A GENERAL METHOD OF DETECTING THE ADDITIVE, DOMINANCE AND EPISTATIC VARIATION THAT INBRED LINES CAN GENERATE USING A SINGLE TESTER

G. S. CHAHAL and J. L. JINKS

Department of Genetics, University of Birmingham, Birmingham B15 2TT

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SUMMARY

A new experimental design and analysis for investigating samples of inbred lines is described which avoids the difficulties that can arise in choosing adequate testers and the consequences of using inadequate testers while retaining most of the advantages of the triple test-cross design. The new design requires only a single inbred tester and providing that the sample of inbreds is in linkage equilibrium it can have any genotype. The design consists of producing the F_1 crosses between each inbred line in the sample and the tester, and then backcrossing each F_1 to its two inbred parents. Three sets of comparisons among the resulting parental, F_1 and backcross families provide tests for epistasis and in its absence equally reliable and direct tests and estimates of the additive D and dominance H components of variation.

If the sample of inbred lines is not in linkage equilibrium, the estimates of D and H may be differentially inflated or deflated, but this can be minimised if dominance is largely unidirectional by choosing a tester with an extreme phenotype. The design itself provides a test of the direction of the dominance.

The new analysis is illustrated by the reanalysis of portions of the F_1 , F_2 , B_1 and B_2 families of a diallel set of crosses between eight inbred lines of *Nicotiana rustica* grown in 1952 and 1953 which have the appropriate structure. The results show good agreement with those of previous analyses based upon more extensive data and suggest that the new analysis is a viable alternative to other test-cross designs.

1. INTRODUCTION

THE breeding of autogamous crop plants usually starts from a collection of inbred lines from different sources. These may be utilised directly for cultivation after screening, used to synthesise commercial hybrids or to create new genetic variability for the selection of superior inbreds. The first involves the direct exploitation of the additive genetic variance while the choice between the other two should depend on the relative magnitudes of the additive and dominance components of gene action. Indeed, if estimates of the additive, dominance and epistatic components of variability are available we can not only objectively assess the relative advantages and disadvantages of hybrids versus inbreds but we can also predict the probabilities of obtaining inbreds which are superior to the hybrids or to the original inbreds (Jinks and Perkins, 1972; Jinks and Pooni, 1976; Pooni and Jinks, 1977; Pooni, Jinks and Cornish, 1977).

The best method currently available for assessing these components of variation and in addition the magnitude and direction of genotype $\times$ environment interactions and linkage is the triple test-cross of Kearsey and Jinks (1968) and its extensions by Jinks and Perkins (1970) and Perkins and Jinks (1970, 1971). This approach has been simplified and modified in a

number of ways for the restricted case of populations of inbred lines (Jinks, Perkins and Breese, 1969; Virk and Jinks, 1977; Jinks and Virk, 1977). However, for an arbitrary collection of inbred lines, choosing adequate testers, testing their adequacy and overcoming any biases introduced by their inadequacies can raise difficulties during the design, analysis and interpretation levels (Jinks and Virk, 1977). Furthermore, should the analyses reveal inadequacies in the testers many of the advantages of this design relative to others are reduced. These advantages can always be regained, of course, by adopting a more elaborate design extending over a number of generations, for example, a partial or complete diallel set of crosses continued to the F_2 and first backcrosses, B_1 and B_2 , of each of the initial crosses (Jinks, 1956; Jinks and Stevens, 1959). But in practice what is required is a design that retains as far as possible the simplicity of the triple test-cross consistent with providing direct tests of the significance of all of the components and independent estimates of each component with comparable reliability. Such a compromise has been found from within the more complex analysis of Jinks and Stevens (1959).

2. EXPERIMENTAL DESIGN

Each line (P_i) of a sample n of a collection of inbred lines is crossed to one arbitrarily chosen inbred line (P_c) to produce an array of $n F_{1i}$ families. Each F_{1i} family is then backcrossed to each of its parents one of which is unique to that family (P_i) and one of which is common to all families (P_c) to produce two first backcross families $B_{1i}(F_{1i} \times P_i)$ and $B_{ci}(F_{1i} \times P_c)$, respectively. The experiment then consists of the P_c family and n of each of the P_i , F_{1i} , B_{1i} and B_{ci} families giving a total of $4n+1$ families, each of which is replicated by raising either r plots or preferably r individuals in a randomised block design. Alternatively we can include nr replicates of P_c , r of which are allocated at random to each of the n sets of other families for making the various comparisons (see Section 3).

