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STATISTICAL GENETIC THEORY AND PROCEDURES USEFUL IN STUDYING VARIETIES AND INTER-VARIETAL CROSSES IN MAIZE

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STATISTICAL GENETIC THEORY AND PROCEDURES USEFUL IN STUDYING VARIETIES AND INTERVARIETAL CROSSES IN MAIZE[1]

C. O. Gardner and J. E. Lonnquist[2]

An understanding of the fundamental nature of gene action involved in the phenomenon of heterosis and in the inheritance of quantitative characters in general is very important to the plant breeder. Although many questions remain unanswered, a great deal of progress has been made in the last decade in gaining a better understanding of the inheritance of yield and other quantitative characters in corn. The utilization of biometrical genetic techniques has been found extremely useful in studying corn populations to determine the relative importance of additive gene action, dominance, overdominance, and epistasis in the inheritance of yield.

The purpose of this paper is to summarize biometrical genetic theory pertaining to panmictic populations and to indicate procedures that appear to be useful in studying open-pollinated varieties of corn, their intercrosses and other derived populations.

Review of Literature

Maize is a naturally cross-pollinated species, and scientists who have studied racial development in maize consider the intercrossing of various types and related species to have been of tremendous importance in providing the variability necessary for the development of more productive types (28, 46, 47). The modern dent corn varieties of the United States Cornbelt are generally considered to have arisen from both intentional and unintentional crossing of distinct types (1). The development of the germplasm pool which gave rise to Cornbelt dent varieties was followed by a long period of selection (mass and ear-to-row) by experiment station workers and hundreds of farmers wherever corn was grown. This activity continued over a period of more than 100 years and resulted in the development of a large number of varieties which differed from one another in various characteristics. Some of these varieties were quite outstanding, and the better known ones, frequent winners of corn shows, became widely distributed. The infiltration of new germplasm into these varieties together with continued selection resulted in a vast array of substrains, each retaining the variety designation (e.g. Reid, Midland, Leaming, etc.) but differing somewhat in yield and other agronomic characteristics.

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The selection practiced by corn growers and the early breeders was largely that of choosing an ear type considered by each to be ideal. The "ideal" type may have been generated by what was thought necessary to win corn shows or perhaps by other ideas the grower may have had concerning traits which may or may not have been important. The major accomplishment was that of selection for superior adaptation of the varieties to the particular areas in which they were being grown. In the early part of the present century, attempts to improve yielding ability of adapted strains by mass selection and ear-to-row selection procedures were generally believed to be unsuccessful.

The possibility of increasing yields of corn by growing F_1 variety crosses was first reported by Beal (3). These findings resulted in rather extensive studies of variety crosses, many of which were summarized by Richey (36). In 244 studies summarized by Richey, 82.4% of the crosses exceeded the midparent yield and 55.7% exceeded the high parent. Greatest heterotic response was obtained where the varieties crossed differed greatly in endosperm type. This is borne out in the study reported by Hayes and Olson (16) in which Minnesota 13 (a dent) was used as the common male parent. The average yields of crosses relative to the yield of the Minnesota 13 parent were 110.4% for the dent variety crosses, 115.7% for the flint crosses, and 132.5% for a single flour variety cross. Relative to the midparent the values were 110.6%, 114.9% and 134.1% respectively. Apparently the increased diversity represented by types differing in endosperm constitution was reflected in higher heterotic response in crosses. Inasmuch as flint and flour types were generally not available in the central Cornbelt and would have been unacceptable to farmers, their possible use in crosses was limited to the more northern areas of the country. Recent results in Brazil (32) are not in accord with these earlier reported findings. Sufficient diversity existed within endosperm types to provide as great or greater heterotic response from crosses within types as that obtained from crosses between types. In the Cornbelt, variety crosses proved to be inconsistent with respect to the heterotic response obtained when crosses between certain named varieties were made by individuals in different areas (44, 27). This was obviously a reflection of the rather substantial differences present among different substrains of any given variety designation. The failure to realize consistent heterotic responses for crosses of any two named varieties resulted in a decline in interest among breeders in variety crosses as a method for improving corn yields. The possibility of maintaining and increasing stocks which provided suitable heterotic response for general distribution and use did not occur to workers of that time. This together with a growing interest in development of inbred lines and formation of double cross hybrids about 1920 resulted in a shift in emphasis in breeding methods.

In recent years there has been a renewal of interest in studying varieties and variety crosses in maize. This has resulted partly from a desire to determine the type of gene action responsible for heterosis. Robinson et al. (38, 41) reported results from all possible intercross combinations of six southern prolific varieties. The F_1 generation was found to average about 20% more than the midparent and

11.5% more than the better parent in yield. The range in superiority relative to the midparent value was shown to be from 4.6% to 46.2%. Relative to the high parent, the F_1 yields ranged from -7.5 to 32.2%. The results obtained were surprisingly large and indicative of substantial diversity among the varieties studied.

A study of variety crosses involving varieties typical of those found in the central Cornbelt was reported by Lonnquist and Gardner (27). Twelve varieties or varietal composites were used resulting in a total of 66 F_1 crosses which were compared with the parents. Results from a total of four test locations (2 locations in each of 2 years) showed the F_1 crosses to average 8.5% over the midparent and 2.8% over the high parent. Taken as a whole, the range in superiority relative to the midparent was -4.2 to 30.9% and relative to the high parent -4.7 to 11.1%. One variety used as a parent was extremely low in yield presumably due to inbreeding, and two other varieties (second cycle synthetics) which showed improvement over their original parent varieties were included in these tests. When crosses involving these three varieties were excluded from the comparisons, the average heterotic response was shown to be 4% relative to the midparent and 2.3% relative to the high parent, a substantially lower response than that reported for the southern prolific varieties.

Moll, et al. (31) crossed varieties from three different regions --southeastern United States, the central Cornbelt, and Puerto Rico. Although the study was extremely limited in scope, it did indicate that heterosis, expressed in percent of the parental mean, increases with increased genetic diversity. Average heterosis for crosses of varieties from the same region was only 4% compared to 24% heterosis observed in crosses of varieties from different regions. Crosses between varieties of southeastern United States and the central Cornbelt exhibited less heterosis than crosses between any of the United States varieties and the Puerto Rican varieties. This response is in agreement with expectation based upon our knowledge of the relationship between varieties in the two regions of the United States. Although the Puerto Rican varieties were low in yield, they appear to possess genes for increased yield not currently present in varieties in this country; consequently, they may be of potential value in the improvement of grain yield in corn in the United States.

