climatic conditions of the Corn Belt of the United States of America,or South-East England or what you will, have led to the development of a branch of genetics to which the name biometrical genetics is frequently applied. The branch is also referred to sometimes as population genetics, though this is hardly appropriate because population genetics also includes the mathematical theory of population dynamics with specified Mendelian systems and the purely biological examination of natural populations. It is also referred to sometimes as the theory of quantitative inheritance. It seems rather likely that the term 'biometrical genetics', which is due, it is believed, to K. Mather, will emerge as the generally accepted name for the area.

That there should be deep general interest in biometrical genetics is not surprising because the two main tools for genetic improvement of economic species are the identification of genotypes and what methodology we have for unidentifiable genotypes. It is also not surprising that the interest in biometrical genetics among biometricians is both widespread and deep, because the tools of biometrical genetics are mathematics and biometry.

It was with this background that the organizers of the Third International Biometrics Congress decided to open the congress with an International Symposium on Biometrical Genetics.

The program of the symposium was arranged by a committee consisting of R. E. Comstock, J. F. Crow, C. R. Henderson, O. Kempthorne (Chairman), J. L. Lush and K. Mather. The general arrangements were under the supervision of the secretary of the Biometrics Society, M. J. R. Healy. Excellent local arrangements at Ottawa were made by Canadian groups specifically those associated with G. B. Oakland and J. W. Hopkins. The International Union of Biological Sciences made a grant towards the publication of the proceedings of the Symposium. The great bulk of editorial work and the preparation of the index have been done by Mr. Neeti Ranjan Bohidar, to whom considerable gratitude is due. The cooperation of the Pergamon Press is most gratefully acknowledged.

It only remains to hope that this volume of papers presented at the Symposium will be useful to workers in the field and will stimulate further development in the area of biometrical genetics.

OSCAR KEMPTHORNE

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EXPERIMENTAL RESULTS

THEORETICAL GENETICS

DOMINANCE, GENOTYPE–ENVIRONMENT INTERACTION, AND HOMEOSTASIS

By R. E. COMSTOCK
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A FORMIDABLE operational problem in the study of variance patterns of quantitative characters arises because the environments pertinent to a genetic population are not distributed randomly in time and space. Instead, the environments associated with a specific point or region in the time-space habitat of a population comprise a unique stratum or sub-set that can never be fully representative of the totality of environments that have had to do with the evolution of the population or will have to do with its future.

Because genotype and environment have interacting (not independent) effects on phenotype, the data from any finite experiment pertain unambiguously to the genetic variance of the population with reference only to the sub-set(s) of environment actually sampled by the experiment. In theory, and to a degree in practice, the difficulty can be overcome by replication of the experiment at different points within the pertinent regions of time and space. However, this is expensive business, subject ordinarily to severe restriction.

For the sake of a specific point of reference and to remain on ground familiar to me, what follows will be related to work with corn. During my association with the quantitative genetics research program at North Carolina State College, a major objective was estimation of the total and relative magnitudes of components of the total variance in grain yield of corn populations. By now, the procedures used can be referred to as routine. They consisted of replicated comparisons of random families from populations being studied. Inclusion of two types of families (full-sib and half-sib) in the same experiment allowed estimation of two components of the genetic variance. At North Carolina such experiments were designed to yield simultaneous estimates of the additive and dominance portions of the genetic variance (see Robinson *et al*, 1955). Primary interests were (1) magnitude of the additive component and (2) the relative magnitudes of the additive and dominance components.

The problems involved in getting estimates (unbiased by variance of genotype–environment interaction effects) of variance in genetic effects defined as averages for the totality of environments pertinent to the destiny of a genetic population have become familiar to workers in the field. They were noted by Comstock and Robinson (1952) and reviewed in greater detail by

Comstock (1955). Attention here will be confined to relative magnitudes of estimates of different sorts of genetic variance.

From the beginning of the North Carolina work, we recognized that, to avoid bias in estimates of the absolute size of genetic variances in which we were interested, contributing data would need to be collected in two or more years and two or more locations. At the same time I argued (wishfully, perhaps) that estimates from experiments not meeting this requirement would be satisfactory for evaluating relative magnitudes, e.g. that

$$\frac{V_g}{V_d} = \frac{B_g}{B_d}$$

where V_g = additive genetic variance
V_d = dominance variance
B_g = bias in the estimate of V_g, and
B_d = bias in the estimate of V_d.