3. MODEL AND ANALYSIS

The analysis may be divided into two parts, the first concerned with the test for epistasis, the second with testing for and estimating the additive and dominance components of variation should epistasis prove to be absent.

(i) *Test for epistasis*

The test for epistasis is based on the standard backcross scaling tests (Mather and Jinks, 1971). In table 1 are given the expectations for these scaling tests for the two backcrosses, B_{1i} and B_{ci} , for each of the four possible genotypes for P_i in respect of two loci, and for the the corresponding four possible genotypes for the tester, P_c . In the absence of epistasis these tests have an expectation of zero irrespective of the genotype of the inbred line, P_i or of the tester, P_c . This will be true irrespective of the number of pairs of loci since all possibilities in respect of any pair of loci are represented in table 1.

We could proceed by carrying out the $2n$ individual scaling tests or preferably the n joint scaling tests combining B_{1i} and B_{ci} for the i th set for

TABLE 1

The contribution of additive, dominance and digenic interaction parameters to the comparisons, (i) $2\bar{B}_{1i} - \bar{F}_{1i} - \bar{P}_i$ and (ii) $2\bar{B}_{ci} - \bar{F}_{1i} - \bar{P}_c$ for all possible P_c genotypes in respect of two pairs of genes

P_i			Possible P_c genotypes			
Frequency*	Genotype	Comparison	$AABB$	$AAbb$	$aaBB$	$aabb$
α	$AABB$	(i)	0	0	0	$\frac{1}{2}(-i_{ab} + j_{ab} + j_{ba} - l_{ab})$
		(ii)	0	0	0	$\frac{1}{2}(-i_{ab} - j_{ab} - j_{ba} - l_{ab})$
β	$AAbb$	(i)	0	0	$\frac{1}{2}(i_{ab} + j_{ab} - j_{ba} - l_{ab})$	0
		(ii)	0	0	$\frac{1}{2}(i_{ab} - j_{ab} + j_{ba} - l_{ab})$	0
γ	$aaBB$	(i)	0	$\frac{1}{2}(i_{ab} - j_{ab} + j_{ba} - l_{ab})$	0	0
		(ii)	0	$\frac{1}{2}(i_{ab} + j_{ab} - j_{ba} - l_{ab})$	0	0
δ	$aabb$	(i)	$\frac{1}{2}(-i_{ab} - j_{ab} - j_{ba} - l_{ab})$	0	0	0
		(ii)	$\frac{1}{2}(-i_{ab} + j_{ab} + j_{ba} - l_{ab})$	0	0	0

* $\alpha + \beta = u_a$, $\alpha + \gamma = u_b$, $\gamma + \delta = v_a$ and $\beta + \delta = v_b$

$i = 1$ to n (Mather and Jinks, 1971). The former would lead to $2n$ individual t tests the latter to n individual χ^2 tests each for 2 degrees of freedom. A test which combined all these individual tests of significance is clearly preferable and this can be achieved within the context of an analysis of variance.

Taking first the n sets of comparisons of the kind

$$2\bar{B}_{1i} - \bar{F}_{1i} - \bar{P}_i = A_i$$

from the sum of A_i^2 over the n sets $\left(\sum_{i=1}^n A_i^2\right)$ we can obtain a mean square for n degrees of freedom. This can be tested against the mean square derived from pooling the corresponding variances of A_i which are obtained in the usual way from the variances of the B_{1i} , F_{1i} and P_i family means (Mather and Jinks, 1971).

