Genetic Analysis of Plant Mixtures

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ABSTRACT

Plant mixtures are difficult to analyze genetically because of possible interactions between neighboring plants (i.e., between plants in the same biological group). However, a genetic modeling scheme has been devised which, theoretically, can accommodate such interactions. This study was an attempt to put the theoretical modeling procedure to an experimental test. To this end an experimental procedure was devised that generated biological groups from a well defined base population. A cultural system was used which permitted growing plant mixtures in controlled environmental facilities. This allowed the experiment to be conducted over a wide range of temperature and nutrient conditions. Application of the theoretical gene model to the experimental data permitted identification of those classes of gene effects that were responsible for genetic variation exhibited by the mixtures. Adequacy of the genetic modeling description was corroborated by precise prediction of an independent genetic response. The genetic analyses also identified statistically significant temperature- and nutrient-dependent forms of heterosis. It was concluded that the study demonstrated the suitability of the theoretical group gene model for describing complexities inherent in plant mixtures.

USE of plant mixtures in a variety of forms is becoming of increasing interest in agronomic and horticultural practice. This is largely due to the fact that plant mixtures have a number of potential advantages in comparison with genetically homogeneous plantings. Some of these advantages are: (i) a potentially greater utilization of the total environmental space, (ii) a greater stability (homeostasis) over a variety of environmental conditions (including periods of stress), and (iii) the possibility of greater overall resistance to plant pathogens and insect pests (Clay and Allard 1969; Barrett 1981). Because of these potential advantages there is an increasing interest in the improvement of mixtures through plant breeding (Mayo 1980).

In order to manipulate the genetic composition of mixtures through plant breeding, it is necessary to be able to model the system genetically. However, the main problem with the genetic characterization of mixture performance is that interactions invariably occur among neighboring individuals (i.e., among members of biological groups). The ways that growth of one plant can influence that of another are numerous, e.g., competition for limited environmental space, physical interference (shading), biochemical interference (through production of allelochemicals), etc. Accommodation of such interactions presents an insurmountable dilemma from the point of view of classical (noninteraction) quantitative genetic methodology.

The objective of this study is to demonstrate that a genetic analysis of responses due to interacting genotypes in biological groups can be made in terms of a group genetic model specifically designed for such a purpose (Griffing 1967, 1981a).

A model biological system was used to produce the experimental framework for the study. This system involved growing pairs of plants (Arabidopsis thaliana) in test tubes. The plants were derived from two homozygous parental races and their hybrid. The genetic relationship among plants in the base population provided the basis for a genetic analysis. Growing pairs of plants in test tubes produced the smallest sized groups in which individual plants interact for limited nutrient resources. Use of the Arabidopsis test tube culture system made it possible to include a large number of plants in controlled environment facilities. These facilities permitted the inclusion of a graded series of two important growth conditions; temperature and nutrients. In this way it was possible to assess the effect of temperature and nutritional stress on the performance of interacting genotypes.

The task of the study, then, is to use the data from the above experimental procedure to test the ability of the group genetic model to fully describe the complex genetic variability as it occurs in a living system.

MATERIALS AND METHODS

Experimental procedures: The experimental procedures of this study were similar to those described in an earlier publication (Griffing and Zsiros 1971). The genetic materials consisted of two races of A. thaliana and their F2. The races used were CHI (a race collected in Schisdra, Russia) and DI (a race from Dijon, France). Since A. thaliana is an obligatorily self-fertilized species, the races are homo-
TABLE 1
Genetic design involving all possible combinations of genetic types

<table>
<thead>
<tr>
<th>Genetic type</th>
<th>CHI</th>
<th>F₁</th>
<th>DI</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHI (X₁₁, X₁₂)</td>
<td>(X₁₂, X₂₁)</td>
<td>(X₁₃, X₂₃)</td>
<td></td>
</tr>
<tr>
<td>F₁ (X₂₁, X₂₂)</td>
<td>(X₂₂, X₃₁)</td>
<td>(X₂₃, X₃₂)</td>
<td></td>
</tr>
<tr>
<td>DI (X₃₁, X₃₂)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

TABLE 2
Direct-associate arrangement of mixture component means

<table>
<thead>
<tr>
<th>Direct genotypes</th>
<th>CHI</th>
<th>F₁</th>
<th>DI</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHI X₁₁ X₁₂ X₁₃</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F₁ X₂₁ X₂₂ X₂₃</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DI X₃₁ X₃₂ X₃₃</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The F₂, rather than the F₁ generation, was used because of the difficulty of obtaining sufficient quantity of F₁ seed. Plants were grown aseptically in test tubes on nutrient solution solidified with agar. The cultural method was that given by Langridge (1957). Plants were grown for 17 days under continuous light and with constant temperature and humidity conditions in LB growth cabinets of the C.S.I.R.O. (Canberra, Australia) phototron. The experimental observations consisted of entire plant weights. Individual plants were removed from the agar and weighed after the roots had been blotted to remove the excess agar. Then the plant weights were transformed to logarithms prior to statistical analyses.

The experiment involved plants grown at all treatment combinations of two temperatures (25° and 28°) and four nutrient levels. The nutrient levels were obtained as dilutions of a normal strength nutrient solution and designated as (1/8)n, (1/4)n, (1/2)n and n. Two seeds were planted in each test tube to produce a genetic design which yielded all possible combinations of genetic types, as illustrated in Table 1. In this table, parentheses denote the domain of a test tube, and X values represent yields of genotypes within the tube. Two replications of the genetic design were grown at all the eight temperature-nutrient regimes. Each replication consisted of 20 test tubes for every genetic combination. Hence for any environmental regime, there were as many as 80 observations for each pure stand and 40 observations for every component of the mixed stands.

Analytic procedures: A planting design which involved two plants per tube was used in order to permit a genetic analysis to be made in the context of biological group theory (Griffing 1967, 1981a). Groups of size two are the smallest groups which may involve interactions between group members. Therefore groups of size two were used in this study and were generated by growing two plants in the same tube.

In order to facilitate the group theoretic approach, the means of the mixture components are separated and arranged in the form of a direct-associate matrix as given in Table 2. In this representation, for example, X₁₁ = mean yield of the direct genotype, CHI, when grown with the associate phenotype, DI. This arrangement of data provides maximum information with respect to yields of genetic types in the various mixture combinations.

It is now possible to state the objectives of the study clearly and concisely in the direct-associate pattern of data. These objectives are: (i) to characterize a system of genetic parameters that can accurately describe the variability among the means; (ii) to provide a methodology that will determine statistical significance with regard to the different classes of genetic parameters; (iii) to provide a method of estimation for those parameters that yield statistical significance; and (iv) to interpret the above genetic characterization in terms of an appropriate gene model.

