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QUANTITATIVE INHERITANCE IN WHEAT

A THESIS SUBMITTED FOR THE DEGREE OF
DOCTOR OF PHILOSOPHY
IN THE
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By

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QUANTITATIVE INHERITANCE IN WHEAT

by

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The inheritance of yield, yield components and some morpho-physiological traits such as, plant height, spike length, and flag leaf length was studied in six New Zealand wheat cultivars. Three biometrical methods namely, the Half Diallel (Morley Jones, 1965), the Scaling Tests (Mather and Jinks, 1971) and the General Methods for Detecting Epistasis (Chahal and Jinks, 1978) were used for the genetical analyses of these traits. The genetical control of flour protein was also studied by The Half and Full Diallel analyses.

Three principal components of yield, spikes per plant, grains per spike and 1000 grain weight were found to be under complex genetical control involving epistasis. However, grains per spike can be reduced into its basic subcomponents of grains at the individual spikelet position and spikelet, per spike to explore the role of multiplicative epistasis. In exact algebraic expression, Grains per Spike = $\sum_{i=1}^N G_i$ where G_i is grain number at a specific spikelet position and N is the number of spikelets per spike. Using this rationale, an attempt was made to resolve the role of multiplicative epistasis in grains per spike by studying the nature of gene

action of this trait and its two subcomponents. Grain number at a single spikelet position was shown to be under relatively simple additive gene control and spikelets per spike was under additive and dominance gene control. The simpler inheritance of these two subcomponents compared to grains per spike, which was shown to express epistasis, has enabled a selection strategy to be proposed. This selection procedure involves the direct exploitation of mainly additive genes for grains at a single spikelet position in early generations. A selection response can conceivably be achieved through this procedure instead of trying to fix the elusive large spike controlled by epistasis in early generation.

The genetical analysis of morpho-physiological traits have revealed plant height and spike length to be under control of additive and dominance genes for all cultivars studied except for plant height in the cultivar Atlas 66. Epistasis for plant height was exhibited by the families derived from Atlas 66 and Karamu. The significance of this record of duplicate type epistasis was discussed in the light of the 'tall dwarf' breeding strategy. It was concluded that while the 'tall dwarf' selection model could be adopted for crosses between the semidwarf and the standard height cultivars studied, difficulty could be anticipated in crosses involving Atlas 66 and the semidwarf cultivar, Karamu, because of epistasis.

The Diallel analyses of flour protein content in the F1 and F2 families indicated the genetical control for this

trait to be mainly of the additive and dominance type. High narrow sense heritability was recorded, emphasizing considerable prospects for early generation response to selection in crosses between the high protein cultivars. However, epistasis was detected in the F1 Half Diallel analysis involving the cultivar, Karamu. The failure to record similar epistasis in the F2 Half Diallel analysis suggested existence of genotype environmental interaction for flour protein.

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CHAPTER 1

INTRODUCTION

The science of breeding autogamous crop, such as wheat, can broadly be divided into three phases: assembly or creation of a pool of variable germplasm, selection of superior individuals from the pool and utilization of the selected individuals to produce synthetic hybrids or to create a superior selection. However,^a more recent attitude is to consider hybrid wheat production as not practical (Lupton, 1979). The practice of these crop breeding strategies demands a thorough knowledge of the nature of gene actions. The selection of superior individuals involves direct exploitation of the additive genetic variance, while the choice between a synthetic hybrid or the selection of a superior inbred, following the standard crossing technique, depends on the relative magnitudes of the additive and dominance components of gene action. Presence of large dominance component would favour the production of commercial hybrid while traits with mainly additive gene control can be improved by selection of superior segregants. Standard hybridization and selection procedures could also take advantage of epistasis, if it is of the additive type (additive x additive, additive x additive x additive, etc.), while other types of epistasis (additive x dominance, dominance x dominance, etc.) are not fixable by selection under self fertilisation and therefore would not favour the development of pure

line cultivars. However, the masking effects of epistasis are of no consequence if selection is delayed until virtual homozygosity since only additive type of epistasis are present in pure lines.

Therefore, if estimates of the additive, dominance and epistatic components of variability are available, one can decide objectively not only the breeding and selection procedures but one can also impose early or late generation selection for optimal advancement. The knowledge of the nature of epistasis can be of immeasurable help, and can save the enthusiastic breeder from chasing the elusive traits that are controlled by non-additive type epistasis at early generations of selfing.

The mating designs used in this study are chosen to provide the best methods currently available for assessing these components of genetic variation and also to optimise the use of all the generations available in this study. The Half Diallel Analysis of Jones (1965) and, the Variance - Covariance ($V_r - W_r$) analysis of Jinks (1954) and Mather and Jinks (1971) are used to extract informations from the F_1 and F_2 families, some of which are produced to the specific requirements of the general methods for detecting the additive, dominance and epistatic variation that inbred lines can generate using a single tester (Chahal and Jinks, 1978). This design of Chahal and Jinks (1978) shall be referred to as the New Triple Test Cross Design in this thesis. The Half Diallel Analysis can provide estimates of both additive and dominance components of variation and also a test for the adequacy of this restrictive additive and dominance model. However, no

unambiguous test for epistasis is available in this Half Diallel analysis. The New Triple Test Cross is designed specifically as an unambiguous test for epistasis and can, in its absence, provide equally reliable and direct tests and estimates of the additive and dominance components of variation. The Scaling Tests and the Cavalli's Joint Scaling Test (Mather and Jinks, 1971) are used to test for the presence of epistasis in each of the individual sets of cross in the New Triple Test Cross Design and, in the presence of epistasis, estimates the magnitude of the non-allelic i, j, l type interactions.

In New Zealand, the objectives of the wheat breeding programme have been for advancement in yield, milling and baking quality and disease resistance (Wright, 1978). In this study, emphasis is placed on understanding the inheritance of yield and yield components. The yield component approach to selection has been subject to a considerable amount of interest. This is because of the high heritabilities of some of the yield components (Kronstad and Foote, 1964; Fonseca and Patterson, 1968; Ketata, Edwards and Smith, 1976a; Rahman, Halloran and Wilson, 1977). Selections based on yield components have been found to be effective in changing yield (Knott and Talukdar, 1971; McNeal, 1978). This is in spite of the negative inter-component correlations between some yield components due to compensation. Yield component compensation is not necessarily complete (Knott and Talukdar) and a genetic increase in one component may well result in a yield increase.

The simpler genetic control and higher heritabilities

of yield components compared to yield *per se* has been attributed to the removal of multiplicative epistasis (Hayman, 1960; Moll, Kojima and Robinson, 1962; Grafius, 1964). Our study also attempts to remove multiplicative epistasis in grain number per spike. Grain number per spike is a multiplicative derivation of grain number at each individual spikelet position and total number of spikelet per spike. By studying grain number at a particular spikelet position, it is hoped to remove any multiplicative epistasis present.

Agronomic traits not directly contributing to yield but playing a significant role in the availability of photosynthate for grain yield are included in this study. As destructive sampling could not be practised, *in situ* measurements could only be made with sufficient precision on plant height, head length and flag leaf length. Genetical analysis of flour protein content, a crucial quality trait, forms the final part of this study.

CHAPTER 2

MATERIALS AND METHODS

2.1 CHOICE OF PARENTS

The six parental cultivars used in this study were chosen to reflect the diversity of wheat cultivars available in New Zealand for commercial production and use ^{by} (of) plant breeders. These six cultivars represent a range of maturity types, plant heights, yielding capabilities and quality characteristics. The parents were cultivars Hilgendorf 61, Kopara 73, Oroua, Ruru (Line 2288,03), Karamu and Atlas 66, all made available by the Crop Research Division, D.S.I.R., Lincoln.

cv. Hilgendorf

Cultivar Hilgendorf 61 was an improved selection of the original cv. Hilgendorf and was obtained by backcrossing India 241 selection Desi a total of seven times to cv. Hilgendorf (Coles and Wrigley, 1976). The original cultivar Hilgendorf was a selection from cross 140 of Tainui x Cross 7 (Frankel and Hullet, 1947). Cultivar Hilgendorf 61 is a standard-height New Zealand wheat (being short-strawed compared with tall wheats of Mexican origin). This awnless cultivar is characterised by low yielding capability, short spike, low spikelet number per spike and low fertility of florets but high 1000-grain weight (Langer, 1965). The superior quality of cv. Hilgendorf 61 as a premium wheat

(Meredith, 1970) is still being acknowledged when McEwan and Cross (1978) stated that cv. Hilgendorf 61 has set high standards for baking quality. Cultivar Hilgendorf 61 shall be referred to as Hilgendorf in the subsequent parts of this thesis.

cv. Kopara 73

Cultivar Kopara 73 is a reselection from the bulk line originally numbered 1020,01 which was released in 1971 as Kopara. Kopara or 1020,01 was a selection from the cross 1020 which ^{combined} had the following two parent ^{F₁ lines} combinations: Arawa x Gabo, Atson x Selkirk, Arawa x Selkirk and Aotea x Hilgendorf ^{grand} as parents (Copp and Cawley, 1973). Cultivar Kopara 73 is another awnless standard height wheat capable of high yield (McEwans and Cross, 1978). It is characterised by long spike with high spikelet number per spike. Cultivar Kopara 73 has been shown to have less variable quality and higher mechanical dough development ^{baking quality} than cv. Karamu over a range of agronomic treatments (Dougherty et al., 1978). Cultivar Kopara 73 shall be referred to in this thesis as Kopara.

cv. Oroua

Oroua, or line 74,02, is derived from the cross 66RN395 x Skemer. It is a short-strawed, awned wheat of similar stature and maturity as cv. Karamu when spring sown, but taller than cv. Karamu when winter sown. Its yielding capacity is below that of cv. Karamu in some areas, but as high as cvs. Karamu and Kopara in other areas (McEwan and Hadfield, 1978; Wright, 1980). The baking quality of Oroua has been shown to be superior to cvs. Karamu or Kopara (McEwan,

Vizer and Douglas, 1979).

cv. Ruru

Cultivar Ruru or line 2288,03, is derived from the cross (Gaines x 1018,01) x (Opal x Hilgendorf 61). Line 1018,01 was a selection from the cross ((Orawa x Gabo) x (Aotea x Onas 53)) x ((Arawa x Selkirk^k) x (Aotea x Hilgendorf 61)). It is a short-strawed, awned, late maturing and is capable of high yield. It has long spike, high spikelet number per spike and good tillering potential.

It is, however, of low^{quality} ^{millage, baking quality, and was not released} and ~~has been classed as a feed~~ ^{for commercial production} wheat (Wright, pers. comm., 1977).

cv. Karamu

Cultivar Karamu was introduced from the Wagga Wagga Research Institute of Australia as WW15. It was a ^{Cimmyt} selection of the cross (Lerma Rojo x Norin 10 - Brevor) x Andes³ (McEwan, 1973). The high harvest index of this selection has been described by Syme (1970, 1972). This awned cultivar has gained widespread acceptance as a high yielder since its release in New Zealand in 1972 (McEwan and Vizer, 1972). Its high yielding potential is mainly attributed to better grain set per spikelet and formation of more spikelet[↑] per spike (Dougherty et al., 1977). It is of semidwarf habit and early maturing. However, this Mexican derivative has been troubled with extremely variable quality since its release (Langer, 1977). Cross and Haslemore (1979) have found Karamu to be inferior in both total grain protein and grain protein concentration. They attributed this to a smaller pool of vegetative N potentially available for re-

distribution and ultimate grain protein synthesis.

cv. Atlas 66

Cultivar Atlas 66 was derived from a cross between a high protein Brazilian wheat Frondoso and Redhart - 3 - Noll 28 (Middleton *et al.*, 1954). It is a tall, awnless, late flowering wheat and compared to modern wheat cultivars, it is low yielding (Gill *et al.*, 1977). Its superior protein content is well recognised (Middleton *et al.*, 1954; Seth *et al.*, 1960; Johnson *et al.*, 1967).

2.2 FIELD EXPERIMENTS

This study involves three seasons of field experimentation at Lincoln College, Canterbury, New Zealand (1977-1980).

Season I : August 1977 - February 1978

Five parental cultivars, Hilgendorf, Kopara, Oroua, Ruru and Karamu were sown at weekly intervals over a period of five weeks to provide enough of male and female materials for the pollination programme. Between ten to fifteen crosses (heads) were made in every possible combination amongst these five parents, except the reciprocals to meet the requirements of the Half Diallel Design of Jones (1965). During the same season, between fifteen to twenty crosses were made between cultivars Atlas 66 and Karamu in the field of the DSIR, Lincoln. These pollinations produced the following F₁ families.

- | | |
|------------------------|-----------------------|
| 1. Hilgendorf x Kopara | 7. Kopara x Karamu |
| 2. Hilgendorf x Oroua | 8. Oroua x Ruru |
| 3. Hilgendorf x Ruru | 9. Oroua x Karamu |
| 4. Hilgendorf x Karamu | 10. Ruru x Karamu |
| 5. Kopara x Oroua | 11. Atlas 66 x Karamu |
| 6. Kopara x Ruru | |

Season II : August 1978 - February 1979

A further season of cross pollination was carried out to meet the requirements of the New Triple Test Cross Design. This crossing programme involved the production of the five F1 families with Karamu as the common parent and the back-crosses of all the five F1 families (produced in Season I) with Karamu as the common parent to their common parent and their respective unique parent. Between ten to fifteen heads were pollinated for each cross and these were achieved through the same strategy involving weekly sowing of parents and F1's materials over a period of five weeks. The following families were produced.

- | | |
|-------------------------------------|-------------------------------------|
| 1. Karamu x Hilgendorf | 9. Karamu x Oroua x Oroua |
| 2. Karamu x Hilgendorf x Karamu | 10. Karamu x Ruru |
| 3. Karamu x Hilgendorf x Hilgendorf | 11. Karamu x Ruru x Karamu |
| 4. Karamu x Kopara | 12. Karamu x Ruru x Ruru |
| 5. Karamu x Kopara x Karamu | 13. Karamu x Atlas 66 |
| 6. Karamu x Kopara x Kopara | 14. Karamu x Atlas 66 x
Karamu |
| 7. Karamu x Oroua | 15. Karamu x Atlas 66 x
Atlas 66 |
| 8. Karamu x Oroua x Karamu | |

During this season, the Half Diallel F1 families and

parents were grown. This crop constitutes Experiment I of this study. The ten F1 families together with the five parents, were planted in two replicate blocks. These families were randomised within block. A row of buffer cultivar was planted at the end of each block. Within each block, each family was represented by twenty five plants. These plants were planted to a single row. They were spaced five cm between plants and $\frac{3}{70}$ cm between rows.

Season III : June 1979 - February 1980

Two experiments, Experiment ~~I~~^{II} and Experiment III, were grown during this season. The F2 families of the Half Diallel studies formed Experiment ~~I~~^{II}. The same experimental design and layout as Experiment I was adopted for Experiment II. Fifty plants per family per block were grown.

Experiment III consisted of the F1, backcrosses and parents of the New Triple Test Cross Design. An additional generation, the F2 of the Karamu families of the New Triple Test Cross Design was also planted to meet the requirements of the Scaling Test Analysis (Mather and Jinks, 1971). The thirty families were planted to three replicate blocks. The thirty families were made up of five sets of Pi, Pc, Bli, Bci, Fli and F2i. A set of Pc is grown for each set of families having a common parent so that the Bi comparison of the test for epistasis will be uncorrelated (Chahal and Jinks, 1978). Each family was represented by twenty five plants per replicate block. The seeds were sown to the same spacing as Experiments I and II.

2.3 BIOMETRICAL METHODS

2.3.1 *The Half Diallel Analysis*

The statistical analysis of the diallel table has been a subject of many studies since the publication by Yates (1947). This analysis has since been modified in various ways to satisfy various experimental mating designs e.g. presence and absence of parental means and reciprocal crosses (Hayman, 1954a, b; Griffing, 1956; Jones, 1965) and unbalanced diallels in which reciprocals or selfs do not occur in arbitrary numbers (England, 1974; Keuls and Garretsen, 1977). Frequently, reciprocal differences are absent and only one of each pair of reciprocal is raised, and this had prompted Jones (1965) to propose the Half Diallel Analysis. Kearsey (1965), in a comparison of five experimental designs, had concluded that the Half Diallel Analysis of Jones (1965) was to be preferred because of the large amount of precise information it could provide about the components of variation.

The appropriate statistical model required to adequately describe the variation in an entry, Yrs, in the full diallel table with maternal and reciprocal effects was proposed by Hayman (1954a) as

$$Yrs = m + jr + js + l + lr + ls + lrs + kr - ks + krs$$

n x A, are suffixes
uncomplete
g j

where m = grand mean

jr = mean dominance deviation

lr = further dominance deviation due to the r th parent

lrs = remaining discrepancy in the r sth reciprocal sum

$2kr$ = difference between the effects of the r th
parental line used as male parent and as
female parent

$2krs$ = remaining discrepancy in the r sth reciprocal
difference.

Jones' (1965) Half Diallel Analysis is based on the same linear model except for the assumption of absence of both maternal and reciprocal effects. The table for the analysis of variance of the half diallel of Jones (1965) is as follows:

	Constant	Sum of Squares	Degree of Freedom
a	j_r	$\frac{1}{(n+2)} \text{dev}^2 U_r$	$n-1$
b	$1 + l_r + l_s$	$b_1 + b_2 + b_3$	$\frac{1}{2}n(n-1)$
b1	1	$\frac{(2y_{..} - (n+1)y.)^2}{n(n^2-1)}$	1
b2	l_r	$\frac{1}{(n^2-4)} \text{dev}^2 t_r$	$n-1$
b3	l_{rs}	Total -- a - b1 - b2	$\frac{1}{2}n(n-3)$

y_{rs} - entry in the r th row and s th column of the diallel table

y_{rr} - value of the r th self

$y_r.$ - row sum

$y_{..}$ - $\sum y_r.$

$y.$ - $\sum y_{rr}$

U_r - $y_r. + y_{rr}$

t_r - $2 y_r. - n y_{rr}$

If the additive and dominance model is adequate, then the mean square^Δ_λ in the analysis of variance of the diallel can be interpreted as follows: The 'a' item tests the additive effect

while the 'b' item the dominance effect. The 'b' item can further be subdivided into 'b1', 'b2' and 'b3'. The 'b1' item tests the mean deviation of the F1's from the mid-parental values. Its significance indicates presence of directional dominance. The 'b2' item tests whether the mean dominance deviation of the F1 from their mid-parental values within each array differs over arrays. Its significance implies some parents contain more dominant alleles than others. The 'b3' item tests the presence of specific combining ability. It will be significant if some ^{specific} F1 perform considerably better or worse than ^{other} the parents.

2.3.2 The Covariance (W_r) - Variance (V_r) Analysis of the Diallel Table

The theory and analysis of the diallel crosses, as presented by Jinks (1954), Hayman (1954b) and Mather and Jinks (1971) have one of its features the W_r , V_r distribution which is used to assess the genetical assumptions underlying this theory. If all the assumptions are met, then the line of regression of W_r , the covariance between the offsprings of the r th parents and their non recurrent parents, on V_r , the variance of all these offsprings, would have a slope of one. In the regression form this can be expressed as $W_{ri} = \frac{1}{2}(D - H_1) + bV_{ri}$ where $b = 1$. Therefore, a line of unit slope through the origin is that of complete dominance. Movement of this line of unit slope upward or downward, relative to the line of complete dominance would reveal decreasing or increasing dominance respectively. The intercept of the line on the ordinate is then a measure of the average degree of dominance.

The distance of this intercept from the origin was $\frac{1}{2} (D - H1)$. Therefore, when $D > H1$ the intercept is positive, $D = H1$ where the line passes through the origin and $D < H1$ when the intercept is negative. Moreover, the relative order of the points along the regression line indicates the distribution of the dominant and recessive genes among the parents. The points nearest the origin are from the arrays with most dominant genes and the points furthest away from the origin from arrays with most recessive genes. A correlation coefficient of the sum of $(Wri + Vri)$ values with the phenotypic values of the common parents, Pi , can also indicate the dominance and recessive nature of the parents. A high negative correlation of the $(Wri + Vri)$ with the Pi means dominance of the increasing phenotype. A high positive correlation means the reverse is true.

2.3.3 *Testing the Adequacy of the Additive and Dominance Model*

Two tests proposed by Mather and Jinks (1971) to test the goodness of fit for the additive and dominance model were conducted in the programme Binhalf (Appendix I). The two tests are an analysis of variance to test the consistency of the $Wr - Vr$ over arrays and a joint regression analysis of Wr on Vr to test the agreement between blocks and the agreement of the joint regression slope with unity. Since both these tests are approximate, only when both indicated a significant disagreement with the model ($P < 0.05$) was it concluded that the data did not confirm to one or more of the basic assumptions of the model (Gibori et al., 1978). In this thesis, the joint

regression analysis of W_r and V_r is used as the sole test for the adequacy of the assumptions of the model.

2.3.4 *The Perfect Fit Estimates of the Components of Variation*

If the additive and dominance model is deemed adequate, then perfect fit estimates of the components of variation can be attempted. The following second degree statistics are used for estimating the various components of variation:

V_p = variance of the parents,

\bar{V}_r = mean of V_r over all arrays,

$\bar{V}\bar{r}$ = variance array means around overall progeny mean,

\bar{W}_r = mean of W_r over all arrays,

E = block interaction of the family means.

The equations for estimating the various components of variation are:

$$D = V_p - E$$

$$H1 = 4 \bar{V}_r + V_p - 4 \bar{W}_r - \frac{3n - 2}{n} E$$

$$H2 = 4 \bar{V}_r - 4 \bar{V}\bar{r} - \frac{2(n^2 - 1)}{n^2} E$$

$$F = 2V_p - 4 \bar{W}_r - \frac{2(n - 2)}{n} E$$

$$\bar{u}\bar{v} = \sqrt{(H1/D)}$$

$$\text{Narrow sense Heritability} = \frac{\frac{1}{2}D + \frac{1}{2}H1 - \frac{1}{2}H2 - \frac{1}{2}F}{\frac{1}{2}D + \frac{1}{2}H1 - \frac{1}{4}H2 - \frac{1}{2}F + E}$$

$$\text{Broad sense Heritability} = \frac{\frac{1}{2}D + \frac{1}{2}H1 - \frac{1}{4}H2 - \frac{1}{2}F}{\frac{1}{2}D + \frac{1}{2}H1 - \frac{1}{4}H2 - \frac{1}{2}F + E}$$

For the F2 generation, the contribution of dominance component to family and generation means is correspondingly halved, the coefficients of H1 and H2 must be quartered (terms in h^2) and the coefficient of F (term in h) halved. As the computer programme Binhalf (Appendix I) was written for the F1 analysis, minor alterations of the coefficients of H1, H2 and F is necessary for the F2 analysis.

There has been, however, much criticism of the validity of the assumptions involved in the diallel model (Gilbert, 1958; Sokol and Baker, 1977; Baker, 1978).

The genetical assumptions required of the model are:

- (i) diploid segregation,
- (ii) no difference between reciprocal crosses,
- (iii) independent action of non-allelic genes,
- (iv) no multiple allelism,
- (v) homozygous parents,
- (vi) genes independently distributed between parents.

Sokol and Baker (1977), in their theoretical studies using computer simulation, demonstrated that general combining ability includes effects due to additive, epistatic and, when gene frequencies are not equal to 0.5, dominant gene action. They were of the opinion that most diallel experiments were of little value due to these inabilities of meeting the assumptions of gene frequencies of 0.5 and absence of epistasis. Baker (1978) pointed out that the assumption that genes are distributed independently in the parents of the diallel is not realistic. Nassar (1965) emphasized that the correlation of gene distribution is inevitable in any small sample of parental genotypes, and may be a

frequent source of deviation from expectations provided by the diallel model in the absence of other failure in the list of assumptions. The assumption of the absence of epistasis prompted Gilbert (1958) to state that such an assumption cannot be justified from our knowledge of the biochemical pathways in plants. Crumpacker and Allard (1962), in their diallel analysis of heading date in wheat, were of the opinion that the assumptions of no epistasis, no multiple alleles, and uncorrelated gene distribution were not strictly valid. They, however, emphasized that the $V_r - W_r$ graphs were not distorted indicating that these failures were unlikely to be a significant source of bias and seemed unlikely to be large enough to disturb a genetic analysis of the data. Johnson (1963), in both theoretical and practical considerations of the application of the diallel cross technique to plant breeding, was of the opinion that the diallel analysis could provide invaluable genetical information and guidance in practical plant breeding. Hayman (1963) in his discussion of the use of the small diallel crosses, emphasized that when the parent number used is less than ten, then none of the components of variation, either statistical or genetical, in the diallel cross can be significant estimates of the population parameters. The information available from the small diallel cross is that there are certain differences between the parents, between the crosses, or between the general or specific combining abilities of the parents.

2.3.5 *The New Triple Test Cross Analysis*

Most genetic models have as one of their simplifying assumptions the absence of epistasis or non-allelic gene interaction. Among the many multiple mating designs such as the North Carolina Model I, II and III (Comstock and Robinson, 1952) and the Diallels (Hayman, 1954a; Morley Jones, 1965; Kearsey, 1965) only the Diallels provide a test for the adequacy of the additive and dominance model. Moreover, most breeding designs have a much larger standard error for the dominance components compared to the additive components except for the North Carolina Model III of Comstock and Robinson (1952).

Kearsey and Jinks (1968) in their effort to overcome these deficiencies, proposed the Triple Test Cross Analysis. The Triple Test Cross is essentially a simple extension of the North Carolina Model III. The extended design includes the F₁ in the backcrosses. This modification provides not only a more efficient estimate of the dominance component, but also an unambiguous test for epistasis. It has been adequately emphasized by Kearsey and Jinks (1968), in their proposal of the original triple test cross design, that the two inbred lines L₁ and L₂ must be extreme genotypes if the estimates of the additive and dominance variation are to be detected with precision. Since estimates of additive variation have meaning only if L₁ and L₂ are extreme selections, heritability estimates can only be obtained after selection has taken place. Thus, this heritability estimate is of little value to breeders seeking a selection response. Therefore, it must be emphasized that the major contribution of this

technique is only for furthering an understanding of the gene action of the traits under study.

The original ^Ttriple ^Ttest ^Ccross of Kearsey and Jinks (1968) has been simplified by Jinks, Perkins and Breese (1969). In the simplified version, the F₁ has been replaced by P_i families. The P_i families are selfed of the pure breeding population under study. The analysis can yield unambiguous results only if the L₁ and L₂ pure breeding testers differ at all the K loci at which individuals in the population may differ. The consequences of using inadequate testers in the simplified triple test cross has been discussed by Virk and Jinks (1977) and a modified analysis to test and allow for inadequate testers has been proposed by Jinks and Virk (1977). In the modified analysis, proposals have been made to correct the resulting biases, due to inadequate testers, that is testers with common loci. Jinks and Perkins (1970) have further modified the Triple Test Cross of Kearsey and Jinks (1968). In the modified analysis, all comparisons among the three kinds of progeny means namely \bar{L}_1i (P₁ x F₂), \bar{L}_2i (P₂ x F₂) and \bar{L}_3i (F₁ x F₂), are orthogonal to one another. Moreover, in the absence of epistasis, the \bar{L}_3i means are used to estimate the additive component of variation instead of being discarded. The modified analysis of Jinks and Perkins (1970) have been applied to wheat, and epistasis has been found to be important for all five characters studied (Singh and Singh, 1976).

In the most recent design proposed by Chahal and Jinks (1978), the need to choose adequate testers with extreme genotypes has been completely relaxed. The new design can

accommodate inbreds of any genotypes. It thus removes all the difficulties and limitations of the original, simplified and modified triple test cross (Kearsey and Jinks, 1968; Jinks et al., 1969; Virk and Jinks, 1977; Jinks and Virk, 1977). The New Triple Test Cross Analysis of Chahal and Jinks (1978) is as follows:

2.3.6 *Mating and Field Design* ⁰

A sample of n inbred lines, P_i , and, a single tester line P_c , are chosen from a population of inbred lines. The inbred lines are then individually crossed to P_c to produce n F_{li} families,

i.e. $n F_{li} = P_i \times P_c$ where $i = (1, \dots, n)$.

The n F_{li} families are then backcrossed to each of their parents to produce two series of backcross families, B_{li} ($F_{li} \times P_i$) and B_{ci} ($F_{li} \times P_c$).

The experiment then consists of the following families P_c , $n P_i$, $n F_{li}$, $n B_{li}$ and $n B_{ci}$. There are, therefore, $4n + 1$ families. Each family is replicated with r individuals in a randomised block design. In this study, the five P_i inbred lines are: P_1 - Hilgendorf, P_2 - Kopara, P_3 - Oroua, P_4 - Ruru and P_5 - Atlas 66. The single tester, P_c , is Karamu.