In attempting to provide an explanation for the heterotic response from crosses of lines or varietal populations, the biometrical geneticists have derived expectations of means and variances based upon relatively simple genetic models. Most of the results obtained in corn can be explained in terms of additive gene action with dominance without resorting to overdominance (14). Epistasis does not appear to be an important source of genetic variation in varieties but may be very important in hybrid combinations (2, 7, 14, 33, 37, 43, 45). More information is needed on the epistatic contribution to heterotic response.

Genetic Structure of an Open-Pollinated Variety

An open-pollinated variety of corn may be viewed as a random-mating population that has reached equilibrium. At equilibrium, the forces of mutation and natural selection balance one another and the population gene frequencies do not change from generation to generation. Even though the genes are linked on the chromosomes, the genotypic frequencies are those expected with random mating and independent assortment among loci.

The simplest genetic model which one might consider is a population (variety) mating at random and segregating for a single pair of genes, B_i, b_i, at the ith locus. The letter "i" is simply used as a subscript in a general term which can be made specific by assigning "i" one of the numbers 1, 2, 3, ... n, where these numbers identify the n loci involved in the inheritance of a specific quantitative character. For the time being, we are assuming that genes at n-1 of the loci are fixed in homozygous state and that segregation is occurring at only one locus. Consequently, the "i" subscript can be disregarded temporarily.

Let p be the frequency of the more favorable gene B and (1-p) be the frequency of the less favorable gene b. Let 2a be the difference between the two homozygous genotypes and let d be the degree of dominance. If Z is used to indicate the average value of the individuals homozygous for the less favorable gene, the genotypes in a segregating population, their frequencies, and their genotypic values may be represented as follows:

Genotypes	Frequency (f)	No. of favorable genes (X)	Genotypic value	
			Uncoded (Y')	Coded (Y=Y'-Z-a)
BB	p^2	2	Z + 2a	a
Bb	2p(1-p)	1	Z + a + da	da
bb	$(1-p)^2$	0	Z	-a

From the above table, the scale for the degree of dominance becomes obvious by examining the genotypic values for the three genotypes. When d = 0, Bb = (BB + bb)/2, and no dominance exists. When d = 1, Bb = BB, and the B gene is completely dominant over b. When d = -1, Bb = bb, and the b gene is completely dominant over B. The complete scale can be summarized as follows:

Value of d	Degree of dominance
$d = 0$	No dominance
$0 < d < 1$	Partial dominance of B over b
$d = 1$	Complete dominance of B over b
$d > 1$	Overdominance (bb < Bb > BB)
$-1 < d < 0$	Partial dominance of b over B
$d = -1$	Complete dominance of b over B
$d < -1$	Underdominance (Bb < bb)

The genetic parameters that can be calculated from the above frequency distribution table are: (1) the population mean, (2) the additive effect of the favorable gene B, (3) the total genetic variance, (4) the additive genetic variance and (5) the variance due to dominance deviations from the additive scheme. In a similar manner, frequency distribution tables can be formulated for other populations derived from the base population by crossing, selfing, or random mating and their genetic parameters can be determined.

The mean of the jth variety (j being an identifying subscript indicating a specific variety when j is assigned some number) is obtained by multiplying each genotypic value by its respective frequency and summing over the three genotypes. Since the sum of the frequencies, $\sum_{i=1}^{3} f_i = p^2 + 2p(1 - p) + (1 - p)^2$, is equal to 1.0, the mean and the sum of the genotypic values are identical. The variety mean is

$$V_j = \bar{Y} = \sum_{i=1}^{3} f_i Y_i \Big/ \sum_{i=1}^{3} f_i = \sum_{i=1}^{3} f_i Y_i$$

$$= p^2 a + 2p(1 - p) da + (1 - p)^2(-a)$$

$$= [(2p - 1) a + 2p(1 - p) da] \qquad (1)$$

The contribution of a given locus to the variety mean has two terms. One, $(2p - 1)a$, is attributable to homozygotes in the population and the other, $2p(1 - p)da$, is attributable to heterozygotes. When dominance is lacking ($d = 0$), the second term is zero and the mean is directly proportional to gene frequency, $V_j = (2p - 1)a$. When dominance exists, the mean is proportional to the square of the gene frequency, and when dominance is complete, $V_j = (-1 + 4p - 2p^2)a$.

The additive effect of the gene B can be calculated as its average substitution value in the population or as the average change in genotypic value per unit change in number of favorable genes at each locus in the genotype. This is the ordinary least squares linear regression coefficient where Y = coded genotypic value and X = number of favorable genes. The regression coefficient can be calculated as the ratio of the covariance between X and Y to the variance of X and is calculated from the following relationships:

$$\bar{X} = \sum_{i=1}^{3} f_i X_i \Big/ \sum_{i=1}^{3} f_i = \sum_{i=1}^{3} f_i X_i$$

$$= 2p^2 + 2p(1 - p) = 2p$$

$$\bar{Y} = V_j = (2p - 1)\, a + 2p(1 - p)\, da \text{ (See equation}$$

The covariance between X and Y is

$$\sigma_{xy} = \sum_{i=1}^{3} f_i X_i Y_i - \sum_{i=1}^{3} f_i X_i \sum_{i=1}^{3} f_i Y_i$$

$$= 2p^2 a + 2p(1 - p)\, da - (2p) \left[(2p-1)\, a + 2p(1 - p)\, da\right]$$

$$= 2p(1 - p) \left[a + (1 - 2p)\, da\right]$$

The variance of X is

$$\sigma_x^2 = \sum_{i=1}^{3} f_i X_i^2 - \left(\sum_{i=1}^{3} f_i X_i\right)^2$$

$$= 4p^2 + 2p(1 - p) - (2p)^2$$

$$= 2p(1 - p)$$

The additive effect of the gene B is the linear regression coefficient

$$\beta_{yx} = \sigma_{xy} / \sigma_x^2 = \frac{2p(1 - p) \left[a + (1 - 2p)\, da\right]}{2p(1 - p)}$$

$$= \left[a + (1 - 2p)\, da\right] = \left[1 + (1 - 2p)\, d\right] a \qquad (2)$$

Equation (2) indicates that when dominance is lacking ($d = 0$), the additive effect of the favorable gene is constant and is independent of gene frequency. However, if dominance exists, the additive effect of the favorable gene will be highest when its frequency is low and lowest when its frequency is high.