The direct-associate pattern of observations is amenable to a factorial ANOVA which provides the basis for characterizing and statistically testing the genetic parameters of interest. The structure of the ANOVA yielding the desired information is that in which the total sum of squares is subdivided into orthogonal partitions each of which is associated with a single degree of freedom as in Table 3. The underlying comparisons of means that form the basis for sums of squares associated with individual degrees of freedom listed in Table 3 constitute the genetic parameters of interest. The ANOVA, itself, provides statistical tests for the significance of these parameters. Estimation of those parameters that yield statistical significance is provided by the comparisons, themselves.

Interpretation of the eight comparisons in terms of gene effects in a group gene model will be given after the experimental data are reviewed. Suffice it to say that "linear" comparisons are functions of additive gene effects, "quadratic" comparisons are functions of nonadditive effects generated by alleles in the same genotype, and the four interaction (DXA) comparisons are functions of nonadditive effects generated by genes in different genotypes within the same group. Direct and associate dimensions of mixture yields are analyzed first. These are followed by analyses of homogeneous and heterogeneous mixture responses. Finally, the highly significant and complex pattern of genetic variability is interpreted in terms of a group gene model.

RESULTS

Analyses of mixture components; a specific example: This section is devoted to analyses of mixture components in terms of direct and associate dimensions of gene activity. The analyses are introduced by a specific numerical example which constitutes the responses to a single environmental regime [28°, (1/2)n]. The purpose of this example is to provide a typical direct-associate table of means, for a single environmental regime, and present figures which graphically illustrate the relative responses of mixture components.
The mixture components for plants grown at the \([28^\circ, (1/2)N]\) regime are recorded in a direct-associate table of means (Table 4). Means for the two replications are included. The means in Table 4 are presented graphically in Figure 1. Figure 1A gives the means in terms of an “associate” representation. The three points on a specific vertical graph are those for mean values of the corresponding column of Table 4. Figure 1B depicts the means in a “direct” representation. In this case the points on a vertical graph correspond to values of the appropriate row of Table 4.

It is clear from Table 4 and Figures 1A and 1B that for this specific environmental regime there are large differences in yield among the different genetic types when grown in various mixture combinations. A closer scrutiny of the data indicates that most of the genetic variation is nonadditive. The ANOVA for the above example is given in Table 6 under the column headed by \((1/2)N\). The ANOVA results indicate that highly significant genetic variation exists in both direct and associate dimensions and that this variation is almost entirely nonadditive.

Analyses of mixture components; all treatments:

This section deals with the analyses of mixture components for all regimes. Tables 5 and 6 present the direct-associate ANOVAs for each of the eight temperature-nutrient combinations. These ANOVAs are approximate in the sense that unequal numbers, and the possibility of correlation among individual error terms associated with the two plants within tubes, may exist. However, these problems are minimized when the ANOVAs are calculated on means involving substantial numbers of observations, and the tests involve the \(RX\) interaction mean square as denominators. This assertion was tested by recalculating all ANOVAs from an abridged set of data in which only one plant per tube was used. The patterns of statistical significance for the two kinds of ANOVAs were almost identical. Hence the total data-set was used for all analyses.

The entire set of ANOVAs produce three strikingly consistent patterns of results: (i) The direct \((D)\) and associate \((A)\) sources of variation are uniformly highly significant over all environmental regimes. Therefore, further partitioning of direct and associate sums of squares is warranted. Such partitioning yields the second consistent pattern of results; (ii) For both direct and associate sources of variation all linear mean squares are essentially nonsignificant, but all quadratic mean squares are highly significant. Finally, (iii) the analyses are consistent in demonstrating a complete lack of \(D\times A\) interaction effects. Therefore, further partitioning of the \(D\times A\) sums of squares is not warranted.

The analyses suggest that clarification of the complex nature of the genetic variation exhibited in this study depends entirely on the direct and associate quadratic comparisons of genetic variation. These quadratic comparisons measure the extent of the deviation of the hybrid mean from that of its midparental value. They are defined as follows:

Direct \((D)\) nonadditive measures:

\[
Q(D) = \sum_{i} X_{i1} - \sum_{i} X_{i3}.
\]

Associate \((A)\) nonadditive measures:

\[
Q(A) = \sum_{i} X_{i1} - \sum_{i} X_{i3}.
\]

In the following presentation, the estimation and subsequent analyses of these quadratic values are discussed with those for the \(Q(D)\) values presented first. The analysis of variance for the direct nonadditive measure involves separate \(Q\) values calculated for each
genetic type within each temperature-nutrient regime. This ANOVA is presented in Table 7. It demonstrates that both temperature and nutrient environmental factors generate highly significant mean squares. Also, the $T \times N$ interaction mean square is significant at the $P < 0.02$ level. The mean values for $Q(D)$ when averaged over the three genetic types are plotted for the eight regimes in Figure 2A. It is clear from this figure that $Q(D)$ values for $28^\text{o}$ are consistently greater than the corresponding values for $25^\text{o}$. Thus the $Q(D)$ parameter displays a temperature-dependent form of genetic nonadditivity. Validity of this temperature-dependent response is supported by the highly significant temperature mean-square. Also the

![Figure 1](image_url)

**TABLE 5**

Mean squares of the direct-associate ANOVAs for the four nutrient regimes at $25^\text{o}$

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>$(1/8)N$</th>
<th>$(1/4)N$</th>
<th>$(1/2)N$</th>
<th>$N$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Replications: (R)</td>
<td>1</td>
<td>0.00075</td>
<td>NS</td>
<td>0.00520</td>
<td>0.00627*</td>
</tr>
<tr>
<td>Genotypes: (G)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Direct: (D)</td>
<td>2</td>
<td>0.00748**</td>
<td>0.02994**</td>
<td>0.05248***</td>
<td>0.6745***</td>
</tr>
<tr>
<td>Linear: $l(D)$</td>
<td>1</td>
<td>0.00020 NS</td>
<td>0.00018 NS</td>
<td>0.00018 NS</td>
<td>0.00031 NS</td>
</tr>
<tr>
<td>Quadratic: $q(D)$</td>
<td>1</td>
<td>0.01476***</td>
<td>0.05070***</td>
<td>0.10315***</td>
<td>0.13458***</td>
</tr>
<tr>
<td>Associate: (A)</td>
<td>2</td>
<td>0.00253**</td>
<td>0.00920**</td>
<td>0.00845***</td>
<td>0.01106***</td>
</tr>
<tr>
<td>Linear: $l(A)$</td>
<td>1</td>
<td>0.00009 NS</td>
<td>0.00053 NS</td>
<td>0.00003 NS</td>
<td>0 NS</td>
</tr>
<tr>
<td>Quadratic: $q(A)$</td>
<td>1</td>
<td>0.00497***</td>
<td>0.01787***</td>
<td>0.01686***</td>
<td>0.02210***</td>
</tr>
<tr>
<td>$D \times A$</td>
<td>4</td>
<td>0.00011 NS</td>
<td>0.00015 NS</td>
<td>0.00058*</td>
<td>0.00006 NS</td>
</tr>
<tr>
<td>$R \times G$</td>
<td>8</td>
<td>0.00027</td>
<td>0.00018</td>
<td>0.00011</td>
<td>0.00067</td>
</tr>
</tbody>
</table>