2.3.7 *The Test of Epistasis*

The test of epistasis is based on the standard backcross Scaling Test of Mather and Jinks (1971). The expectations of the scaling tests are summarised by Chahal and Jinks (1978) and a derivation is shown in Table 2.1. In the absence of epistasis

these comparisons have expectation of zero irrespective of the genotype of the inbred line, P_i or tester P_c . The test of epistasis is effected by three methods. The first of which is a 3 n (comprising n sets of A, B, C Scaling Tests) individual Scaling Tests. The second method is the n Joint Scaling Tests or the Cavalli's Scaling Test analysis (Mather and Jinks, 1971; Tan, 1974; Gale, Mather and Jinks, 1977; Rowe and Alexander, 1980). Both these tests are executed with the computer programme Bintest (Appendix IV). A test combining all the individual tests of significance is achieved in the context of the analysis of variance and is effected with the programme Bintri (Appendix III). The theory of this analysis of variance is as follows:

Considering the comparison

$$2 \bar{B}_{li} - \bar{F}_{li} - \bar{P}_i = A_i.$$

The sum of square of A_i is simply

$$\left(\sum_{i=1}^n A_i^2 \right) \text{ for } i = 1 \text{ to } n.$$

This has n degree of freedom and therefore the mean square is

$$\left(\sum_{i=1}^n A_i^2 \right) / n.$$

For the test of significance, this mean square can be tested against the mean square derived from pooling the corresponding variances of A_i . The variance of A_i is obtained thus

$$V A_i = 4 V \bar{B}_{li} + V \bar{F}_{li} + V \bar{P}_i$$

where $V \bar{B}_{li}$, $V \bar{F}_{li}$ and $V \bar{P}_i$ are the variances of the B_{li} , F_{li} and P_i family means.

For the second comparison of

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

\bar{P}_c appears in every set of $i = 1$ to n . So nP_c sets are grown for each replicate so that the nB_i values will be independent. The analysis is exactly as for A_i and is achieved by the Call Statement to the same subroutine (PVAR) of the programme Bintri for the analysis of the A_i comparison.

2.3.8 Tests and Estimates of the Additive and Dominance Components

In the absence of epistasis, two further orthogonal comparisons provide unique tests and estimates for the additive and dominance components of variation. They are

$$\bar{B}_{ci} - \bar{B}_{li} - \bar{P}_c + \bar{P}_i \text{ (Additive) and}$$

$$\bar{B}_{ci} + \bar{B}_{li} - \bar{P}_c - \bar{P}_i \text{ (Dominance).}$$

The additive comparison has the following genotypic value for P_i (AABB) and P_c (aabb)

$$\begin{array}{cccc} \text{da} & \text{db} & \text{jab} & \text{jba} \\ -1 & -1 & -\frac{1}{2} & -\frac{1}{2} \end{array}$$

Therefore, in the absence of epistasis, the comparison has value of $-lda -ldb$. The dominance comparison has genotypic value of

$$\begin{array}{cccc} \text{ha} & \text{hb} & i & l \\ 1 & 1 & \frac{1}{2} & \frac{1}{2} \end{array}$$

Table 2.1 Derivation of the expectations of the epistatic comparisons.

	da	db	ha	hb	i	jab	jba	l
P1 AABB	1	1	-	-	1	-	-	-
Pc aabb	-1	-1	-	-	1	-	-	-
F11 AaBb			1	1				1
ABAB	$\frac{1}{4}$	$\frac{1}{4}$	-	-	$\frac{1}{4}$	-	-	-
abAB	-	-	$\frac{1}{4}$	$\frac{1}{4}$		-	-	$\frac{1}{4}$
AbAB	$\frac{1}{4}$	-	-	$\frac{1}{4}$		$\frac{1}{4}$		
aBAB	-	$\frac{1}{4}$	$\frac{1}{4}$			-	$\frac{1}{4}$	-
B11	$\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{4}$	$\frac{1}{4}$	$\frac{1}{4}$	$\frac{1}{4}$
2 B11	1	1	1	1	$\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{2}$
2 B11 - F11 - P1	-	-	-	-	$-\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{2}$	$-\frac{1}{2}$

Therefore 2B11 - F11 - P1 is $-\frac{1}{2}i + \frac{1}{2}jab + \frac{1}{2}jba - \frac{1}{2}l$

	abAB		$+\frac{1}{4}$	$+\frac{1}{4}$				$\frac{1}{4}$
BC1	abab	$-\frac{1}{4}$	$-\frac{1}{4}$		$\frac{1}{4}$			
	abAb		$-\frac{1}{4}$	$\frac{1}{4}$			$-\frac{1}{4}$	
	abaB	$-\frac{1}{4}$	-	-	$\frac{1}{4}$	$-\frac{1}{4}$		
BC1		$-\frac{1}{2}$	$-\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{2}$	$-\frac{1}{4}$	$-\frac{1}{4}$	$\frac{1}{4}$
2BC1		-1	-1	1	1	$\frac{1}{2}$	$-\frac{1}{2}$	$\frac{1}{2}$
2BC1 - F11 - Pc					$-\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{2}$	$-\frac{1}{2}$

Therefore 2BC1 - F11 - Pc is $-\frac{1}{2}i + \frac{1}{2}jab + \frac{1}{2}jba - \frac{1}{2}l$

where i, j, and l are the epistatic items.

Therefore, in the absence of epistasis, the comparison has value of $lha + lhb$.

The appropriate analysis of variance for testing the significance of these comparisons is carried out by the subroutine, Canova, in the programme Bintri. Estimates of the additive (D) and dominance (H) effects are also made by this programme. An example is illustrated in Section 2.4.2.

2.4 COMPUTATIONAL PROCEDURES

The following computer programmes were written by the author to facilitate the data processing. The programmes were written in Fortran IV for use in the Burroughs B6700 computer.

2.4.1 *The Half and Full Diallel Analyses - Binhalf and Bindial*

The Half Diallel analysis as described by Morley Jones (1965) and the Full Diallel analysis of Hayman (1954a) were translated into Fortran and their complete listings are given in Appendices I and II. These programmes also have the Covariance (Wr) and Variance (Vr) analysis described by Mather and Jinks (1971) and allowed for the perfect fit estimates of the genetic parameters to be made. As the same subroutines for these computations were used in both the Full and Half Diallels, only one set of the subroutines is included in the listings of Appendices I and II.

Data Input

Card 1 Is the title card and can be in alphanumeric

punched in column 1 to 80.

Card 2 For the Half Diallel Programme, Binhalf, this card contains two integer numbers N and IBLK. For the Full Diallel programmes, three integers numbers IM, N and IBLK are read in. The numbers are in format I4. N or IM is the number of parents used in the Diallel crosses. IBLK is the number of replicate block for the experiment.

Card 3 to (N + 3) contains the family means of the Half or Full Diallel table for the first block. This is followed by another N cards for the family means of the second block.

Data Output

The programmes output include:

- (i) The Full or Half Diallel table of the family means for Block 1, Block 2 and the Mean of Blocks 1 and 2. The Diallel analyses of variance for each block and the mean of the two blocks are also presented (pages 26-28, 32, 33).
- (ii) The overall Full or Half Diallel analysis with the accompanying block interaction terms. This analysis also provides test of significance for each item of the Diallel analysis of variance table (pages 29, 34).
- (iii) The joint regression analysis of the W_r - V_r regression (pages 30, 35).
- (iv) The ANOVA for the sum of $W_r + V_r$ and the difference of W_r - V_r (pages 30, 35).
- (v) The perfect fit estimates of the genetic parameters (pages 31, 36).

INPUT VALUES OF HALF DIALLEL

106.7900	114.6200	110.0600	106.8500	99.5300	
	110.6600	112.1400	104.3500	105.0500	
		99.1400	102.4500	90.3200	
			85.8800	83.1000	
				87.1800	
106.790	114.620	110.060	106.850	99.530	
114.620	110.660	112.140	104.350	105.050	
110.060	112.140	99.140	102.450	90.320	
106.850	104.350	102.450	85.880	83.100	
99.530	105.050	90.320	83.100	87.180	
SUM OF ROW	537.8500	546.8200	514.1100	482.6300	465.1800
SUM OF COLUMN	537.8500	546.8200	514.1100	482.6300	465.1800
SUM OF ROW+PARENT	644.6400	657.4800	613.2500	568.5100	552.3600
SUM OF TR	541.7500	540.3400	532.5200	535.8600	494.4600

1518.1200	489.6500	155087.4170
SUM OF SQUARE	DF	MEAN SQUARE

1210.1092	4	302.5273
80.5896	1	80.5896
73.5057	4	18.3764
77.3235	5	15.4647
1441.5280	14	

INPUT VALUES OF HALF DIALLEL

105.2800	115.4200	110.8700	106.1100	101.7400
	111.0400	104.8100	103.2700	106.4800
		97.9600	98.3900	94.2700
			88.1000	90.9000

				88.3100
105.280	115.420	110.870	106.110	101.740
115.420	111.040	104.810	103.270	106.480
110.870	104.810	97.960	98.390	94.270
106.110	103.270	98.390	88.100	90.900
101.740	106.480	94.270	90.900	88.310

SUM OF ROW	539.4200	541.0200	506.3000	486.7700	481.7000
SUM OF COLUMN	539.4200	541.0200	506.3000	486.7700	481.7000
SUM OF ROW+PARENT	644.7000	652.0600	604.2600	574.8700	570.0100
SUM OF TR	552.4400	526.8400	522.8000	533.0400	521.8500

1522.9500	490.6900	155612.5351
SUM OF SQUARE	DF	MEAN SQUARE

833.7195	4	208.4299
86.2925	1	86.2925
30.0632	4	7.5158
37.3465	5	7.4693
987.4216	14	

THE MEAN VALUES OF THE HALF DIALLED INPUT

106.0350	115.0200	110.4650	106.4800	100.6350	
	110.8500	108.4750	103.8100	105.7650	
		98.5500	100.4200	92.2950	
			86.9900	87.0000	
				87.7450	
106.035	115.020	110.465	106.480	100.635	
115.020	110.850	108.475	103.810	105.765	
110.465	108.475	98.550	100.420	92.295	
106.480	103.810	100.420	86.990	87.000	
100.635	105.765	92.295	87.000	87.745	
SUM OF ROW	538.6350	543.9200	510.2050	484.7000	473.4400
SUM OF COLUMN	538.6350	543.9200	510.2050	484.7000	473.4400
SUM OF ROW+PARENT	644.6700	654.7700	608.7550	571.6900	561.1850
SUM OF TR	547.0950	533.5900	527.6600	534.4500	508.1550
1520.5350	490.1700	155308.3220			
SUM OF SQUARE		DF	MEAN SQUARE		
1006.0747		4	251.5187		
83.4167		1	83.4167		
38.4490		4	9.6122		
45.2691		5	9.0538		
1173.2096		14			

SOURCE OF VARIATION	DF	MEAN SQUARE	VRA	PROB	VRT	PROB
A	4	503.0374	63.5164	0.0007	85.3324	0.0000
B1	1	166.8334	3423.2738	0.0109	28.3006	0.0001
B2	4	19.2245	2.8832	0.1548	3.2611	0.0436
B3	5	18.1077	3.7518	0.0865	3.0717	0.0415
B	10	33.4270	6.5735	0.0032	5.6704	0.0019
THE BLOCK INTERACTION MEAN SQUARE						

	DF	INTERACTION MEANSQUARE
BA	4	7.9198
BD1	1	0.0487
BD2	4	6.6677
BD3	5	4.8263
BB	10	5.0851
BT	14	5.8950
THE HOMOGENEITY OF VARIANCE BY BARTLETT IS		0.3221

THE Y INTERCEPT IS 121.5775

ITEM	MS	DF	VR	PROB
JOINT REGRESSION	5211.3910	1	178.4081	0.0000
HETEROGENEITY OF REGRESSION	105.8984	1		
REMAINDER	29.3787	6		

REGRESSION COEFFICIENT IS 0.8012 SE 0.0600 SIGNIFICANT FROM 1.0 0.4480 SIGNIFICANT FROM 0.0 0.0170

CORRELATION COEF. OF WRVR AND PI IS -0.9805 SE 0.1135 IT IS 0.0033

THE ANALYSIS OF VARIANCE OF WRVR

SOURCE OF VARIATION	DEGREES OF FREEDOM	SUM OF SQUARES	MEAN SQUARE	F	PROBABILITY
REPLICATION	1	30.3050	30.3050	1.3585	0.3361
TREATMENT	4	348.3911	87.0978		
RESIDUALS	4	256.4564	64.1141		

SOURCE OF VARIATION	DEGREES OF FREEDOM	SUM OF SQUARES	MEAN SQUARE	F	PROBABILITY
REPLICATION	1	4941.0939	4941.0939	6.0546	0.054
TREATMENT	4	22977.3553	5744.3388		
RESIDUALS	4	3795.0192	948.7548		

ESTIMATES OF COMPONENTS OF VARIATION

STATISTICS	BLK1	BLK2	MEAN	MODEL
VP	125.6954	103.7131	114.7043	D+E
VEN	66.7516	42.7822	54.7669	$1/4D+1/4H1-1/4F+5/9E$
WRH	76.6033	56.1156	66.3594	$1/2D-1/4F+1/9E$
W	49.0385	31.7656	40.4021	$1/4D+1/4H1-1/4H2-1/4F+5/81E$
E	5.8950	5.8950	5.8950	E

PERFECT FIT ESTIMATES OF THE COMPONENTS

COMPONENT	BLK1	BLK2	MEAN
D	119.8004	97.8181	108.8092
H1	70.9616	35.0527	53.0071
H2	70.8525	44.0665	57.4595
F	-62.0965	-24.1101	-43.1033
E	5.8950	5.8950	5.8950
SRH1/D	0.7696	0.5986	0.6641
UV	0.2496	0.3143	0.2820
HEPAR	0.7940	0.7695	0.7818
HERBD	0.9486	0.9197	0.9341

FULL DIALED ANALYSIS OF PROTEIN CONTENT OF PLANTING ONE

THE DIALED ANALYSIS FOR BLOCK 1

15.4600	14.0300	15.8700	13.2000	
14.0600	13.4500	14.7400	11.9600	
16.7100	15.3700	13.7900	14.4700	
13.2500	12.1200	14.2300	12.3600	
SUM OF MALE ROW=		58.5600	54.2100	60.3700
SUM OF FEMALE COLUMN=		59.5100	54.3700	59.6300
SUM OF MALE ROW FEMALE COLUMN=		118.0700	109.1000	119.0000
DIFF OF MALE ROW FEMALE COLUMN		-0.9500	-0.7600	1.7400
PARENTAL DEVIATION=		56.2300	55.3800	63.8400
	-0.0300	-0.8700	-0.0500	
		-0.6300	-0.1600	
			0.2400	
	SUM OF SQUARE		DF	MEAN SQUARE
A	19.6737	3		6.5579
B	7.4041	6		1.2340
C	0.5636	3		0.1879
D	0.0566	3		0.0189
B1	0.4921	1		0.4921
B2	6.9053	3		2.3018
B3	0.0067	2		0.0033
T	27.6980	15		1.8465

THE DIALED ANALYSIS FOR BLOCK 2

15.8000	14.5900	15.6500	12.2700	
14.0600	14.6200	14.3700	13.0200	
16.4000	14.2200	15.4700	13.0500	
12.6900	11.8100	13.4400	11.2700	
SUM OF MALE ROW=		59.3100	56.0700	59.1400
SUM OF FEMALE COLUMN=		58.9500	55.2400	58.9100
SUM OF MALE ROW FEMALE COLUMN=		117.2600	111.3100	118.0700
DIFF OF MALE ROW FEMALE COLUMN		-0.0400	0.8300	0.2100
PARENTAL DEVIATION=		54.0600	52.8300	56.1900
	0.5300	-0.7500	-0.4200	
		0.1500	1.2100	
			-0.3900	
	SUM OF SQUARE		DF	MEAN SQUARE
A	29.6360	3		9.8787
B	2.8732	6		0.4789
C	0.1628	3		0.0543
D	1.1664	3		0.3888
B1	0.7277	1		0.7277
B2	0.7565	3		0.2528
B3	1.3870	2		0.6935
T	33.8385	15		2.2559

THE DIALLIED ANALYSIS FOR BLOCK 1+2			
15.6300	14.3100	15.7600	12.7350
14.0600	14.0350	14.5550	12.4900
16.5700	14.7950	14.6300	13.7600
12.9700	11.9650	13.8350	11.8150
SUM OF MALE ROW=	58.4350	55.1400	52.7550
SUM OF FEMALE COLUMN=	59.2300	55.1050	58.7800
SUM OF MALE ROW FEMALE COLUMN=	117.6650	110.2450	118.5350
DIFF OF MALE ROW FEMALE COLUMN	-0.7950	0.0350	0.9750
PARENTAL DEVIATION=	55.1450	54.1050	60.0150
	0.2500	-0.8100	-0.2350
		-0.2400	0.5250
		-0.0750	
	SUM OF SQUARE	DF	MEAN SQUARE
A	23.8187	3	7.9396
B	3.3633	6	0.5606
C	0.2038	3	0.0679
D	0.3526	3	0.1175
B1	0.0057	1	0.0057
B2	2.9831	3	0.9944
B3	0.3745	2	0.1872
T	27.7383	15	1.8492

CORRECTED VALUES FOR DIALLEL ANALYSIS OF BLOCK 1+2

B	1.1211	6
A	15.8791	3
C	0.1358	3
D	0.2350	3
B1	0.0115	1
B2	1.9887	3
B3	0.3745	2
T	3.6984	15

THE BLOCK INTERACTION MEAN SQUARES

BB	0.5918	6
BA	0.5575	3
BC	0.1063	3
BD	0.1726	3
BB1	1.2083	1
BB2	0.5659	3
BB3	0.3224	2
BI	0.4040	15

BARTLETT TEST IS 1.0000

VRA	PROB	VRT	PROB
1.8945	0.2282	2.7751	0.0509
28.4850	0.0105	39.3059	0.0000
1.2780	0.4225	0.3363	0.7994
1.3616	0.4029	0.5818	0.6360
0.0095	0.9381	0.0284	0.8684
3.5145	0.1647	4.9228	0.0141

THE Y INTERCEPT IS 10.6249

ITEM	MS	DF	VR	PROB
JOINT REGRESSION	1.8008	1	69.0801	0.0011
HETEROGENEITY OF REGRESSION	0.0292	1		
REMAINDER	0.0261	4		

JOINT REGRESSION COEFFICIENT IS 0.8066 SE 0.0971 SIGNIFANCE FROM 1.0 0.1172 SIGNIFICANCE FROM 0.0 0.001
 CORRELATION COEF. OF WR+VR AND PI IS 0.8586SE 0.3625IT IS 0.1414

THE ANALYSIS OF VARIANCE OF WRVR

SOURCE OF VARIATION	DEGREES OF FREEDOM	SUM OF SQUARES	MEAN SQUARE	F	PROBABILI
REPLICATION	1	3.3623	3.3623	2.5054	0.235
TREATMENT	3	0.1694	0.0565		
RESIDUALS	3	0.0676	0.0225		

SOURCE OF VARIATION	DEGREES OF FREEDOM	SUM OF SQUARES	MEAN SQUARE	F	PROBABILI
REPLICATION	1	3.7628	3.7628	3.6992	0.155
TREATMENT	3	7.2160	2.4053		
RESIDUALS	3	1.9507	0.6502		

ESTIMATES OF COMPONENTS OF VARIATION

STATISTICS	BLK1	BLK2	MEAN	MODEL
VP	1.6490	4.3006	2.9748	D+E
VPB	1.4367	1.4743	1.4555	1/4D+1/4H1-1/4F+5/9E
WRH	0.9443	2.2784	1.6113	1/2D-1/4F+1/9E
4	0.8197	1.2348	1.0273	1/4D+1/4H1-1/4H2-1/4F +5/81E
E	0.4040	0.4040	0.4040	E

PERFECT FIT ESTIMATES OF THE COMPONENTS

COMPONENT	BLK1	BLK2	MEAN
D	1.2450	3.8966	2.5708
H1	2.6089	0.0742	1.3416
H2	1.7105	0.2003	0.9554
F	-0.8831	-0.9163	-0.8997
E	0.4040	0.4040	0.4040
SRH1/D	1.4476	0.1380	0.7928
UV	0.1639	0.6748	0.4194
BERAR	0.5453	0.8377	0.7415
BERBD	0.8277	0.8556	0.8417

2.4.2 *The New Triple Test Cross Analysis - Bintri*

This programme is based on the paper entitled "A General Method of Detecting the Additive, Dominance and Epistatic Variation that Inbred Lines can Generate Using a Single Tester" (Chahal and Jinks, 1978). This programme enables an unambiguous test for the presence of epistasis to be made. It can also test for the significance of both the additive and dominance effects and in the absence of epistasis allowed unbiased estimates of the additive and dominance effects. The complete listing of this programme is given in Appendix III.

Data Input

Card 1 Contains three integer numbers, IBLK, IF, IS in format 3I4;
 IBLK is number of replicate blocks,
 IF is number of parents (P_i),
 IS is sample size for each family in each block.

This programme uses equal sample sizes for all the P_1 , P_2 , F_1 , BC_1 and BC_2 families. It can handle four replicate blocks, 20 parental families ($P_i = 20$) and the sample size of 50 plants per family per replicate block.

Card 2 The subsequent cards are raw data cards.
 Data from replicate Block 1 are read first.
 The data in each block are arranged in the following order:
 Data from parent 1 ($P_i: i = 1$), P_c the common tester, the backcrosses (BC_1 and BC_2) and F_1 generated from these two parents. When all

the data from Parent 1 and Pc and their derived families have been read, the data for Parent 2, Pc and crosses derived from them are read in the same order as those of Parent 1. This process is repeated until all the Parents and their families (in this study IF = 5) have been exhausted.

Data from the families of the second replicate block are read next until all the replicate blocks (in this study IBLK = 3) have been exhausted.

Data Output

The programme output is as follows:

- (i) Printout of raw data for each block, parent and families. A representative section of this print out is shown in page 39).
- (ii) The mean values for the parents, the common tester Pc, their backcrosses (BC1 and BC2) and the F1 are printed out. On the same print out, are the four comparisons for each of the parental groups (in this study IF = 5). The comparisons are the two epistatic comparisons (Ai and Bi) and the additive and dominance comparisons. These values are calculated on a replicate block basis and the values for each replicate block are printed out separately (pages 40-42).
- (iii) The analyses of variance (ANOVA) for each of the generation, for all the parents are printed out next. The analysis groups all the families from the same generation to extract the variance of the particular generation. The print out shown in page 43 illustrates the ANOVA for all the

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*
*   THE NEW TRIPLE TEST CROSS ANALYSIS
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INPUT VALUE OF RAW DATA

BLOCK 1

PARENT PI 1

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*****
PI
101.9 108.7 104.5 107.9 106.3 114.8 103.6 107.5 105.0 105.8 111.2 108.3 111.4 105.6 107.9
PC
86.3 89.4 87.9 98.9 89.2 89.7 89.5 89.3 90.2 92.3 92.2 90.7 89.6 87.2 88.3
B1I
95.3 99.6 97.5 100.4 106.5 101.2 109.2 95.2 93.5 116.6 120.9 114.6 112.3 95.5 91.6
BCI
98.5 84.4 111.2 88.0 104.2 85.9 95.6 97.2 110.6 113.4 107.5 95.2 93.7 100.3 109.4
F1I
104.1 109.2 108.2 107.7 106.9 109.3 107.7 107.1 105.2 103.4 104.5 112.6 108.6 108.7 108.4

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PARENT PI 2

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*****
PI
110.3 106.5 105.4 114.4 113.5 107.8 113.7 109.2 109.9 105.2 109.9 104.7 99.6 95.9 112.6
PC
90.1 87.4 86.0 87.4 87.9 90.0 86.0 88.5 92.4 85.5 98.4 97.2 91.2 83.7 89.1
B1I
106.6 103.5 101.2 104.2 93.2 118.4 106.5 104.0 109.2 116.5 98.9 110.4 115.6 103.8 91.6
BCI
113.9 89.3 85.7 82.1 110.2 111.2 90.8 94.5 88.7 122.3 97.8 119.3 92.0 117.8 117.9
F1I
112.2 111.9 112.4 111.9 113.4 112.1 111.7 110.0 113.5 114.8 113.9 115.3 115.4 112.0 107.1