The total genotypic variance among individuals in the population as calculated from the frequency table is

$$\sigma_G^2 = p^2 a^2 + 2p(1 - p)\, d^2 a^2 + (1 - p)^2 (-a)^2 - \left[(2p - 1)\, a + 2p(1 - p)\, da\right]^2$$

$$= 2p(1-p)\left[1 + 2(1-2p)\,d + (1-2p+2p^2)\,d^2\right]a^2 \qquad (3)$$

The total genetic variance can be subdivided into two parts: (1) the additive genetic variance and (2) variance due to dominance deviations.

Additive genetic variance is the variance due to the additive effect of the favorable gene or the variance in genotypic values that can be explained by linear regression on number of favorable genes in the genotype.

$$\sigma_A^2 = (\sigma_{xy})^2 / \sigma_x^2 = \{2p(1-p)\left[a + (1-2p)\,da\right]\}^2 / 2p(1-p)$$
$$= 2p(1-p)\left[1 + (1-2p)\,d\right]^2 a^2 \qquad (4)$$

Additive genetic variance can also be defined as the variance due to deviations in breeding values, where breeding value is defined as the mean value of the progeny of a genotype when the genotype is mated at random to other members of the population. Breeding value is also the predicted value of the genotype using the equation

$\hat{Y}_i = \overline{Y} + \beta_{yx}\,(X_i - \overline{X})$; hence, additive genetic variance is

$\sum_{i=1}^{3} f_i\,(\hat{Y} - \overline{Y})^2 = (\sigma_{xy})^2 / \sigma_x^2$ as above.

The remainder of total genetic variance is that which cannot be explained by linear regression and it arises as a consequence of dominance. It may be calculated as a difference $\sigma_G^2 - \sigma_A^2 = \sigma_D^2$ or it may be calculated directly as the variance due to deviations from regression, i.e. deviations of genotypic values from breeding values,

$$\sigma_D^2 = \sum_{i=1}^{3} f_i\,(Y_i - \hat{Y})^2 = \sum_{i=1}^{3} f_i\,(Y_i - \overline{Y})^2 - \sum_{i=1}^{3} f_i\,(\hat{Y} - \overline{Y})^2$$
$$= 2p(1-p)\left[1 + 2(1-2p)\,d + (1-2p+2p^2)\,d^2\right]a^2$$
$$- 2p\,(1-p)\left[1 + (1-2p)\,d\right]^2 a^2$$
$$= 4p^2\,(1-p)^2\,d^2\,a^2 \qquad (5)$$

Although the above calculations are all based on segregation at a single locus, the extension to several loci is possible if the genes at different loci are assumed to act independently. That is, if there is no epistasis, the total genotypic effect is simply the sum of the effects of individual loci. If there are n loci involved, each

segregating for two genes and if p_i is used to represent the frequency of the more favorable gene, $(1 - p_i)$ the frequency of the less favorable gene, $2a_i$ the difference between the two homozygotes, and d_i the degree of dominance at the ith locus, the previously calculated genetic parameters can be rewritten as follows:

Variety mean

$$V_j = \sum_{i=1}^{n} [(2p_i - 1)\, a_i + 2p_i\, (1 - p_i)\, d_i\, a_i] \qquad (6)$$

Additive effect of favorable genes

$$\beta_{yx} = \sum_{i=1}^{n} [1 + (1 - 2p_i)\, d_i]\, a_i \qquad (7)$$

Total genetic variance

$$\sigma_G^2 = 2 \sum_{i=1}^{n} p_i\, (1 - p_i) [1 + 2(1 - 2p_i)\, d_i + (1 - 2p_i + 2p_i^2)\, d_i^2] a_i^2 \qquad (8)$$

Additive genetic variance

$$\sigma_A^2 = 2 \sum_{i=1}^{n} p_i\, (1 - p_i) [1 + (1 - 2p_i)\, d_i]^2\, a_i^2 \qquad (9)$$

Dominance variance

$$\sigma_D^2 = 4 \sum_{i=1}^{n} p_i^2\, (1 - p_i)^2\, d_i^2\, a_i^2 \qquad (10)$$

The above values are appropriate if the population is at equilibrium with respect to linkage phases or if the loci involved are not linked. If for any reason linkage disequilibrium exists, the distributions and genotypic values would be affected. A more detailed discussion of the derivation of the expectations given above may be found in slightly different notation in books by Falconer (11) and Mather (29) and in a paper by Comstock and Robinson (5).

The maintenance of varieties is important from the standpoint of their use in genetic studies and as a source of germplasm in breeding programs. This is generally done by random mating either by hand pollination in the nursery or by open-pollination in a field isolated from other corn. If the population size is kept large, the various genotypic frequencies and genotypic variances will remain relatively

constant in the absence of selection. However, if small populations are used, inbreeding will occur, the equilibrium state will be upset, and the genotypic frequencies and genetic variances will be altered.

The amount of inbreeding that occurs in the maintenance of varieties is related to sample size. If N is the number of individuals used to advance a population from one generation to the next by random mating, the decrease in heterozygosity will be 1/2N. Hence, if the number of individuals grown each generation is known, the amount of inbreeding in a population relative to some base in earlier generations can easily be calculated. The amount of inbreeding that occurs under random mating in finite populations is given for different population sizes in the following table:

Population size	% inbreeding each generation
25	2.00
50	1.00
100	.50
200	.25
500	.10
1000	.05
5000	.01

The above table emphasizes the need for adequate sample sizes (1000 or more individuals) to maintain the equilibrium genotype frequencies desirable in populations to be used for genetic studies and for reasonable constancy in test performance from one generation to the next.

Means of Populations Derived from Varieties

Individuals in a variety are often self-fertilized to produce inbred lines. This is the most intense form of inbreeding and in one generation reduces the mean of a random set of lines to

$$V_{js} = \sum_{i=1}^{n} (2p_i - 1)\, a_i + \sum_{i=1}^{n} p_i\,(1 - p_i)\, d_i\, a_i \qquad (11)$$

By comparing this equation with (6), one can immediately see that the second term which involves dominance is the only one affected by inbreeding and it is reduced by one-half each generation. Data collected on a set of varieties and their selfed progenies will permit an estimate of the inbreeding depression. If there is no dominance ($d_1 = d_2 = \ldots = d_n = 0$), the second term of equations (6) and (11) will both be zero, and there will be no inbreeding depression under the model considered.

Individuals chosen at random in one variety may be crossed to individuals chosen at random in another variety to form an intervariety cross which will be called the F_1. The F_1 may in turn be advanced to

the F_2 generation by allowing random mating among individuals in the F_1 population. Also randomly chosen individuals in the F_1 population are sometimes self-fertilized to produce what will be symbolized as the F_1s. Data collected on these kinds of populations simultaneously with that collected on the parent varieties permit an estimation of heterosis and inbreeding depression. In this paper heterosis is defined as the difference between the F_1 and the midparent value (mean of the two parent varieties).