*NS = nonsignificant; * $= 0.01 < P < 0.05$; ** $= 0.001 < P < 0.01$; *** $= P < 0.001$.

**TABLE 6**

Mean squares of the direct-associate ANOVAs for the four nutrient regimes at $28^\text{o}$

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>$(1/8)N$</th>
<th>$(1/4)N$</th>
<th>$(1/2)N$</th>
<th>$N$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Replications: (R)</td>
<td>1</td>
<td>0.00269 NS</td>
<td>0.00756**</td>
<td>0.00646***</td>
<td>0.00598*</td>
</tr>
<tr>
<td>Genotypes: (G)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Direct: (D)</td>
<td>2</td>
<td>0.08735***</td>
<td>0.11772***</td>
<td>0.13642***</td>
<td>0.10262***</td>
</tr>
<tr>
<td>Linear: $l(D)$</td>
<td>1</td>
<td>0.00122 NS</td>
<td>0.00183 NS</td>
<td>0 NS</td>
<td>0.00002 NS</td>
</tr>
<tr>
<td>Quadratic: $q(D)$</td>
<td>1</td>
<td>0.17347***</td>
<td>0.23661***</td>
<td>0.27283***</td>
<td>0.20521***</td>
</tr>
<tr>
<td>Associate: (A)</td>
<td>2</td>
<td>0.02471***</td>
<td>0.01894***</td>
<td>0.01994***</td>
<td>0.01268***</td>
</tr>
<tr>
<td>Linear: $l(A)$</td>
<td>1</td>
<td>0.00015 NS</td>
<td>0.00036 NS</td>
<td>0.00011 NS</td>
<td>0.00059 NS</td>
</tr>
<tr>
<td>Quadratic: $q(A)$</td>
<td>1</td>
<td>0.04928***</td>
<td>0.03751***</td>
<td>0.03987***</td>
<td>0.02496***</td>
</tr>
<tr>
<td>$D \times A$</td>
<td>4</td>
<td>0.00060 NS</td>
<td>0.00108 NS</td>
<td>0.00015 NS</td>
<td>0.00108 NS</td>
</tr>
<tr>
<td>$R \times G$</td>
<td>8</td>
<td>0.00068</td>
<td>0.00064</td>
<td>0.00028</td>
<td>0.00071</td>
</tr>
</tbody>
</table>

*NS = nonsignificant; * $= 0.01 < P < 0.05$; ** $= 0.001 < P < 0.01$; *** $= P < 0.001$. 
TABLE 7

ANOVAs for the nonadditive measures Q(D) and Q(A)

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>Q(D) mean squares</th>
<th>Q(A) mean squares</th>
</tr>
</thead>
<tbody>
<tr>
<td>Genetic type (G)</td>
<td>2</td>
<td>0.0022 ns</td>
<td>0.0023 ns</td>
</tr>
<tr>
<td>Temperature (T)</td>
<td>1</td>
<td>0.2528***</td>
<td>0.0320**</td>
</tr>
<tr>
<td>Nutrient (N)</td>
<td>3</td>
<td>0.0290**</td>
<td>0.0005 ns</td>
</tr>
<tr>
<td>G × T</td>
<td>2</td>
<td>0.0002 ns</td>
<td>0.0002 ns</td>
</tr>
<tr>
<td>G × N</td>
<td>6</td>
<td>0.0011 ns</td>
<td>0.0019 ns</td>
</tr>
<tr>
<td>T × N</td>
<td>3</td>
<td>0.0118*</td>
<td>0.0052 ns</td>
</tr>
<tr>
<td>G × T × N</td>
<td>6</td>
<td>0.0015</td>
<td>0.0021</td>
</tr>
</tbody>
</table>

NS = nonsignificant; * = 0.01 < P < 0.05; ** = 0.001 < P < 0.01; *** = P < 0.001.

Q(D) values tend to increase with increased nutrients, and the mean square for nutrient levels is highly significant. Therefore the Q(D) data also illustrate a nutrient-dependent form of genetic nonadditivity. The significant T×N interaction mean square indicates that temperature responses are not entirely consistent at various nutrient levels. From Figure 2A, it is apparent that the discrepant value appears to be at the highest nutrient level, N.

The analysis of variance for the associate measure of nonadditivity, Q(A), is presented in Table 7. The only significant mean square is that due to temperature. Thus the associate form of genetic nonadditivity is temperature-dependent. Figure 2B presents the average responses of Q(A) for each of the temperature-nutrient regimes. This figure illustrates that all Q(A) values are negative, and that the values at 28°C are more extreme than those at 25°C.

It is clear from Figure 2, A and B, that the Q(D) and Q(A) values differ in qualitative and quantitative ways: Q(D) is positive and Q(A) is negative; Q(D) is of greater magnitude than Q(A). However, there is one important characteristic that they have in common. In the ANOVAs for both Q(D) and Q(A), the mean squares for “genetic type” are nonsignificant (see Table 7). This implies that Q values estimated for each genetic type (CHI, F2, and DI), within each temperature-nutrient regime, are essentially equal. Hence, apparently, the genetic type does not influence the Q(D) (or Q(A)) value with which it is associated.

Analyses of total mixture responses: In previous sections, the individual mixture components were analyzed. In this section the components are combined to yield total mixture responses as the basis for analysis.

To introduce the notion of total mixture response, consider the example given in Table 4. The mixture component means of Table 4 are combined to give the appropriate total mixture means as listed in Table 8. There are two classes of mixtures represented in Table 8: (i) homogeneous (pure-stand) mixtures; these include CHI, F2, and DI pure stands, the means of which are: CHI = 1.361, F2 = 1.510 and DI = 1.360, and (ii) heterogeneous mixtures; these include the (CHI, F2), (CHI, DI), and (F2, DI) combinations. Means for heterogeneous mixtures are (CHI, F2) = 1.439, (CHI, DI) = 1.547 and (F2, DI) = 1.438.