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PARENT PI 3

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*****
PI
95.2 93.6 104.0 91.7 111.2 106.5 102.4 104.0 102.2 99.6 99.4 98.5 104.5 101.8 92.0
PC
87.7 89.2 87.4 99.3 84.7 90.0 97.7 92.0 90.6 87.3 89.8 92.7 88.9 88.1 83.6

```

BLOCK 1

THE MEAN VALUE FOR PARENTS PI

107.3600 107.9067 100.4400 86.6000 143.3200

THE MEAN VALUE FOR THE PARENT PC

90.0467 89.3867 90.2667 88.8933 90.5067

THE MEAN VALUES FOR B11

103.3267 105.5733 94.3200 94.1867 141.5533

THE MEAN VALUES FOR BCI

99.6733 102.2333 91.6867 93.9333 108.6600

THE MEAN VALUES FOR F11

107.4400 112.5067 99.9000 94.6400 130.2667

THE VALUE FOR THE EPISTASIS CONTRAST AI

-8.1467 -9.2667 -11.7000 7.1333 9.5200

THE VALUE FOR THE EPISTASIS CONTRAST B1

1.8600 2.5733 -6.7933 4.3333 -3.4533

THE VALUE FOR THE THE ADDITIVE CONTRAST

13.6600 15.1800 7.5400 -2.5467 19.9200

THE VALUE FOR THE THE DOMINANCE CONTRAST

5.5933 10.5133 -4.7000 12.6267 16.3867

BLOCK 2

THE MEAN VALUE FOR PARENTS PI

105.2667 107.4133 100.7933 86.7067 142.8000

THE MEAN VALUE FOR THE PARENT PC

89.3867 90.2667 88.8933 88.7867 90.5067

THE MEAN VALUES FOR B1I

103.9867 107.8400 94.9467 95.8933 138.1200

THE MEAN VALUES FOR BCI

97.0800 101.5533 94.4467 90.7267 109.6200

THE MEAN VALUES FOR F1I

105.5400 112.1867 99.2400 94.8667 128.7000

THE VALUE FOR THE EPISTASIS CONTRAST AI

-2.8333 -3.9200 -10.1400 10.2133 4.7400

THE VALUE FOR THE EPISTASIS CONTRAST BI

-0.7667 0.6533 0.7600 -2.2000 0.0333

THE VALUE FOR THE THE ADDITIVE CONTRAST

8.9733 10.8600 11.4000 -7.2467 23.7933

THE VALUE FOR THE THE DOMINANCE CONTRAST

6.4133 11.7133 -0.2933 11.1267 14.4333

BLOCK 3

THE MEAN VALUE FOR PARENTS PI

107.2800 107.3467 100.7333 88.1733 145.1800

THE MEAN VALUE FOR THE PARENT PC

90.0467 89.9733 87.8600 89.0000 87.4467

THE MEAN VALUES FOR B1I

104.7733 104.4933 98.9000 89.6467 136.4667

THE MEAN VALUES FOR BCI

103.7333 97.2467 93.7800 90.0133 116.4467

THE MEAN VALUES FOR F1I

103.7667 110.4000 98.8400 92.6200 128.7667

THE VALUE FOR THE EPISTASIS CONTRAST AI

-1.5000 -8.7600 -1.7733 -1.5000 -1.0133

THE VALUE FOR THE EPISTASIS CONTRAST BI

13.6533 -5.8800 0.8600 -1.5933 16.6800

THE VALUE FOR THE THE ADDITIVE CONTRAST

16.1933 10.1267 7.7533 -0.4600 37.7133

THE VALUE FOR THE THE DOMINANCE CONTRAST

11.1800 4.4200 4.0867 2.4867 20.2867

THE ANALYSIS OF VARIANCE FOR B11 FAMILY

SOURCE OF VARIATION	DEGREES OF FREEDOM	SUM OF SQUARES	MEAN SQUARE	F	PROB
REPLICATION	2	67.5759	33.7879	161.9018	.0000
FAMILY	4	59529.2776	14882.3194		
SAMPLING ERROR	210	29910.7108	142.4320		
EXPERIMENTAL ERROR	8	735.3752	91.9219		

THE SUM OF SAMPLE FOR EACH FAMILY

1549.9000 1583.6000 1414.8000 1412.8000 2123.3000

THE MEAN OF EACH FAMILY

103.3267 105.5733 94.3200 94.1867 141.5533

THE SUM OF SAMPLE FOR EACH FAMILY

1559.8000 1617.6000 1424.2000 1438.4000 2071.8000

THE MEAN OF EACH FAMILY

103.9867 107.8400 94.9467 95.8933 138.1200

THE SUM OF SAMPLE FOR EACH FAMILY

1571.6000 1567.4000 1483.5000 1344.7000 2047.0000

THE MEAN OF EACH FAMILY

104.7733 104.4933 98.9000 89.6467 136.4667

THE ANALYSIS OF VARIANCE FOR BCI FAMILY

SOURCE OF VARIATION	DEGREES OF FREEDOM	SUM OF SQUARES	MEAN SQUARE	F	PROB
REPLICATION	2	93.6881	46.8441	18.6767	.0004
FAMILY	4	11176.7734	2794.1934		
SAMPLING ERROR	210	35163.6107	167.4458		
EXPERIMENTAL ERROR	8	1196.8701	149.6088		

THE SUM OF SAMPLE FOR EACH FAMILY

1495.1000	1533.5000	1375.3000	1409.0000	1629.9000
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THE MEAN OF EACH FAMILY

99.6733	102.2333	91.6867	93.9333	108.6600
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THE SUM OF SAMPLE FOR EACH FAMILY

1456.2000	1523.3000	1416.7000	1360.9000	1644.3000
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THE MEAN OF EACH FAMILY

97.0800	101.5533	94.4467	90.7267	109.6200
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THE SUM OF SAMPLE FOR EACH FAMILY

1556.0000	1458.7000	1406.7000	1350.2000	1746.7000
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THE MEAN OF EACH FAMILY

103.7333	97.2467	93.7800	90.0133	116.4467
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THE ANALYSIS OF VARIANCE FOR PC FAMILY

SOURCE OF VARIATION	DEGREES OF FREEDOM	SUM OF SQUARES	MEAN SQUARE	F	PROB
REPLICATION	2	36.7159	18.3580	0.6671	.6325
FAMILY	4	37.1499	9.2875		
SAMPLING ERROR	210	3072.3534	14.6303		
EXPERIMENTAL ERROR	8	111.3698	13.9212		

THE SUM OF SAMPLE FOR EACH FAMILY

1350.7000	1340.8000	1354.0000	1333.4000	1357.6000
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THE MEAN OF EACH FAMILY

90.0467	89.3867	90.2667	88.8933	90.5067
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THE SUM OF SAMPLE FOR EACH FAMILY

1340.8000	1354.0000	1333.4000	1331.8000	1357.6000
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THE MEAN OF EACH FAMILY

89.3867	90.2667	88.8933	88.7867	90.5067
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THE SUM OF SAMPLE FOR EACH FAMILY

1350.7000	1349.6000	1317.9000	1335.0000	1311.7000
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THE MEAN OF EACH FAMILY

90.0467	89.9733	87.8600	89.0000	87.4467
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THE ANALYSIS FOR P1 FAMILY

SOURCE OF VARIATION	DEGREES OF FREEDOM	SUM OF SQUARES	MEAN SQUARE	F	PROB
REPLICATION	2	49.4035	24.7017	2373.4794	.0000
FAMILY	4	79330.2243	19832.5561		
SAMPLING ERROR	210	7147.6227	34.0363		
EXPERIMENTAL ERROR	8	66.8472	8.3559		

THE SUM OF SAMPLE FOR EACH FAMILY

1610.4000 1618.6000 1506.6000 1299.0000 2149.8000

THE MEAN OF EACH FAMILY

107.3600 107.9067 100.4400 86.6000 143.3200

THE SUM OF SAMPLE FOR EACH FAMILY

1579.0000 1611.2000 1511.9000 1300.6000 2142.0000

THE MEAN OF EACH FAMILY

105.2667 107.4133 100.7933 86.7067 142.8000

THE SUM OF SAMPLE FOR EACH FAMILY

1609.2000 1610.2000 1511.0000 1322.6000 2177.7000

THE MEAN OF EACH FAMILY

107.2800 107.3467 100.7333 88.1733 145.1800

THE ANALYSIS OF VARIANCE FOR F1I FAMILY

SOURCE OF VARIATION	DEGREES OF FREEDOM	SUM OF SQUARES	MEAN SQUARE	F	PROB
REPLICATION	2	162.8376	81.4188	1210.1021	.0000
FAMILY	4	33340.0333	8335.0083		
SAMPLING ERROR	210	2141.8440	10.1993		
EXPERIMENTAL ERROR	8	55.1028	6.8879		

THE SUM OF SAMPLE FOR EACH FAMILY

1611.6000	1687.6000	1498.5000	1419.6000	1954.0000
-----------	-----------	-----------	-----------	-----------

THE MEAN OF EACH FAMILY

107.4400	112.5067	99.9000	94.6400	130.2667
----------	----------	---------	---------	----------

THE SUM OF SAMPLE FOR EACH FAMILY

1583.1000	1682.8000	1488.6000	1423.0000	1930.5000
-----------	-----------	-----------	-----------	-----------

THE MEAN OF EACH FAMILY

105.5400	112.1867	99.2400	94.8667	128.7000
----------	----------	---------	---------	----------

THE SUM OF SAMPLE FOR EACH FAMILY

1556.5000	1656.0000	1482.6000	1389.3000	1931.5000
-----------	-----------	-----------	-----------	-----------

THE MEAN OF EACH FAMILY

103.7667	110.4000	98.8400	92.6200	128.7667
----------	----------	---------	---------	----------

THE MEAN SQUARED DEVIATION FROM ZERO

THE BLOCK SUM OF THE CONTRAST

THE BLOCK SUM SQUARED OF THE CONTRAST 14.7467 -2.6533 -5.1733 0.5400 13.2600

THE VALUE OF SUM OF EPISTSTATIC CONTRAST IS 217.4642 7.0402 26.7634 0.2916 175.8276

THE VALUE OF THE CONTRAST MEAN IS 9.4975

THE BLOCK SUM OF THE CONTRAST

THE BLOCK SUM SQUARED OF THE CONTRAST -12.4800 -21.9467 -23.6133 15.8467 13.2467

THE VALUE OF SUM OF EPISTSTATIC CONTRAST IS 155.7504 481.6562 557.5895 251.1168 175.4742

THE VALUE OF THE CONTRAST MEAN IS 36.0353

THE POOLED VARIANCE OF AI CONTRAST IS 13.6436

THE POOLED VARIANCE OF BI CONTRAST IS 15.4358

THE F RATIO FOR AI CONTRAST IS 2.6412PROB.0244
THE F RATIO FOR THE BI CONTRAST IS 0.6153PROB.6883

THE ANALYSIS OF VARIANCE FOR THE ADDITIVE

444.6223

SOURCE OF VARIATION	DF	SS	MS	F	PROB
MEAN	1	498.0097	498.0097	83.6441	0.0000
CTRAST	5	855.2921	171.0584	28.7304	0.0001
DEV	4	357.2824	89.3206	15.0020	0.0009
BLOCK	2	14.9825	7.4912	1.2582	0.3349
ERROR	8	47.6313	5.9539	*****	*****
TOTAL	15	917.9059	*****	*****	*****

THE ADDITIVE VALUE IS 444.6223

THE TOTAL ADDITIVE VALUE IS 1100.6966

THE AVERAGE ADDITIVE VALUE IS 220.1393

THE ANALYSIS OF VARIANCE FOR DOMINANCE

123.7471

SOURCE OF VARIATION	DF	SS	MS	F	PROB
MEAN	1	265.7492	265.7492	51.8108	0.0001
CTRAST	5	379.0764	75.8153	14.7810	0.0007
DEV	4	113.3272	28.3318	5.5236	0.0197
BLOCK	2	0.2312	0.1156	0.0225	0.9778
ERROR	8	41.0338	5.1292	*****	*****
TOTAL	15	420.3415	*****	*****	*****

THE DOMINANCEVALUE IS 123.7471

THE TOTAL DOMINANCE VALUE IS 471.2404

THE AVERAGE DOMINANCE VALUE IS 94.2481

backcross (BCli) families. This ANOVA is repeated for all the five generations (Pi, Pc, BCl_i, BC2_i and Fli) to extract their respective family variances (pages 43-47).

(iv) The tests of significance for epistatic comparisons (Ai and Bi) are shown next. The mean squares of each of the Ai and Bi are derived and tested against their respective pooled variances. Their significances are indicated by the probability of the F tests. (Page 48).

(v) The analysis of variance for testing the significance of both the dominance and additive components are presented next and their corresponding estimates made. These estimates are unbiased when the test of epistasis is not significant. (Page 49).

2.4.3 *The Scaling Test Analysis - BINTEST*

The Scaling Test analysis as described by Mather and Jinks (1971) was translated into Fortran and the complete Fortran listing is given in Appendix IV. Included in this programme are the A, B, C Scaling Tests, the S (6) Scaling Test used by Law (1977), and the Cavalli's Joint Scaling Test. The six parameter model for the estimates of the m d, h, i, j and l effects is also included.

Data Input

Card 1	Contains five integer values, IBLK, IS, IQ, IR, N, in format 5I4.
	IBLK - is number of replicate blocks,
	IS - sample size per replicate block of P1, P2 and F1 families,
	IQ - sample size per replicate block of BCl and BC2 families,

IR - sample size per replicate block of F2 family,

N - is the number three and is the size of the matrix to be inverted.

This programme can handle five replicate blocks and 50 observations for each trait per family per block. IS, IQ and IR can assume any interger values below 50. This allows different sample size for the non-segregating and segregating families.

Card 2 Is the title card. Alphanumeric title can be used and can be punched in column 1 - 80.

Cards 3-8 The next six cards contain the coefficients of the six equations for the least square solution of m , (d) and (h) .
The coefficients are in the format (3F4.2).

Cards 9 - Card 9 onwards are raw data cards read in the following order - Replicate Block 1 is read first. The families within each block are read in the following order P1, P2, BC1, BC2, F1 and F2. All the observations within each family for each trait are punched in the horizontal format of F 8.4. When all the data are exhausted, the next replicate block is read until all the replicate blocks are exhausted.

Data Output

The programme output includes:

- (i) Print out of the raw data for each family in each block.

SCALING TEST ON PLANT HEIGHT IN ATLAS 66

BLOCK 1

THE INPUT VALUES FOR GENERATION

145.9	148.1	147.5	140.7	127.8	147.6	146.4	148.9	149.5	125.4	125.0	149.3	151.0	148.6	148.1
148.7	139.3	125.5	143.4	137.5	134.0	141.6	142.6	140.0	134.4	160.5	142.1	138.6	151.2	143.9
124.2	126.4	128.9	126.4	127.1	127.2	129.1	128.8	131.5	136.5	129.4	139.5	130.8	132.5	135.7
110.7	103.4	99.7	122.8	96.8	102.5	136.0	121.2	135.1	133.4	142.7	104.8	125.2	122.5	74.8
117.3	100.7	115.7	109.7	122.4	101.2	103.5	128.5	126.8	86.0	100.2	126.7	95.3	90.1	105.8
85.5	83.5	91.6	94.2	86.2	95.4	89.1	93.2	88.5	99.2	91.5	93.5	89.7	90.0	86.5

BLOCK 2

THE INPUT VALUES FOR GENERATION

137.2	100.8	146.9	145.6	143.0	149.3	144.9	143.4	148.2	150.3	142.0	149.6	146.7	145.8	148.3
139.6	139.2	143.8	143.5	138.4	123.0	146.6	146.8	121.1	124.5	137.4	142.0	156.2	133.9	135.8
129.1	127.7	135.2	124.5	132.1	126.8	127.3	130.5	125.1	131.7	131.6	113.2	134.5	132.7	128.5
120.3	119.9	117.2	96.0	111.5	123.1	131.6	135.0	87.7	112.6	119.9	141.8	105.7	125.7	124.7
112.7	104.2	106.5	105.7	104.8	120.7	87.7	119.5	116.2	113.5	120.8	97.5	110.7	101.4	122.4
85.5	83.5	91.6	94.2	86.2	95.4	89.1	93.2	88.5	99.2	91.5	93.5	89.7	90.0	86.5

BLOCK 3

THE INPUT VALUES FOR GENERATION

138.5	139.8	143.7	139.1	147.4	148.9	148.7	145.1	142.7	147.6	150.2	146.9	142.8	145.5	150.8
133.5	132.7	138.6	138.4	136.0	135.3	142.4	126.7	148.2	148.0	124.2	134.3	133.7	127.0	148.0
120.0	120.5	126.2	129.0	128.8	128.1	130.0	127.9	130.0	131.4	126.7	129.2	132.1	133.2	138.4
109.3	123.0	72.6	129.0	99.7	137.5	111.6	125.9	116.5	122.1	109.5	119.0	119.3	147.2	129.0
114.6	98.4	99.5	123.8	111.3	118.7	131.2	106.7	124.2	123.3	97.2	118.9	128.1	128.6	122.2
87.4	85.7	93.3	94.4	82.6	82.7	84.8	86.3	86.0	90.9	91.4	89.7	84.2	85.1	87.2

P1 MEAN

P2 MEAN

F1 MEAN

B1 MEAN

B2 MEAN

F2 MEAN

143.7667

89.4867

129.2444

138.7133

111.5756

117.2333

THE ANALYSIS OF VARIANCE FOR THE P1 GENERATION

SOURCE OF VARIATION	DF	SS	MS	F	PROB
TOTAL	44	3516.2600	79.9150	*****	*****
BLOCK	2	46.9720	23.4860	0.2843	.7540
ERROR	42	3469.2880	82.6021	*****	*****

THE ANALYSIS OF VARIANCE FOR THE B1 GENERATION

SOURCE OF VARIATION	DF	SS	MS	F	PROB
TOTAL	44	3214.9120	73.0662	*****	*****
BLOCK	2	201.9773	100.9887	1.4078	.2560
ERROR	42	3012.9347	71.7365	*****	*****

THE ANALYSIS OF VARIANCE FOR THE F1 GENERATION

SOURCE OF VARIATION	DF	SS	MS	F	PROB
TOTAL	44	968.9911	22.0225	*****	*****
BLOCK	2	23.5445	11.7722	0.5230	.5966
ERROR	42	945.4467	22.5106	*****	*****

THE ANALYSIS OF VARIANCE FOR THE F2 GENERATION

SOURCE OF VARIATION	DF	SS	MS	F	PROB
TOTAL	44	11851.6200	269.3550	*****	*****
BLOCK	2	72.4360	36.2180	0.1291	.8792
ERROR	42	11779.1840	280.4568	*****	*****

THE ANALYSIS OF VARIANCE FOR THE B2 GENERATION

SOURCE OF VARIATION	DF	SS	MS	F	PROB
TOTAL	44	6329.9031	143.8614	*****	*****
BLOCK	2	540.7858	270.3929	1.9617	.1533
ERROR	42	5789.1173	137.8361	*****	*****

THE ANALYSIS OF VARIANCE FOR THE P2 GENERATION

SOURCE OF VARIATION	DF	SS	MS	F	PROB
TOTAL	44	778.9320	17.7030	*****	*****
BLOCK	2	93.6360	46.8180	2.8694	.0679
ERROR	42	685.2960	16.3160	*****	*****

THE SCALING TEST ANALYSIS ON THE GENERATION

SCALING TEST	VALUE	STANDARD ERROR	T	SIGNIFICANCE
A	4.4156	2.9517	1.4959	.1372
B	4.4200	3.6215	1.2205	.2246
C	-22.8089	10.1940	-2.2375	.0266
D	27.1400	0.7413	36.6106	.0000
H	84.7422	23.9160	3.5433	.0005
I	31.6444	10.8787	2.9088	.0043
J	-0.0044	4.5636	-0.0010	.9992
L	-40.4800	13.3578	-3.0304	.0027
M	84.9822	10.9040	7.7937	.0000
S	-7.9111	2.7197	-2.9088	.0043

THE CAVALLI SCALING TEST ANALYSIS

THE WEIGHTS-1/RECIPROCAL OF GENERATION MEAN

0.5448	0.6273	1.9991	0.1605	0.3265	2.7579
--------	--------	--------	--------	--------	--------

THE WEIGHTED MEANS

78.3213	87.0142	258.3668	18.8104	36.4266	246.7982
---------	---------	----------	---------	---------	----------

THE PRODUCT OF WT. AND COEF. OF EQUATION

0.5448	0.5448	0.0000	0.6273	0.3136	0.3136
1.9991	-0.0000	1.9991	0.1605	0.0000	0.0802
0.3265	-0.1632	0.1632	2.7579	-2.7579	0.0000

THE WT.COEF.*COEF. OF M

0.5448	0.5448	0.0000
0.6273	0.3136	0.3136
1.9991	0.0000	1.9991
0.1605	0.0000	0.0802
0.3265	-0.1632	0.1632
2.7579	-2.7579	0.0000

THE WT. COEF.*COEF OF D

0.5448	0.5448	0.0000
0.3136	0.1568	0.1568
0.0000	0.0000	0.0000
0.0000	0.0000	0.0000
-0.1632	0.0816	-0.0816
-2.7579	2.7579	0.0000

THE WT. COEF.*COEF OF H

0.0000	0.0000	0.0000
0.3136	0.1568	0.1568
1.9991	0.0000	1.9991
0.0802	0.0000	0.0401
0.1632	-0.0816	0.0816
0.0000	0.0000	0.0000

THE COEF*WT.MEAN

78.3213	87.0142	258.3668	18.8104	36.4266	246.7982
78.3213	43.5071	0.0000	0.0000	-18.2133	-246.7982
0.0000	43.5071	258.3668	9.4052	18.2133	0.0000

THE INFORMATION MATRIX

6.4160	-2.0627	2.5562
-2.0627	3.5412	0.0752
2.5562	0.0752	2.2776

THE S MATRIX

725.7374	-143.1831	329.4924
----------	-----------	----------

THE INVERSE OF THE INFORMATION MATRIX-

0.4456	0.2703	-0.5090
0.2703	0.4466	-0.3182
-0.5090	-0.3182	1.0208

THE ESTIMATE OF M D H AND TEST OF GOODNESS OF FIT

THE ESTIMATE OF M IS 116.9440IT S SE IS0.667500

THE ESTIMATE OF D IS 27.4207IT S SE IS0.668304

THE ESTIMATE OF H IS 12.5139IT S SE IS1.010339

GENERATION	EXPECTED	OBSERVED	DEVIATION
P1	144.3647	143.7667	0.1949
B1	136.9113	138.7133	2.0370
F1	129.4579	129.2444	0.0911
F2	123.2010	117.2333	5.7141
B2	109.4906	111.5756	1.4192
P2	89.5233	89.4867	0.0037

THE CHI SQUARE VALUE IS 9.4600PROBABILITY.0238

THE HERITABILITY ESTIMATE BY WARNER METHOD 0.6264

The family means for the whole experiment are also presented (page 52).

(ii) Analysis of variance table for the six generations. This analysis of variance is mainly for extracting the error mean squares and their respective degrees of freedom for the Scaling Tests. (Page 53).

(iii) The A, B, C and S Scaling Tests and the six estimates of m , d , h , i , j and l , their standard errors, t tests and probabilities of their significance (page 54).

(iv) The intermediate steps in the weighted least square solution to the Cavalli's Joint Scaling Tests (page 55).

(v) The estimates of m , d , h and χ^2 test of goodness of fit for the three parameters model (page 56).

2.4.4 Plot of $W_r - V_r$ Graphs - Bingraph

Computer plot of $W_r - V_r$ graphs is achieved by the use of the subroutine graph written by Pearson and McArthur (1975). The subroutine is available at the Lincoln College Computing Centre and is therefore not listed here. The main programme shown in Appendix V is written by the author.

Data Input

- | | |
|--------|--|
| Card 1 | An integer number indicating the number of families is read in format I4. |
| Card 2 | The W_{ri} values are read in Format F8.4. |
| Card 3 | A title card with alphanumeric letters which can be punched in column 1 to 80. |
| Card 4 | The V_{ri} values are read in Format F8.4. |

Card 5 The values of the parental variance, y intercept,
joint regression coefficient and its standard
error in Format F8.4.

Output

- (i) A plot of Wri against Vri.
- (ii) A plot of the limiting parabola as given by
 $Wri^2 = Vp \times Vri.$

CHAPTER 3

INHERITANCE OF YIELD COMPONENTS IN WHEAT

3.0 REVIEW OF LITERATURE

Engledow and Wadham (1923), in their attempt to interpret yield in terms of governing factors, were the first to propose the now classical description of yield in terms of its contributing components. They suggested that yield per plant be represented as the product of the mean number of ear bearing tillers per plant; mean number of grains per ear and mean weight of a single grain. Their farsighted objective of breaking up yield into its components was not only meant to help the agronomist by relating yield to its components, but was also a simplifying measure to aid the geneticist to work out the mode of inheritance of these component characters. The usefulness of the component approach in the analysis of the genetics of the complex trait, yield, was illustrated by Hayman (1960). In his consideration of the genetical basis of fruit yield in tomatoes, he found the heterotic effect on yield to be the result of accumulation of favourable dominants of the two components of yield, fruit numbers and mean fruit weight. Further credibility to this component approach was given by Grafius (1964) when he stated that this method could help remove epistatic interactions due to components. Evidence that such interactions exist for the complex trait, yield has been provided by various workers (Whitehouse *et al.*, 1958;

Lupton, 1961; Chapman and McNeal, 1971; Ketata *et al.*, 1976b; Singh and Singh, 1976). Moll *et al.* (1962), while agreeing that multiplicative epistasis affecting yield might be avoided by treating the components separately, questioned the advantage of this method. They showed that the epistatic effect could be small compared with the additive and dominance effect in a multiplicative model. They emphasized that the advantage of the yield component approach, based on a better agreement with a simple model, was small. They further suggested that this advantage could probably be less than the disadvantages due to the accumulation of errors and other difficulties in a joint interpretation of multiplicative components. Leng (1963) in his work on yield components inheritance in maize, found that this study could lead to simpler and more tenable conclusions about gene action than a study of the character 'yield'. The heritability of yield was found to be so low, that prediction of hybrid performance from yields of parental lines was difficult in maize breeding programme. The relevance of this component approach in wheat was again highlighted by Rahman *et al.* (1977), when they emphasized that genetic control of individual yield component was worthy of intensive study. This study can provide the possibility of improving yield through genetic manipulation of yield affecting characters which were likely to be under much simpler genetic control and with higher heritability than total yield. The usefulness of this approach was demonstrated by McNeal *et al.* (1978) when they found that selection for yield components can be an effective force in changing grain yield. Both kernel weight and kernel numbers per spike were found to be useful

for indirect selection for yield improvement. Number of ear was, however, found not adequate for selection for yield. The merit of this yield component approach for breeding and selection has been challenged on the ground that yield tends to be stabilised by compensation among yield components (Adams, 1967). Leng (1963) found no real advantage in the component approach in maize because the components were negatively correlated with each other and were poorly correlated with yield. Grafius (1964), however, stated that there is no way in which yield can be changed without changing one or more of the components. Although the changes may compensate each other, resulting in no variation in yield, any increase in yield must be accompanied by changes in one or more of the components. Knott and Talukdar (1971) also emphasized that this compensation need not necessarily be complete. A genetic increase in one component may well result in an increase in yield. They obtained increased yield by increasing grain weight by backcrossing.