The reasoning behind this argument was that the definition of genetic variance components is not related to the physiology of gene action, that the various kinds of genetic effects (statistically defined) involve comparable sorts of physiological pathways and hence should not differ in sensitivity to varying environment.

Lerner (1954) proposed that greater developmental homeostasis is associated with more heterozygous genotypes; that, on the average, phenotypes associated with a heterozygous genotype vary less as a consequence of environment than those associated with a more homozygous genotype. He reviewed pertinent evidence and other evidence has since been reported (e.g. Dobzhansky and Levene, 1955). There is no need here to review all that has been written concerning Lerner's proposal or to debate its merits. More pertinent is examination of what ' homeostasis of heterozygotes ' could mean with respect to biases of variance component estimates.

For illustration, let us consider what happens if one or the other homozygote (at any locus) is superior to the heterozygote in almost all sub-sets of environments but the heterozygote, on the average for all pertinent sub-sets of environments, is superior to both homozygotes. (For simplicity two alleles are assumed per locus.) In such a situation, it is obvious that the ratio of expectations of additive genetic and dominance variance estimates from experiments confined to one sub-set of environments would be characteristic of partial to complete dominance, while the same ratio in an adequate experiment would be characteristic of overdominance. Symbolically this can be stated as follows:

$$\frac{V_d + B_d}{V_g + B_g} < \frac{V_d}{V_g}$$

which means that

$$\frac{B_d}{B_g} < \frac{V_d}{V_g} \quad \text{and} \quad \frac{B_d}{V_d} < \frac{B_g}{V_g}.$$

Bias in experiments that fail to sample the whole of the pertinent environment distribution arise from genotype–environment interaction, i.e.

$$B_d = V_{dE} \quad \text{and} \quad V_g = V_{gE}.$$

Here V_{dE} and V_{gE} are portions of the genotype–environment interaction variance that get confounded with V_d and V_g, respectively, in results from experiments conducted within one stratum (sub-set) of environments.

Now let us look at the problem in terms of a simple genetic model, which will not serve for comprehensive analysis, but will provide enough insight for present purposes. Consider one locus with segregation of two alleles. In any sub-set of environments (the i^{th}) let the average phenotypic difference between *AA* and *aa* homozygotes be $Y_{2.i} - Y_{0.i} = 2x_i$ which may be positive or negative. Let the deviation of the average for heterozygotes from the mean of homozygote averages be

$$Y_{1.i} - \frac{Y_{2.i} + Y_{0.i}}{2} = h_i.$$

Then expectations of contributions of this locus to estimates from an experiment confined to one sub-set of environments will be

$$V_{gi} = 2pq[x_i - (p-q)h_i]^2$$

and

$$V_{di} = 4p^2q^2h_i^2$$

where $p = 1 - q =$ frequency of allele, *A*.

Corresponding averages over all possible sub-sets of environments are

$$\bar{V}_g = 2pq[(\bar{x}^2 + \sigma_x^2) - 2(p-q)(\bar{x}\bar{h} + \sigma_{xh}) + (p-q)^2(\bar{h}^2 + \sigma_h^2)]$$
$$\bar{V}_d = 4p^2q^2(\bar{h}^2 + \sigma_h^2).$$

Here $\bar{x}$ and $\bar{h}$ represent means over all environment sub-sets; σ_x^2, σ_h^2 and σ_{xh}, the variances and covariance of x and h.

Contributions to the variances (for genetic effects defined as averages over the totality of environments) which were the real objects of interest in the North Carolina studies would in this case be

$$V_g = 2pq[\bar{x}^2 - 2(p-q)\bar{x}\bar{h} + (p-q)^2\bar{h}^2]$$
$$V_d = 4p^2q^2\bar{h}^2$$

$\bar{V}_g$ and $\bar{V}_d$ are V_g and V_d plus genotype–environment interaction components, i.e.

$$\bar{V}_g = V_g + V_{gE} \qquad \bar{V}_d = V_d + V_{dE}.$$

Hence

$$V_{gE} = \bar{V}_g - V_g = 2pq[\sigma_x^2 - 2(p-q)\sigma_{xh} + (p-q)^2\sigma_h^2]$$
$$V_{dE} = \bar{V}_d - V_d = 4p^2q^2\sigma_h^2.$$