In the following analyses, which include all eight environmental regimes, the homogeneous and heterogeneous mixtures are presented separately. Homogeneous mixtures are those in which the two plants in a test tube are of the same genetic type. Designation of the three types is shortened to CHI, F2 and DI pure stands. Mean yields of the pure stands for each nutrient level at 25°C are given graphically in Figure 3A, and at 28°C in Figure 3B. These figures indicate that a very considerable amount of genetic nonadditivity is expressed among the homogeneous mixtures. A more critical analysis of the nonadditive responses is made by use of the Q measure which is calculated as follows:

\[ Q(PS) = 2(F2,F2) - [(CHI, CHI) + (DI, DI)]. \]

A factorial ANOVA for the Q(PS) values is given in Table 9. It is clear that the mean squares for temperatures and nutrient-levels are significant. Therefore, nonadditivity measured among pure stands is both temperature- and nutrient-dependent. The increased Q values at higher temperatures, and also at higher nutrient levels, can be observed in Figure 3C, in which Q(PS) values are plotted for each temperature-nutrient regime.

Heterogeneous mixtures are defined as those in which the two mixture components are derived from different genetic types. The experimental results indicate that heterogeneous mixture responses are fundamentally different from those of homogeneous mixtures. Homogeneous mixtures exhibit a large amount of genetic nonadditivity; heterogeneous mixtures exhibit complete additivity. As an illustration, consider yields of (CHI, F2) and its component pure stands for all nutrient levels at 28°C, which are plotted in Figure 4A. The (CHI, F2) mixture yields are strictly intermediate. Similar responses occur for all heterogeneous mixtures in the various environmental regimes. A Q measure can be used again to analyze, more critically, the extent of nonadditivity of mixture responses. For example, the Q measure for (CHI, F2) is defined as follows:

\[ Q(M) = 2(CHI, F2) - [(CHI, CHI) + (F2, F2)]. \]

A factorial ANOVA for heterogeneous Q values is given in Table 9. The nonsignificant mean square for mixtures in the ANOVA indicates that all heterogeneous mixtures exhibit a strictly additive relationship with respect to the pure stands of their component genetic types. Furthermore, heterogeneous mixture responses remain additive at all temperature-nutrient regimes; as indicated by nonsignificant temperature and nutrient mean squares. In fact, the remarkable aspect of the ANOVA for heterogeneous mixtures in Table 9 is that there are no significant mean squares.
Experimental results requiring a genetic interpretation: With regard to the component mixture analyses, the facts are that the \(Q(D)\) values are positive and statistically significant, and the \(Q(A)\) values are negative and statistically significant. However, the curious aspect of the \(Q\) values (for both \(Q(D)\) and \(Q(A)\)) is that they are essentially equal when calculated with regard to the three different genetic types within each of the eight environmental regimes. This assertion is corroborated by the fact that the "genetic type" mean squares are not significant in the ANOVAs for \(Q(D)\) and \(Q(A)\). Hence the question that an explanatory gene model must answer is: In view of the highly significant variation among \(Q(D)\) and \(Q(A)\) values over the entire experiment, why is it that, within a single environmental regime, \(Q\) values calculated independently with respect to the three genetic types are essentially equal?

With regard to the total mixture analyses, the facts are that the homogeneous mixtures exhibit highly significant, positive genetic nonadditivity as measured by \(Q(PS)\), whereas the heterogeneous mixtures exhibit almost complete genetic additivity as indicated by the \(Q(M)\) values which are essentially equal to zero. These results hold true for all eight temperature-nutrient regimes. Hence it is clear that in constructing an explanatory genetic model, it must be able to accommodate both homogeneous and heterogeneous mixture results which, at first sight, appear to be inconsistent.

Finally once a gene model has been constructed that satisfactorily explains the component and total mixture results, then it will be put to a further test involving the prediction of \(Q(PS)\), which is a total mixture parameter, by use of \(Q(D)\) and \(Q(A)\), which are component mixture parameters. This final test will be discussed in more detail later.

Construction of the group representation: In this section, a group genetic model is developed in order to answer the questions raised above. This model involves groups of interacting genotypes generated at a single locus. Extension to more than one locus is...
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The genetic design of this study incorporates two homozygous races and their F₂. With regard to a single locus, the design dictates use of a model involving two equally frequent alleles. Hence the genotypic array for the conceptual base population is:

\[(1/4)(A₁A₁ + A₁A₂ + A₂A₁ + A₂A₂)\].

Since the genetic design also utilizes groups of size two, it is necessary to generate a population of random groups of size two from the base population. This can be accomplished by "squaring" the base array as follows:

\[
[(1/4)(A₁A₁ + A₁A₂ + A₂A₁ + A₂A₂)]^2.
\]

The direct-associate matrix of genotypic values for such a population of groups is illustrated in Table 10, where, for example; \(\hat{G}_{M} = \) genotypic value for \(A₄A₄\) when associated with \(A₄A₄\) in groups of size two.

**Relationships between experimental and group representations:** In order to interpret the experimental results in terms of group genetic parameters, it is necessary to establish relationships between experimental and group genotypic values. In working out
The next step is to evaluate the various measures of genetic nonadditivity in terms of group genotypic values. The expected genetic nonadditivity as expressed with pure stands is:

\[ E(Q(PS)) = E[2X_{22} - (X_{11} + X_{33})] \]

Expectations for the three values of \(i\) are:

\[
\begin{align*}
E(Q(A_1)) &= 2(G_{11}) - (G_{11} + G_{22}), \\
E(Q(A_2)) &= 2(...G) - (...G_{11} + ...G_{22}), \text{ and} \\
E(Q(A_3)) &= 2(G_{22}) - (G_{11} + G_{22}).
\end{align*}
\]

The genetic nonadditivity as expressed with pure stands is:

\[
E(Q(PS)) = E[2X_{22} - (X_{11} + X_{33})] = [2(...G) - (G_{11} + G_{22})].
\]

Q values for the three heterogeneous mixtures are evaluated as follows:

(i) (CHI, DI) mixture:

\[ E(Q(M)) = E[(X_{13} + X_{31}) - (X_{11} + X_{33})] = (G_{22} + 2G_{11}) - (G_{11} + G_{22}) \]

(ii) (CHI, F2) mixture:

\[ E(Q(M)) = E[(X_{12} + X_{21}) - (X_{11} + X_{22})] = (G_{22} + ...G_{11}) - (G_{11} + ...G) \]

(iii) (DI, F2) mixture:

\[ E(Q(M)) = E[(X_{32} + X_{23}) - (X_{22} + X_{33})] = 2G_{22} + ...G - (2G_{22} + ...G) \].