Encouraged by this simplified approach, many workers have directed their efforts to study the inheritance of the component characters together with yield *per se*. Whitehouse *et al.* (1958) were among the earliest workers to carry out detail genetical studies on yield components. In their diallel studies they found all the components of yield to be controlled by a simple additive and dominance genetic relationship. Improvement for individual component was indicated and specific crosses were identified for the improvement of each component. However, Lupton (1961), in a more elaborate study involving six cultivars, found evidences of epistatic effects in all yield components studied. Removal of individual array

c.a. approach not distinct in relation to existing data

removed epistatic effect. Bhatt (1971) in a diallel cross analysis of spring wheat, concluded that spikelet per spike, grains per spikelet and weight per grain were all under significant G.C.A. effect, whereas spike per plant was under significant S.C.A. effect. This underlines the role of additive effect for the three components with high G.C.A. and the importance of dominance and epistasis for spike per plant. More evidence of the absence of epistatic interaction and the importance of the additive effect in grain weight was provided by Bhatt (1972). Satisfactory fit for the three parameter model, of mean, additive and dominance, was found for grain weight. Good success for selecting for higher grain weight was also indicated. The importance of the large additive effect of yield components was further provided by the study of Hsu and Walton (1970b). They found dominance to have only a small effect in the inheritance of grains per ear, grain weight and yield itself. Overdominance, however, was found for number of ears per plant. Chapman and McNeal (1971) also reported absence of epistatic effect for number of spikelets and grain weight. Additive gene action had the greatest effect on the number of spikelets and both additive and dominance influenced grain weight. No epistasis was detected for number of spike, spikelet per spike and grains per spike by Ketata et al. (1976a).

Narrow sense heritability estimates of yield components and yield in wheat have also been made by various workers. The high variance estimates provided by these workers are summarised in Table 3.0. The low narrow sense heritability estimates provided by some workers could be attributed to the

Table 3.0 Narrow sense heritability estimates of yield and yield components.

Source	Yield	Grain Wt./ grain	Spike/ plant	Spikelet/ spike	Grain/ spikelet	Grain/ spike
Kronstad and Foote (1964)	0.259	0.472	0.407	0.607	0.478	-
Johnson et al. (1966)	0.102 <u>+0.203</u>	0.547 <u>+0.189</u>	0.034 <u>+0.099</u>	-	-	-
Ketata et al. (1976)	0.16 <u>+0.19</u>	0.65 <u>+0.12</u>	0.36 <u>+0.18</u>	0.09 <u>+0.20</u>	0.28 <u>+0.18</u>	0.15 <u>+0.20</u>
Fonseca and Patterson (1968)		F1 - 1962-1963				
	0.17 <u>+0.07</u>	0.15 <u>+0.13</u>	-	-	-	0.47 <u>+0.16</u>
		F1 - 1963-1964				
	0.49 <u>+0.14</u>	0.51 <u>+0.08</u>	-	-	-	0.89 <u>+0.08</u>
		F2 - 1963-1964				
	0.28 <u>+0.15</u>	0.44 <u>+0.11</u>	-	-	-	0.79 <u>+0.12</u>
		Drilled Plots F2 - 1963-1964				
	0.27 <u>+0.10</u>	0.55 <u>+0.17</u>	-	-	-	0.85 <u>+0.18</u>

presence of epistasis and dominance. Evidence of the presence of epistasis was provided by Ketata *et al.* (1976b) in their studies designed specifically for its detection. They found epistasis to be present in the traits 'grains per spikelet', and 'grain yield'. Epistasis was also recorded for grain weight in one experiment but not the other. Further indication of the role of epistasis was provided by Singh and Singh (1976). In their studies using the triple test cross model of Jinks and Perkins (1970), designed for detection of epistasis, they found epistasis in characters such as spikelets per spike, grains per spike and yield per plant. Chapman and McNeal (1971) also found spikes per plant and yield to be under complex interaction gene control or epistasis.

These differences in the expression of the genetic effects of additive, dominance and epistasis reported by various workers could mainly be attributed to the dissimilarities in the genetic background of the parents used. This was adequately brought out by Ketata *et al.* (1976a) when they found epistasis to be influenced by particular cultivars. Evidence for the contribution of the parental cultivars to epistasis was provided by Lupton (1961) when he detected epistatic interactions when all six cultivars were studied together. As pointed out earlier, removal of individual arrays removed epistasis in the diallel analysis. It is, therefore, apparent that the components approach has not completely removed gene-gene interactions or epistasis. While the simple additive and dominance model is adequate to explain inheritance in yield components in some studies, other studies have shown presence of epistasis.

In New Zealand, the component approach to studies in yield has been the subject of intensive agronomic studies. Scott *et al.* (1977) concluded that a high yielding wheat crop in Canterbury results from the production of a large number of grains per unit area. They emphasized that the most obvious method of achieving this number is by improving grains per spikelet and maintaining high ear densities. The importance of the number of ears and grain set per spikelet on yield was further highlighted by Langer (1978). He stressed that the critical time to influence plant yield is at the stage when these two components are being determined developmentally. Langer (1976) also noted great variation in grain set of fertile spikelet and suggested great scope for improvement in this area. Genetical factors were thought to be involved because cultivar such as Karamu, originating from the Mexican breeding programmes, tends to set more grain per spikelet. Karamu has been able to out-yield other New Zealand wheat by as much as 20% because of better grain set per spikelet and formation of more spikelet per ear (Dougherty *et al.*, 1975). Inherent cultivar differences and environmental effects on spikelet production have also been recorded by Langer and Dougherty (1976). The cultivar Hilgendorf consistently produced fewer spikelet than cultivar Arawa.

The agronomist has, to a great extent, been able to achieve yield increases by influencing yield components through his agronomic practices. It is clear that if this component approach to breeding is to be fruitful, a detailed understanding of gene action associated with the various yield components must be known. Estimates of gene effects

have a direct influence on breeding and selection methods. The presence and magnitude of additive effect are particularly useful for early response to selection in the development of inbred pure lines such as wheat. The presence of dominance and epistasis can be utilised for hybrid seed production. Standard hybridisation and selection procedures can take advantage of epistasis if it is of the additive type only (additive X additive, etc.). Other type of epistasis (additive X dominance, dominance X dominance) are not fixable by selection under self fertilisation and therefore would not be favourable for developing pure line cultivars. Moreover, estimates of additive and dominance ^{effects} can be biased if procedure assuming no epistasis is used. More elaborate experiments that include several generations and environments can provide information relating to the magnitudes of the different types of non-allelic interactions. The detection of epistasis followed by the determination of the types and magnitudes of these interactions could ultimately lead to the development of more efficient breeding procedures. The diallel experiments, the New Triple Test Cross and the associated Scaling Test studies conducted by the writer can help to identify these gene effects.

3.1 INHERITANCE OF GRAIN NUMBER IN THE SPIKE AND SPIKELET OF WHEAT: INTRODUCTION

The use of the classical method of dividing yield into its components has to a great extent removed multiplicative epistasis so often encountered in yield analysis. However,

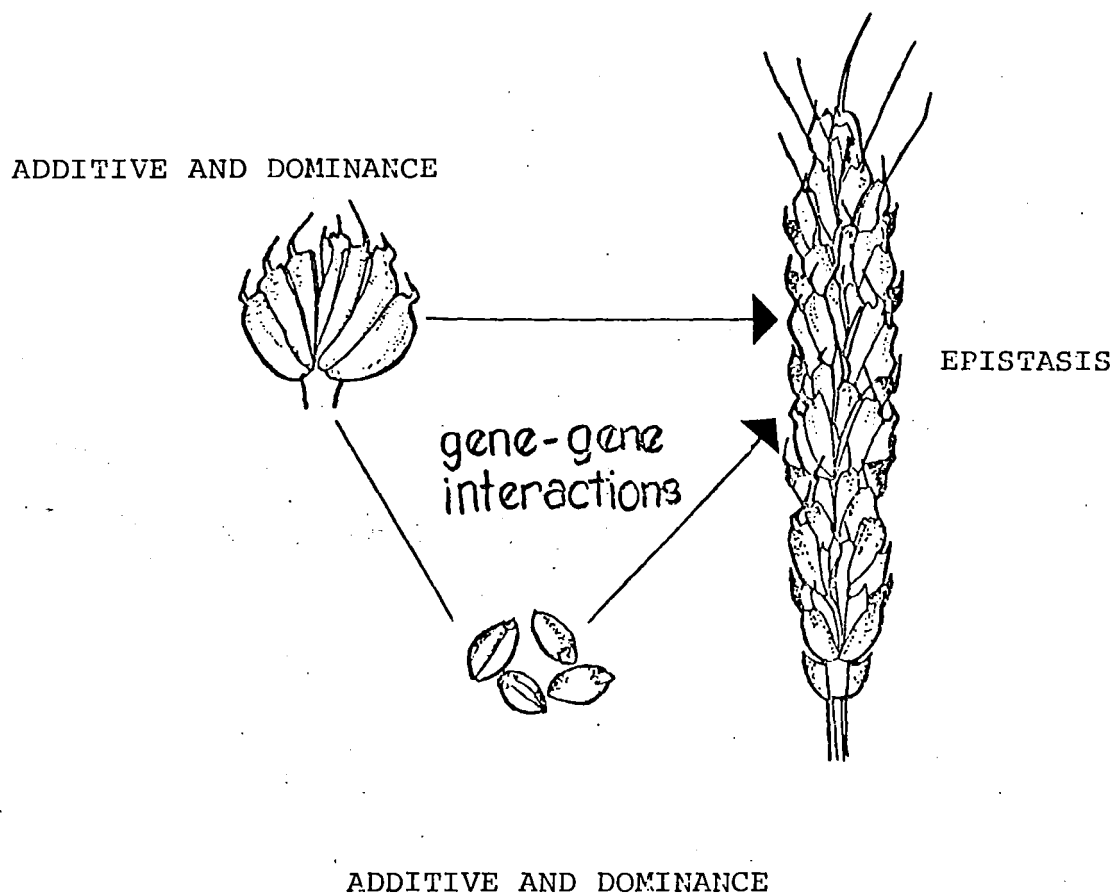


FIG. 3.1 Model for multiplicative epistasis in grains per spike.

not all the yield components have been reduced to its very basic physical level. Grain number per spike and grain number per spikelet are still the multiplicative results of two traits, namely grain number at individual spikelet level and number of spikelet. This study attempts to divide further the yield component, 'grains per spikelet' into its components of grain number at the individual spikelet level and number of spikelet. This yield component, 'grains per spikelet', together with its sub-components are subjected to genetical analysis to reveal the role of multiplicative epistasis.

3.2 MATERIAL AND METHODS

Ten individual plants per family per block of the Half Diallel (Experiment I) were used in this yield component study. Details of the experiment layout and procedure are given in Chapter 2. The ten plants were individually tagged on first emergence of the main stem. The main stems, tiller one and tiller two of each plant were identified with a different colour tag as they emerged. The plants were harvested individually and counts were made of the total number of spikelet and number of grain on the main stem, tiller one and tiller two of each plant. Mean number of grains per spikelet was obtained by dividing grain number per spike by number of spikelets per spike. The number of grains in spikelet position ten, P10 (from the base of each spike), was also recorded for the main stem, tiller one and tiller two. The data for each trait recorded on the mainstem, tiller one and tiller two were pooled and a mean value for the three tillers was calculated. Thirty plants per family per block were

sampled in the F2 study of Experiment II and similar data records were made on the main stems of these plants. Fifteen plants per family per block were sampled in Experiment III and similar counts were made on the main stems of these plants. Means of each of the traits were calculated using Wilson's (1979) Teddybear Programme. Duncan multiple range test was used to test the difference of the family means. The family means of each block of both Experiments I and II were used for the Half Diallel Analysis of Morley Jones (1965) and the Variance-Covariance Analysis described in Mather and Jinks (1971). The analysis was carried out using the computer programme Binhalf (Appendix I) and the W_r - V_r graphs plotted by the graph programme Bingraph (Appendix V). The raw data recorded in Experiment III were used for the New Triple Test Cross and Scaling Test analyses. These analyses were executed with the programmes Bintri and Bintest (Appendices III and IV).

3.3 RESULTS AND DISCUSSION

3.3.1 *F1 Half Diallel Analysis on Spikelets Per Spike*

The number of spikelet per spike was found to be under simple additive and dominance genetic control. The adequacy of this restrictive model was demonstrated by the linear regression coefficients of the W_r - V_r graphs (Figure 3.2) shown in Table 3.10. The linear regression coefficient of the W_r - V_r graph for the mean of three spikes under study was not significantly different from one but highly significant from zero.

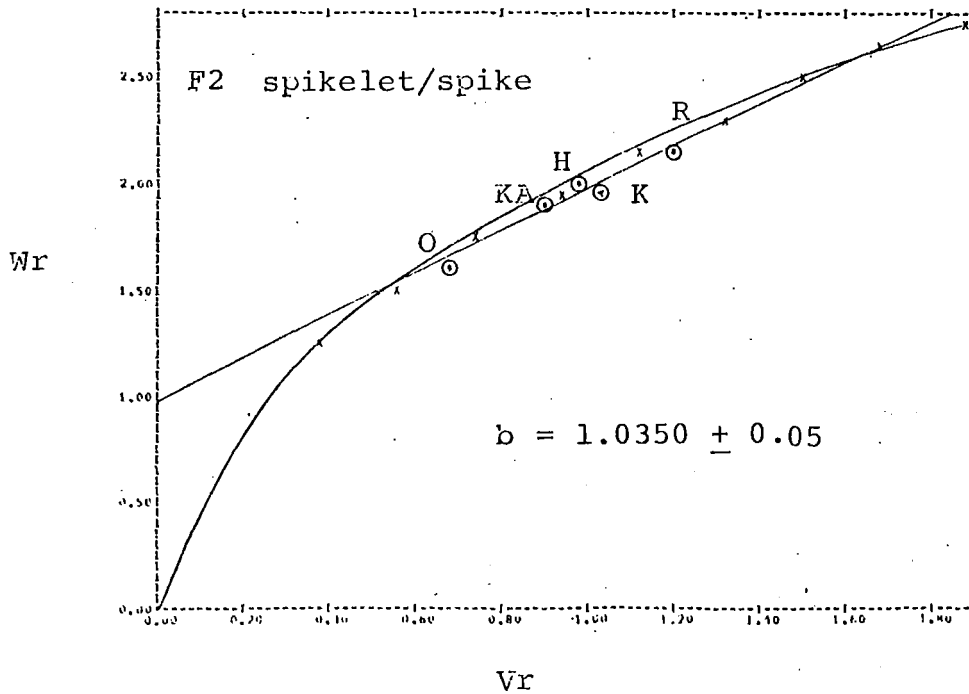
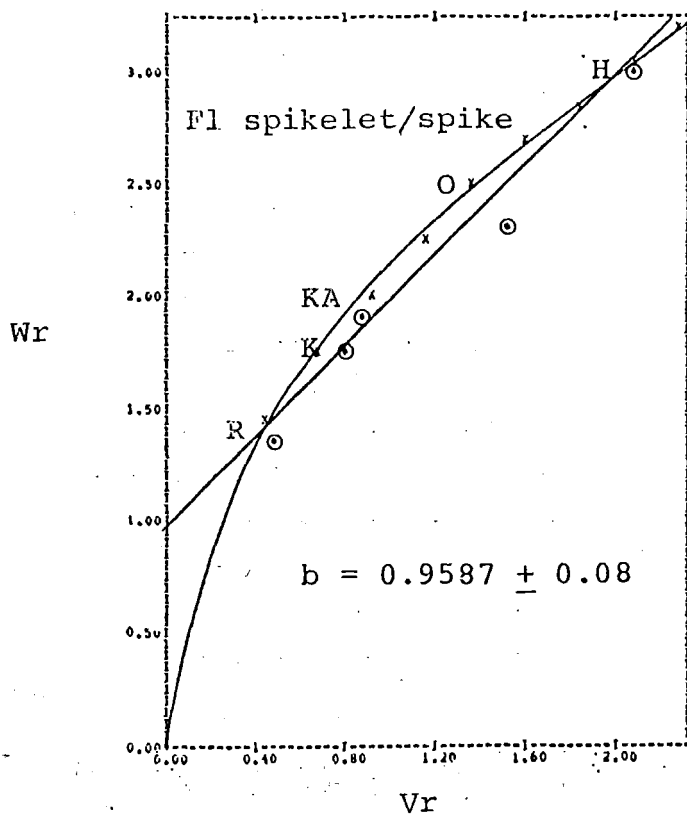


FIG. 3.2 The relationship between W_r and V_r for the F1 and F2 generations for spikelets per spike.
 H = Hilgendorf, K = Kopara, KA = Karamu,
 O = Oroua, R = Ruru.

Parents with high spikelet number were found to possess more dominant alleles, whereas parents with lower spikelet number had more recessive alleles. Cultivars such as Ruru, Karamu and Kopara, had their W_r-V_r array values nearest the origin whereas cultivars such as Hilgen-dorf and Oroua had their values furthest away (Figure 3.2). However, dominance of high spikelet number was not complete. The regression line of the W_r-V_r graph approximated the limiting parabola. Evidence of the dominance of high spikelet number under 24 hour photoperiod had been provided by Rahman *et al.* (1978). The analysis of variance of the Half Diallel (Table 3.9) showed highly significant 'a' or additive effect for this trait. In addition, the 'b' and 'bl' items were both highly significant. This is indicative of the presence of directional dominance.

3.3.2 *F2 Half Diallel Analysis on Spikelets*

Per Spike

The F_2 Half Diallel analysis of spikelets per spike confirmed the F_1 analysis carried out in the previous section. Table 3.12 showed the joint linear regression coefficient of 1.0305 which was not significantly different from one ($P = 0.8932$) but highly significant from zero ($P = 0.0032$). This supported the earlier conclusion of the adequacy of the additive and dominance model.

However, the analysis of variance of the Half Diallel shown in Table 3.11 showed only significance for the 'a' or additive effect. The 'b' item and its component 'bl' were both not significant. This contradicts the results of the

F1 analysis discussed earlier. While the 'a' item in the F2 analysis maintained a similar magnitude as that of the F1, the absolute values of the 'b' and its components were appreciably reduced resulting in their non-significance. These reductions in the magnitudes of the dominance components in the F2 may be explained by the halving of the dominance in advancing from F1 to F2 generation.

The loss of dominance in the F2 generation was again reflected in the order and arrangement of the parents in the Wr-Vr graph of Figure 3.2. Cultivars Ruru, Karamu and Kopara which possess high spikelet number and are shown to carry most dominant alleles in the F1 studies were not near the origin but randomly distributed along the regression line.

3.3.3 *The New Triple Test Cross and Scaling Test Analyses on Spikelets Per Spike*

The New Triple Test Cross Analysis showed an absence of significance for the Ai and Bi comparisons (Table 3.14), indicating absence of epistasis in the trait. These tests lend support to the results of the F1 and F2 Diallel analyses. These conclusions, from the Diallel analyses and the New Triple Test Cross, were generally confirmed by the A and B individual Scaling Test of Table 3.15. Only two of the ten, A and B tests, of the five family arrays showed significance. The parental array Hilgendorf showed significance for the A test, while the Kopara array showed significance for the B test. However, the C, S and the Cavalli's Joint Scaling tests failed to confirm the results of the Diallel and New Triple Test Cross Analyses and the A and B Scaling Tests.

Two of the family arrays, namely Kopara and Ruru, were found to have significant C, S and Cavalli's Joint Scaling Tests (Table 3.15). These results are indicative of the absence of fit for the simple additive model of m, (d) and (h) with evidence for epistasis.

It is not clear why the results of the C, S and Cavalli's Joint Scaling Tests were at variance to those of the A and B Scaling Tests. It could be speculated that the difficulty in obtaining a sufficiently precise F2 mean with the present sample size, for each of the Kopara and Ruru families, could be causing the deviation. The C, S and Cavalli's Joint Scaling Tests used the F2 means in their estimations, whereas no F2 mean was used in the A and B tests. The difficulty in achieving comparable conclusion in the Scaling Test Analysis was also recorded by Ketata *et al.* (1976a). They stated that since each of the Scaling Tests has its own expectation in term of the type and magnitude of epistatic effects, agreement should not necessarily be expected among these tests.

3.3.4 *F1 Half Diallel Analysis on Grain Number at P10*

The number of grain at spikelet position ten (P10) was also found to be under simple genetical control. No evidence of epistatic effect was detected in this trait. This was demonstrated by the joint linear regression coefficients of Table 3.10. The joint linear regression coefficient of the W_r - V_r graph of Figure 3.3 was not significantly different from one but significantly different from zero. The analysis

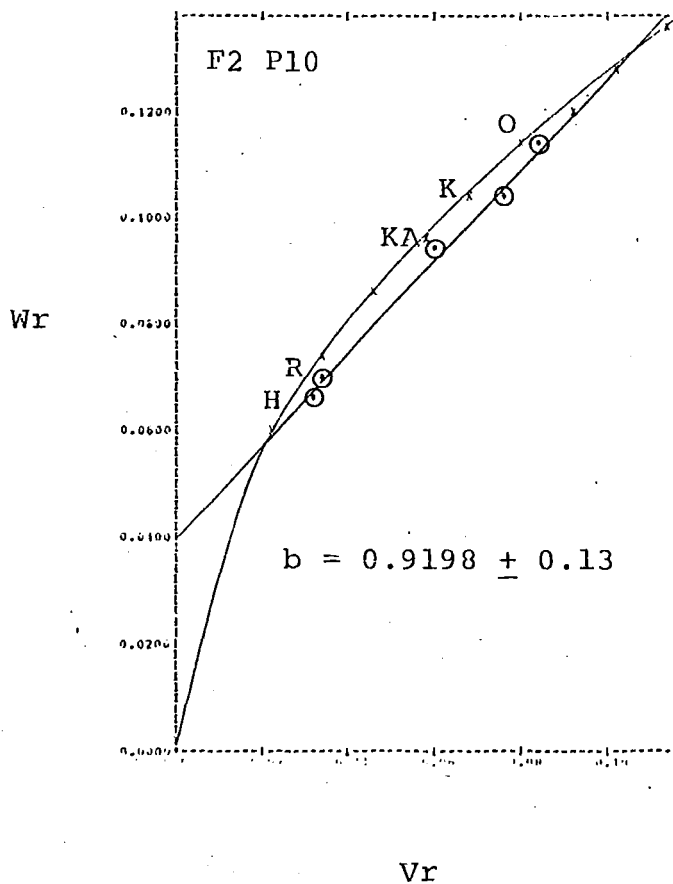
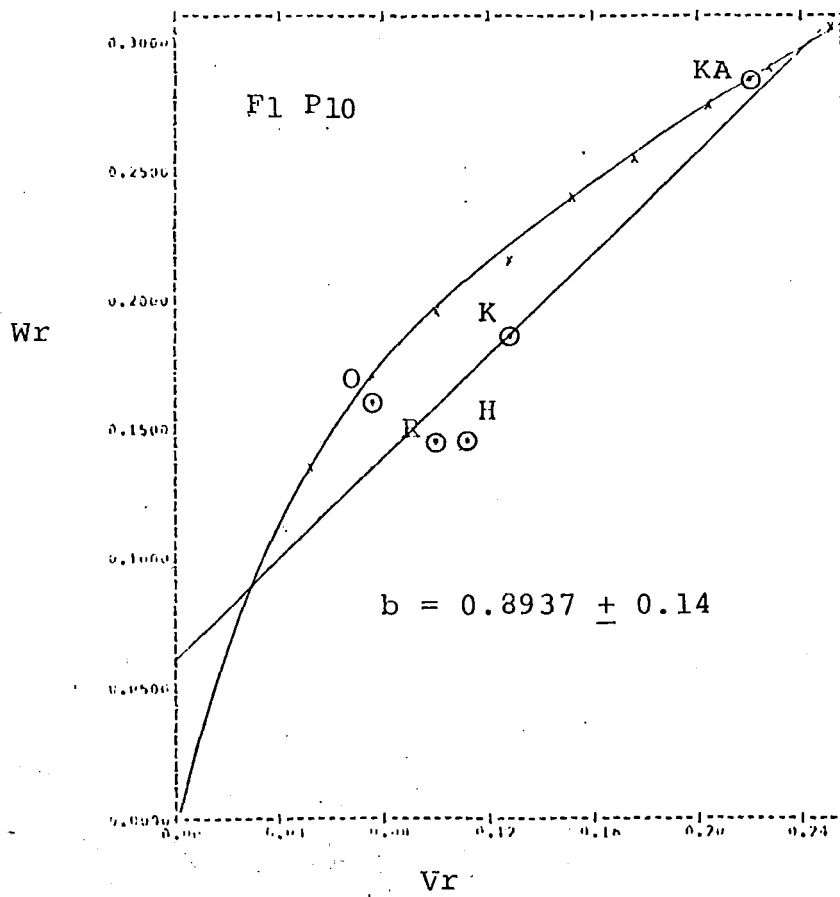


FIG. 3.3 The relationship between W_r and V_r for the F1 and F2 generations for P10.

of the half diallel showed the genetic variation of grain number at P10 to be mainly of the additive type. Dominance was found to be significant but the magnitude of the dominance effect was small compared to the additive effect (Table 3.9).

3.3.5 *F2 Half Diallel Analysis on Grain Number at Spikelet Position Ten (P10)*

The absence of complex inheritance for grain number at spikelet position ten (P10) was again demonstrated by the Diallel Analysis in the F2 generation. The joint regression coefficient for the W_r-V_r graph (Figure 3.3) shown in Table 3.12 was not significantly different from one but significantly different from zero. This allowed the additive and dominance model to be accepted as adequate. The Half Diallel analysis of variance as presented in Table 3.11 on P10 gave further support to the results of the F1 generation which indicated the presence of large and significant additive effect.

3.3.6 *New Triple Test Cross and Scaling Test Analyses on Grain Number at P10*

Additional evidence for absence of epistasis in this trait was provided by the New Triple Test Cross Analysis. Both the A_i and B_i tests were not significant. *(Table 3.13)*

The A, B, C and S Scaling Tests of all the five family arrays were generally in agreement on the absence of epistasis except for the B test in the Ruru array. This absence of fit was again reflected in the Cavalli's Joint Scaling Test where only the Ruru array showed an absence of fit for the three parameters model of m , (d) and (h) .

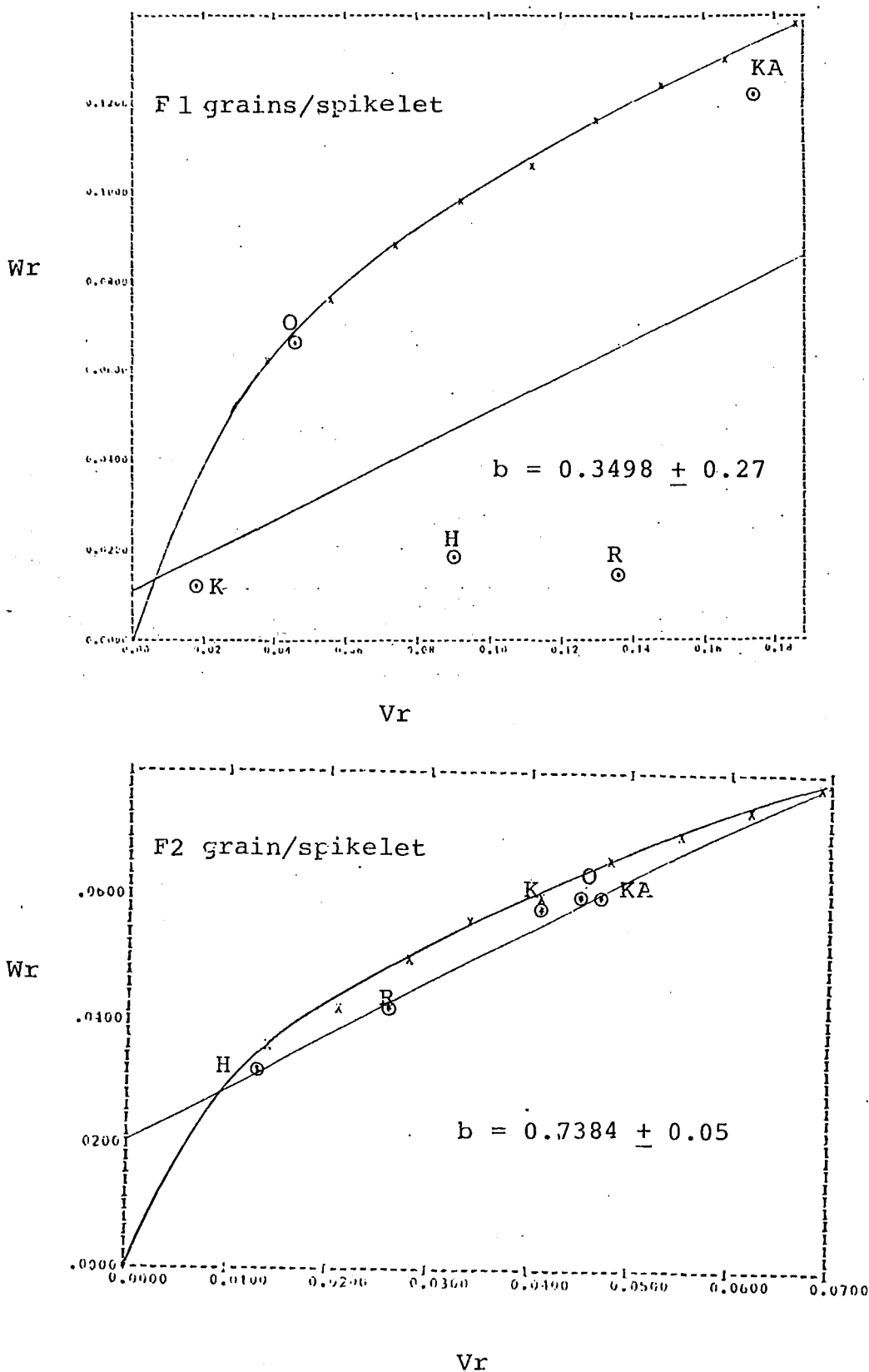


FIG. 3.4 The relationship between W_r and V_r for the F1 and F2 generations for grains per spikelet.

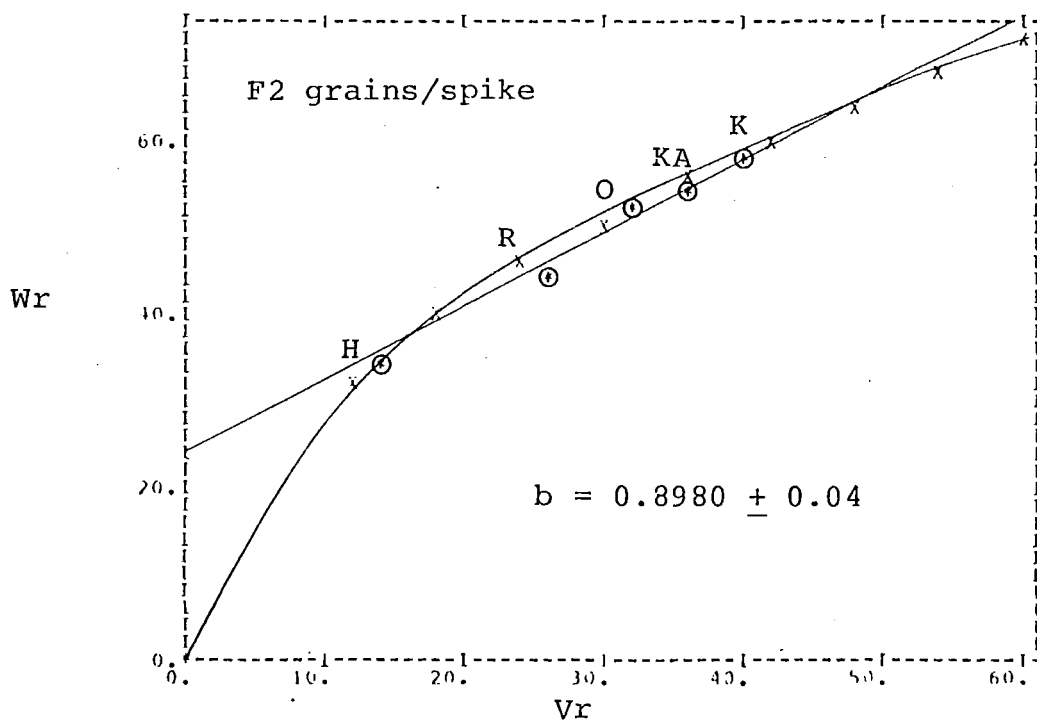
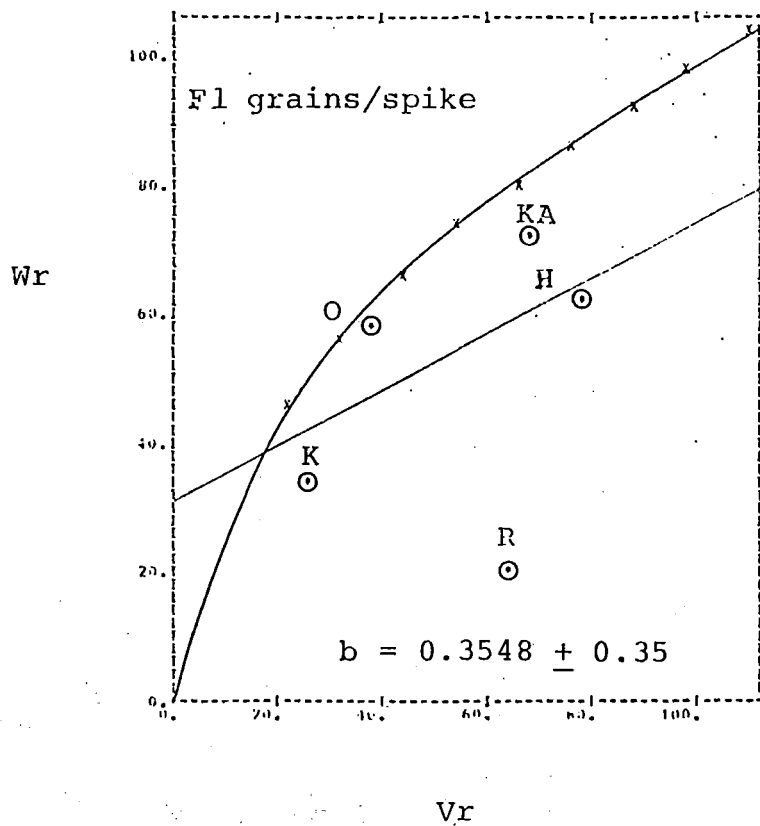


FIG. 3.5 The relationship between Wr and Vr for the F1 and F2 generations for grains/spike.

3.3.7 *F1 Half Diallel Analysis on Grain Number*

Per Spikelet and Grain Number Per Spike

Grain number per spike and grain number per spikelet showed strong evidence of epistatic interaction. The joint linear regression coefficients of both these traits were not significantly different from zero nor one (Table 3.10). The Wr-Vr points of Figures 3.4 and 3.5 were distributed at random. This is implicit of the presence of interacting genes or epistasis. Similar epistatic effects have been reported by various workers (Lupton, 1961; Ketata, 1976b; Singh and Singh, 1976). The Half Diallel analysis of variance (Table 3.9) on grains per spikelet and grains per spike showed a similar pattern with significant additive and dominance items.

3.3.8 *F2 Half Diallel Analysis on Number of Grains*

Per Spike and Number of Grains Per Spikelet

The F2 Half Diallel Analysis failed to reflect the results of the F1 generation. The joint regression coefficients of the Wr-Vr graphs (Figures 3.4 and 3.5) for grains per spikelet and grains per spike were shown in Table 3.12. These coefficients were not significantly different from one but significantly different from zero. The additive and dominance model is therefore considered satisfactory. The Half Diallel analysis of variance shown in Table 3.11 provided similar significance for the additive or 'a' effect, to that of the F1 generation. However, the 'b' and its component items were all not significant. As discussed in the spikelet study and, possibly due to loss of dominance, these

items have all been reduced in absolute values. The relative values of these items, however, reflected a similar pattern as those of the F1 generation.

This reduction in dominance could explain the failure to reproduce the results of the F1 generation in the test of the adequacy of the additive and dominance model by the joint regression coefficient. With the loss of dominance, the detection of F2 families exceeding the parental means, is considerably more difficult with the present sample size. Without a F2 progeny family mean exceeding (positively or negatively) the parental mean significantly, the joint regression coefficient will tend to conform to the value of one, making the additive and dominance model adequate.

3.3.9 *New Triple Test Cross and Scaling Test*

*Analyses on Number of Grains Per Spike and
Number of Grains Per Spikelet*

The New Triple Test Cross Analysis on grains per spikelet, shown in Table 3.20, indicated both the Ai and Bi tests for epistasis to be significant. Moreover, there is evidence for the presence of epistasis for grains per spike. This is indicated by the New Triple Test Cross Analysis on this trait shown in Table 3.23. The Ai comparison showed a highly significant F test. A closer evaluation of Tables 3.20 and 3.23 of the components of the sum of squares from each parental array, for each of the two traits, showed a dominant contribution of the Ruru array to the total sum of squares. Such evidence for the presence of epistasis in the Ruru array for both these traits was supported by the Scaling Test analyses.