For the n sets of comparisons of the kind

$$2\bar{B}_{ci} - \bar{F}_{1i} - \bar{P}_c = B_i$$

$\bar{P}_c$ appears in every set and hence unless we raise n independently randomised sets of r replicates of P_c , each allocated to a particular one of the n sets (Section 2), the n B_i values will not be independent. Given these n sets of P_c , the analysis of the B_i values proceeds exactly as for the corresponding A_i 's. Alternatively, however, if we have only one estimate of $\bar{P}_c$ we can rewrite this comparison as

$$2\bar{B}_{ci} - \bar{F}_{1i} = B_i + \bar{P}_c = B_i + \text{constant}$$

Our test is then based on the mean square derived from the sum of squares of the deviations of the n values of $(2\bar{B}_{ci} - \bar{F}_{1i})$ around their own mean for $n-1$ degrees of freedom. This can be tested against the mean square derived in the usual way from the variances of the B_{ci} and F_{1i} family means.

(ii) Additive and dominance components

If epistasis is absent two further orthogonal comparisons provide unique tests for the additive and dominance components of variation. These are summarised in table 2 for all possible genotypes in respect of two loci for P_i and for the corresponding possible genotypes for P_c . For each comparison we can compute a sum of squares of deviations of the n values from their own

TABLE 2

The contribution of additive and dominance parameters to the comparisons (i) $\bar{B}_{ci} - \bar{B}_{1i} - \bar{P}_c + \bar{P}_i$ and (ii) $\bar{B}_{ci} + \bar{B}_{1i} - \bar{P}_c - \bar{P}_i$, for all possible P_c genotypes in respect of two pairs of genes

P_i		Possible P_c genotypes							
		$AABB$		$AAbb$		$aaBB$		$aabb$	
Frequency	Genotype	(i)	(ii)	(i)	(ii)	(i)	(ii)	(i)	(ii)
α	$AABB$	0	0	d_b	h_b	d_a	h_a	$d_a + d_b$	$h_a + h_b$
β	$AAbb$	$-d_b$	h_b	0	0	$d_a - d_b$	$h_a + h_b$	d_a	h_a
γ	$aaBB$	$-d_a$	h_a	$-d_a + d_b$	$h_a + h_b$	0	0	d_b	h_b
δ	$aabb$	$-d_a - d_b$	$h_a + h_b$	$-d_a$	h_a	$-d_b$	h_b	0	0

mean for $n-1$ degrees of freedom. We can also compute a corresponding sum of squares for the replicate error whose exact form and degrees of freedom will depend on the experimental design. The expected mean squares in all cases would reduce to the following form, where r is the number of replications.

Comparison	Expected mean square
1. $\bar{B}_{ci} - \bar{B}_{1i} - \bar{P}_c + \bar{P}_i$ (Additive)	$\sigma^2 + 4r\sigma_1^2$
2. $\bar{B}_{ci} + \bar{B}_{1i} - \bar{P}_c - \bar{P}_i$ (Dominance)	$\sigma^2 + 4r\sigma_2^2$
Error	σ^2

Hence we can test the significance of, and estimate σ_1^2 and σ_2^2 independently and with equal precision. Furthermore, reference to tables 2 and 3 shows that in all circumstances σ_1^2 reflects only the additive genetic effects (d) while σ_2^2 reflects only the dominance effects (h). Where we have only a single estimate of P_c for these comparisons, this, being a constant in all the

TABLE 3

Estimates of the additive and dominance components of variation and tests of epistasis for final height in 1952 and 1953 from previous analyses and from the new analysis

Year	Component	Previous analyses (Jinks 1954, 1956)					New analysis	
		Complete F_1 diallel	Complete F_1 , F_2 , B_1 and B_2 diallel	F_1 , F_2 , B_1 and B_2 of V38 array			$P_c = V38$	$P_c = V12$
		Significant*	Significant	Non-significant			Non-significant	Significant
1952	Epistasis	Significant*	Significant	Non-significant			Non-significant	Significant
	D	43.00	37.13 ± 3.72	44.59 ± 3.20			46.62	120.07
	H	108.99	76.74 ± 20.68	38.15 ± 20.19			37.72	132.35
	$\sqrt{H/D}$	1.59	1.44	0.92			0.90	1.05
1953	Epistasis	Significant*	Significant	Non-significant			Non-significant	
	D	64.66	55.25 ± 5.07	61.20 ± 5.12			57.75	
	H	231.04	222.98 ± 28.16	94.41 ± 32.28			81.26	
	$\sqrt{H/D}$	1.94	2.01	1.24			1.19	

* These significances could result from epistasis, linkage disequilibrium or both but in all other analyses they test specifically epistasis only.

comparisons, can be omitted and the expectations amended accordingly, the coefficients of σ_1^2 and σ_2^2 both becoming $3r$.