The ratios of bias to variance are

$$\frac{V_{gE}}{V_g} = \frac{\sigma_x^2 - 2(p-q)\sigma_{xh} + (p-q)^2\sigma_h^2}{\bar{x}^2 - 2(p-q)\bar{x}\bar{h} + (p-q)^2\bar{h}^2}$$

and

$$\frac{V_{dE}}{V_d} = \frac{\sigma_h^2}{\bar{h}^2}.$$

Of concern to us are conditions that lead to equality or inequality of the two ratios.

Consider, first, a special case. Given $\bar{h} > \bar{x}$, i.e. over dominance in average effects of the genotypes, and $p = (\bar{x} + \bar{h})/2\bar{h}$, the denominator of V_{gE}/V_g would be zero. The ratio itself would go to infinity and would obviously exceed V_{dE}/V_d.

For more general treatment let

$$a = \bar{h}/\bar{x} \quad \text{and} \quad b = \sigma_h/\sigma_x.$$

Then $\frac{V_{gE}}{V_g} \gtreqless \frac{V_{dE}}{V_d}$ when

$$(a^2 - b^2) \gtreqless 2(p-q)ab(ar-b)$$

where r is the correlation between x and h. There is no loss of generality if $\bar{x}$ is limited to positive values. This amounts only to deciding that the allele which in homozygous state has the higher average phenotype shall be designated as A. It is reasonable also to consider only positive values of h; because in characters related to fitness the regression of phenotype on heterozygosity is ordinarily positive. Under these conditions, a will always be positive; as will b, since standard deviations are always positive. Then

$$\frac{V_{gE}}{V_g} > \frac{V_{dE}}{V_d}$$

when

(1) $a > b$, $p = q$ or $r = b/a$

(2) $a > b$, $p > q$ and $r < b/a$

(3) $a > b$ $p < q$ and $r > b/a$.

The reverse will be true when

$$(4)\ a<b \text{ and } p\leqq q$$

$$(5)\ 1-a^2/b^2>2(p-q)a \text{ and } a<b.$$

These sets of conditions are, of course, sufficient rather than necessary. The intent is merely to demonstrate that inequality of V_{gE}/V_g and V_{dE}/V_d, in either direction, is possible.

It is proper to inquire next whether any of the conditions that make $V_{gE}/V_g > V_{dE}/V_d$, and vice-versa, are, on biological grounds, at all probable. First, consider case (2). Given overdominance a is greater than 1.0 and might easily be greater than b. The frequency, p, of the more favorable allele would often be larger than q, frequency of the less favorable allele. Finally, r might easily be smaller than b/a. This situation is of special interest in connection with inference concerning level of dominance from the ratio of estimates of dominance and additive genetic variance. It is easily seen that upward bias in the estimate of dominance variance could be less than in the estimate of additive genetic variance when estimates are based on data from a single sub-set of environments. Then interpretation assuming equal bias would under-evaluate the true level of dominance.

On the other side of the picture, consider case (5). Given a character for which affecting genes exhibit little dominance, a rather small and considerably less than b is not improbable. In this case condition (5) could easily be met. However, the chance of critical misinterpretation as a consequence of disproportion in biases appears less likely here than in case (2).

The prime issue is that for biases under discussion the ratio,

$$\frac{\text{Bias}}{\text{Variance estimated}}$$

is not necessarily the same for all kinds of genetic variance. Hence ratios of genetic variance components cannot be inferred with complete assurance from data collected in one sub-set of environments, say, in one year at one location.

Experimental evidence on biases actually encountered is scanty. Rojas and Sprague (1952) estimated V_g, V_d and components of genotype–environment interaction variance in corn yield. They reported that interaction variance associated with dominance effects was larger relative to dominance variance than interaction variance associated with additive effects relative to additive genetic variance. Somewhat different results were reported by Comstock *et al.* (1957) in a summary of data then available from the North Carolina studies. Their data were also on corn yield but did not allow complete separation of genotype–environment interaction variances from genetic variances. In one series of experiments, replicated over years but not over places, V_g+V_{gy}, $V_{gy}+V_{gyr}$, V_d+V_{dy} and $V_{dy}+V_{dyr}$ could be estimated. In

another series, replicated in some instances over years but not over places and in other instances over places but not over years, the quantities listed above or the quantities, $V_g + V_{gy}$, $V_{gp} + V_{gpy}$, $V_d + V_{dy}$ and $V_{dp} + V_{dpy}$ could be estimated. Here