Table 3.1 Mean number of spikelets per spike of
parents and their F1 families - Experiment I.

	1	2	3	4	5
1. Hilgendorf	15.5	18.5	17.9	19.4	17.5
2. Kopara		20.0	20.0	20.8	19.3
3. Oroua			18.2	20.5	18.1
4. Ruru				21.1	19.9
5. Karamu					18.5

Standard Error of Mean = 0.221

Coefficient of Variation = 5.16%

Table 3.2 Mean number of spikelets of the mainstem of
the parents and their F2 families - Experiment II.

	1	2	3	4	5
1. Hilgendorf	17.2	19.5	19.1	19.6	18.5
2. Kopara		21.8	21.2	21.9	20.8
3. Oroua			20.6	20.8	20.0
4. Ruru				22.4	20.8
5. Karamu					20.0

Standard Error of Mean = 0.1558

Coefficient of Variation = 5.95%

Table 3.3 Mean number of grains at spikelet P10 of parents and their F1 families - Experiment I.

	1	2	3	4	5
1. Hilgendorf	2.8	3.6	3.2	3.6	3.2
2. Kopara		4.4	3.9	4.4	4.4
3. Oroua			3.8	3.7	3.8
4. Ruru				3.7	3.8
5. Karamu					4.2

Standard Error of Mean = 0.121

Coefficient of Variation = 14.46%

Table 3.4 Mean number of grains at spikelet position ten (P10) of the mainstem of the F2 and parental families - Experiment II.

	1	2	3	4	5
1. Hilgendorf	2.88	3.02	3.02	3.10	3.37
2. Kopara		3.53	3.40	3.52	3.77
3. Oroua			3.47	3.40	3.83
4. Ruru				3.35	3.57
5. Karamu					4.00

Standard Error Mean = 0.0798

Coefficient of Variation = 18.10%

Table 3.5 Mean number of grains per spikelet of parents and their F1 families - Experiment I.

	1	2	3	4	5
1. Hilgendorf	2.5	3.1	2.7	3.1	2.5
2. Kopara		3.3	3.2	3.5	3.4
3. Oroua			2.9	2.9	3.2
4. Ruru				2.7	2.5
5. Karamu					3.2

Standard Error of Mean = 0.09

Coefficient of Variation = 13.96%

Table 3.6 Mean number of grains per spikelet of the mainstem of the F2 and parental families - Experiment II.

	1	2	3	4	5
1. Hilgendorf	2.26	2.39	2.41	2.44	2.57
2. Kopara		2.72	2.71	2.81	2.98
3. Oroua			2.81	2.81	2.99
4. Ruru				2.60	2.79
5. Karamu					3.07

Standard Error of Mean = 0.0521

Coefficient of Variation = 15.01%

Table 3.7 Mean number of grains per spike of parents
and their F1 families - Experiment I.

	1	2	3	4	5
1. Hilgendorf	39.8	58.4	48.1	60.3	44.4
2. Kopara		66.3	64.1	72.4	65.2
3. Oroua			53.2	59.9	57.7
4. Ruru				57.6	50.1
5. Karamu					60.1

Standard Error of Mean = 1.94

Coefficient of Variation = 15.32%

Table 3.8 Mean number of grains per spike of the F2 and
parental families - Experiment II.

	1	2	3	4	5
1. Hilgendorf	38.8	46.4	46.0	48.0	47.7
2. Kopara		59.2	57.3	61.5	62.1
3. Oroua			58.1	58.4	59.9
4. Ruru				58.2	57.9
5. Karamu					61.6

Standard Error of Mean = 1.1466

Coefficient of Variation = 16.23%

Table 3.9 Half Diallel analysis of spikelets per spike, grains at P10, grains per spikelet and grains per spike after Morley Jones (1965) - F1 generation.

Trait		Spikelets/spike	Grains at P10	Grains/spikelet	Grains/spike
Item	df	M.S.	M.S.	M.S.	M.S.
a	4	14.1707***	1.1310***	0.4009***	392.3544***
b	10	0.4501***	0.0634**	0.1339***	56.7051***
b1	1	1.7579***	0.0002	0.0282	47.7755*
b2	4	0.4813**	0.0908**	0.1194**	63.7168**
b3	5	0.1636	0.0541*	0.1666***	52.8816**
Bxa	4	0.0349	0.0152	0.0177	8.9630
Bxb	10	0.0679	0.0120	0.0197	8.3159
Bxb1	1	0.0443	0.0522	0.0317	19.4029
Bxb2	4	0.1335	0.0039	0.0233	9.8346
Bxb3	5	0.0196	0.0105	0.0145	4.8836
Block inter- action	14	0.0583	0.0129	0.0192	8.5008

* P < 0.05 ** P < 0.01 *** P < 0.001

Table 3.10 Joint regression analysis for testing the adequacy of the additive and dominance model on spikelets per spike, grains at P10, grains per spikelet and grains per spike - F1 generation.

Trait		Spikelets/spike	Grains at P10	Grains/spikelet	Grains/spike
Item	df	M.S.	M.S.	M.S.	M.S.
Joint Regression	1	3.2155***	0.0267***	0.0043 ns	611.3181 ns
Heterogeneity of Regression	1	0.0030 ns	0.0001 ns	0.0004 ns	189.4900 ns
Remainder	6	0.0239	0.0006	0.0025	592.2909
Joint Regression Coefficient		0.9587	0.8937	0.3498	0.3548
Standard Error		0.0826	0.1386	0.2655	0.3492
Significant From 1.0		0.8903	0.7848	0.2538	0.3168
Significant From 0.0		0.0157	0.0532	0.5225	0.5702

* $P < 0.05$ ** $P < 0.01$ *** $P < 0.001$

Table 3.11 F2 Half Diallel analysis of spikelet number, grain number per spike and grain number per spikelet and P10 of the mainstem of the F2 generation after Morley Jones (1965).

Item	df	Spikelet No. M.S.	Grains/spike M.S.	Grains/spikelet M.S.	P10 M.S.
a	4	13.0687***	360.0132***	0.3761***	0.6604***
b	10	0.0953	5.2072	0.0124	0.0152
b1	1	0.1967	3.1268	0.0001	0.0156
b2	4	0.1167	5.0405	0.0137	0.0089
b3	5	0.0579	5.7566	0.0138	0.0203
Bxa	4	0.0699	1.9240	0.0073	0.0024
Bxb	10	0.0653	2.7818	0.0082	0.0175
Bxb1	1	0.0562	2.6886	0.0017	0.0002
Bxb2	4	0.0447	3.5285	0.0137	0.0358
Bxb3	5	0.0836	2.2031	0.0057	0.0064
Block Interaction	14	0.0666	2.5367	0.0079	0.0132

* P < 0.05 ** P < 0.01 *** P < 0.001

Table 3.12 Joint linear regression coefficient for testing adequacy of the additive and dominance model for the mainstem of F2 and parental generation.

Trait	Linear Regression Coefficient	SE + -	Probability From 1.0	<i>for</i> Significance of difference From 0.0
Spikelet Nos.	1.0305	0.0473	0.8932	0.0032
Grains/spikelet	0.7384	0.0497	0.2849	0.0161
Grains/spike	0.8980	0.0374	0.6168	0.0035
Grains at P10	0.9198	0.1301	0.8315	0.0435

Table 3.13 Mean spikelet per spike of the mainstem of parents (Pi), common tester (Pc), F1, F2 and backcrosses (Bci, Bc2) - Experiment III.

Parents Generation	Hilgendorf	Kopara	Oorua	Ruru	Atlas 66
Pi	17.2	21.7	20.2	22.5	21.2
Pc	20.4	20.6	20.5	20.5	20.6
B1	17.4	21.6	20.1	22.2	21.2
B2	19.5	20.9	20.1	20.5	20.4
F1	18.7	21.4	19.8	21.3	20.7
F2	18.8	20.7	20.0	20.7	20.4

Standard Error of Mean = 0.1623

Coefficient of Variation = 5.34%

Table 3.14 Sums of squares of comparisons for detecting epistasis on number of spikelet per spike on the mainstem.

Parent (Pi)	Ai Comparison Sums of Squares	Bi Comparison Sums of Squares
Hilgendorf	12.9600	0.1111
Kopara	0.0178	0.6400
Oroua	0.2178	0.0178
Ruru	3.0044	6.0844
Atlas 66	1.9600	2.1511
Total	18.1600	9.0044
Mean Square	0.4036	0.2000
Pooled Variance	0.2247	0.1625
F Ratio	1.7962 ns	1.2316 ns

ns non significance

Table 3.15 Scaling Tests and estimates of parameters for number of spikelets per spike on the mainstem.

Parents (Pi)	Hilgendorf	Kopara	Oroua	Ruru	Atlas 66
Test					
A	-1.2000**	0.0444	0.1556	0.5778	0.4667
B	-0.1111	0.2667	-0.0444	-0.8222	-0.4889
C	0.0108	-1.8724**	-0.3735	-2.9556***	-1.4000
S	0.3305	-0.5459***	-0.1211	-0.6778***	-0.3444
Estimates					
m	20.1108***	18.7720***	19.8821***	18.8111***	19.5000***
d	-1.5889***	0.7111**	-0.1444	1.0111***	0.3000**
h	-4.0326*	5.1226*	0.4692	4.9444*	2.5667
i	-1.3219	2.1835	0.4846	2.7111***	1.3778
j	-1.0889*	-0.2222	0.2000	1.4000*	0.9556
l	2.6330	-2.4946	-0.5957	-2.4667	-1.3556
Joint Tests					
m	18.77 + 0.06	20.18 + 0.15	20.36 + 0.08	21.34 + 0.10	20.86 + 0.10
d	-1.61 + 0.07	0.78 + 0.16	-0.14 + 0.08	0.98 + 0.10	0.33 + 0.10
h	-0.09 + 0.10	0.55 + 0.20	-0.62 + 0.14	-0.40 + 0.17	-0.19 + 0.14
X ² (3)	7.485 ns	9.7153*	0.621 ns	29.4756***	5.8164 ns
* P < 0.05	** P < 0.01	*** P < 0.001			

Table 3.16 Mean number of grains at spikelet position ten (P10) of the mainstem of parents (Pi), common tester (Pc), F1, F2, Bc1 and Bc2 families - Experiment III.

Parents Generation	Hilgendorf	Kopara	Oroua	Ruru	Atlas 66
Pi	2.6	3.5	3.6	3.4	3.4
Pc	4.0	4.1	4.3	4.1	4.0
B1	3.0	4.0	3.8	3.4	3.5
B2	3.6	4.1	4.0	4.0	4.0
F1	3.0	4.2	3.9	3.1	3.9
F2	3.2	4.0	3.9	3.6	3.7

Standard Error of Mean = 0.0998

Coefficient of Variation = 18.04%

Table 3.17 Sums of squares of comparisons for detecting epistasis on grain number at P10 of the mainstem.

Parent (Pi)	Ai Comparison Sum of Squares	Bi Comparison Sum of Squares
Hilgendorf	1.6044	0.4444
Kopara	0.7511	0.3600
Oroua	0.0000	0.2178
Ruru	1.0000	5.4444
Atlas 66	1.2844	0.1111
Total	4.6400	6.5778
Mean Square	0.1032	0.1462
Pooled Variance	0.0586	0.0636
F Ratio	1.7592 ns	2.2992 ns

ns non significance

Table 3.18 Scaling Tests and estimates of parameters for grain number at P10 of the mainstem.

Parents (Pi)	Hilgendorf	Kopara	Oroua	Ruru	Atlas 66
Test					
A	0.4222	2.889	0.0000	0.3333	-0.3778
B	0.2222	-0.2000	-0.1556	0.7778**	0.1111
C	0.2158	-0.2624	-0.3147	0.4588	-0.5778
S	-0.1072	-0.0878	-0.0398	-0.1631	-0.0728
Estimates					
m	2.8713***	3.4710***	3.8075***	3.1032***	3.3778***
d	-0.7444***	-0.3111***	-0.3222***	-0.3333***	-0.2667***
h	1.2240	1.2136	0.1072	1.7935	0.6000
i	0.4287	0.3513	0.1591	0.6523	0.3111
j	0.2000	0.4889	0.1556	-0.4444	-0.4889
l	-1.0731	-0.4401	-0.0036	-1.7634	-0.0444
Joint Test					
m	3.32 + 0.05	3.80 + 0.07	3.95 + 0.06	3.81 + 0.06	3.68 + 0.06
d	-0.73 + 0.05	-0.28 + 0.07	-0.31 + 0.05	-0.38 + 0.05	-0.32 + 0.06
h	-0.16 + 0.11	0.43 + 0.13	-0.08 + 0.11	-0.45 + 0.12	0.22 + 0.10
χ^2 (3)	4.01 ns	1.91 ns	0.89 ns	10.29*	4.76 ns
* P < 0.05 ** P < 0.01 *** P < 0.001					

Table 3.19 Mean number of grains per spikelet of the main stem of parents (Pi), common tester (Pc), F1, F2 and backcrosses (Bc1, Bc2) - Experiment III.

Parents Generation	Hilgendorf	Kopara	Oroua	Ruru	Atlas 66
Pi	2.29	2.68	3.04	2.57	2.56
Pc	3.11	3.16	3.22	3.20	3.21
B1	2.43	3.10	2.89	2.65	2.76
B2	2.82	3.26	3.15	3.00	3.09
F1	2.40	3.24	2.94	2.31	3.09
F2	2.55	3.31	3.09	2.72	2.84

Standard Error of Mean = 0.0634

Coefficient of Variation = 14.77%

Table 3.20 Sums of squares of comparisons for detecting epistasis on number of grains per spikelet on the mainstem.

Parent (Pi)	Ai Comparison Sums of Squares	Bi Comparison Sums of Squares
Hilgendorf	0.3166	0.1414
Kopara	0.7476	0.1419
Oroua	0.3434	0.9880
Ruru	1.5658	2.2741
Atlas 66	0.1648	0.1500
Total	3.1383	3.6954
Mean Square	0.0798	0.0822
Pooled Variance	0.0199	0.0263
F Ratio	3.5120**	3.1255**

Table 3.21 Scaling Tests and estimates of parameters for number of grains per spikelet on the mainstem.

Parents (Pi)	Hilgendorf	Kopara	Oroua	Ruru	Atlas 66
Test					
A	0.1876	0.2882	-0.1953	0.4171*	-0.1353
B	0.1253	0.1256	0.3313*	0.5027**	-0.1291
C	0.0324	0.9040*	0.4116	0.5144	-0.6200
S	-0.0701	0.1266	0.0689	-0.1013	-0.0889
Estimates					
m	2.4138	3.4113***	3.3060***	2.4757***	2.5326***
d	-0.4122***	0.0432***	0.0060	-0.3137***	-0.3244***
h	0.5789	0.9621	-0.5098	1.1601	0.6544
i	0.2804	-0.4902	-0.2756	0.4053	0.3556
j	0.0622	0.1627	-0.5267	-0.0856	-0.0062
l	-0.5933	0.0764	0.1396	-1.3257	-0.0911
Joint Test					
m	2.70 + 0.03	2.96 + 0.04	3.04 + 0.04	2.93 + 0.04	2.86 + 0.03
d	-0.41 + 0.03	-0.24 + 0.04	-0.04 + 0.04	-0.33 + 0.04	-0.32 + 0.03
h	-0.27 + 0.06	0.34 + 0.07	-0.06 + 0.07	-0.53 + 0.06	0.19 + 0.06
χ^2 (3)	2.3029 ns	8.163*	10.5234*	13.6685**	4.4756 ns
* P < 0.05 ** P < 0.01 *** P < 0.001					

Table 3.22 Mean number of grains per spike of the mainstem of parents (Pi), common tester (Pc), F1, F2 and backcrosses (Bc1, Bc2) - Experiment III.

Parents	Hilgendorf	Kopara	Oroua	Ruru	Atlas 66
Generation					
Pi	39.2	57.7	59.7	57.9	54.0
Pc	63.3	65.3	66.0	65.6	66.1
B1	42.2	66.9	57.1	58.9	57.8
B2	54.8	68.2	62.3	61.0	62.9
F1	44.9	69.3	58.5	47.2	64.1
F2	47.5	65.1	60.6	56.7	57.8

Standard Error of Mean = 1.3601

Coefficient of Variation = 15.53%

Table 3.23 Sums of squares of comparisons for detecting epistasis on number of grains per spike on the mainstem.

Parent (Pi)	Ai Comparison Sums of Squares	Bi Comparison Sums of Squares
Hilgendorf	1.0000	16.5378
Kopara	405.3511	33.6400
Oroua	142.4044	0.0400
Ruru	11464.3378	784.0000
Atlas 66	50.8844	170.7378
Total	2063.9778	1004.9556
Mean Square	45.8662	22.2324
Pooled Variance	10.6544	11.6970
F Ratio	4.3049***	1.9092 ns

ns non significance

* $P < 0.05$

** $P < 0.01$

*** $P < 0.001$

Table 3.24. Scaling Tests and estimates of parameters for number of grains per spike on the mainstem.

Parents (Pi)	Hilgendorf	Kopara	Oroua	Ruru	Atlas 66
Test					
A	0.3333	6.7111	-3.9778	12.7556**	-2.3778
B	1.3556	1.9333	0.0667	9.3333*	-4.3556
C	-2.4645	-1.2767	0.0014	9.1914	-16.9556**
S	-1.0384	-2.4803	0.9781	-3.2244	-2.5556
Estimates					
m	47.1355***	51.5789***	66.7348***	48.8136***	49.8111***
d	-12.0444***	-3.7667***	-3.1556***	-3.8444***	-6.0556***
h	3.6179	36.2756*	-16.0695	33.3283*	17.7667
i	4.1534	9.9211	-3.9125	12.8975	10.2222
j	-1.0222	4.7778	-4.0444	3.4222	1.9778
l	-5.8423	-18.5656	7.8237	-34.9864**	-3.4889
Joint Tests					
m	51.26 + 0.62	61.74 + 0.83	62.57 + 0.79	62.53 + 0.79	59.12 + 0.75
d	-12.04 + 0.65	-3.50 + 0.85	-3.52 + 0.79	-3.95 + 0.80	-5.76 + 0.74
h	-6.44 + 1.14	8.14 + 1.47	-4.47 + 1.50	-12.60 + 1.62	3.70 + 0.30
χ^2 (3)	0.6243 ns	4.1474 ns	1.5592 ns	12.3929**	8.3872*
* P < 0.05	** P < 0.01	*** P < 0.001			

The Scaling Test analyses for both these traits showed significance for the A, B and Cavalli's Joint Scaling Tests (Tables 3.21 and 3.24).

3.4 DISCUSSION

The Half Diallel, the New Triple Test Cross and the A and B Scaling Test Analyses have provided sufficient evidence to show the adequacy of the additive and dominance model for spikelets per spike. The absence of epistasis for this trait has been well documented (Whitehouse *et al.*, 1958; Bhatt, 1971; Chapman and McNeal, 1971; Ketata *et al.*, 1976a, b; Edwards *et al.*, 1976; Rahman *et al.*, 1977). On the other hand, Lupton (1961), Singh and Singh (1976), Gill *et al.* (1977) have suggested presence of epistasis for spikelets per spike. Such contradiction in the results could be explained by the differences in the genetic background of the parents studied, and this has been adequately illustrated by Lupton (1961). The observation in the F1 Half Diallel analysis on the significance of the dominance effect, is reinforced by the preponderance of the dominance effect (h) in the estimates provided by the Scaling Test analyses (Table 3.15). Similar evidences on the presence of dominance for high spikelet number under lengthening photoperiod have been provided by Rahman *et al.* (1977) who recorded such dominance under a 24-hour photoperiod.

The inheritance of grain number at a single spikelet position, for example P10, has not been reported previously. However, our conclusions from the F1, F2 Half Diallel, the New Triple Test Cross and Scaling Test analyses are all in common

agreement on the absence of gene interaction for this trait. Our observation, based on the Half Diallel analyses on the presence of the additive control, has also been substantiated by the estimates of (d) and (h) in the Scaling Test analysis. Table 3.18 shows the presence of highly significant (d) terms but non-significant (h) estimates.

The genetical analyses of grains per spikelet and grains per spike discussed in Sections 3.3.7, 3.3.8 and 3.3.9 are by no means conclusive. The difficulty of achieving a common conclusion from such analyses is increased by the complexity of the traits under study. This could be attributed to the large environmental interaction with an increasingly complex trait. However, the presence of significant tests of epistasis, and the significant Scaling Tests for the Ruru array for grains per spike and grains per spikelet, are further indication of absence of fit for the additive and dominance model as observed in the F1 Half Diallel analysis of the previous season. Results obtained by other workers are conflicting. Lupton (1961) and Singh and Singh (1976) recorded epistasis for grains per spike but Hsu and Walton (1970) and Ketata *et al.* (1976a, b) have provided contrasting evidences.

With the available information, it is therefore not unduly speculative for us to propose the presence of gene interactions for grains per spike in our genetic material. The presence of epistasis for this trait will be consistent with the expression of multiplicative epistasis as discussed by Moll *et al.* (1962) and Grafius (1964). A simple model shown in Figure 3.1 is presented to illustrate the likely role of multiplicative epistasis in grains per spike. Grain number per spike is the multiplicative character of grain number

at individual spikelet position and number of spikelet. In exact algebraic expression: Grain Number Per Spike is $\sum_{i=1}^N G_i$ where G_i is grain number at individual spikelet position and N is the number of spikelet. From this algebraic expression, it is obvious that grain number per spike is the result of the interaction of two traits. The interacting gene system or epistasis detected in grains per spike could be explained by the interaction of genes controlling spikelet number with the genes controlling grain number at individual spikelet position.

The evidence provided above on the genetics of grain number may have important implications in the methods of breeding and selection of inbred crop, such as wheat. The epistasis found in grain number per spike, unless of the additive x additive type, is not fixable. This could explain the elusive spike with a large number of grains and the futile efforts in trying to select for it. The expression of high grain number per spike in a segregating generation would be lost when the additive x dominance and dominance x dominance epistasis segregate to homozygosity in advance generations. The nature and magnitude of the epistasis on grains per spike in the cultivar Ruru suggests the existence of such undesirable epistasis. The presence of the duplicate type epistasis for this trait (Table 3.24) is shown by significant (h) and (l) effects of opposing sign (Mather, 1967). Epistasis of this nature is particularly unsuitable for selection of inbreds.

On the other hand, if selection is based on grain number at the individual spikelet level, response to selection would

be ensured because of the absence of epistatic interference. Moreover, the presence of the mainly additive effect would make early response to selection possible. However, selection for the other yield component, spikelets per spike, would be difficult because of significant dominance effect in this trait. A positive response to selection of grains at individual spikelet level could conceivably increase grains per spike if component compensation is not complete.

CHAPTER 4

GENETICAL STUDIES OF AVERAGE 1000 GRAIN WEIGHT,
NUMBER OF SPIKES PER PLANT AND GRAIN YIELD
PER PLANT IN WHEAT

4.0 REVIEW OF LITERATURE

Grain yield in wheat has been shown to have low narrow sense heritability (McNeal, 1960; Kronstad and Foote, 1964; Johnson *et al.*, 1966; Fonseca and Patterson, 1968; Ketata *et al.*, 1976b). These low narrow sense heritability estimates were attributed to dominance and complex gene interactions of the non-additive type epistasis governing this trait (Chapman and McNeal, 1971; Singh and Singh, 1976; Ketata *et al.*, 1976b). Paroda and Joshi (1970) have shown grain yield to be controlled mainly by the non-fixable dominance component of the genetic variation, whereas Whitehouse *et al.* (1958) and Hsu and Walton (1970) have found yield to be governed largely by overdominance. Duplicate type epistasis was recorded by Ketata *et al.* (1976b). The complex genetic nature of this trait, yield, means that it cannot be reliably used as a selection criterion in early generations. The problem of selecting for this polygenic trait, yield, which could be controlled by more than 21 loci, was discussed by Snee (1977). Knott (1972) in testing differences of F3 from F2 selection, recorded a gain too small to be of value in a breeding programme. McGinnis and Shebeski (1968) in testing the progenies of selected and

random F2 plants found no difference in their yield. There was also an absence of correlation between F2 and F3 yields. Depauw and Shebeski (1973) obtained significant but low correlation coefficient of only 0.39 between the F3 and F4 generations. Briggs and Shebeski (1971) obtained contradictory results of high correlations of the F5 on F3 for one year, but no significant correlations in two subsequent years. Similarly, Knott and Kumar (1975) obtained low intergeneration correlation of only 0.14 and 0.29 between the F3 and F5 yield. O'Brien *et al.* (1978), however, obtained response in the F4 and F5 generations for two of the four crosses. They emphasized that the effectiveness of early generation selection depended greatly on the presence of large genetic variances and the influence of the environment. Knott (1979) is of the opinion that selection in the F3 for yield had limited effect unless carried out in extensively replicated trials. He suggested the use of the single seed decent method instead of the laborious and ineffective early generation selection. Wright and Thomas (1976) in their evaluation of the single seed decent method found no real advantage and advocated the continuation of the pedigree method for New Zealand wheat breeding programme.

The principal component of yield, spikes per plant, unfortunately, expresses similarly complex inheritance as yield. Epistasis has been detected for this trait by Chapman and McNeal (1971), Bhatt (1971), Ketata *et al.* (1976b), Singh and Singh (1976) and Gill *et al.* (1977). McNeal *et al.* (1978) reported no useful response to selection based on this trait. They explained that the lack of response to selection was due either to the presence of adverse linkages to other

yield related traits as in oats (Chandhanamutta and Frey, 1973) or annual differences in tillering that could have affected the expression of genetic differences so that selection was ineffective in some years. The latter reason appeared to be more plausible because of the extreme sensitivity of tillering capability and eventual spike formation to environmental factors (Evans and Wardlaw, 1976; Langer, 1974), and soil nitrogen status (Langer, 1978). This difficulty of separating the genetic variation from the environmental effect on tillering and spike formation could also help explain the expression of epistasis recorded by various workers.