If the subscripts j and k are used as the general terms to identify the varieties used in an intervariety cross, p_{ji} may be used to represent the frequency of the more favorable gene at the ith locus in the jth variety and p_{ki} may be used to represent the frequency of the same gene in the kth variety. Then the parent varieties, the midparent value, the selfed progeny of the parent varieties, the F_1 hybrid between the varieties, the F_2 generation, and the selfed progeny of the F_1 have the following genotypic mean values:

Variety means

$$V_j = \sum_{i=1}^{n} (2p_{ji} - 1)\, a_i + 2 \sum_{i=1}^{n} p_{ji}\,(1 - p_{ji})\, d_i\, a_i \qquad (12)$$

$$V_k = \sum_{i=1}^{n} (2p_{ki} - 1)\, a_i + 2 \sum_{i=1}^{n} p_{ki}\,(1 - p_{ki})\, d_i\, a_i \qquad (13)$$

Midparent value

$$MP_{jk} = (V_j + V_k) / 2$$

$$= \sum_{i=1}^{n} (p_{ji} + p_{ki} - 1)\, a_i + \sum_{i=1}^{n} (p_{ji} + p_{ki} - 2p_{ji}\, p_{ki})\, d_i\, a_i$$

$$- \sum_{i=1}^{n} (p_{ji} - p_{ki})^2\, d_i\, a_i \qquad (14)$$

Mean of selfed progeny of varieties

$$V_j s = \sum_{i=1}^{n} (2p_{ji} - 1)\, a_i + \sum_{i=1}^{n} p_{ji}\,(1 - p_{ji})\, d_i\, a_i \qquad (15)$$

$$V_k s = \sum_{i=1}^{n} (2p_{ki} - 1)\, a_i + \sum_{i=1}^{n} p_{ki}\,(1 - p_{ki})\, d_i\, a_i \qquad (16)$$

Mean of the F_1 cross of two varieties

$$F_{1.jk} = \sum_{i=1}^{n} (p_{ji} + p_{ki} - 1)\, a_i + \sum_{i=1}^{n} (p_{ji} + p_{ki} - 2p_{ji}p_{ki})\, d_i\, a_i \qquad (17)$$

Mean of the F_2 generation produced by random mating F_1 plants

$$F_{2.jk} = \sum_{i=1}^{n} (p_{ji} + p_{ki} - 1)\, a_i + \sum_{i=1}^{n} (p_{ji} + p_{ki} - 2p_{ji}\, p_{ki})\, d_i\, a_i - \frac{1}{2} \sum_{i=1}^{n} (p_{ji} - p_{ki})^2\, d_i\, a_i \qquad (18)$$

Mean of the selfed progeny of the F_1 generation

$$F_{1.jk}s = \sum_{i=1}^{n} (p_{ji} + p_{ki} - 1)\, a_i + \frac{1}{2} \sum_{i=1}^{n} (p_{ji} + p_{ki} - 2p_{ji}\, p_{ki})\, d_i\, a_i \qquad (19)$$

Each of the equations (12) through (19) can be written in simplified form by making the following substitutions:

Let $\mu + \alpha_j = \sum_i (2p_{ji} - 1)\, a_i$, the contribution of homozygotes to a variety mean.

Let $\delta_j = 2\sum_i p_{ji}\,(1 - p_{ji})\, d_i\, a_i$, the contribution of heterozygotes to a variety mean.

Let $h_{jk} = \sum_i (p_{ji} - p_{ki})^2\, d_i\, a_i$, the amount of heterosis or the difference between the mean of the F_1 hybrid of varieties j and k and the mean of the two parents (midparent value).

Thus μ is defined as the mean of random inbred lines that could be developed from the varieties and $\mu + \alpha_j$ is the mean of random lines that could be developed from the jth variety. Substituting the μ, α_j's, δ_j's and h_{ij}'s into equations (12) through (19) provides the following set of equations:

$$V_j = \mu + \alpha_j + \delta_j \qquad (12a)$$

$$V_k = \mu + \alpha_k + \delta_k \qquad (13a)$$

$$MP_{jk} = \mu + (\alpha_j + \alpha_k + \delta_j + \delta_k)/2 \qquad (14a)$$

$$V_j s = \mu + \alpha_j + \delta_j/2 \qquad (15a)$$

$$V_k s = \mu + \alpha_k + \delta_k/2 \qquad (16a)$$

$$F_{1.jk} = \mu + (\alpha_j + \alpha_k + \delta_j + \delta_k)/2 + h_{jk} \tag{17a}$$

$$F_{2.jk} = \mu + (\alpha_j + \alpha_k + \delta_j + \delta_k)/2 + h_{jk}/2 \tag{18a}$$

$$F_{1.jk^s} = \mu + (\alpha_j + \alpha_k)/2 + (\delta_j + \delta_k)/4 + h_{jk}/2 \tag{19a}$$

Any set of v varieties can be used to produce the populations indicated in equations (12) through (19). If the number of varieties used is v, the following populations can be evaluated in a replicated test:

Population	No. of population means evaluated
V_j	v
V_j^s	v
$F_{1.jk}$	v(v - 1)/2
$F_{2.jk}$	v(v - 1)/2
$F_{1.jk^s}$	v(v - 1)/2
Total	v(3v + 1)/2

By equating the population means to their expected values indicated in equations (12) through (19), one has a set of v(3v + 1)/2 equations involving (v + 1)(v + 2)/2 unknown parameters. These equations can be used to obtain a set of normal equations from which least squares estimates of the parameters can be obtained (see Mather (29) pp. 60-63). In order to get a unique solution, one restriction must be used because μ and the α_i are not independent. The restriction used is

$$\sum_{i=1}^{v} \alpha_i = 0.$$

One of the α_j's must be eliminated from the equations. If we eliminate the last one (α_v), we simply substitute $-\sum_{i=1}^{v-1} \alpha_i$ for α_v wherever α_v appears in the equations. The normal equations can then be calculated and solved for the unknown constants.

The information obtained in experiments of this kind involving varieties and other populations derived from them is useful in arriving at conclusions concerning the kinds of gene action involved in the inheritance of quantitative traits and in understanding heterosis and inbreeding depression.