V_{gy} is variance due to interaction of additive genetic effects with year,
V_{gp} is variance due to interaction of additive effects with places,
V_{gpy} is variance due to second order interaction of additive effects with year and place,

and V_{dy}, V_{dp} and V_{dpy} have analogous meaning in terms of dominance effects. Averaging over experiments the ratios of the purely interaction variance estimate to the estimate of the quantity including V_g or V_d, e.g. estimate of $V_{gy} + V_{gpy}$ divided by estimate of $V_g + V_{gp}$, were as follows:

	Estimates involving additive effects	Estimates involving dominance effects
Series 1	0.73	0.50
Series 2	0.73	0.16

In contrast to results reported by Rojas and Sprague, the North Carolina data suggested greatest bias from genotype–environment interaction variance in estimates of V_g. This contrast could be interpreted in two ways: (1) as indicating that the ratio of interaction to genetic variance is sometimes higher for one kind of genetic effects and in other cases higher for another kind, or (2) as the result of high sampling error of the estimates involved. The nature of the sampling errors is such that their magnitude can only be approximated in terms of the observed variation of a series of estimates of the quantities (ratios) in question. In this respect the evidence cited does not suffice for confident interpretation.

The foregoing and its implications may be summarized as follows:

1. If estimates of genetic variances and ratios among them are to be useful in analysis of forces responsible for the present status of populations and in prediction of future changes, they must pertain to genetic effects defined meaningfully in terms of the totality of environments having to do with the history and destiny of the population.
2. At best such variances are elusive quantities. A reasonable approach to unbiased estimates of their absolute magnitudes requires data from experiments replicated in both time and space. Places employed should of course be properly related to the normal 'habitat' of the population.
3. Inferences from relative magnitudes of estimates of different kinds of genetic variances could be based on biased estimates if it were safe to assume, for all genetic variances, a constant ratio of bias to variance estimated.
4. The notion that developmental homeostasis is a function of hetero-

zygosity suggests that this is not a safe assumption. The analysis presented here demonstrates, if that were necessary, that the ratio of bias to variance estimated may conceivably be either higher or lower for dominance variance than for additive genetic variance. Of special interest is the fact that inequality of this ratio might have a critical bearing on inference concerning level of dominance based on the relative magnitude of estimates of dominance and additive genetic variance.

5. The prime implication is that information about the relative magnitude of different sorts of genetic variance obtained from an experiment confined to one stratum of the totality of pertinent environments needs to be supplemented by at least some information on magnitude of bias from genotype–environment interaction variance.
6. Replication (of the whole experiment providing estimates of two or more genetic variances) in time and place is desirable but often impossible due to cost considerations. A supplementary experiment providing information on magnitude of bias in estimation of only one genetic variance will sometimes suffice. For example, in the work of Robinson *et al.* (1955) the smallness of the ratio of the estimate of dominance variance to that of additive genetic variance was the critical finding. The critical feature of the result could have been the result of estimation bias only through greater upward bias in additive genetic variance than in dominance variance. Adjusting the original estimate of additive genetic variance downward in accord with later information on bias in the estimate of that component and accepting the original estimate of dominance variance as unbiased provides a severe test of the original interpretation. When this is done, the ratio of the estimate of dominance variance to the estimate of additive genetic variance is still small enough so that interpretation of the finding is unchanged, though the force of the evidence is a little less.