As reviewed in Section 3.0, average 1000 grain weight has been shown to be under less complex genetic control. Knott and Taludar (1971) were able to recover lines approximating the grain weight of cultivar Selkirk in their back-crossing programme, and McNeal *et al.* (1978) had found selection for grain weight effective for yield improvement. However, evidence of complex genetical control, involving epistasis, has been recorded by Lupton (1961) and Ketata *et al.* (1976a). Lupton (1961) was able to remove epistatic effect by removing individual array in his diallel analysis, thus emphasizing the role of the genetic background of the parents in the expression of this trait. Ketata *et al.* (1976a) detected epistasis in only one of his two experiments underlining the effect of environment in the expression of this trait.

4.1 INTRODUCTION

An understanding of the inheritance of spikes per plant and 1000 grain weight is important because spikes per plant is a principal component of yield while 1000 grain weight is an important yield and quality trait. The knowledge of the genetics of tillering is also desirable because of the need to adapt different tillering genotypes to different environments. Uniculu wheat under high seeding density has attracted considerable attention in some countries, while in New Zealand heavy tillering cultivars are needed. This is because of the need to compensate for any loss during establishment caused by drowning. The Half Diallel, the New Triple Test Cross and the Scaling Tests analyses are used to study the genetics of these two yield components and yield per plant.

4.2 MATERIAL AND METHODS

Ten plants per family per block of Experiment I were harvested individually. Number of spikes per plant was counted and only spikes bearing mature grains at harvest were counted. The spikes of each individual plant were then threshed to extract the grains. The grain number per plant was counted with a seed counter. The grains from each individual plant were then weighed. The grain weights were corrected to 14 per cent moisture, and 1000 grain weights were calculated as the mean single grain weight \times 1000 for each family in each block.

Thirty plants per family per block of Experiment II were

harvested and the mature spikes were counted, as in Experiment I. The spikes (except that of mainstems) from each of the family of each block were then pooled and bulk threshed with a mechanical thresher. The mainstems were individually hand threshed for the studies of Chapter 3, after which these grains were pooled with the bulk threshed grains. The pooled grains were weighed and the weights corrected to 14 per cent moisture. The resultant moisture corrected weights were divided by 30 to give the mean grain yield of each individual plant. Thousand grain weight of each family of each block was obtained by weighing five randomly counted 200 grains samples from the pooled grains. Fifteen plants per family per block of Experiment III were harvested and the mature spikes per plant were counted as in Experiments I and II. In this experiment, the mature spikes were then uniformly cut just below the last spikelet. The air dried spikes of each individual plant were weighed to give an estimate of single plant yield (spike weight). The mainstems which had been identified with tags soon after emergence were individually hand threshed and the grains weighed and counted. Thousand grain weight of each mainstem was estimated by dividing the grain weight, corrected to 14 per cent moisture, by the grain number of the mainstem multiplied by 1000.

The individual values for each trait were subjected to the calculation of means and test of significant differences using Wilson's (1979) Teddy Bear Programme. The family means of each block of both Experiments I and II were then used for the Half Diallel analysis of Morley Jones (1965) and the Variance-Covariance analysis of Mather and Jinks (1971).

These analyses were executed with the computer programme Binhalf (Appendix I) and the Wr-Vr graph plotted with the programme Bingraph (Appendix V). The fifteen individual values for each trait of each family of the three replicate blocks of Experiment III were used in the New Triple Test Cross and the Scaling Test Analyses. These analyses were executed with the computer programmes Bintri and Bintest respectively (Appendices III and IV).

4.3 RESULTS

4.3.1 F1 Half Diallel Analysis of 1000 Grain Weight

The comparison of means in Table 4.1 shows that the 1000 grain weights of the parents differed significantly. Cultivar Hilgendorf had the highest 1000 grain weight among the parents studied. Cultivar Karamu, on the other hand, had the lowest 1000 grain weight, but this could partly have been due to the presence of leaf rust (*Puccinia recondita* Rob. ex. Desm.) which could also have reduced the total yield of this cultivar substantially. Cultivars Kopara, Oroua and Ruru possessed similar 1000 grain weights. Some of the F1 plants exhibited greater 1000 grain weight than the parents, indicating the presence of hybrid vigour for this trait. The analysis of variance of the Half Diallel shown in Table 4.2 indicated the presence of significant dominance (item b) and also significant directional dominance (item bl). The dominance was towards larger grain weight as reflected in the 1000 grain weight of the F1 which either exceeded or approximated the parents with greater 1000

Table 4.1 Estimates of number of spikes per plant, 1000 grain weight, and total grain weight per plant in the F1 Half Diallel Crosses - Experiment I.

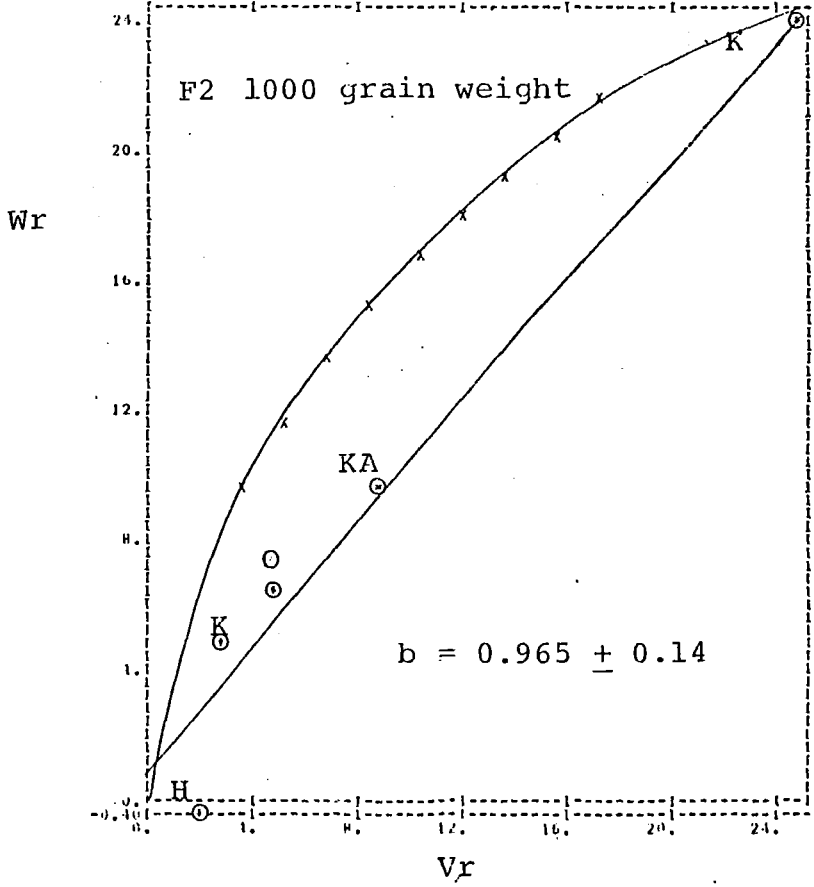
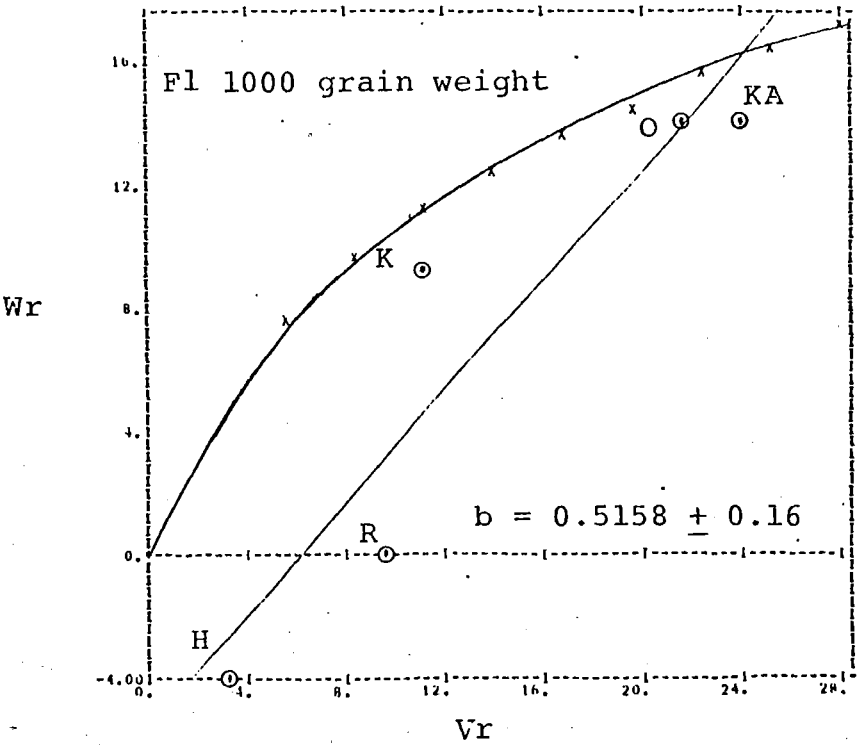
	Spikes/plant	Average 1000 grain wt. (gms)	Grain wt./plant (gms)
Hilgendorf x Kopara	6.4	40.7	13.4
Hilgendorf x Oroua	5.9	43.6	10.4
Hilgendorf x Ruru	6.7	39.6	13.7
Hilgendorf x Karamu	5.9	41.1	9.6
Kopara x Oroua	6.6	37.2	13.7
Kopara x Ruru	5.6	34.2	12.2
Kopara x Karamu	5.7	32.6	10.7
Oroua x Ruru	6.9	39.4	12.7
Oroua x Karamu	6.0	33.2	10.6
Ruru x Karamu	6.3	39.9	10.5
Hilgendorf	6.2	38.7	8.3
Kopara	5.8	33.1	10.2
Oroua	7.0	32.2	9.3
Ruru	7.3	33.7	11.4
Karamu	5.5	29.8	8.2
S.E. of Mean	0.30	0.01	0.63
C.V.	21.1%	11.4%	25.7%

grain weight. This was further confirmed by the correlation coefficient of $W_{ri} + V_{ri}$ with P_i . The correlation coefficient was -0.9087 ± 0.2410 and it was highly significant. Dominance for higher grain weight found in the present study have also been shown by Bhatt (1972) and Paroda and Joshi (1970). The additive effect or 'a' item was also significant.

The W_r - V_r relationships are shown in Figure 4.1. The graph showed a random distribution of the W_r - V_r values and the joint regression coefficient was 0.5158 ± 0.1629 . This is indicative of the lack of fit for the additive and dominance model. Non-allelic interaction may be present. This absence of fit for the additive and dominance model and the implicit presence of epistasis was mainly because of the expression of hybrid vigour in some of the F_1 families.

4.3.2 *F2 Half Diallel Analysis of 1000 Grain Weight*

The comparison of means for Experiment II as shown in Table 4.4 confirmed the high 1000 grain weight of cultivar Hilgendorf. However, cultivar Ruru which exhibited moderately high 1000 grain weight during the previous growing season (Experiment I), produced grain of extremely low 1000 grain weight for this season. This can be attributed to the serious occurrence of 'speckled leaf blotch' caused by *Septoria tritici* (Gaunt, pers. comm. 1979). The other three cultivars showed similar relative values, although differing in their absolute values to those of Experiment I. The analysis of variance of the Half Diallel shown in Table 4.5 confirmed the significance of the 'a' and 'b' items. The importance of directional dominance was reinforced by the highly significant



$K \propto R$

FIG. 4.1 The relationship between W_r and V_r for the F1 and F2 generations for 1000 grain weight.

Table 4.2 F1 Half Diallel Analysis of spikes per plant, 100 grain weight and grain weight per plant after Morley Jones (1965).

Item	df	Spikes/plant MS	1000 Grain Wt. MS	Grain Wt. MS
a	4	1.3484*	55.6002**	9.2057**
b	10	0.2785	25.2730*	5.6236**
b1	1	0.2042	144.0570***	33.9122***
b2	4	0.1080	7.6397	2.0411
b3	5	0.4298	15.6228	2.8319
B x a	4	0.2731	10.2153	1.4833
B x b	10	0.2982	6.0354	1.3240
B x b1	1	0.0482	0.4806	0.6020
B x b2	4	0.5296	11.0932	1.9963
B x b3	5	0.1632	3.1002	0.9305
Block Interaction	14	0.2910	7.2297	1.3695

* P < 0.05 ** P < 0.01 *** P < 0.001

Table 4.3 Linear regression coefficient for testing the adequacy of the additive and dominance model for spikes per plant, 1000 grain weight and grain weight per plant.

Expt.	Trait	Linear Regression coefficient	SE ±	Probability from 1.0	Significance from 0.0
I	Spikes/plant	0.5835	0.2801	0.4612	0.3125
	1000 grain wt.	0.5158	0.1629	0.2756	0.2485
	Grain wt./plant	0.2685	0.2113	0.1626	0.5804
II	Spikes/plant	0.3311	0.0324	0.0099	0.1155
	1000 grain wt.	0.9652	0.1358	0.9279	0.0396
	Grain wt./plant	0.1548	0.0541	0.0109	0.5304

Table 4.4 Estimates of number of spikes per plant, 1000 grain weight, and total grain weight per plant in the F2 Half Diallel Crosses - Experiment II.

	**Spike/plant	*Average 1000 grain wt. (gms)	*Total Grain Wt./ plant (gms)
Hilgendorf x Kopara	6.5	42.6	9.1
Hilgendorf x Oroua	7.7	43.3	10.4
Hilgendorf x Ruru	7.2	45.6	11.2
Hilgendorf x Karamu	6.8	44.8	9.4
Kopara x Oroua	6.3	42.9	10.2
Kopara x Ruru	7.0	40.2	11.1
Kopara x Karamu	6.2	39.1	9.7
Oroua x Ruru	8.7	40.8	13.6
Oroua x Karamu	6.6	39.3	9.9
Ruru x Karamu	7.2	40.3	10.8
Hilgendorf	6.7	45.9	8.2
Kopara	6.5	40.6	10.5
Oroua	7.7	38.5	10.9
Ruru	7.7	31.8	10.2
Karamu	6.3	36.7	8.5
S.E.M.	0.26	1.18	0.83
C.V.	29.10%	4.09%	11.52%

* Derived from replicate block means.

** Derived from single plant counts.

Table 4.5 F2 Half Diallel analysis of spikes per plant, 1000 grain weight and total grain weight per plant after Morley Jones (1965).

Item	df	Spikes/plant	1000 Grain Wt.	Total Grain Wt.
		MS	MS	MS
a	4	2.6454**	58.4736***	6.9573**
b	10	0.3534	14.3644**	1.9179
b1	1	0.0116	68.3051***	5.3485*
b2	4	0.3043	12.4636*	2.4455
b3	5	0.4611	5.0908	0.8098
B x a	4	0.1110	1.6735	0.7955
B x b	10	0.4824	3.2380	1.6350
B x b1	1	1.0578	13.9472*	0.6341
B x b2	4	0.0808	2.4818	1.3090
B x b3	5	0.6887	1.7011	2.0961
Block Interaction	14	0.3763	2.7910	1.3952

* P < 0.05

** P < 0.01

*** P < 0.001

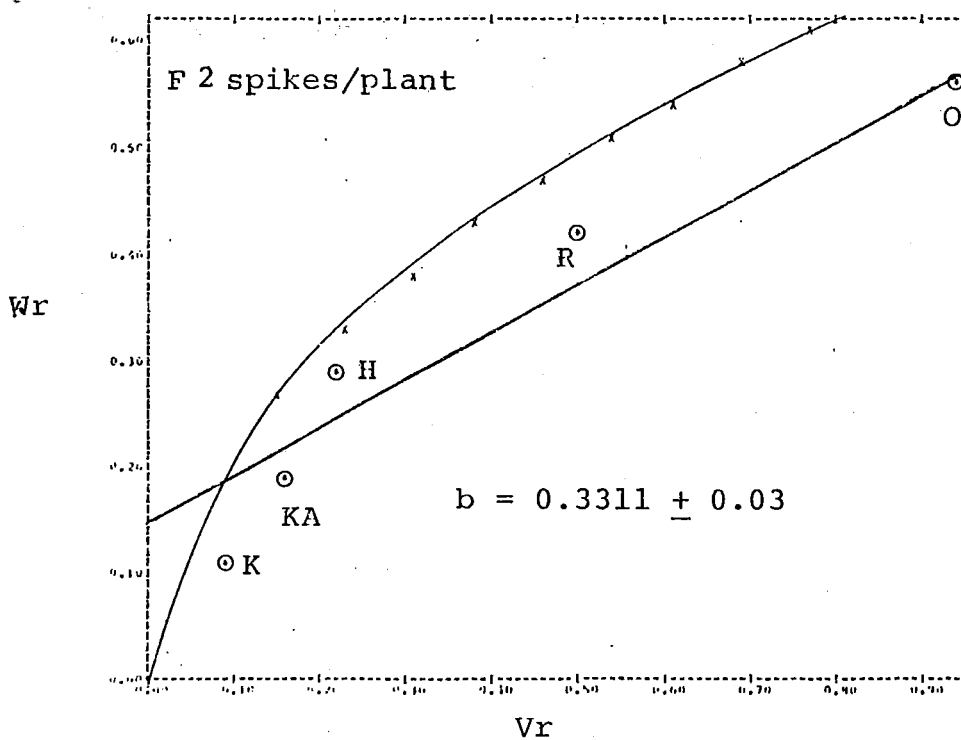
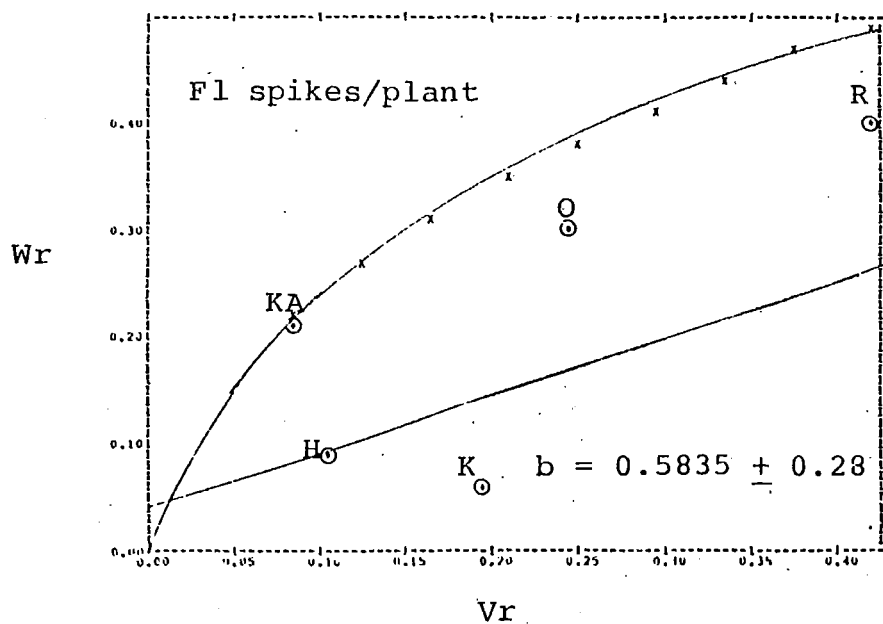


FIG. 4.2 The relationship between W_r and V_r for the F1 and F2 generations for spikes per plant.

'b1' items. As observed in the F1 Diallel analysis, the directional dominance was towards larger grain size and this was emphasized by the correlation coefficient of Wri + Vri with Pi which was of the order of -0.9106 ± 0.2386 ($P = 0.0316$).

The regression analysis of the Wr-Vr graph (Figure 4.1) for Experiment II is not in agreement with that of the F1 generation of the previous season (Experiment I). While the results of Experiment I showed an absence of fit for the additive and dominance model, the test of fit for the additive and dominance model was found to be adequate for Experiment II. The joint regression coefficient as shown in Table 4.3 was 0.9652 ± 0.1358 which was not significant from one but highly significant from zero. The discrepancy of results in the two seasons was due to an absence of F2 progeny mean significantly above the parents. The F1 hybrid vigour resulting in crosses with higher 1000 grain weights than their parents gave rise to an absence of fit for Experiment I, while the absence of such vigour in the F2 generation of Experiment II had resulted in the failure to duplicate the F1 results.

4.3.3 *New Triple Test Cross and Scaling Test Analyses of 1000 Grain Weight*

The presence of epistasis in 1000 grain weight as suggested by the F1 Diallel analysis is also shown by both the New Triple Cross and Scaling Test Analyses. The two epistastic comparisons, Ai and Bi, showed significant tests for epistasis. A review of the contributions to the sum of

Table 4.6 Mean 1000 grain weight of mainstem of parents (Pi), common tester (Pc), F1, F2, Bc1 and Bc2 families - Experiment III.

Parents Generation	Hilgendorf	Kopara	Oroua	Ruru	Atlas 66
Pi	46.3	39.6	38.4	40.7	37.8
Pc	40.5	41.8	42.3	40.6	40.3
B1	45.5	39.3	39.3	43.9	40.7
B2	48.4	41.3	42.8	52.0	44.8
F1	48.3	41.8	41.7	45.3	42.3
F2	45.2	40.7	41.1	42.9	41.2

Standard Error of Mean = 0.9021

Coefficient of Variation = 14.17%

Table 4.7 Sums of squares of comparisons for detecting epistasis on 1000 grain weight of the mainstem.

Parent (Pi)	Ai Comparison Sums of Squares	Bi Comparison Sums of Squares
Hilgendorf	31.88	40.34
Kopara	68.36	173.59
Oroua	40.88	372.28
Ruru	430.87	826.18
Atlas 66	36.61	120.68
Total	608.59	1533.07
Mean Square	13.52	34.06
Pooled Variance	4.32	5.99
F Ratio	3.13**	5.68***

* P < 0.05 ** P < 0.01 *** P < 0.001

Table 4.8 Scaling Tests and estimates of parameters for 1000 grain weight on the mainstem.

Parent (Pi)	Hilgendorf	Kopara	Oroua	Ruru	Atlas
Test					
A	1.88	2.76	2.13	-6.92*	2.02
B	2.12	-4.39	-6.43***	-9.58**	-3.66
C	-3.02	-1.11	-1.95	-23.37***	-2.98
S	-1.75	0.13	0.59	-1.72	-0.34
Estimates					
m	36.38***	41.20***	42.68***	33.75***	37.72***
d	2.86***	-1.09*	-1.92***	0.06	-1.26**
h	23.04	-2.07	-6.51	13.47	6.73
i	7.02	-0.52	-2.35	6.87	1.34
j	-0.24	7.15*	8.56***	2.66	5.56*
l	-11.02	2.16	6.65	9.63	0.30
Joint Test					
m	43.59 + 0.45	40.73 + 0.18	39.931 + 0.46	40.46 + 0.39	39.13 + 0.41
d	2.94 + 0.44	-0.78 + 0.49	-1.02 + 0.46	0.13 + 0.38	-0.79 + 0.40
h	5.17 + 0.83	0.53 + 0.77	2.48 + 0.78	8.88 + 1.31	5.72 + 0.60
χ^2 (3)	3.59 ns	4.32 ns	16.27***	11.75**	6.59
* P < 0.05	** P < 0.01	*** P < 0.001			

squares of the Ai and Bi epistasis comparisons (Table 4.7) indicated the role of the two parents Ruru and Oroua in the expression of epistasis. Cultivar Ruru contributed to more than half of the total sums of squares for both the Ai and Bi comparisons and cultivar Oroua provided a significant contribution to the Bi comparison. The significant contribution of the cultivars Ruru and Oroua to the epistatic sums of squares was further reinforced by the individual Scaling Tests shown in Table 4.8. The Scaling Test Analysis showed cultivar Ruru to be significant in the A, B and C Scaling Tests and to have a highly significant χ^2 test in the Cavalli's Joint Scaling Test. This emphasized the inadequacy of the three parameter model of m, (d) and (h) for this trait, in the crosses involving cultivar Ruru. Cultivar Oroua, on the other hand, showed significance in only the B Scaling Test. The χ^2 test of goodness of fit for the three parameter model of m, (d) and (h) was also highly significant indicating an absence of such fit.

4.3.4 *F1 Half Diallel Analysis of Spikes Per Plant*

Cultivars Ruru and Oroua produced the highest number of spikes per plant (Table 4.1). The other three parents were not significantly different in their ability to produce spikes. In field populations, Karamu has been shown to be able to produce more tillers per plant than Kopara (Fraser and Dougherty, 1977). The inability to discriminate between these three parents highlights the difficulty of estimating this trait in this study where only ten plants per block were used. The major problem was the extreme variability encountered

where some plants within the same family had more than double the mean number of spikes. The Diallel analysis based on these means must then be interpreted with utmost caution. The Half Diallel analysis of variance shown in Table 4.2 showed only significance for the additive effect. There was an absence of significance for the dominance effect. However, the Wr-Vr graphical analysis showed the Wr-Vr points to be distributed at random and the joint regression coefficient was 0.5835 ± 0.2811 . This is indicative of the lack of fit for the additive dominance model with implication of non-allelic interaction or epistasis. The presence of epistasis was due to F1 producing fewer spikes per plant than their parents. The conclusion of the presence of epistasis could be accepted if this trait had been determined with sufficient precision. However, in the analysis of this trait, where the expression of spikes per plant is not sufficiently accurately determined, this conclusion must be deemed inappropriate. The difficulty of genetical work with this trait has been emphasized by McNeal *et al.* (1978). For the F2 Half Diallel studies in the next section, the sample size per block has been increased to thirty, in the hope of obtaining a more accurate estimate for this trait.

4.3.5 F2 Half Diallel Analysis on Spikes Per Plant

The mean spike counts of Experiment II, as shown in Table 4.4, confirmed the high spike forming potential of cultivars Oroua and Ruru recorded in Experiment I. Both these parents produced significantly more mature spikes than cultivars Hilgendorf, Kopara and Karamu. The Half Diallel analysis

again showed significance of only the 'a' or additive effects. The 'b' and its component items were all not significant due mainly to the large block interaction mean square used for testing their significances (Table 4.5). The Wr-Vr graphical analysis and the joint regression analysis contradict the Diallel analysis of variance discussed above. The Wr-Vr points were distributed at random and the joint regression coefficient was 0.3311 ± 0.0324 which was highly significant from one but not from zero (Figure 4.2). This is indicative of a lack of fit to the additive and dominance model. This absence of fit is caused by the mean value of the cross Oroua x Ruru which significantly exceeded both parents in their mean values. This could have been reflected as a significance for the 'b3' item had the block interaction mean square been of a lower magnitude. The high sampling variability encountered in Experiment I continued to be a problem in Experiment II. This is despite the larger sample size of 30 plants per block. The within family variances of the parental cultivars were similar to those of the segregating F2 families. This emphasized the difficulty of separating the genetic differences from those due to the environment. The coefficient of variation for Experiment II was of the order of 29.10 per cent and this accentuated the difficulty of interpreting the results of the Diallel analysis for this trait.

4.3.6 *The New Triple Test Cross and Scaling Test Analyses on Spikes Per Plant*

The high coefficient of variation of 26.7 per cent for spikes per plant as recorded in Experiment III (Table 4.9) continued to affect the genetical analysis by increasing the within family variances and the difficulty of making accurate estimates of family means. This is clearly reflected in both the New Triple Test Cross and the Scaling Test analyses, both of which used single plant datum for the derivation of their family means and variances. The sums of squares of the epistatic comparisons on spikes per plant of the New Triple Test Cross analysis are shown in Table 4.10. Both the Ai and Bi comparisons gave rise to mean squares which, when tested against their respective large pooled variances, gave F ratios which were marginally significant.

A closer scrutiny of Table 4.10, however, showed a disproportionate contribution of each of the family arrays to the total sums of squares of both the Ai and Bi comparisons. For the Ai comparison, the Oroua and Atlas 66 arrays were the dominant contributors to the total sum of squares, whereas for the Bi comparison, only the Hilgendorf array was the dominant contributor. There is therefore, suggestion of epistasis in some of the families arrays as indicated by their larger contribution to the sums of squares in both the Ai and Bi comparisons.

The evidence for the presence of epistasis in some of the families is further strengthened by the Scaling Test analysis shown in Table 4.11. Significant Scaling Tests were

Table 4.9 Mean number of spikes per plant of parents (Pi) and common tester (Pc), F1, F2, Bc1 and Bc2 families - Experiment III.

Parents Generation	Hilgendorf	Kopara	Oroua	Ruru	Atlas 66
Pi	7.0	5.9	8.0	7.2	6.5
Pc	5.9	6.2	5.9	6.0	6.2
B1	6.7	6.7	6.8	6.9	7.5
B2	7.0	6.6	7.0	6.7	6.7
F1	6.8	6.3	7.2	6.5	6.8
F2	7.4	6.1	6.5	7.0	6.8

Standard Error of Mean = 0.2693

Coefficient of Variation = 26.97%

Table 4.10 Sums of squares of comparisons for detecting epistasis on spikes per plant.

Parent (Pi)	Ai Comparison Sum of Squares	Bi Comparison Sum of Squares
Hilgendorf	1.44	22.40
Kopara	11.11	4.27
Oroua	20.55	5.76
Ruru	0.00	6.42
Atlas 66	25.00	1.78
Total	58.10	40.63
Mean Square	1.30	0.90
Pooled Variance	0.43	0.38
F Ratio	3.02*	2.37*

* $P < 0.05$

Table 4.11 Scaling Tests and estimates of parameters for number of spikes per plant.

Parent (Pi) Test	Hilgendorf	Kopara	Oroua	Ruru	Atlas 66
A	-0.40	1.11	-1.51*	0.00	1.67*
B	1.58*	0.69	0.80	0.84	0.44
C	3.34**	-0.39	-2.38*	1.91	0.91
S	0.54	-0.55	-0.41	0.27	-0.30
Estimates					
m	8.62***	3.87***	5.29***	7.66***	5.14***
(d)	0.57**	-0.14	1.04***	0.64***	0.17
(h)	-2.85	6.43**	2.86	-1.34	4.97
(i)	-2.16	2.19	1.66	-1.1	1.20
(j)	-1.98*	0.42	-2.31**	-0.84	1.22
(l)	0.99	-3.99*	-0.95	0.22	-3.31
Joint Test					
m	6.52 + 0.17	6.12 + 0.16	6.77 + 0.17	6.67 + 0.15	6.46 + 0.15
(d)	0.43 + 0.16	-0.11 + 0.16	0.79 + 0.16	0.63 + 0.15	0.26 + 0.14
(h)	0.64 + 0.33	0.28 + 0.27	0.13 + 0.33	0.12 + 0.29	0.65 + 0.29
χ^2 (3)	13.06**	4.51 ns	12.01**	4.78 ns	6.24 ns
* P < 0.05	** P < 0.01	*** P < 0.001			

recorded in families with disproportionately high sum of squares in the New Triple Test Cross analysis. For example, the Hilgendorf, Oroua and Atlas 66 family arrays have at least one of the A, B or C Scaling Test showing significance. Moreover, the Cavalli's Joint Scaling Test was found to be significant for both the Hilgendorf and Oroua families. The significance of this χ^2 test, for absence of fit in the three parameter model of mean, additive and dominance, indicated presence of non-allelic interactions.