It is clear from table 2 that while σ_1^2 and σ_2^2 depend solely on the d and h effects of the genes, respectively, their expectations will depend on the relative frequencies with which the different P_i genotypes are included in the sample of inbreds and in some circumstances on the genotype of the tester, P_c also. For example, if we assume that the sample is in linkage equilibrium ($\alpha = u_a u_b$, $\beta = u_a v_b$, $\gamma = v_a u_b$ and $\delta = v_a v_b$) but with arbitrary gene frequencies of u_a for A , v_a for a and u_b for B and v_b for b , then for a single pair of loci the expectations are

$$\begin{aligned}\sigma_1^2 &= fu_a v_a d_a^2 + fu_b v_b d_b^2 \\ \sigma_2^2 &= fu_a v_a h_a^2 + fu_b v_b h_b^2,\end{aligned}$$

where $f = \frac{1}{18}$ if $\bar{P}_c$ is included and $\frac{1}{9}$ if $\bar{P}_c$ is excluded from the comparisons irrespective of the genotype of P_c itself.

For many such pairs of loci

$$\begin{aligned}\sigma_1^2 &= f \sum u_i v_i d_i^2 = f \frac{1}{4} D \\ \sigma_2^2 &= f \sum u_i v_i h_i^2 = f \frac{1}{4} H\end{aligned}$$

for all testers.

If we now relax the assumption of linkage equilibrium so that $\alpha \neq u_a u_b$, $\beta \neq u_a v_b$, $\gamma \neq v_a u_b$ and $\delta \neq v_a v_b$ the expectations of σ_1^2 and σ_2^2 will depend on the genotype of the tester P_c . For example, if the tester is $AABB$

$$\sigma_1^2 = fu_a v_a d_a^2 + fu_b v_b d_b^2 + 2f(\delta - v_a v_b) d_a d_b$$

and

$$\sigma_2^2 = fu_a v_a h_a^2 + fu_b v_b h_b^2 + 2f(\delta - v_a v_b) h_a h_b$$

whereas if the tester is $AAbb$

$$\sigma_1^2 = fu_a v_a d_a^2 + fu_b v_b d_b^2 - 2f(\gamma - v_a u_b) d_a d_b$$

$$\sigma_2^2 = fu_a v_a h_a^2 + fu_b v_b h_b^2 + 2f(\gamma - v_a u_b) h_a h_b$$

The expectation for the other testers, $aabb$ and $aaBB$ are obtained by substituting $(\alpha - u_a u_b)$ for $(\delta - v_a v_b)$ and $(\beta - u_a v_b)$ for $(\gamma - v_a u_b)$ in these expectations, respectively. Since at equilibrium $\delta = v_a v_b$, $\gamma = v_a u_b$, $\alpha = u_a u_b$ and $\beta = u_a v_b$ these additional terms are simple functions of the linkage disequilibrium. In the presence of linkage disequilibrium, however, the form of the expectations differs according to whether the single tester P_c has the pairs of genes in association ($AABB$ or $aabb$) or in dispersion ($AAbb$ or $aaBB$). Furthermore, the coefficients of the d terms in σ_1^2 and of the h terms in σ_2^2 are no longer necessarily identical. All testers therefore, will do equally well when the P_i sample is in equilibrium. When this is not so a tester with the maximum number of gene pairs in association and hence with an extreme phenotype is preferable because the most critical decisions usually arise when there is strong directional dominance leading to heterosis (see Introduction). We can in fact obtain a good indication as to whether the dominance is mainly unidirectional or ambidirectional from the signs

of the deviations obtained from the dominance comparison (table 2). If all n values for this comparison have the same sign there is clearly a strong unidirectional component. If on the other hand some are positive and some negative the unidirectional element must be weak or even absent if the positive and negative deviations exactly balance one another.