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THE BALANCE-SHEET OF VARIABILITY

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PROVIDED that there has been no selection and in the absence of interactions between non-allelic genes, the genetic variability in the segregating generations descended from a cross between two homozygous lines appears in three components. First, there is the D component, depending on differences between individuals homozygous for allelic genes, and second, there is the H component, depending on departures of heterozygotes from the average of the two corresponding homozygotes. Both of these contribute to the variation within each generation, measured round its own mean. Thirdly, there is the departure of the generation mean from the mid-parent value, found as the average of the two parental homozygous lines, and this is a function of $S(h)$, the notation being that of Mather (1949a). Since genetic variation measured round the mid-parent value is the sum of the variance round the generation mean and the squared deviation of this mean from the mid-parent, it may be written in terms of D, H and $S^2(h)$ each component taking a coefficient appropriate to the generation. These coefficients must sum to unity (Mather, 1949b). Thus if we write the variation of any generation as $xD+yH+zS^2(h)$, then $x+y+z=1$. For example in the F_1, $x=y=0$ and $z=1$, while in the F_2 generation $x=\frac{1}{2}$, $y=\frac{1}{4}$ and $z=\frac{1}{2}^2=\frac{1}{4}$. In F_3, we must add $V_{1F2}=\frac{1}{2}D+\frac{1}{16}H$ and $V_{2F3}=\frac{1}{4}D+\frac{1}{8}H$, these being the genetic variances between and within families, to obtain the genetic variance round the generation mean, which is itself $\frac{1}{4}S(h)$. Then $x=\frac{1}{2}+\frac{1}{4}=\frac{3}{4}$, $y=\frac{1}{16}+\frac{1}{8}=\frac{3}{16}$ and $z=\frac{1}{4}^2=\frac{1}{16}$. Similarly for the S_3 generation obtained by sibmating in F_2, $x=\frac{1}{4}+\frac{1}{4}=\frac{1}{2}$, $y=\frac{1}{16}+\frac{3}{16}=\frac{1}{4}$, and $z=\frac{1}{2}^2=\frac{1}{4}$.

The total variability as measured from the phenotypes will vary with the generation according to the relative magnitudes of D, H and $S^2(h)$. But in a deeper sense the differences are no more than a redistribution among the components. Genetically the total variability is constant, as is indeed required by the permanence of genetic differences. A similar relation holds for the components of variation depending on genotype–environment interactions, and a similar balance sheet may be struck for them (Mather and Morley Jones, 1958).

The situation is changed in detail, though not in principle, if selection is practised. Some of the gene differences will then be lost because certain alleles will be fixed in the sense that every member of the population will become homozygous for them. This will be reflected in a corresponding and

permanent departure of the mean from the mid-parent, and this departure will thus come to contain a further component depending on the d increments of the fixed genes as well as the h increments of those still unfixed.

Returning, however, to the case where selection is absent, the visible variability as measured by $xD+yH+zS^2(h)$ is not the total of the variability contained in the cross. Indeed it may be only a small part of it. Let us consider a very simple example where the two parent lines differ in k unlinked genes, each contributing the same d increment and all with $h = 0$. The maximum difference obtainable is where all the increasing alleles are associated in one line and all the decreasing alleles in another. Each line then departs by $S(d)$ from the mid-parent so that the total variation is $S^2(d) = (kd)^2 = kD$. Now in the F_2 from crossing two such lines, half the variation is present in the heterozygous state and will be reflected in the H and $S^2(h)$ components. The other half is present in differences between homozygotes and is measured by the D component. But half the total variability is $\frac{1}{2}kD$ of which $\frac{1}{2}D$ constitutes only the fraction $1/k$. The fraction $(k-1)/k$ of the variability is concealed, obviously as a result of balancing combinations of homozygous genes of the type *AAbb* etc. The more genes there are the greater will be this concealed fraction $(k-1)/k$. This concealed fraction is the homozygotic potential variability of Mather (1943). With linkage the concealed fraction is increased where the repulsion phase is preponderant and decreased where coupling relations preponderate. It is not difficult to accommodate inequality of the k values of d in the calculation, but the simple case outlined will serve to illustrate the point.

Two balance sheets of variability may thus be struck. The first relates to the immediately measureable variability which may be redistributed among the three components and this may be represented by the relation $x+y+z = 1$ in the expression $xD+yH+zS^2(h)$. The second balance sheet goes deeper, for it covers all the variability, potential as well as free, in the cross and it depends on the additional parameter k. In principle, k can be estimated in various ways by quantities generally denotable as K, the number of effective factors (Mather, 1949a). Such estimates are, however, difficult to arrive at reliably, and little attention has so far been paid to their calculation. Nevertheless without reliable estimation of the number of effective factors the balance of potential and free variability cannot be struck and the magnitude of ultimate response to selection cannot be calculated. The derivation of reliable methods of estimation is long overdue.

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