4.3.7 *F1 Half Diallel Analysis of Yield Per Plant*

Yield per plant as measured by grain weight showed significant variation among parents (Table 4.1). Cultivar Ruru had the highest yield, followed by Kopara, Oroua, Hilgendorf and Karamu. Karamu showed an abnormally low yield, probably because of the serious occurrence of leaf rust. This cultivar has been shown to be capable of producing high yields (Smith, 1974; McEwan, 1973). Most of the F1 plants exceeded the parents in their yielding capacity and exhibited varying degrees of hybrid vigour.

The Half Diallel analysis of variance showed significant additive and dominance variation for this trait. The dominance variation was mainly of the directional type, with item 'b1' highly significant. The Wr-Vr graphical analysis showed the absence of fit for the additive and dominance model. The joint regression coefficient was neither significant from zero nor from one (Table 4.3), confirming the presence of genetic variation other than of the additive and dominance type. The presence of non-allelic interaction is well accepted in

yield. The absence of fit for the additive and dominance model confirmed the presence of epistasis. This emphasized the difficulty of trying to fix the high yielding segregants by early generation selection.

4.3.8 *F2 Half Diallel Analysis on Yield Per Plant*

The results of the second season (Experiment II) as shown in Table 4.4 demonstrated the high yielding potential of cultivar Oroua. Cultivars Kopara and Ruru retained their relative high yielding standing, as was recorded in Experiment I. They exceeded cultivars Hilgendorf and Karamu by approximately two grammes per plant. Cultivar Karamu continued to exhibit the low yield recorded in the previous season. This could be indicative of the poor yielding capability of this cultivar under spaced planted condition. The high yielding potential of this cultivar, under conventional planting, has been well documented and has been discussed in Section 4.3.7. Cultivar Hilgendorf again produced the lowest grain yield recorded in the parents. The cross, Oroua x Ruru, exceeded both parents in their grain yielding capacity.

The Half Diallel analysis of variance of Table 4.5 showed significant 'a' and 'bl' effects, indicating presence of both additive effect and directional dominance. The Wr-Vr graphical analysis of Figure 4.3 showed the points distributed at random. Moreover, the joint regression coefficient of the Wr-Vr graph was neither significant from zero nor one (Table 4.3). This is indicative of the inadequacy of the additive and dominance model and suggests the presence of non-allelic

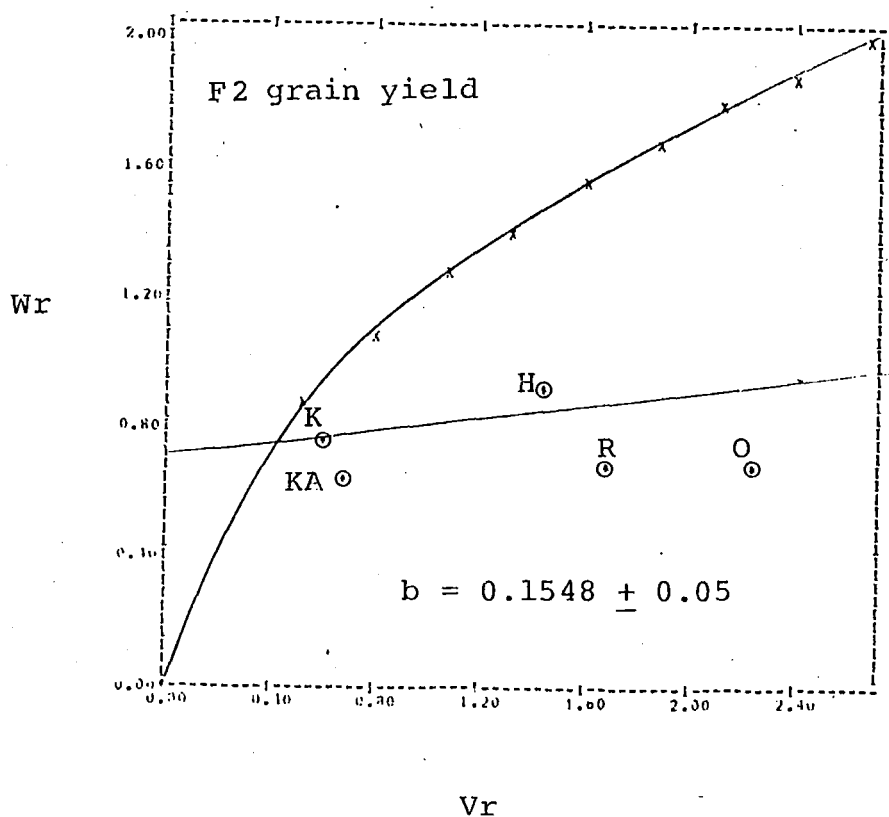
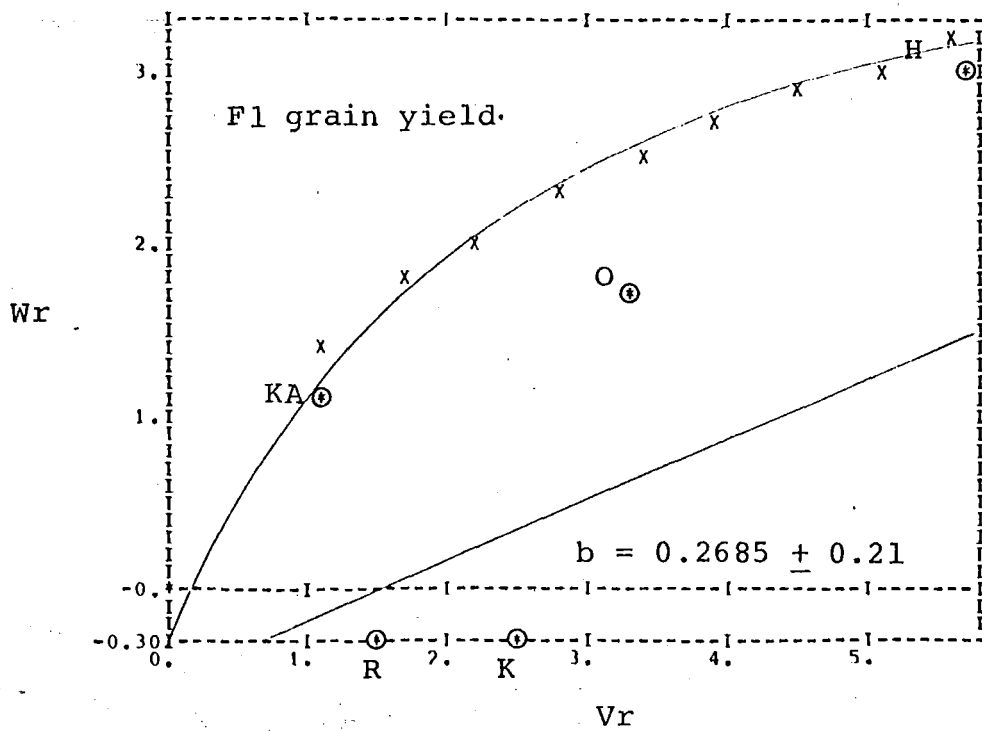


FIG. 4.3 The relationship between W_r and V_r for the F1 and F2 generations for grain yield.

Table 4.12 Mean yield (spike weight per plant) of parents (Pi) and common tester (Pc), F1, F2, Bc1 and Bc2 families - Experiment III.

Parents Generation	Hilgendorf	Kopara	Oroua	Ruru	Atlas 66
Pi	15.2	17.0	20.2	19.7	14.5
Pc	16.2	18.1	17.7	17.1	16.9
B1	16.8	21.7	18.9	20.6	19.4
B2	19.1	19.3	18.1	20.4	18.6
F1	18.8	21.6	19.4	19.6	20.1
F2	19.4	18.3	17.4	19.0	18.1

Standard Error of Mean = 0.9935

Coefficient of Variation = 35.63%

Table 4.13 Sums of squares of comparisons for detecting epistasis on yield (spike weight per plant).

Parent (Pi)	Ai Comparison Sum of Squares	Bi Comparison Sum of Squares
Hilgendorf	0.62	97.13
Kopara	211.20	14.36
Oroua	34.23	6.67
Ruru	28.74	143.70
Atlas 66	148.21	0.02
Total	423.00	261.88
Mean Square	9.40	5.82
Pooled Variance	6.02	5.31
F Ratio	1.56 ns	1.10 ns

ns non significance

Table 4.14 Scaling Tests and estimates of parameters for yield (spike weight per plant).

Parent (Pi)	Hilgendorf	Kopara	Oroua	Ruru	Atlas 66
Test					
A	-0.26	4.84	-1.95	1.79	4.06
B	3.29	-1.26	-0.86	3.99	0.04
C	8.83*	-5.27	-7.16	-0.14	0.66
S	1.45	1.15*	-1.09	-1.48	-0.86
Estimates					
m	21.47	8.72*	14.60***	12.47**	12.29
d	-0.49	-0.56	1.29*	1.33*	-1.19*
h	-5.50	25.33*	6.37	18.87	15.39
i	-5.80	8.85*	4.35	5.92	3.44
j	-3.55	6.11	-1.09	-2.21	4.01
l	2.78	-12.43	-1.53	-11.71	-7.55**
Joint Test					
m	15.90 + 0.50	17.48 + 0.66	18.57 + 0.57	18.54 + 0.58	15.91 + 0.49
d	-0.75 + 0.49	-0.11 + 0.67	1.06 + 0.56	1.25 + 0.57	-0.91 + 0.48
h	4.02 + 1.09	3.92 + 1.17	-0.20 + 1.15	1.81 + 1.21	4.85 + 1.03
X ²	5.90 ns	6.13 ns	3.39 ns	2.72 ns	3.46 ns

* P < 0.05

** P < 0.01

*** P < 0.001

interaction.

4.3.9 *New Triple Test Cross and Scaling Test*

Analyses on Yield Per Plant

The New Triple Test Cross analysis of Experiment III on spike weight per plant as shown in Table 4.13 provided no evidence for the presence of epistasis. The A_i and B_i comparisons testing the presence of epistasis were not significant. The absence of significance for the A_i and B_i comparisons was further reinforced by the A, B, C and S Scaling Tests and the Cavalli's Joint Test. The results of these tests are shown in Table 4.14. There is a general agreement on the adequacy of the three parameter model of m , (d) and (h). The results of the New Triple Test Cross and the Scaling Test analyses are therefore in disagreement with those of the F_1 and F_2 Diallel analyses. The Half Diallel analyses have provided evidence of absence of fit for the simple additive and dominance model. These differences in the outcome of these three biometrical genetical models epitomize the sensitivity or insensitivity of each of the models which is discussed in Section 4.4.1.

4.4 DISCUSSION

4.4.1 *Yield and Spikes Per Plant*

The complex inheritance of yield has been well documented (Whitehouse et al., 1958; Hsu and Walton, 1970; Paroda and Joshi, 1970; Chapman and McNeal, 1971; Singh and Singh, 1976; Ketata et al., 1976b). Low narrow sense heritability of this

trait as recorded by McNeal (1960), Kronstad and Foote (1964), Johnson et al. (1966) and Fonseca and Patterson, (1968), testified to the difficulty of achieving a selection response. McNeal et al. (1978) and Knott (1979) have recorded such poor response for direct selection for yield, whereas O'Brien et al. (1978) have managed to achieve a selection response for two of their four crosses.

Reports on the inheritance of the yield component, spikes per plant are conflicting. There were reports of simple additive and dominance inheritance to more complex inheritance involving epistasis (Whitehouse et al., 1958; Lupton, 1961; Panigrahi, 1962; Kronstad and Foote, 1964; Paroda and Joshi, 1970; Singh and Anand, 1971; Ketata et al., 1976a, b; Gill et al., 1977). These differences have been attributed to differences in the genetic background of the parents (Lupton, 1961), and the presence of genotype environmental interactions was not discounted by Ketata et al. (1976b). Our results from the Half Diallel analysis, the New Triple Test Cross and Scaling Tests analyses on these two traits are not in agreement. While it is convenient to attribute the conflicting results to genotype environmental interactions and differences in the genetical background of the parents, we are of the opinion that the absence of agreement in our results could be explained by the sensitivity of each biometrical model to the presence of high sampling variation. In the test for the adequacy of the additive and dominance model of the Half Diallel analyses, the covariances (W_{ri}) and variances (V_{ri}) of the family means are used. In a situation where high within family variances are present, estimates of the family means will be correspondingly biased.

The use of the biased family means (simulating genetic effects) will inflate the covariances (W_{ri}) and variances (V_{ri}), resulting in the random distribution of the W_r - V_r points. This will result in an absence of fit for the additive and dominance model which must necessarily be misleading. On the other hand, the high within family variances which are pooled for testing the presence of epistasis in the New Triple Test Cross and the Scaling Test analyses will increase the difficulty of achieving significant epistasis. It is therefore, obvious that the genetical interpretations, derived from different biometrical models of traits showing great variations, should be viewed with caution. This study may have failed to produce a uniform conclusion on the nature of gene actions for these traits. However, it may have added another dimension to the explanation of the contradictory results produced by the genetical analyses of highly variable traits. Difficulty in achieving consistent genetical results may simply be attributed to micro-environmental influences rather than genotype environmental interactions. Differing micro-environments caused by the inability to control the spatial relationship between plants, due to diseases or mortality, could play an important role in determining the variability of spike formation and yield. Difficulty of direct selection for these two traits have been discussed by McNeal *et al.* (1978). Prospects for genetical advancement of these traits by direct selection will continue to be weakened by the complication from epistasis, genotype environmental interactions and micro-environmental influence on these traits.

4.4.2 1000 Grain Weight

The different conclusions reached in the F1 and F2 generations of the Half Diallel analyses on 1000 grain weight could possibly be explained by the halving of the dominance effect in the F2 generation. This loss of dominance will result in reduced hybrid vigour. Our inability to record an F2 family mean exceeding significantly the parental means may explain the failure to obtain the same result as the F1 generation analysis. However, the evidence provided by both the New Triple Test Cross and the Scaling Test analyses is in agreement with that of the F1 generation analysis. These supports for the presence of epistasis are in line with reports by Lupton (1961) and Ketata *et al.* (1976a), although simpler genetic control has been recorded by various workers (Hsu and Walton, 1970; Bhatt, 1971; Chapman and McNeal, 1971). Response to selection for this trait has been discussed by Knott and Taludar (1971) and McNeal *et al.* (1978). Expression of epistasis in our study is confined to crosses involving cultivars, Oroua and Ruru. It is conceivable that grain size improvement could be achieved in the context of breeding among our other parental cultivars. Cultivar Hilgendorf, a high 1000 grain weight cultivar, could conceivably be crossed with cultivar Kopara for 1000 grain weight selection.

CHAPTER 5

THE GENETICAL CONTROL OF PLANT HEIGHT,
SPIKE LENGTH AND FLAG LEAF LENGTH IN WHEAT

5.0 REVIEW OF LITERATURE

Plant Height

Plant height in wheat has been a subject of intense interest to wheat breeders prior to the 1940's. This is because of the strong association between height and grain yield. The belief that only tall wheat had potential for high yield was accepted as fact by most breeders during the 1940's (Briggle and Vogel, 1968). However, plant breeders never lost sight of the advantages of shorter wheats. The lodging resistance and early maturity types of shorter wheats were advantages that plant breeders were keen to incorporate into their new cultivars. In Britain, sturdy efforts by breeders brought the height down 30 cm from 130 cm over a period of 50 years (Lupton, 1975). This was done by selecting shorter strawed progeny from crosses between taller cultivars, taking advantage of a range of minor genes which determines height. No major breakthrough in breeding for height was recorded until the introduction of the semidwarf growth habit of Norin 10 into the wheat breeding programme of the Washington State University. This was acclaimed as the beginning of new records in the production efficiency of winter wheat (Vogel *et al.*, 1956). Norin 10 was crossed with Brevor and selection 14 from this cross has been extensively used as the basic material for the development of semidwarf wheats

throughout the world. This pioneering work on the introduction of the dwarfing genes of Norin 10 has resulted in the profound impact of the "Green Revolution". New Zealand's Karamu and the newly released cultivars Oroua and Rongotea have all been developed using the Mexican dwarfing derivatives of the original Norin 10 (McEwan and Viger, 1972; McEwan and Cross, 1978; McEwan and Hadfield, 1978).

The excitement over the outstanding success of the dwarfing genes in the development of modern wheat cultivars in the past twenty years has led breeders to intensify their selection for even shorter strawed wheats. The publication of the work on the genetical relationship between plant height and yield by Law *et al.* (1978) should serve as a timely warning to enthusiastic breeders pushing the straw length to its dwarf limit and therefore forfeiting any genetical association between yield and height. This warning was also carried by Stoskopf and Fairey (1975) when they stressed the growing awareness of special grain yield problems in progressively shorter strawed genotypes.

The genetical control of height in wheat can be divided into the major dwarfing genes and the numerous other quantitatively inherited genes. Two major dwarfing genes, namely *Rht1* and *Rht2*, have been identified as being responsible for the semidwarf characteristic of modern wheats which were derivatives of the Norin 10 (Vogel *et al.*, 1963; Hermsen, 1963; Allan *et al.*, 1968; Reddi and Heyne, 1970; Fick *et al.*, 1973). Two gibberellic acid insensitive genes *Gail* and *Gai2* have been identified and were thought to be located on the same chromosomes as *Rht1* and *Rht2* (Gale *et al.*, 1975; Gale and

Marshall, 1976). A third gibberellic acid insensitive gene, Gai3, found in another source of dwarfism, Tom Thumb, has been recorded (McVille *et al.*, 1978). This work also indicated that the genes Rht1/Gai1, Rht2/Gai2 and Rht3/Gai3 were probably not three pairs of linked genes but only three single genes expressing pleiotropic effects for dwarfism and gibberellic acid insensitivity.

Apart from the two major dwarfing genes, height has been shown to be a quantitative trait by Anwar and Chowdhry (1967) and genes affecting height have been located in seventeen of the twenty-one pairs of chromosomes (Snape *et al.*, 1977). F2 distribution studies by Novoa (1973) showed a small number of genes to be involved in the inheritance of height. Fick and Qualset (1973) indicated that four independently segregating loci accounted for most of the differences in height among the four cultivars. Halloran (1974), after eliminating the differential responses of photoperiod and vernalization, recorded genetical control of culm length by three genes. Quantitative genetical studies on plant height have revealed simple additive and dominance control to complex epistatic interactions. However, additive gene effects have been found to be the major component of the genetic variation (Johnson *et al.*, 1966; Bitzer *et al.*, 1971; Fick and Qualset, 1973; Bhatt, 1972; Gill *et al.*, 1973). The expression of dominance has been found to be towards loci increasing height (Merkle and Atkins, 1964; Allan *et al.*, 1968; Bhatt, 1972). In some crosses, epistatic effects were an important component. Epistasis in the inheritance of height has been recorded by Singh and Singh (1976, 1978). Snape *et al.* (1977) found epistatic effect of

the additive x additive type in their study of the cross Chinese Spring and Capella^l-Desprez. Fick and Qualset (1973) found that the primary source of genetical variation in one of the six crosses they studied was due to epistasis with additive x additive and dominance x dominance effects of major importance. Chapman and McNeal (1971) in their studies of plant height inheritance studied over two seasons, found height to have significant epistatic effect of the dominance x dominance type in one season and absence of epistatic effect in the second season.

Length of Spike and Flag Leaf

Plant physiologists have highlighted the role of photosynthetic area in the determination of grain yield. Watson et al. (1963) have stressed the contribution of leaf area and leaf area duration to grain yield. Thorne (1966) was of the opinion that photosynthetic area above the flag leaf was the major contributor to grain yield. Thorne (1965), in ^{her}his study of the photosynthesis of ears and flag leaves of wheat, found that seventeen per cent of the photosynthate would be assumed to come from the ear and the rest from the photosynthetic parts of the flag leaf and shoot. The importance of the stem and the ear was again emphasized by Evans and Wardlaw (1976) when they stated that an important characteristic of some cereals is the substantial photosynthetic contribution made by both the stems and inflorescences, particularly in the latter stages of grain growth when stem and ear photosynthesis can become a major source of current assimilate.

Walton (1969) and Hsu and Walton (1970a, b) were among the first workers to investigate the genetic nature of these

morpho-physiological traits discussed by plant physiologists. They were of the opinion that these traits could offer good prospects as additional basis to the conventional yield components as selection criteria for high yield. They were able to show that flag leaf area and flag leaf breadth were associated with yield. However, more recent work on near isogenic populations selected on the basis of flag leaf area have shown little difference in grain yield, and flag leaf area by itself did not appear to be a good index of grain yield (McNeal and Berg, 1977). Similar observations have been made by Watson *et al.* (1963). In their limited comparisons of old and new cultivars, they were able to demonstrate that continued selection and breeding to improve the grain yield of wheat had not altered the efficiency of the photosynthetic mechanism of the leaves, nor had it altered the leaf area of the plant in ways beneficial to yield. The reason why leaf area index had not been affected was not obvious to them, but they suggested that improvement of yield through change in leaf area index might have been prevented by simultaneous selection for characters other than yield, e.g. short, stiff straw to counter lodging. New cultivars yielded more grain partly because their ears emerged sooner and this lengthened the period of grain growth and the ears were able to photosynthesize more. They believed yield could be increased further by breeding cultivars with maximum leaf area index within the period of grain growth after the ears had emerged either by promoting still earlier emergence or by delaying senescence and the death of leaves.

The genetics of spike length have been studied by various workers. Novoa (1973) in his studies on the F₂ popu-

lation showed that the trait had a trimodal distribution and he concluded that this trait was controlled by a few genes.

Hsu and Walton (1970a) and Gill *et al.* (1973) have all demonstrated significant additive genetic effect for this trait. In another study, Gill *et al.* (1977) detected significant additive and dominance effect in one cross and only additive effect in another cross. Test for epistasis was found not significant. Evidence of partial dominance was provided by Hsu and Walton (1970a), while Singh and Singh (1976) detected epistasis in spike length. The inheritance of flag leaf area has been studied by Walton (1969) and by Balalic (1973), whereas flag leaf length was studied by Hsu and Walton (1970b). Partial dominance for leaf area was recorded by Walton (1969) and Balilic (1973). They also reported large and significant additive effect of this trait. Flag leaf length expressed both significant additive and dominance effect (Hsu and Walton, 1970b).

5.1 INTRODUCTION

Genetic studies of the traits plant height, flag leaf length and spike length are of considerable importance. This is mainly because of the direct association between height and yield and the contributions to the photosynthetic surfaces of the latter two characters. Direct measurement of the photosynthetic area of the flag leaf and spike would involve destructive sampling which cannot be carried out in the F₁ and the backcrosses because of severe limitations of seed numbers. The F₁ Half Diallel analysis was carried out as a preliminary study of the inheritance of these traits. More detailed under-

standing of the inheritance of these traits is provided by our studies of the F₂, the Triple Test Cross and the Scaling Test analysis.

5.2 MATERIALS AND METHOD

Ten plants per family per block of Experiment I were randomly tagged. The main stems of these ten plants which had been identified with colour tags soon after seedling emergence, were used for the studies on plant height, flag leaf length and spike length. Plant height was measured at harvest and was taken as the distance from the base of the stem to the tip of the spike excluding the awns. Length of spike was measured at harvest and was the length from the first spikelet node to the top of the last spikelet excluding the awns. Flag leaf length was measured soon after spike emergence and was taken as the length of the lamina from the position of the auricles to the tip of the leaf. The same methods of measurement on plant height, spike length and flag leaf length were used in Experiments II and III. Thirty plants per block were sampled in Experiment II and fifteen plants per block in Experiment III. The data for each trait were tested for family and block differences using the programme Teddy Bear (Wilson, 1979). The Half Diallel analyses (Morley Jones, 1965) for the F₁ and F₂ generations were carried out using the programme Binhalf (Appendix I). The individual datum measured on each plant for each trait was used in the New Triple Test Cross and the Scaling Test Analyses. These analyses were executed by the programmes Bintri and Bintest respectively (Appendices III and IV).