The method outlined above was applied to data of Lonnquist and Gardner (unpublished) to interpret the means obtained from four varieties (two open-pollinated varieties and two synthetic varieties derived from them) and other populations developed from the varieties as indicated in equations (12) through (19). Fitting the complete model accounted for 99.96 percent of the total variation in grain yield among the population means. This indicates that the model involving additive gene action with dominance is adequate to explain variation in grain yield. If epistasis exists it was not detectable in the population means. This is not too surprising when one considers the heterogeneous nature of the individuals comprising such populations and the cancelling effects of the different kinds of epistasis that could occur in population means. Least squares estimates of heterosis ranged from 4.6 to 11.9 percent and averaged 7.8 percent. Heterosis must therefore be attributed to dominance of favorable genes and differing gene frequencies in the varieties used.

A similar model considered by Robinson and Cockerham (37) included multiple alleles, but this extension does not alter the results. They considered theoretical means of two varieties, their F_1 and F_2 and selfed progenies of these three populations. They expressed their population means in terms of three parameters A, B, and C which can be expressed in terms of symbols used in this paper as

$$A' = (a_j + a_k)/2$$

$$B' = (\delta_j + \delta_k + h_{jk})/2$$

$$C' = (h_{jk})/2$$

In their experiment, the varieties Jarvis and Indian Chief, the F_1 cross, the F_3 generation (assumed to be the same as the F_2) and selfed progeny of the varieties were included. Six levels of heterozygosity were considered from a theoretical standpoint: $(V_1 + V_2)/2$, F_1, F_2, $(V_1s + V_2s)/2$, F_1s, F_2s, but in their experiment the F_2s were not included and the F_3 was substituted for the F_2. Thus, only 5 levels of heterozygosity were actually tested. The data conformed to the model with additive gene action and dominance and no evidence of epistasis was observed. Heterosis was manifested for yield but not for ear height.

Genetic Variances in Open-Pollinated Varieties and Derived Populations

Genetic variances in open-pollinated varieties and other populations derived from them provide additional valuable information on heterosis and the nature of gene action involved in determining yield of corn. The use of "biparental progenies" as outlined by Comstock

and Robinson (5, 6) and sometimes referred to as "Design I Experiments" have been found very useful in obtaining genetic information from corn varieties. In such experiments plants chosen at random and designated as females are divided into groups of equal size, usually 4 per group, and each group is mated to a separate randomly chosen plant designated as a male since it provides the pollen. Such a mating system results in 4 full-sib families within each half-sib family, and data collected on the families in a number of environments can be analyzed by analysis of variance technique which permits the estimation of genetic and environmental components of variance and of genotype-environment interaction components. The analysis of variance in its most elemental form is given in Table 1.

The components of particular interest are σ_m^2 and σ_f^2 because they arise as a result of genetic differences among males and among females mated to the same male, respectively; hence they have a genetic interpretation. The within plots component σ_w^2 is also of some interest because it is partially genetic in origin. If the experiment is designed in such a way as to permit an estimation of the within plot environmental variance, the σ_w^2 may be partitioned into a genetic part and an environmental part. Comstock and Robinson (5, 6) derived the expectations of these genetic components of variance using a model in which epistasis was assumed to be absent. A number of genetic studies involving F_2 generations of crosses between homozygous lines as well as open-pollinated varietal populations were interpreted under this model. Both southern dent varieties and Cornbelt varieties have been investigated (26, 38, 39, 40, 42).

In terms previously defined in this paper, the expectations of the genetic components are as follows when both the males and the females are from the same variety (intravariety cross)

$$\sigma_m^2 = \frac{1}{2} \sum_{i=1}^{n} p_i (1 - p_i) [1 + (1 - 2p_i) d_i]^2 a_i^2 \qquad (21)$$

$= \frac{1}{4} \sigma_A^2$ where σ_A^2 is the additive genetic variance (Equation 9)

$$\sigma_{f(m)}^2 = \frac{1}{2} \sum_{i=1}^{n} p_i (1 - p_i) [1 + 2(1 - 2p) d_i + (1 - 2p_i + 2p_i^2) d_i^2] a_i^2$$

$= \sigma_m^2 + \sum_{i=1}^{n} p_i^2 (1 - 2p_i)^2 d^2 a^2 = \frac{1}{4} \sigma_A^2 + \frac{1}{4} \sigma_D^2$ where σ_D^2 is the dominance variance (Equation 10)

Consequently, the following estimates can be obtained

$$\hat{\sigma}_A^2 = 4 \hat{\sigma}_m^2 \qquad \text{and}$$

Table 1. Analysis of variance form for progenies in a Design I experiment.

Source of variation	d.f.	Mean square	Parameters estimated
Environments	$e - 1$		
Reps in environments	$e(r - 1)$		
Males	$m - 1$	M_m	$\sigma_w^2/k + \sigma_p^2 + r\sigma_{f(m)e}^2 + re\sigma_{f(m)}^2 + rf\sigma_{me}^2 + rfe\sigma_m^2$
Females in males	$m(f - 1)$	M_f	$\sigma_w^2/k + \sigma_p^2 + r\sigma_{f(m)e}^2 + re\sigma_{f(m)}^2$
Males x environments	$(m-1)(e-1)$	M_{me}	$\sigma_w^2/k + \sigma_p^2 + r\sigma_{f(m)e}^2 + rf\sigma_{me}^2$
Females in males x environments	$m(f-1)(e-1)$	M_{fe}	$\sigma_w^2/k + \sigma_p^2 + r\sigma_{f(m)e}^2$
Error	$e(mf-1)(r-1)$	M_p	$\sigma_w^2/k + \sigma_p^2$
Plants within plots	$\sum_i (k_i - 1)$	M_w	σ_w^2

e = Number of environments.

r = Number of replications per environment.

m = Number of males.

f = Number of females per male.

k_i = Number of plants within the ith plot.

σ_w^2 = Component of variance due to variation among plants within plots.

σ_p^2 = Component of variance due to variation among plots within replications.

$\sigma_{f(m)e}^2$ = Component of variance due to the interaction of female genotypes with environment.

σ_{me}^2 = Component of variance due to the interaction of male genotypes with environment.

$\sigma_{f(m)}^2$ = Component of variance due to variation among female genotypes mated to the same male.

σ_m^2 = Component of variance due to variation among male genotypes.

$$\hat{\sigma}^2_D = 4(\hat{\sigma}^2_{f(m)} - \hat{\sigma}^2_m)$$

where the caret (^) denotes an estimate of the parameter.