4. ALTERNATIVE SETS OF COMPARISONS

In Section 3 we presented one set of comparisons among the P_c , P_i , F_{1i} , B_{ci} and B_{1i} families which satisfies all our requirements. In all, however, we have examined 63 different comparisons, if we count different signs and coefficients, among these families and have found only one further orthogonal pair which can provide independent tests of significance and estimates of D and H ; these are $\bar{B}_{ci} + \bar{B}_{1i} - \bar{P}_i - \bar{F}_{1i}$ and $\bar{B}_{ci} - \bar{B}_{1i} + \bar{P}_i - \bar{F}_{1i}$, respectively. In terms of the simple theory we have developed there is no reason to prefer either one set or the other for estimating D and H . However, because of the symmetry of the earlier set of comparisons it will in general be more robust in the presence of complications. For example, with the earlier set of comparisons it is sufficient in the presence of maternal effects to use P_i as the maternal parent of B_{1i} and P_c as the maternal parent of B_{ci} to eliminate these effects from the estimates of D and H . Furthermore, with these comparisons the biases in the estimates of D and H in the presence of interactions are the familiar ones whose properties are well understood (see Mather and Jinks, 1971, Chapter 7).

5. EXAMPLE

For illustration we shall reanalyse data for final height from a diallel set of crosses between eight inbred lines of *Nicotiana rustica* carried out by Jinks between 1950 and 1953. The parents and F_1 's of this diallel set were grown in 1951 and again in 1952 and 1953 along with their F_2 and first backcross (B_1 and B_2) families (see Jinks, 1954 and 1956, for details). The data for final height and for a number of other characters have been analysed as F_1 diallels (Jinks, 1954), as combined parental, F_1 , F_2 , B_1 and B_2 families of a diallel set (Jinks, 1956) and by a set of comparisons which separate additive, dominance and epistatic components of variation (Jinks and Stevens, 1959). The results of some of these earlier analyses are summarised in the first three columns of table 4. The first is the analysis of the complete F_1 diallel for 1952 and 1953. This provides perfect fit estimates of D and H (as well as of other components which are not relevant) and tests of the adequacy of the additive, dominance model which in this context is a joint test of the independence of the genes in action (no epistasis) and in distribution (no linkage disequilibrium). The second is the analysis of the combined parental, F_1 , F_2 , B_1 and B_2 families for 1952 and 1953. This provides least squares estimates of D and H and of other components, and specific tests for epistasis as well as for other causes of inadequacy of the simple model. The third is a joint analysis of all the families belonging to one array of the diallel set, that with common parent $V38$, the only one within which the additive, dominance model is adequate, using the single array analysis of Jinks (1955). This array is the obvious first choice for illustrating our new analysis.

TABLE 4

The mean final height in inches and the number of plots of five plants on which the mean is based for the tester line, P_c , the seven P_i lines and their F_{1i} , B_{ci} and B_{1i} families grown in 1952

P_c	V38	35.90 (2)	$F_{1i}(P_i \times P_c)$	$B_{ci}(F_{1i} \times P_c)$	$B_{1i}(F_{1i} \times P_i)$
$P_i \ i = 1$	V2	33.50 (2)	34.55 (4)	32.55 (4)	33.70 (4)
2	V5	38.40 (2)	40.80 (4)	36.05 (4)	41.90 (4)
3	V7	27.40 (2)	35.40 (4)	34.10 (4)	32.90 (4)
4	V12	51.30 (2)	50.60 (4)	40.95 (4)	50.35 (4)
5	V14	35.80 (2)	36.35 (4)	34.70 (4)	40.35 (4)
6	V29	39.50 (2)	35.30 (4)	32.80 (4)	36.45 (4)
7	V41	33.30 (2)	33.70 (2)	33.65 (4)	33.70 (4)