**Construction of the group gene model:** In order to interpret the \(Q\) values (listed above) in terms of genetic effects, it is necessary to construct a genetic model to be associated with the group genotypic values \((g_i a_k)\). These values can be characterized by genetic models at two levels (i.e., at genotypic and gene levels).

Genotypic model:

\[ g_{ik} = \mu + d_{ij} + a_{jk} + (DA)_{ijk}, \]

where

\[ \mu = G_\text{mean}, \]

<table>
<thead>
<tr>
<th>Direct genotypes</th>
<th>(A_1A_1)</th>
<th>(A_1A_2)</th>
<th>(A_2A_1)</th>
<th>(A_2A_2)</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>(A_1A_2)</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>(A_1A_2)</td>
<td>1</td>
<td>1</td>
<td>1</td>
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</tr>
<tr>
<td>(A_1A_2)</td>
<td>1</td>
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<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>(A_1A_2)</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Mean</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
</tbody>
</table>

These relationships it must be remembered that the \(F_2\) rather than the \(F_1\) generation is used. The relationships are as follows (\(E\) symbolizes “expectation” in the statistical sense).

\[
\begin{align*}
E(X_{11}) &= G_{11} \\
E(X_{12}) &= (1/4)(G_{11} + G_{12} + G_{21} + G_{22}) = G_{11} \\
E(X_{13}) &= G_{12} \\
E(X_{21}) &= (1/4)(G_{11} + G_{12} + G_{21} + G_{22}) = G_{11} \\
E(X_{22}) &= G_{12} \\
E(X_{31}) &= G_{11} \\
E(X_{32}) &= (1/4)(G_{11} + G_{12} + G_{21} + G_{22}) = G_{22} \\
E(X_{33}) &= G_{22}.
\end{align*}
\]

The next step is to evaluate the expected values of group genotypic values. The expected \(Q(D_i)\) value when calculated with respect to the \(k\)th associate background is:

\[ E(Q(D_i)) = E[2X_{22} - (X_{11} + X_{33})]. \]

Expectations for the three values of \(k\) are:

\[
\begin{align*}
E(Q(D)) &= 2(G_{11}) - (G_{11} + G_{22}), \\
E(Q(D2)) &= 2(...G) - (G_{11} + ...G_{22}), \text{ and} \\
E(Q(D3)) &= 2(G_{22}) - (G_{11} + G_{22}).
\end{align*}
\]
$D_{ij} = \eta G_{ij} - \mu = \text{direct effect of genotype } A_i A_j,$

$A_{il} = \eta G_{il} - \mu = \text{associate effect of genotype } A_i A_l$, and

$(DA)_{ijkl} = \eta G_{ijkl} - \mu - D_{ij} - A_{il} = \text{interaction effect between direct genotype } A_i A_j$ and associate genotype $A_i A_l$.

**Gene model:** The genotypic model can be partitioned further into a gene model as follows:

$$
\eta G_{ijkl} = \mu + d_i + d_j + (dd)_{ij} + a_i + a_j + (aa)_{ij} + (da)_{ij} + (dd)_{ij} + (daa)_{ijkl} + (ddaa)_{ijkl}
$$

where,

$$
d_i = \eta G_{i} - \mu = \text{direct additive effect of } A_i,
$$

$$
(dd)_{ij} = \eta G_{ij} - \mu - d_i - d_j = \text{direct dominance effect of } A_i A_j,
$$

$$
a_i = \eta G_{i} - \mu = \text{associate additive effect of } A_i,
$$

$$
(aa)_{ij} = \eta G_{ij} - \mu - a_i - a_j = \text{associate dominance effect of } A_i A_j.
$$

All remaining effects in the gene model are interaction effects between direct and associate genes and/or genotypes.

With two equally frequent alleles, the restrictions on direct and associate main effects are simply:

$$
d_i + d_j = 0, \Sigma_d(dd)_{ij} = 0, \text{ for all } i, \text{ and } \Sigma_i(dd)_{ij} = 0, \text{ for all } j.
$$

$$
a_i + a_j = 0, \Sigma(aa)_{ij} = 0, \text{ for all } i; \text{ and } \Sigma_i(aa)_{ij} = 0, \text{ for all } j.
$$

Elements in the two models can be related as follows:

$$
D_{ij} = d_i + d_j + (dd)_{ij},
$$

$$
A_{il} = a_i + a_l + (aa)_{il}.
$$

$$(DA)_{ijkl} = (da)_{ija} + \cdots + (ddaa)_{ijkl} = \text{sum of all interaction effects.}
$$

For a further elaboration of the group model see Griffing (1981a).

**Evaluation of Q values and interpretation of experimental results:** Before evaluating the Q values, it is useful to simplify the group gene model, (3), in accordance with the experimental results. The basic information required for judging which elements should be included in the model is given in the ANOVAs listed in Tables 5 and 6. The patterns of statistical significance for mean squares, MS(D), MS(A), and MS(DA), over all eight regimes, are as follows:

1. Direct mean squares, MS(D), are consistently highly significant. Therefore,

$$
D_{ij} = d_i + d_j + (dd)_{ij},
$$

should be included in the model.

2. Associate mean squares, MS(A), are consistently highly significant. Therefore,

$$
A_{il} = a_i + a_l + (aa)_{il},
$$

should be included.

3. However, direct × associate mean squares, MS(DA), are nonsignificant in essentially all treatment regimes. This implies that the interaction effects,

$$
(DA)_{ijkl} = (da)_{ija} + \cdots + (ddaa)_{ijkl},
$$

can be ignored.

Based on these statistical results, the group genotypic model simplifies to,

$$
iG_{ijkl} = \mu + D_{ij} + A_{il},
$$

and the gene model becomes.

$$
iG_{ijkl} = \mu + d_i + d_j + (dd)_{ij} + a_i + a_j + (aa)_{ij} + (da)_{ij} + (dd)_{ij} + (daa)_{ijkl} + (ddaa)_{ijkl}.
$$

This model could be simplified even further by considering the linear and quadratic components of MS(D) and MS(A). However, models (4) and (5) are used in order to provide a more generalized result, which demonstrates independence of additive and nonadditive (dominance) effects in the Q measures.