The genetic variance among individuals within families, estimates $\frac{1}{2}\sigma^2_A + \frac{3}{4}\sigma^2_D$ in the absence of epistasis. Hence if σ^2_w can be su divided into two parts, $\sigma^2_{w_g}$, a genetic portion, and $\sigma^2_{w_e}$, an environmental portion, then

$$\sigma^2_{w_g} = 3\,\sigma^2_{f(m)} - \sigma^2_m$$

Any departure of estimates from this equality could be interpreted t be a consequence of epistasis. Comstock et al. (7) used F_1 hybrids estimate $\hat{\sigma}^2_{w_e}$ and calculated $\hat{\sigma}^2_{w_g} = \hat{\sigma}^2_w - \hat{\sigma}^2_{w_e}$. Then they made the comparison $\hat{\sigma}^2_{w_g} - (3\,\hat{\sigma}^2_{f(m)} - \hat{\sigma}^2_m)$. They concluded that the variance whic could be attributed to epistasis was not more than 10 percent of the total genetic variance.

In general the genetic variance studies involving varieties ind cate that additive genetic variance is greater than variance due to dominance deviations for all quantitative characters studied includi grain yield. The magnitude of the additive genetic variance observe for grain yield suggests that considerable progress in the direction of higher yield should be possible by any of the methods of mass or ear-to-row selection. Gardner's results (13) using mass selection i the Hays Golden variety substantiate this conclusion. Overdominance does not appear to be an important cause of genetic variation in cor varieties. For a summary of the genetic studies conducted on open-pollinated varieties see Gardner (14).

When plants from variety j (males) are crossed to plants of another variety k (females), the intervariety male component of variance has the expectation

$$\sigma^2_{m_{jk}} = \frac{1}{2}\sum_{i=1}^{n} p_{ji}\,(1 - p_{ji})\,[1 + (1 - 2p_{ki})\,d_i]^2\,a_i^2 \qquad (23)$$

The same component in the reciprocal cross has the expectation

$$\sigma^2_{m_{kj}} = \frac{1}{2}\sum_{i=1}^{n} p_{ki}\,(1 - p_{ki})\,[1 + (1 - 2p_{ji})\,d_i]^2\,a_i^2 \qquad (24)$$

The corresponding intervariety female components of variance are

$$\sigma^2_{f(m)_{jk}} = \frac{1}{2} \sum_{i=1}^{n} p_{ki} (1 - p_{ki}) [1 + 2(1 - 2p_{ji}) d_i + (1 - 2p_{ji} + 2p_{ji}^2) d^2] a^2$$

$$= \sigma^2_{m_{kj}} + \sum_{i=1}^{n} p_{ki} (1 - p_{ki}) p_{ji} (1 - p_{ji}) d_i^2 a_i^2 \quad (25)$$

$$\sigma^2_{f(m)_{kj}} = \frac{1}{2} \sum_{i=1}^{n} p_{ji} (1 - p_{ji}) [1 + 2(1 - 2p_{ki}) d_i + (1 - 2p_{ki} + 2p_{ki}^2) d^2] a^2$$

$$= \sigma^2_{m_{jk}} + \sum_{i=1}^{n} p_{ki} (1 - p_{ki}) p_{ji} (1 - p_{ji}) d_i^2 a_i^2 \quad (26)$$

The utilization of the above approach to gain an understanding of open-pollinated varieties was reported by Robinson et al. (43). They calculated the following additional quantities:

1. The difference between the mean intravariety and mean intervariety male components of variance

$$\frac{1}{2} (\sigma^2_{m_{jj}} + \sigma^2_{m_{kk}}) - \frac{1}{2} (\sigma^2_{m_{jk}} + \sigma^2_{m_{kj}})$$

$$= \sum_{i=1}^{n} (p_{ji} - p_{ki})^2 (p_{ji} + p_{ki} - 1) [1 - (p_{ji} + p_{ki} - 1) d_i] d_i a_i^2 \quad (27)$$

2. The ratio of the intravariety: intervariety male components of variance

$$(\sigma^2_{m_{jj}} + \sigma^2_{m_{kk}}) / (\sigma^2_{m_{jk}} + \sigma^2_{m_{kj}}) \quad (28)$$

3. The difference between the mean intravariety and the mean intervariety female components of variance

$$\frac{1}{2} (\sigma^2_{f(m)_{jj}} + \sigma^2_{f(m)_{kk}}) - \frac{1}{2} (\sigma^2_{f(m)_{jk}} + \sigma^2_{f(m)_{kj}})$$

$$= \frac{1}{2} \sum_{i=1}^{n} (p_{ji} - p_{ki})^2 (p_{ji} + p_{ki} - 1) [2 - (p_{ji} + p_{ki} - 1) d_i] d_i^2 a_i^2 \quad (29)$$

The contribution of an individual locus to equation (27) will be positive when $p_{ji} \neq p_{ki}$ or $p_{ji} \neq (1 - p_{ki})$ and either

(1) $d > 0$ and $1 < (p_{ji} + p_{ki}) < (1 + 1/d)$ or

(2) $d < 0$ and $(1 + 1/d) < (p_{ji} + p_{ki}) < 1$.

When equation (27) is positive, the ratio given by (28) will exceed one. The contribution of an individual locus to equation (29) will be positive when $p_{ji} \neq p_{ki}$ or $p_{ji} \neq (1 - p_{ki})$ and either

(1) $d > 0$ and $1 < (p_{ji} + p_{ki}) < (1 + 2/d)$ or

(2) $d < 0$ and $(1 + 2/d) < (p_{ji} + p_{ki}) < 1$.

Robinson et al. (42) calculated theoretical ratios for equation (28) but they were based on a single locus. Observed ratios which are a consequence of summation over all loci where gene frequencies, gene effects, and degree of dominance may vary were compared to theoretical ratios. Observed ratios were found to be very plausible for the additive model with partial to complete dominance.

Intercrosses of Random Inbred Lines

Another possibility to be discussed briefly is the utilization of a random set of inbred lines developed from an open-pollinated variety. Such a set of lines may be developed by choosing plants of the open-pollinated variety by some procedure that insures randomness and by self-pollinating these plants and their offspring each generation to provide one line derived from each original open-pollinated plant. One may expect natural selection to eliminate a few of the lines, but the goal should be to have lines from as many of the original plants as possible.

Inbred lines may be crossed in many combinations--single crosses, three-way crosses and double crosses. The theory of the diallel cross (all possible single crosses among a set of inbred lines) has been the subject of numerous publications (15, 17, 18, 19, 20, 24, 30). More recently, the theory of three-way crosses (36) and double crosses (35) has been developed by Rawlings and Cockerham. All three kinds of crosses yield valuable genetic information.