If we now equate the relevant families of this array to those of the new design; V38 is the tester line, P_c , and the remaining seven inbred lines, provide a sample of $n = 7$ of the inbred lines, P_i . There are then seven F_{1i} families ($P_i \times P_c$ for $i = 1$ to 7). In the complete diallel these F_1 crosses were made reciprocally and we shall regard these as replicates in our subsequent analyses. Both reciprocals of each F_{1i} were themselves back-crossed as female parents to P_c and to the corresponding P_i to produce reciprocal (replicate) pairs of B_{ci} and B_{1i} families. All P_c , P_i , F_{1i} , B_{ci} and B_{1i} families were grown simultaneously in 1952 and in 1953 in a randomised block design with two blocks, the unit of randomisation being a plot of five plants of the same family. The total number of plots of each family grown in 1952 are given in table 4 along with the mean final height of each family in inches. For reasons which have been discussed elsewhere we shall use a conservative estimate of the replicate error based upon the differences between reciprocal crosses rather than on the differences between blocks when both are available (Jinks, 1954, 1956).

Since the Wr , Vr analysis and tests of goodness of fit of models to first and second degree statistics (Jinks, 1954, 1956) revealed no evidence of epistasis in any cross involving P_c (V38) it is not surprising that our test for epistasis based on an analysis of variance (Section 3 (i)) was not significant ($P > 0.25$). We can proceed therefore with the fitting of the additive, dominance model.

Using the comparisons described in Section 3 the expression appropriate for testing and estimating the additive and dominance effects in these data are those which omit $\bar{P}_c$ and these and their corresponding mean squares are given in table 5. The 35 degrees of freedom for the error mean square

TABLE 5

Analysis of variance for the additive and dominance comparisons for final height in 1952

Item	d.f.	M.S.	P	EMS
1. Additive ($\bar{B}_{ci} - \bar{B}_{1i} + \bar{P}_i$)	6	12.6447	0.05	$\sigma^2 + 3r\sigma_1^2$
2. Dominance ($\bar{B}_{ci} + \bar{B}_{1i} - \bar{P}_i$)	6	11.1613	0.05-0.10	$\sigma^2 + 3r\sigma^2$
Error	35	4.8751		σ^2

$$\hat{\sigma}_1^2 = 1.2949 = \frac{1}{36} D \quad \hat{D} = 46.62$$

$$\hat{\sigma}_2^2 = 1.0477 = \frac{1}{36} H \quad \hat{H} = 37.72.$$

are made up of 28 for the reciprocal differences within blocks of the seven B_{ci} and seven B_{1i} families (two for each family) plus 7 degrees of freedom for block difference for the seven P_i inbreds. While the dominance component is not formally significant ($P \leq 0.05$) this merely reflects the inefficiency of the experimental designs used at that time so we shall proceed to estimate both of the genetical components. The estimates of D and H (tables 3 and 5) correspond very closely to those obtained from the previous analysis of the $V38$ array. The corresponding 1953 data have also been reanalysed using the new method with the results shown in table 3. They too correspond very closely with the results of previous analyses. Furthermore, in these data the dominance component was significant at the level of $P = 0.05-0.01$.

We can of course use the other arrays from the 8×8 diallel set of crosses to illustrate our new analysis but in so far as previous analyses have shown that they include epistatic crosses our analyses should detect epistasis, and if we then proceed to estimate D and H they will be biased by this epistasis. To illustrate this we have analysed the 1952 data for the array of which $V12$ is the common parent. The W_r , V_r analysis and the joint scaling tests (Jinks, 1954, 1956) have shown that the crosses in this array have a high incidence of epistasis and our analysis of variance for epistasis confirms this ($P = 0.05$). Nevertheless, we have estimated the additive and dominance components both of which are significant ($P < 0.05$) and their values are given in table 3. As we expected they are both greatly inflated relative to the corresponding estimates for the $V38$ array.

6. CONCLUSIONS

In this paper we have shown that for a population of inbred lines the problems created by inadequate testers can be completely avoided. At the cost of an additional generation of breeding, little or no increase is required in the size of the experiment or in the complexity of the breeding programme. While we have yet to test the new design and analysis on material specifically and optimally designed for the purpose, the results of reanalysing existing data which have an appropriate structure are encouraging and reproduce the conclusions drawn from more extensive data analysis. For the special case of populations of inbred lines, the new design and analysis is a viable alternative to the triple test-cross and its various modifications.

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