It is now possible to evaluate the Q values in terms of the simplified genetic models as follows:

The expected Q(D) parameters, in terms of the group genotypic values, are given in (1). For all values of k, these become,

$$
E[Q(D)] = -(D_{i1} + D_{i2})
$$

$$
= -2d_i + (dd)_{i1} + 2d_j + (dd)_{i2},
$$

When the restrictions on gene effects are applied, (6) becomes,

$$
E[Q(D)] = 2(dd)_{i1}, \text{ for } k = 1, 2, 3.
$$

Thus, although the three functions in (1) are different for the three associate backgrounds (i.e., k = 1, 2, 3), they all yield the same result, (7), with the simplified gene model (5). This demonstrates that Q(D) values calculated for each associate background within each of the eight environmental regimes should be approximately equal. Thus one of the puzzling experimental results is solved. The second important point with regard to (7) is that Q(D) values are functions of only direct dominance effects. Perhaps this becomes more clear if the results of (7) are transformed into a different diallelic model in which the direct genotypic values are: $A_1A_1$, $A_1A_2$, and $A_2A_2$, $-d$. In this model the genotypic value, $h_{ij}$, is a measure of the deviation of the direct heterozygote from its midparental value. With gene frequencies equal to one-half, $2(d)_{i1} = h_{ij}$, and (7) becomes simply, $E[Q(D)] = h_{ij}$. Finally, Q(D) values can change from one temperature-nutrient regime to another. Such changes are a reflection of the phenomena of temperature- and/or...
nutrient-dependent heterosis. Both forms of heterosis were found to be highly significant in Table 7.

The expected \(Q(A)\) parameters, in terms of the group genotypic values, are given in (2). In terms of the gene model, these all become,

\[
E[Q(A)] = (A_{11} + A_{22})
\]

\[
= 2(aa)_{i2}
\]

\[
= \hat{h}_i, i = 1, 2, 3.
\]

As with \(Q(D)\) values it is clear that: (i) The result, (8), holds for any of the three direct genetic types; this fact is responsible for the nonsignificant mean square attributable to "genetic type" in the ANOVA for \(Q(A)\) in Table 7, and thus, the second puzzling experimental result is explained. (ii) The result, (8), demonstrates that genetic nonadditivity measured by \(Q(A)\) is strictly a measure of heterosis in the associate dimension of gene activity. (iii) \(Q(A)\) values can, and do, change when measured in different temperature-nutrient regimes. The highly significant mean square in Table 7 indicates that this form of heterosis is temperature-dependent.

The third puzzling aspect of the experimental data was the fact that there was considerable manifestation of genetic nonadditivity in mixture components, the \(Q(M)\) values for heterogeneous mixtures were all essentially equal to zero. Thus heterogeneous mixtures exhibited complete additivity. To explain this phenomenon it is necessary to evaluate the \(Q(M)\)s in terms of the group genetic models [(4) and (5)]:

(i) (CHI, DI) heterogeneous mixture.

\[
[Q(CHI, DI)] = (1, G_{22} + 2G_{11}) - (1, G_{11} + 2G_{22})
\]

\[
= [(\mu + D_{11} + A_{22}) + (\mu + D_{22} + A_{11})]
\]

\[
= -[(\mu + D_{11} + A_{11}) + (\mu + D_{22} + A_{22})]
\]

\[
= 0.
\]

(ii) (CHI, F2) heterogeneous mixture.

\[
E[Q(CHI, F2)] = (1, G_{..} + G_{11}) - (1, G_{11} + G_{..})
\]

\[
= [(\mu + D_{11}) + (\mu + A_{11})]
\]

\[
= 0.
\]

(iii) (DI, F2) heterogeneous mixture.

\[
E[Q(DI, F2)] = (G_{..} + G_{22}) - (G_{22} + G_{..})
\]

\[
= [(\mu + D_{22}) + (\mu + A_{22})]
\]

\[
= 0.
\]

These results indicate that the group genetic model can accommodate any degree of genetic nonadditivity in the direct and associate dimensions of gene expression, while at the same time exhibit complete additivity, with regard to the interaction between genotypes within groups. Hence the model that has been constructed completely describes the experimental results obtained with regard to the \(Q(D), Q(A)\) and \(Q(M)\) parameters.

**Prediction of the \(Q(PS)\) parameter:** Finally, it is possible to put the genetic model to an independent test. This test involves the \(Q\) parameter for homogeneous mixtures, \(Q(PS)\), which is evaluated in terms of the gene model as follows:

\[
E[Q(PS)] = 2(.G.) - (1, G_{11} + 2G_{22})
\]

\[
= [2(\mu)] - [(\mu + D_{11} + A_{11}) + (\mu + D_{22} + A_{22})]
\]

\[
= h_0 + h_1.
\]

If the group theory is correct, and the group gene model is useful in describing genetic nonadditivity in the experimental system, it should be possible to predict \(Q(PS)\) by the following relationship of \(Q\) values:

\[
Q(PS) = Q(D) + Q(A).
\]

Validity of this prediction can be observed in Figure 5A which presents average \(Q\) values for each nutrient level at 25°, and Figure 5B which gives \(Q\) values for each nutrient level at 28°. These figures illustrate the remarkable coincidence of \(Q(PS)\) and its predictor, \([Q(D) + Q(A)]\), over the entire range of temperature-nutrient regimes. Hence the predictive nature of the group genetic model is demonstrated.

**DISCUSSION**

**Restatement of problem and method of attack:**
The basic objective of this study is to provide a genetic analysis of responses to interacting genotypes in biological groups which were subjected to a range of environmental conditions. The experimental procedure involved growing all possible pairwise combinations of plants, from two different races and their hybrid, in test tubes which were subjected to exactly controlled environmental conditions.

Genetic analyses of such mixtures are complicated by the fact that plants within groups may interact, i.e., they may influence each others' growth responses, especially when the plants are competing for limited environmental resources. This implies that a particular genetic type may yield quite differently depending on the associate genetic type with which it is grown. Such a complication creates a difficult problem of interpretation in terms of the classical (noninteraction) quantitative genetic theory.