Perhaps the most useful mating system for a large number of lines is a modification of the diallel cross called Experimental Design II by Comstock and Robinson (5, 6). In this system the lines are divided into groups of 8 or 10. Each group is further subdivided into two subgroups and all possible crosses between the two subgroups are made. Assignment to the groups and subgroups must be at random, so that information from the several groups can be pooled and used to characterize the genetic situation in the original variety. If the sample of lines is to adequately represent the variety, the number must be fairly

large. The number of progenies in a complete diallel cross set would be unmanageable, but the number in a Design II experiment can easily be handled. Listed below is a comparison of the number of progenies produced for testing when n lines are crossed in (1) a complete diallel, (2) three-way crosses, (3) double crosses, and (4) a Design II experiment.

n	Diallel (single crosses)	Triallel (3-way crosses)	Quadriallel (double crosses)	Design II crosses 4 x 4	Design II crosses 5 x 5
10	45	360	630		25
20	190	3,420	14,535		50
40	780	29,640	274,170	80	100
80	3,160	246,480	4,744,740	160	200
160	12,720	2,009,760	78,883,080	320	400

The more lines involved in an experiment of this kind, the less likely are the conclusions concerning genetic parameters to be in error as a consequence of sampling. Other variations of the diallel are presented in detail by Kempthorne and Curnow (25). These may be of value in permitting the use of a larger number of lines without greatly increasing the number of crosses involved and thereby providing better estimates of the population parameters.

If the two subgroups in a Design II experiment are designated as males and females, the analysis of variance in elemental form is given in Table 2.

The genetic components of interest in a Design II experiment are σ_m^2, σ_f^2, and σ_{mf}^2. If the lines used are homozygous, the expectations of these three genetic components are

$$\sigma_m^2 = \sigma_f^2 = \sum_{i=1}^{n} p_i (1 - p_i) [1 + (1 - 2p_i) d_i]^2 a_i^2 \qquad (29)$$

$$\sigma_{mf}^2 = \sum_{i=1}^{n} p_i^2 (1 - p_i)^2 d_i^2 a_i^2 \qquad (30)$$

When compared to population variances given in equations (3), (4), and (5)

$$\sigma_m^2 = \sigma_f^2 = \frac{1}{2} \sigma_A^2 \text{ and } \sigma_{mf}^2 = \sigma_D^2$$

Cockerham (8) has given the expectations of these same genetic components when the lines are at any level of inbreeding as long as they are all at the same level. For any level of inbreeding F, the expectations are

Table 2. Analysis of variance form for progenies in a Design II experiment.

Source of variation	d.f.	Mean square	Parameters estimated
Environments	e-1		
Reps in environments	e(r-1)		
Males	(m-1)	M_m	$\sigma_w^2/k + \sigma_p^2 + r\sigma_{mfe}^2 + re\sigma_{mf}^2 + rf\sigma_{me}^2 + rfe\sigma_m^2$
Females	(f-1)	M_f	$\sigma_w^2/k + \sigma_p^2 + r\sigma_{mfe}^2 + re\sigma_{mf}^2 + rm\sigma_{fe}^2 + rme\sigma_f^2$
Males x females	(m-1)(f-1)	M_{mf}	$\sigma_w^2/k + \sigma_p^2 + r\sigma_{mfe}^2 + re\sigma_{mf}^2$
Males x environments	(m-1)(e-1)	M_{me}	$\sigma_w^2/k + \sigma_p^2 + r\sigma_{mfe}^2 + rf\sigma_{me}^2$
Females x environments	(f-1)(e-1)	M_{fe}	$\sigma_w^2/k + \sigma_p^2 + r\sigma_{mfe}^2 + rm\sigma_{fe}^2$
Males x females x environments	(f-1)(m-1)(e-1)	M_{mfe}	$\sigma_w^2/k + \sigma_p^2 + r\sigma_{mfe}^2$
Error	e(mf-1)(r-1)	M_p	$\sigma_w^2/k + \sigma_p^2$

All symbols have the same meaning as in Table 1. Two new ones need to be defined:

σ_{mf}^2 = Component of variance due to the interaction of male genotypes and female genotypes.

σ_{mfe}^2 = Component of variance due to the interaction of the male genotypes, female genotypes, and environment.

$\sigma_f^2 = \sigma_m^2$

$$\sigma^2_m = \sigma^2_f = \left(\frac{1 + F}{4}\right) \sigma^2_A$$

$$\sigma^2_{mf} = \left(\frac{1 + F}{2}\right)^2 \sigma^2_D$$

where F is the coefficient of inbreeding.

The component of variance for general combining ability and that for specific combining ability in a diallel cross have the same expectations as σ^2_m and σ^2_{mf}, respectively, in the Design II experiment.

Discussion

The most widely accepted hypothesis for the explanation of hybrid vigor is the cumulative action of dominant favorable genes proposed by Davenport (10), Bruce (4), and Keeble and Pellew (23). Although more research is needed to determine the role of epistasis, the results of research on open-pollinated varieties of corn appear to be compatible with the relatively simple model involving only additive gene action with dominance. The question of level of dominance has attracted considerable attention because of Hull's (22) proposal that overdominance was the primary cause of heterosis and of the failure of breeders to make progress by mass selection and ear-to-row selection in open-pollinated varieties even though considerable genetic variation appeared to exist.

The observed heterosis in variety crosses, the effects of inbreeding, and the genetic variances within varieties and in intervariety crosses can be explained on the basis of an additive model with partial to complete dominance and with differing gene frequencies in the varieties crossed. The differences in the means of varieties, their crosses, and other derived populations and the differences in heterosis and inbreeding effects observed could result in part from favorable genes that are fixed or at high frequency in one variety and completely lacking in another. Such fixation could have occurred as a result of inbreeding (a consequence of a finite population size), genetic drift, or mutational changes that may have occurred in non-interbreeding varieties. On the other hand, only those loci that are segregating in the population will contribute to the intravariety variances or to variation among plants in a cross of two varieties.

The fact that data observed in experiments involving open-pollinated varieties fit the additive model with dominance does not exclude the possibility of epistasis existing. The occurrence of different kinds of epistasis and the interaction of numerous gene pairs or multiple sets of genes could conceivably have a cancelling effect so that the means observed by Robinson and Cockerham (38) and by

Lonnquist and Gardner (unpublished) would not deviate substantially from expectation based on an additive model with dominance. The effect on the intravariety variances might be more noticeable because some bias would be expected to result from epistasis. However, if the proportion of total genetic variance attributable to epistasis is no more than 10 percent as suggested by Comstock et al. (7), the amount of bias in estimates of additive genetic and dominance variance based on the simple model would not be serious in the kinds of conclusions drawn.

Horner et al. (21) investigated the effect of some types of non-allelic gene interactions on estimates of additive genetic variance, dominance variance, and degree of dominance made from Design I and Design II experiments when used with F_2 generation plants from a cross between homozygous lines. They concluded that bias from multiplicative gene action was not serious, and bias from the optimum gene number model was not serious except when the optimum point is near half the maximum number of favorable genes and many gene pairs are involved, but bias from complementary and duplicate factor genes could be serious.

Cockerham (8) approached the problem of non-allelic gene interactions from a more general point of view and as a logical extension of the partitioning of genetic variance originally proposed by Fisher (12). The epistatic variance is partitioned into a systematic series of components in terms of which the genetic correlations among relatives and the genetic variances and covariances of individuals and families can be completely specified. The analysis used may be compared to a 3^n factorial experiment, where n is the number of loci involved and each locus has 3 levels (3 genetic phases--BB, Bb, bb or 2, 1, and 0 favorable genes). Each locus can be partitioned into (1) an additive (linear) variance and (2) a dominance (quadratic) variance which when summed over all loci are additive genetic variance σ^2_A, and dominance variance σ^2_D, respectively. Interactions between pairs of loci can be further partitioned into (1) additive x additive (linear x linear) variance, (2) additive by dominance (linear x quadratic) variance, and (3) dominance x dominance (quadratic x quadratic) variance, which when summed over all pairs of loci are the additive x additive epistatic variance σ^2_{AA}, the additive x dominance epistatic variance σ^2_{AD}, and the dominance x dominance epistatic variance σ^2_{DD}, respectively. This can be extended to 3, 4, or any number of factor pairs up to n.

Expectations of the genetic components of variance in Design I and Design II experiments have been derived by Cockerham (9) using a model that includes epistasis as well as additive gene action and dominance. These expectations are as follows

Design I used with an open-pollinated variety.

$$\sigma^2_{m_1} = \frac{1}{4}\sigma^2_A + \left[\frac{1}{16}\sigma^2_{AA} + \frac{1}{64}\sigma^2_{AAA} + \frac{1}{256}\sigma^2_{AAAA} + \ldots\right] \qquad (31)$$

$$\sigma^2_{f(m)} = \frac{1}{4}\sigma^2_A + \frac{1}{4}\sigma^2_D + \left[\frac{3}{8}\sigma^2_{AA} + \frac{1}{8}\sigma^2_{AD} + \frac{1}{16}\sigma^2_{DD} + \frac{7}{64}\sigma^2_{AAA} + \ldots\right] \tag{32}$$

Design II used with random homozygous lines.

$$\sigma^2_{m_2} = \sigma^2_f = \frac{1}{2}\sigma^2_A + \left[\frac{1}{4}\sigma^2_{AA} + \frac{1}{8}\sigma^2_{AAA} + \frac{1}{16}\sigma^2_{AAAA} + \ldots\right] \tag{33}$$

$$\sigma^2_{mf} = \sigma^2_D + \left[\frac{1}{2}\sigma^2_{AA} + \sigma^2_{AD} + \sigma^2_{DD} + \frac{3}{4}\sigma^2_{AAA} + \sigma^2_{AAD} + \sigma^2_{ADD} + \sigma^2_{DDD} + \frac{7}{8}\sigma^2_{AAAA} + \ldots\right] \tag{34}$$

The epistatic components of variance that contribute to bias in estimates of the genetic components of variance based on the additive model with dominance are enclosed in brackets. By using Design I with an open-pollinated variety and Design II with homozygous random lines developed from that same variety and testing the progenies simultaneously, the following comparisons suggested by Cockerham (9) are possible

$$\sigma^2_{m_2} - 2\sigma^2_{m_1} = \frac{1}{8}\sigma^2_{AA} + \frac{3}{32}\sigma^2_{AAA} + \frac{7}{128}\sigma^2_{AAAA} + \ldots \tag{35}$$

$$\sigma^2_{mf} + 2\sigma^2_{m_2} - 4\sigma^2_{f(m)} = \frac{1}{4}\sigma^2_{AA} + \frac{1}{2}\sigma^2_{AD} + \frac{3}{4}\sigma^2_{DD} + \frac{9}{16}\sigma^2_{AAA} + \frac{3}{4}\sigma^2_{AAD} + \frac{7}{8}\sigma^2_{ADD} + \frac{15}{16}\sigma^2_{DDD} + \frac{49}{64}\sigma^2_{AAAA} + \ldots \tag{36}$$

$$\sigma^2_{mf} + 4\sigma^2_{m_2} - 4\sigma^2_{m_1} - 4\sigma^2_{f(m)} = \frac{1}{2}\sigma^2_{AA} + \frac{1}{2}\sigma^2_{AD} + \frac{3}{4}\sigma^2_{DD} + \frac{3}{4}\sigma^2_{AAA} + \frac{3}{4}\sigma^2_{AAD} + \frac{7}{8}\sigma^2_{ADD} + \frac{15}{16}\sigma^2_{DDD} + \frac{7}{8}\sigma^2_{AAAA} + \ldots \tag{37}$$

Each of these comparisons involve substantial amounts of epistatic variance. Equation (35) contains only additive types of epistatic variance while equations (36) and (37) contain some of all types but more of the dominance types. Comparisons such as these would provide some information on the magnitude and importance of epistasis in genetic variation among plants in an open-pollinated variety. Research is underway which may eventually provide more concrete information on the importance of epistasis in open-pollinated varieties of corn and in heterosis in F_1 hybrids between homozygous lines.

Although epistasis does not appear to be an important source of genetic variation in open-pollinated varieties of corn, this does not mean that epistasis is unimportant in corn breeding. Epistasis may be very important indeed in the hybrid produced by crossing two inbred lines.

Summary

In this paper an attempt is made to explain theoretical aspects of the relatively simple model involving additive gene action with dominance as applied to open-pollinated varieties of corn and other populations derived from them. Some discussion of epistasis and its effect on means and variances is also included. Some of the results with varieties, intercrosses among varieties, and other derived populations in which theoretical models have been used are briefly discussed. No attempt has been made to give exhaustive treatment to the subject. Interested persons should read the many references to gain a thorough understanding of current knowledge concerning corn varieties and the nature of gene action involved in determining quantitative characters.

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