An outline of the method of attack used in this study is as follows: (i) Recognize that a group gene model [as set out in (3)] has been specifically designed to accommodate interactions between individuals within groups. This model is suitable for the genetic design in this study. (ii) Recognize that a one-to-one correspondence exists between classes of gene model effects and the eight comparisons responsible for an orthogonal partitioning of the total variation in a \(2^3\) factorial ANOVA of the direct-associate pattern of means. Such a correspondence is illustrated in Table
Genetic Analysis of Plant Mixtures

TABLE 11

<table>
<thead>
<tr>
<th>Gene model effects</th>
<th>ANOVA comparisons</th>
</tr>
</thead>
<tbody>
<tr>
<td>$G_0$</td>
<td>$l(D)$ (direct linear)</td>
</tr>
<tr>
<td>$\mu$</td>
<td>$l(A)$ (associate linear)</td>
</tr>
<tr>
<td>$+(d_d)$</td>
<td>$q(D)$ (direct quadratic)</td>
</tr>
<tr>
<td>$+(a_d)$</td>
<td>$q(A)$ (associate quadratic)</td>
</tr>
<tr>
<td>$+(a_d)$</td>
<td>$l(D) \times l(A)$ (linear $\times$ linear)</td>
</tr>
<tr>
<td>$+(d_d)$</td>
<td>$l(D) \times q(A)$ (linear $\times$ quadratic)</td>
</tr>
<tr>
<td>$+(d_d)$</td>
<td>$q(D) \times l(A)$ (quadratic $\times$ linear)</td>
</tr>
<tr>
<td>$+(d_d)$</td>
<td>$q(D) \times q(A)$ (quadratic $\times$ quadratic)</td>
</tr>
</tbody>
</table>

This ANOVA also provides the appropriate method for statistically testing the contribution of each class of gene effects with respect to genetic variation in the direct-associate pattern of data. It is clear then, that in the above method of attack, the key is the recognition of the one-to-one relationship between ANOVA comparisons and classes of gene effects. Hence the operational procedure is to perform the direct-associate ANOVA and determine which of the eight orthogonal comparisons are significant. Then the statistically significant comparisons, themselves, are used to characterize the contributions of the specific classes of gene effects to the genetic variation in the experimental material.

The above argument holds perfectly for the situation in which the experimental procedure utilizes the $F_1$ generation resulting from crossing two homozygous parents. However when the $F_2$ generation is used, as in this study, certain modifications need to be made in the analysis. Earlier, it was demonstrated that in the absence of direct $\times$ associate interaction effects, quadratic comparisons yielded unbiased estimates of dominance parameters. However, when direct $\times$ associate interaction effects cannot be ignored, the procedure must be slightly modified, when $F_2$ data are used. In this case it is necessary, first, to identify the different possible linear and quadratic comparisons as follows:

**Linear comparisons:**

$L(A_1) = X_{11} - X_{13}$

$L(D_1) = X_{11} - X_{31}$

$L(A_2) = X_{21} - X_{23}$

$L(D_2) = X_{12} - X_{52}$

$L(A_3) = X_{31} - X_{33}$

$L(D_3) = X_{13} - X_{33}$.

**Quadratic comparisons:**

$Q(A_1) = 2X_{12} - (X_{11} + X_{13})$

$Q(D_1) = 2X_{21} - (X_{11} + X_{31})$

$Q(A_2) = 2X_{23} - (X_{21} + X_{25})$

$Q(D_2) = 2X_{22} - (X_{12} + X_{32})$

$Q(A_3) = 2X_{32} - (X_{31} + X_{33})$

$Q(D_3) = 2X_{23} - (X_{13} + X_{33})$. 

**Figure 5.**—Comparison of $Q(PS)$ with the predicted value given by $[Q(D) + Q(A)]$ for each nutrient level: (A) at $25^\circ$, and (B) at $28^\circ$. 

**Table 11**

Correspondence of classes of gene model effects and ANOVA comparisons
Using these linear and quadratic contrasts of experimental means, the eight orthogonal comparisons can be determined which, in turn, provide unbiased estimates of the corresponding class of gene effects. These may be summarized as follows:

**Main effects:**

1. Direct additive, \( (d) \):
\[
I(D) = k_1L(D_2)
\]
2. Associative additive, \( (a) \):
\[
I(A) = k_1L(A_2)
\]
3. Direct dominance, \( [(dd)] \):
\[
q(D) = k_2Q(D_2)
\]
4. Associate dominance, \( [(aa)] \):
\[
q(A) = k_2Q(A_2)
\]

**Interaction effects:**

5. Additive \( \times \) additive, \( [(aa)] \):
\[
I(D) \times I(A) = k_4[L(D_1) - L(D_3)] = k_6[L(A_1) - L(A_3)]
\]
6. Additive \( \times \) Quadratic, \( [(dd)] \):
\[
I(D) \times q(A) = k_4[2L(D_2) - [L(D_1) + L(D_3)]]
\]
\[
= k_6[Q(A_1) - Q(A_3)]
\]
7. Quadratic \( \times \) Additive, \( [(da)] \):
\[
q(D) \times I(A) = k_5[Q(D_1) - Q(D_3)]
\]
\[
= k_6[2L(A_2) - [(L_1) + L(A_3)]
\]
8. Quadratic \( \times \) quadratic, \( [(daa)] \):
\[
q(D) \times q(A) = k_6[2Q(D_2) - [Q(D_1) + Q(D_3)]]
\]
\[
= k_6[2Q(A_2) - [Q(A_1) + Q(A_3)]]
\]

In the above representation, the \( k \)'s are appropriate constants.

Further points of interest with regard to the complete model and use of \( F_2 \) data are:

1. The quadratic measure for homogeneous mixtures can be predicted as:
\[
Q(PS) = Q(D_3) + Q(A_3) + Q(CHI, F_2) + Q(DI, F_2).
\]
2. The quadratic measures for heterogeneous mixtures, \( Q(CHI, F_2), Q(DI, F_2) \) and \( Q(CHI, DI) \), are functions of only direct \( \times \) associate interaction effects. This implies that a greater average yield than the expected interactions between genes in different genotypes within groups.

Finally, the theory developed in the above analyses is based on genotypes segregating at a single locus. When several independent segregating loci contribute to the total genetic variability, the orthogonal comparison for a particular class of gene effects becomes the sum of those effects from each locus.

**Review of experimental results.** With regard to the experimental results of this study, the ANOVAs yielded consistent results over all environmental regimes. These results are summarized briefly as follows:

1. Direct \( (D) \) mean squares were highly significant over all regimes. This implied that genotypes of the three genetic types differed significantly in their direct contributions to mixture yields. Further partitioning of the direct variation into linear and quadratic components, consistently, demonstrated that essentially all of the direct variation was nonadditive.

2. Associate \( (A) \) mean squares were highly significant. This implied that individuals within groups interfered with each other's growth responses, i.e. the three genetic types of associate members differentially influenced yields of the direct group members. Further partitioning of the associate variation, again, indicated that most variation over all regimes was nonadditive.

3. Direct \( \times \) associate \( (D \times A) \) mean squares were consistently nonsignificant. Thus, although yields of all genetic types were differentially influenced by associate genotypes, a direct \( \times \) associate, nonadditive interaction did not exist. The conclusion from these basic analyses was that the only effects in the gene model that significantly contributed to the genetic variation of the experimental results were the direct and associate heterosis effects.

Having identified the classes of gene effects that caused genetic variation in the experimental material, the final step was to characterize these effects in qualitative and quantitative terms. This was accomplished by use of the appropriate quadratic contrast, \( Q(D) \), which measured the total heterosis in the direct dimension, and \( Q(A) \), which did the same in the associate dimension. The two kinds of heterosis differed qualitatively (direct heterosis was positive whereas associate heterosis was negative) and quantitatively (the magnitude of direct heterosis was greater than that of associate heterosis).

**Biological interpretation of the experimental results:** The previous sections summarized the analytical method of attack and the basic experimental results. A biological interpretation of these results is now considered. The positive, significant \( Q(D) \) values indicate that the \( F_2 \) exhibits greater yield than either parental race when grown with any of the three associate genotypes. This is true for all temperature-nutrient regimes. Hence the \( F_2 \) exhibits a higher growth rate than those of the parental races over the given time period for each and every environmental regime. The negative, significant \( Q(A) \) values can be explained in terms of the higher \( F_2 \) growth rate and the fact that nutrients available to the plants in a test tube are limited. When the \( F_2 \) competes with a parental race for the limited supply of nutrients, the \( F_2 \) utilizes more than its share of nutrients and thereby forces the parental race to grow on a reduced nutrient supply. In all cases, this results in an increased \( F_2 \) yield at the expense of that of the
Additional studies were devoted to this effort. The above explanation for positive $Q(D)$ and negative $Q(A)$ values implies that a negative relationship between these two classes of effects should exist. The highly significant correlation coefficient for $Q(D)$ and $Q(A)$ values, averaged over all genetic types for each temperature-nutrient regime, is: $r = -0.88$. Thus there does exist a close, negative relationship. The regression equation expressing this relationship is:

$$Q(A) = -0.041 - 0.32Q(D).$$

Hence the magnitude of the predicted $Q(A)$ values are approximately one-third that of the $Q(D)$ values.

The temperature-dependent nature of the direct and associate expression of heterosis is of special interest. Comparison of Figures 3A and 3B indicate that yields of parental races drop considerably more than yields of the F$_2$, when the temperature is raised from 25° to 28°. This differential response can be examined more critically by recording differences in yield between 25° and 28° for each genetic type at each nutrient level. These data give the average F$_2$ drop as 0.047 and the average decrease for the two races as 0.115. Hence the parental decrease is approximately 2.5 times as great as that for the F$_2$. Furthermore, the difference is, statistically, highly significant. This temperature induced differential decrease in yield, when a hybrid is compared with its parents, suggests that each parent has a set of temperature-sensitive genes which are somewhat different in the two races. In the hybrid, then, some of the temperature-sensitive alleles of one parent are compensated for by non-temperature-sensitive alleles of the other parent. A temperature-dependent heterosis results.

This phenomenon has been reported in Arabidopsis before (LanRIdge and Griffith 1959; Griffith and Langridge 1963; Griffith and ZsIrOs 1971), and in maize (McWilliam and Griffith 1965). The suggested solutions of either replacing temperature-sensitive by non-temperature-sensitive alleles, or, in more complicated cases by making use of hybrids, have been made in the above references.

Further aspects of the modeling process; historical notes: The group genetic model was first proposed (Griffith 1967) in response to a plant breeding dilemma. This dilemma was brought into sharp focus by Wiebe, Petr and Stevens (1963) in an international plant breeding conference. The classical selection theory could not solve the problem because it could not cope with genotypic interactions. The first step of a theoretical solution was to develop a modeling system that could accommodate genotypic interaction, and then to use this modeling system to search for selection procedures that circumvented the dilemma and produced the desired results. A series of additional studies were devoted to this effort (Griffith 1968a,b, 1969; 1976a,b; 1977). Other authors [Gallais (1976) and Wright (1982, 1983, 1986), among others] have used the group genetic model with regard to plant breeding problems.

Another series of studies utilized the same group genetic model in order to examine various aspects of the evolution of social behavior (Griffith 1981-a,b,c,d; 1982a,b,c,d,e,f). These studies explored, in depth, the various kin- and group-selection strategies that are usually considered in the context of the evolution of social behavior.

Further aspects of the modeling process; properties of the group genetic model: In addition to accommodating genotypic interaction, the model was designed specifically to be an extension of the classical (noninteraction) quantitative genetic model. Thus a generalized quantitative genetic modeling system was constructed which ensured that: (i) the group (interaction) model collapsed to the classical (noninteraction) model in the absence of genotypic interaction; (ii) as with the classical model, the group representation accommodated any number of alleles with arbitrary gene frequencies, and any set of dominance parameters for genotypes at a single locus; extension to more loci was, of course, possible; (iii) the group model retained the important property of the classical model with regard to characterizing the results of selection: it exactly identified and isolated appropriate gene effects and variance components that entered into the description of changes in gene frequencies and the population mean. This property was absolutely essential in all of the theoretical studies reported above.

Finally the group modeling scheme was designed to accommodate groups of arbitrary size, and nonrandom as well as random groups.

Rationale of the present study: All studies listed in the above historical account were theoretical in nature. There does not appear to be any study that tests the validity of the model with data from a living system. Such a description should involve effects rather than variance components because qualitative (positive or negative) as well as quantitative (magnitude) attributes of the model are important. Hence the main objective of the present study was to set up an experimental procedure that permitted the examination of the usefulness of the various classes of model effects in describing the complex variability generated by interacting genotypes within a living system.

The simplest experimental procedure was to plant all possible pairwise combinations of two different homozygous parents and their hybrid. Such a design yielded a direct-associate pattern of means that was appropriate for the analysis of any quantitative locus whose genotypes differed in the parental material. Using the orthogonal partitioning associated with this $2^2$ factorial arrangement provided the statistical tools for the identification, testing and estimation of all.
classes of effects in the group genetic model. The present study, then, utilized this experimental approach in an endeavor to test the group genetic model with data from a bona fide living system.

LITERATURE CITED


Griffing, B., 1976b Selection in reference to biological groups. VI. Use of extreme forms of non-random groups to increase selection efficiency. Genetics 82: 723–731.


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