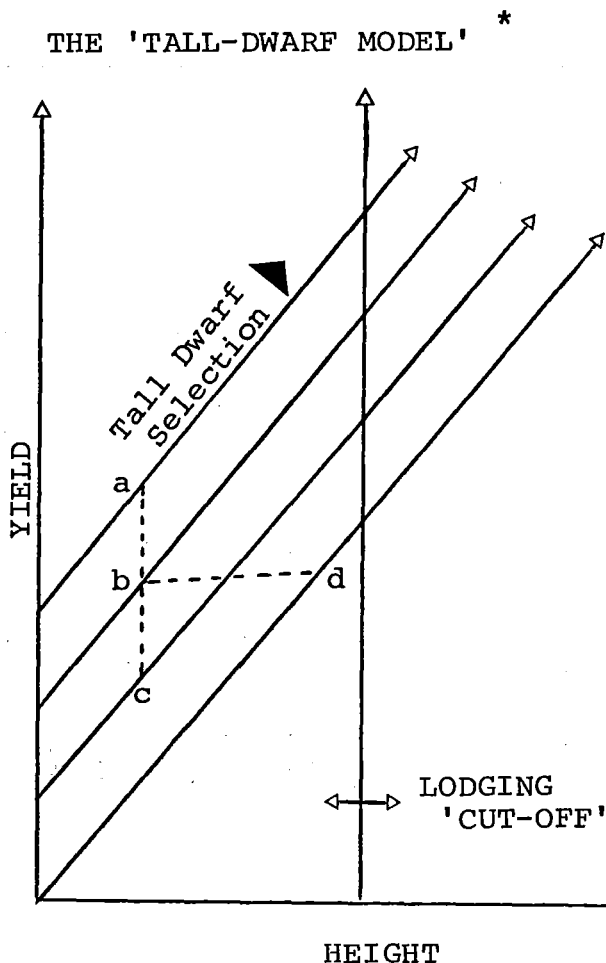


FIG. 5.1 A model showing the effects of selection for increased height in populations into which semi-dwarfing factors have been introduced. The 'lodging cut-off' is the plant height at which losses in yield due to lodging begin to occur.

- (a) indicates the substitution of an allele for dwarfism having a positive effect on yield.
- (b) substitution of an allele for dwarfism whose effect on yield is neutral or identical to the effect of the allele for tallness on yield.
- (c) substitution of an allele for dwarfism whose effect on yield is negative.
- (d) population carrying the tall allele.

*

After Gale, M.D. and Law, C.N. (1976).

Table 5.1 Mean height of parent and F1 plants.

	1	2	3	4	5
1. Hilgendorf	98.0	108.6	104.2	103.7	100.4
2. Kopara		101.1	103.8	100.0	99.9
3. Oroua			85.8	90.1	86.9
4. Ruru				79.1	85.4
5. Karamu					80.7

Standard Error of Mean = 1.04

Coefficient of Variation = 4.87%

Table 5.2 Mean height of parent and F2 families (cm).

	1	2	3	4	5
1. Hilgendorf	106.0	115.0	110.5	106.5	100.6
2. Kopara		110.9	108.5	103.8	105.8
3. Oroua			98.6	100.4	92.3
4. Ruru				87.0	87.0
5. Karamu					87.8

Standard Error Mean = 1.41

Coefficient of Variation = 10.80%

5.3 RESULTS

5.3.1 *F1 Half Diallel Analysis of Plant Height*

The mean heights of the parents and F1 families are shown in Table 5.1. The heights of the five parents can be divided into three different classes. The cultivars, Hilgendorf and Kopara, were the tallest with cultivar Oroua intermediate and cultivars Ruru and Karamu the shortest. Semi-dwarfness in the cultivars Oroua, Karamu (McEwan and Viger, 1973; McEwan and Hadfield, 1978) and Ruru (G.M. *Stall of height* Wright, pers. comm.) are expected because they have been developed from the Mexican dwarfing derivatives of Norin 10. On the other hand, cultivars Hilgendorf and Kopara are standard height wheats and they form the tall parents in this study. All the F1 plants exhibited dominance for tallness. The F1 means either approximated the tallest parents (cvs. Hilgendorf and Kopara) or exceeded them to give rise to a low degree of hybrid vigour for height. The F1 of the shorter parents all exceeded their parental means indicating again some low degree of hybrid vigour.

The Diallel analysis of variance for plant height in Table 5.3 showed highly significant additive and dominance effects for this trait. Moreover, the bl item of the analysis was highly significant indicating the presence of directional dominance for tallness. This directional dominance of the increasing phenotype, tallness was confirmed by the high and negative correlations (-0.9741 ± 0.1305) of the (Wri + Vri) values with Pi, the mean height of the parents. The distribution of the dominant and recessive genes among the parents

Table 5.3 F1 and F2 Half Diallel analysis of variance of plant height after Morley Jones (1965).

Source of Variation	df	Mean Square	
		F1	F2
a	4	455.40***	503.04***
b1	1	587.31***	166.83***
b2	4	17.00**	19.22*
b3	5	14.16**	18.11*
b	10	72.61***	33.43**
B x a	4	3.80	7.92
B x b1	1	1.40	0.05
B x b2	4	1.06	6.67
B x b3	5	3.77	4.83
B x b	10	2.45	5.09
Block Interaction	14	2.83	5.90

Table 5.4 Joint regression analysis of W_r on V_r for estimates from two blocks for F1 and F2 plant height.

Item	df	Mean Square	
		F1	F2
Joint regression	1	13396.87***	5241.39***
Heterogeneity of regression	1	4.47	105.89
Remainder	6	61.33	29.38
Joint regression coefficient		0.9946	0.8012
Standard error		0.0673	0.0600
Probability of significance from 1.0		0.9839	0.4480
Probability of significance from 0.0		0.0086	0.0170

* $P < 0.05$ ** $P < 0.01$ *** $P < 0.001$

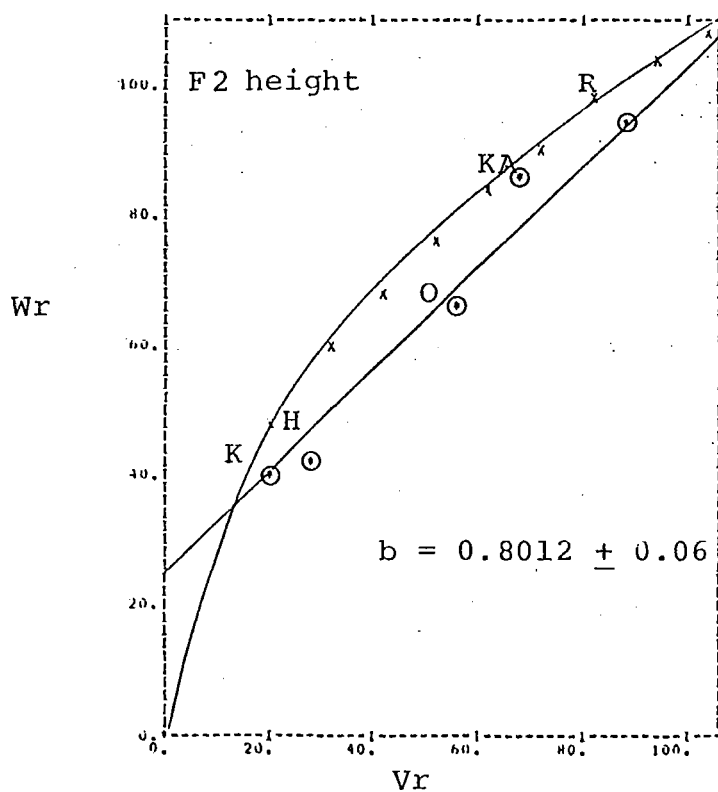
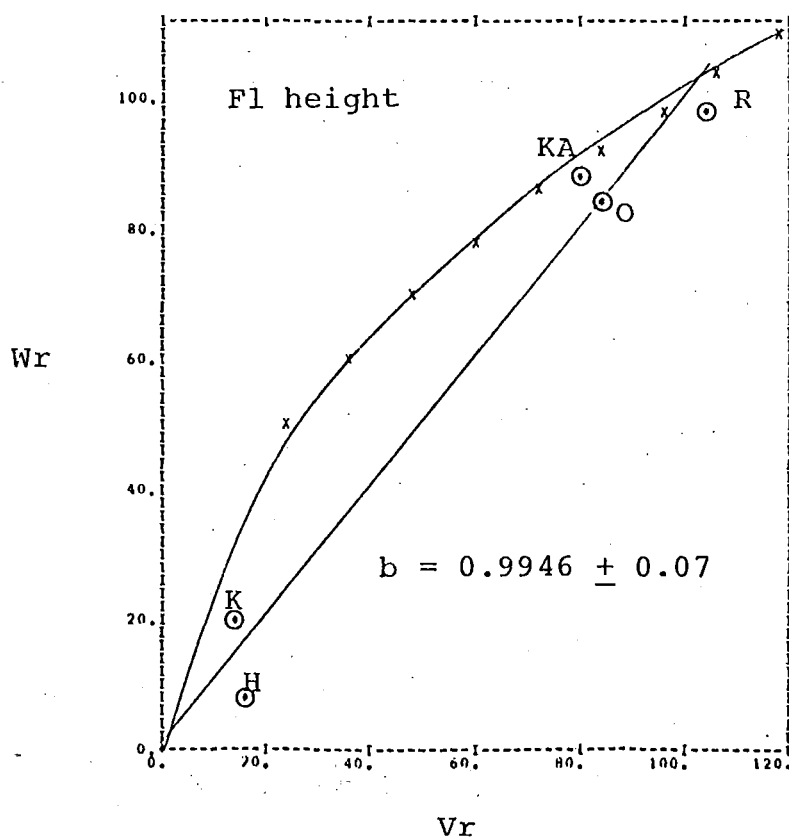


FIG. 5.2 The relationship between W_r and V_r for the F1 and F2 generations for height.

can be determined from the Wr-Vr graph of Figure 5.2. The two tallest cultivars, Hilgendorf and Kopara, had the smallest Wr-Vr values and were positioned nearest the origin, while the other three cultivars had relatively larger Wr-Vr values and were positioned furthest away from the origin of the graph. This confirmed that the two tallest cultivars carried the most dominant genes while the recessive genes were carried by the semidwarf cultivars. This is in agreement with reports on the recessive nature of the dwarfing genes of the Mexican derivatives (Vogel *et al.*, 1963; Ali *et al.*, 1968). The test for the adequacy of the additive and dominance model showed that the joint regression was highly significant and the heterogeneity of regression not significant. The joint linear regression coefficient was 0.9946 ± 0.0673 . This coefficient was not significantly different from one but significantly different from zero (Table 5.4). This is true if the non-additive genetic variation is of the dominance type only. Therefore, the additive and dominance model is adequate for plant height. With the additive and dominance model accepted as sufficient, perfect fit estimates can be made as in Table 5.4. Considering first the value of D and H1, the additive effect, D, is equal to the dominance effect H1 and therefore dominance is complete. The dominance ratio as given by $(H1/D)$ is 1.0067. The full dominance is again confirmed by the graph of Wr-Vr of Figure 5.2 where the regression line passes through the origin. The perfect fit estimates also provide estimates of broad and narrow sense heritabilities. The narrow sense heritability estimate of 0.7093 indicates that early response to selection for this trait is likely, while the high broad sense heritability

estimate of 0.9704 shows presence of the low environmental variance for this trait. Furthermore, the value of H_1 is equal to H_2 and $\frac{1}{4} (H_2/H_1)$ which represents the product $UV = 0.2487$. This indicates $U = V = 0.5$ at all loci for plant height.

5.3.2 *F2 Half Diallel Analysis of Plant Height*

The mean heights of the parents and F_2 families which are shown in Table 5.2 remained in the same relative order as those of Experiment I. The heights of all the families in Experiment II were generally of higher absolute values compared with those of Experiment I. This was mainly due to the early winter sowing of Experiment II compared to the late winter sowing of Experiment I.

The Half Diallel analysis of variance of the F_2 plant height reflected a similar pattern of significance for the 'a', 'b', 'b1', 'b2' and 'b3' items as that of the F_1 analysis of the previous season. However, the absolute values of the 'b' and 'b1' items were considerably reduced in the F_2 analysis compared to those in the F_1 analysis (Table 5.3). Such a reduction is expected because of the halving of the frequency of the heterozygotes in the F_2 generation. The directional dominance for tallness is again confirmed by the highly significant 'b1' item and the highly significant negative correlation of $(W_{ri} + V_{ri})$ values with the P_i values. This correlation coefficient was -0.9805 ± 0.1135 . The dominance of the tall parents is further shown by the W_r - V_r graph of Figure 5.2. The tall parents Hilgendorf and Kopara were all positioned near the origin, whereas the short parents

Table 5.5 Components of Genetic Variation of 5 x 5 Diallel Crosses for plant height.

Generation	F1	F2
Components		
D	99.6625	108.8092
H1	98.8999	212.0280
H2	98.6859	229.8300
$(H1/D)^{\frac{1}{2}}$	1.0067	1.3959
H2/4H1	0.2487	0.2709
E	2.8347	5.8950
Heritability (Narrow sense)	0.7093	0.7818
Heritability (Broad sense)	0.9704	0.9341

Table 5.6 Mean plant height of the parents (Pi), common tester (Pc), F1, F2 and backcrosses (BC1, BC2) - Experiment III.

Parents	Hilgendorf	Kopara	Oroua	Ruru	Atlas 66
Pi	106.6	107.6	98.3	87.2	143.8
Pc	89.8	89.9	89.0	88.9	89.5
BC1	104.0	106.0	96.1	93.2	127.7 138.7
BC2	100.2	100.3	93.3	91.6	122.5 111.6
F1	105.6	111.7	99.3	94.0	129.2
F2	100.2	103.8	93.5	88.5	117.2

Standard Error of Mean = 1.57

Coefficient of Variation = 10.11%

were all distributed away from the origin.

The test for the adequacy of the additive and dominance model shown in Table 5.4 showed a highly significant joint regression coefficient of 0.8012 ± 0.0600 . This regression was not significantly different from one but significant from zero. The additive and dominance model was thus acceptable. This confirmed the conclusion of the F1 analysis of Experiment I and allowed perfect fit estimates of some genetic parameters to be made. The perfect fit estimates for the genetic parameters are shown in Table 5.5. The dominance effect, H1, was considerably higher than the additive effect, D. This gave rise to the dominance ratio of 1.3959. This is higher than the ratio of 1.0067 obtained in the F1 generation. There is therefore, a suggestion of overdominance in the F2 generation. However, this is not supported by the Wr-Vr graph of Figure 5.3 which cut the axis near the origin. Moreover, the additive dominance model had been shown to be sufficient. This overdominance must be regarded as an over-estimate. As in the F1 studies, the narrow and broad sense heritability estimates were high.

5.3.3 *The New Triple Test Cross and Scaling Test Analyses of Plant Height*

The F1 and F2 Half Diallel analyses were conducted with five parents. These five parents were made up of three semidwarf cultivars and two standard height cultivars. In the Triple Test Cross and the Scaling Test analyses, an additional cultivar, Atlas 66, was included. This cultivar can be classified as tall when compared to the two standard height

Table 5.7 Sums of squares of comparisons for detecting epistasis on plant height.

Parent (Pi)	Ai Comparison Sum of Squares		Bi Comparison Sum of Squares	
	With Atlas 66	Without Atlas 66	With Atlas 66	Without Atlas 66
Hilgendorf	155.7504	155.7504	217.4642	217.4642
Kopara	481.6562	481.6562	7.0402	7.0402
Oroua	270.7122 557.5895	270.7122	26.7634	26.7634
Ruru	251.1168	251.1168	0.2916	0.2916
Atlas 66	2760.4516 175.4742	-	6248.3755 x 175.8276	-
Total	3919.6872 1621.45871	1159.2356	6499.9348 427.3869	251.5593
Mean Square	87.1042 36.0353	32.2010	144.4430 ✓ 18.9950 9.4975	6.9878
Pooled Variance	15.3574 13.6436	16.1128	14.7112 15.4358	16.0161
F Ratio	5.718 *** 2.6412**	1.9984 ns 0.6153	9.8186*** 0.6153 ns	0.4362 ns
*** P < 0.001	** P < 0.01	* P < 0.05		

cultivars, Kopara and Hilgendorf. The mean heights of the six generations of P1, P2, F1, F2, BC1 and BC2 generated by the five cultivars (Pi) crossed to the common tester, Karamu (Pc), are shown in Table 5.6. It can be seen that the cultivar Atlas 66 and the families derived from it exceeded all other families in their heights.

The New Triple Cross analysis of Table 5.7 showed highly significant Ai and Bi comparisons.* These significant tests testified to the presence of epistasis in the inheritance of plant height amongst the families studied. An evaluation of Table 5.7 immediately led to the identification of Atlas 66 array as the only dominant contributor to the epistatic sums of squares of the Ai and Bi comparisons. A re-analysis of the data without the Atlas 66 array confirmed the observation on the role of the Atlas 66 array in the expression of epistasis. The F tests of the Ai and Bi comparisons were both insignificant. These tests implied the absence of epistasis for the trait, plant height, for all the families not involving Atlas 66. These results confirmed the conclusions of both the F1 and F2 Half Diallel analyses.

The presence of epistasis in all the families with Atlas 66 parentage was again adequately emphasized by the Scaling Test analysis of Table 5.8. Significances were indicated by all the A, B, C, S and Cavalli's Joint Tests. These tests stressed the importance of non-allelic interactions leading to an absence of fit to the three parameters model of m, (d) and (h). The extent of non-allelic interactions was clearly shown by the significance of all three forms of (i), (j) and (l) epistasis. On the other hand, the Scaling

Table 5.8 Scaling Tests and estimates of parameters for plant height.

Parents (Pi)	Hilgendorf	Kopara	Oroua	Ruru	Atlas 66
Test					
A	-4.1600	-7.3156**	-5.4844	-5.2822	-17.5133***
B	4.9156	-0.8844	-1.7244	0.1800	26.3489***
C	-6.9600	-5.4781	-11.9860*	-10.0006	-22.8099*
S	-1.9289	0.6805	-1.1943	-3.8657	-7.9111
Estimates					
m	90.5156***	101.4375***	88.8606***	72.5638***	84.9822***
d	8.4044***	8.8400***	4.6311***	-0.8667*	27.1400***
h	23.5378	-0.6616	8.0343	42.4036*	84.7422***
i	7.7156	-2.7216	4.7771	15.4629	31.6444**
j	-9.0756*	-6.4311	-3.7600	5.1022	-43.8622***
l	-8.4711	10.9219	2.4317	-20.9251	-40.4800**
Joint Test					
m	98.11 + 0.39	98.37 + 0.48	92.24 + 1.04	87.99 + 0.13	115.55 + 0.69
d	8.24 + 0.39	8.57 + 0.48	3.36 + 1.06	-0.83 + 0.43	25.09 + 0.69
h	7.39 + 0.53	13.08 + 0.67	6.97 + 1.14	6.01 + 0.62	14.18 + 1.01
x ² (3)	5.85 ns	7.05 ns	4.95 ns	3.047 ns	107.01***
* P < 0.05	** P < 0.01	*** P < 0.001			

Table 5.9 The analysis of variance for the additive value for plant height (without the Atlas 66 array).

Source of Variation	df	SS	MS	F	Prob.
Mean	1	147.9582	147.9582	136.1186	0.0000***
Additive	4	275.1850	68.7962	63.2911	0.0000***
Deviation	3	127.2268	42.4089	39.0153	0.0002***
Block	2	11.8972	5.9486	5.4726	0.0444*
Error	6	6.5219	1.0870		
Total	12	293.6041			

The Estimate of the Additive Value = 90.2790

Table 5.10 The analysis of variance for the dominance value for plant height (without the Atlas 66 array).

Source of Variation	df	SS	MS	F	Prob.
Mean	1	141.2017	141.2017	22.1593	0.0033**
Dominance	4	164.6090	41.1523	6.4582	0.0230*
Deviation	3	23.4073	7.8024	1.2245	0.3795
Block	2	7.1678	3.5839	0.5624	0.5972
Error	6	38.2327	6.3721		
Total	12	210.0096			

The Estimate of the Dominance Value = 46.3735

Test analyses for the other parental arrays, were in general agreement on the absence of epistasis. All the A, B, C, S and Cavalli's Joint Tests were not significant except for the significance of one A test involving the Kopara array and one C test involving the Oroua array (Table 5.8). Moreover, in contrast to the Atlas 66 array, all the other estimates of the (i), (j) and (l) epistatic terms except for one, the (j) estimate of the Hilgendorf array, were not significant.

These analyses involving the F1, F2 Half Diallel, the New Triple Test Cross and Scaling Tests have provided conclusive evidence on the adequacy of the additive and dominance model for plant height in families not involving Atlas 66 as a parent. Adequate evidence has also been provided on the role of epistasis in the crosses involving Atlas 66. The absence of the Atlas 66 array in the F1 and F2 Half Diallel analysis is unfortunate, otherwise complementary evidence for the existence of epistasis could be of immense interest for the evaluation of the efficiency of the biometrical genetical models studied here.

In the event of absence of epistasis such as in the analysis without the Atlas 66 array, two further orthogonal comparisons provide unique tests and estimates for the additive and dominance components of variation. These comparisons are $2\bar{B}c1i - \bar{F}1i - \bar{P}i$ for the additive component and $2\bar{B}c2i - \bar{F}1i - \bar{P}c$ for the dominance component. The tests and estimates for both the additive and dominance can be carried out in the context of the analysis of variance shown in Tables 5.9 and 5.10. The theories for these estimates have been discussed in Chapter 2. The analysis of variance for the additive and dominance comparisons was also carried out in the analysis involving the

Table 5.11 The analysis of variance for the additive value for plant height.

Source of Variation	df	SS	MS	F	Prob.
Mean	1	893.1013	893.1013	18.4293	0.0026**
Additive	5	2081.1654	416.2331	8.5890	0.0045**
Deviation	4	1188.0642	297.0160	6.1290	0.0147**
Block	2	158.2632	79.1316	1.6329	0.2543
Error	8	387.6882	48.4610		
Total	15	2627.1168			

The Estimate of the Additive Value = 490.3628

Table 5.12 The analysis of variance for the dominance value for plant height.

Source of Variation	df	SS	MS	F	Prob.
Mean	1	296.7409	296.7409	50.5428	0.0001***
Dominance	5	382.2666	76.4533	13.0220	0.0011***
Deviation	4	85.5257	21.3814	3.6418	0.0566
Block	2	2.8725	1.4362	0.2446	0.7886
Error	8	46.9687	5.8711		
Total	15	432.1078			

The Estimate: for the Dominance Value = 94.1097

Atlas 66 array. Such an analysis would provide insight into the effect of epistasis on the estimates of the additive and dominance component of variation (Tables 5.11, 5.12). High significances were recorded for both components and these significances allowed the estimates of both D and H to be made. The estimates of the additive and dominance components were higher compared to the estimates obtained in the absence of epistasis. These estimates confirmed the inflationary effect of epistasis on the additive and dominance component (Tables 5.11, 5.12).

The nature of epistasis in the Atlas 66 array was revealed by the direct fit estimates for the dominance and epistatic components of (h), (i), (j), and (l). The signs of (h) and (l) were opposite. This indicated that the interactions were predominantly of the duplicate type (Mather, 1967; Jinks and Jones, 1958).

5.3.4 *F1 Half Diallel Analysis of Spike Length*

Large and significant variation in spike length exists for the parents used in this study. Four groups of spike length could be identified (Table 5.13). Cultivar Hilgendorf had the shortest spike, followed by Oroua, Karamu, Kopara and Ruru in order of increasing spike length. The F1 exhibited spike length greater than the mid-parent values but often less than the higher parents. The analysis of the Half Diallel table (Table 5.15) showed high significance for both the additive and dominance effects. Directional dominance was shown by the significance of the 'b1' item while the significance of the 'b2' item showed unequal dominant allele distribution in the parents. Significant specific combining ability shown by the 'b3' item was due to the cross between Kopara and Oroua. This F1 exceeded both parents in spike length.

Table 5.13 Mean spike length of parent and F1 plants (cm).

	1	2	3	4	5
1. Hilgendorf	7.1	9.5	9.6	9.7	8.9
2. Kopara		10.1	10.6	10.5	10.1
3. Oroua			9.5	10.8	9.1
4. Ruru				10.6	10.0
5. Karamu					9.4

Standard Error Mean = 0.13

Coefficient of Variation = 5.78%

Table 5.14 Mean spike length of parent and F2 families (cm).

	1	2	3	4	5
1. Hilgendorf	8.3	9.2	9.8	9.6	9.4
2. Kopara		10.0	10.3	10.7	10.0
3. Oroua			10.3	10.8	10.2
4. Ruru				10.8	10.6
5. Karamu					10.4

Standard Error of Mean = 0.11

Coefficient of Variation = 8.54%

Table 5.15 F1 and F2 Half Diallel analysis of variance of spike length after Morley Jones (1965).

Source of Variation	df	Mean Square	
		F1	F2
a	4	3.695***	3.04***
b1	1	1.115***	0.09
b2	4	0.299***	0.05
b3	4	0.221***	0.07
b	10	0.3414***	0.06
B x a	4	0.027	0.07
B x b1	1	0.017	0.01
B x b2	4	0.036	0.04
B x b3	5	0.013	0.04
B x b	10	0.023	0.05
Block Interaction	14	0.024	0.05

* P < 0.05 ** P < 0.01 *** P < 0.001

Table 5.16 Joint regression analysis of Wr on Vr for estimates from two blocks for F1 and F2 spike length.

Item	df	Mean Square	
		F1	F2
Joint regression	1	0.3764**	0.0994***
Heterogeneity of regression	1	0.0021	0.0020
Remainder	6	0.0128	0.0008
Joint regression coefficient		0.8311	0.9396
Standard error		0.1533	0.0857
Probability of significance from 1.0		0.6814	0.8435
Probability of significance from 0.0		0.0780	0.0184

* P < 0.05 ** P < 0.01 *** P < 0.001

The test of the adequacy of the additive and dominance model for this trait by the joint regression analysis showed the joint regression highly significant and the heterogeneity of regression as not significant (Table 5.16). The joint linear regression coefficient was 0.8311 ± 0.1533 and it was not significantly different from one ($P = 0.6814$). The additive and dominance model was accepted as satisfactory for this trait although the probability of the joint regression being significant from zero was $P = 0.078$. This is due to low joint regression coefficient of 0.8311 and the high standard error of 0.1533 which can be attributed to the F1 between Kopara and Oroua which exceeded their parental means. The Wr-Vr graph of Figure 5.3 showed the parents Kopara and Ruru both with the longest spike located near the origin indicated that long spike is dominant over shorter spike. The shortest spike parent, Hilgendorf, had its Wr-Vr value further away showing that it carried the most recessive genes for this trait. Cultivars Oroua and Karamu were intermediate between the two extreme groups discussed above. Confirmation of the direction of dominance as being towards long spike was given by the correlation between the $(Wri + Vri)$ values and the phenotypic means of the parents (Pi) . The correlation of -0.9608 ± 0.1600 confirmed that dominance was towards longer spike.

With the additive and dominance model adjudged satisfactory, perfect fit estimates of the genetic components could be made and are shown in Table 5.17. The additive effect D is greater than the dominance effect value $H1$, therefore dominance is partial. The degree of partial dominance

Table 5.17 Components of genetic variation of 5 x 5 Diallel Crosses for spike length.

Components	Generation	F1	F2
	D	1.2561	0.8650
	H1	0.6361	0.3616
	H2	0.5573	1.0436
	$(H1/D)^{\frac{1}{2}}$	0.6997	0.6465
	H2/4H1	0.2266	0.7215
	E	0.0237	0.0464
Heritability (Narrow sense)		0.7485	0.7549
Heritability (Broad sense)		0.9629	0.8974

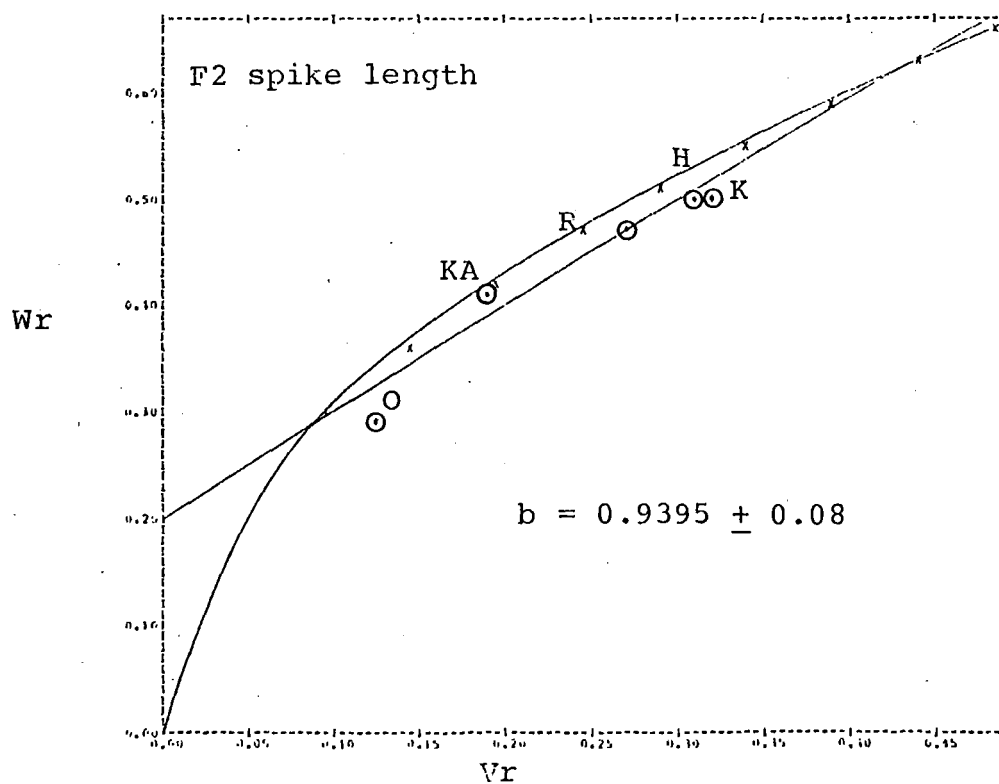
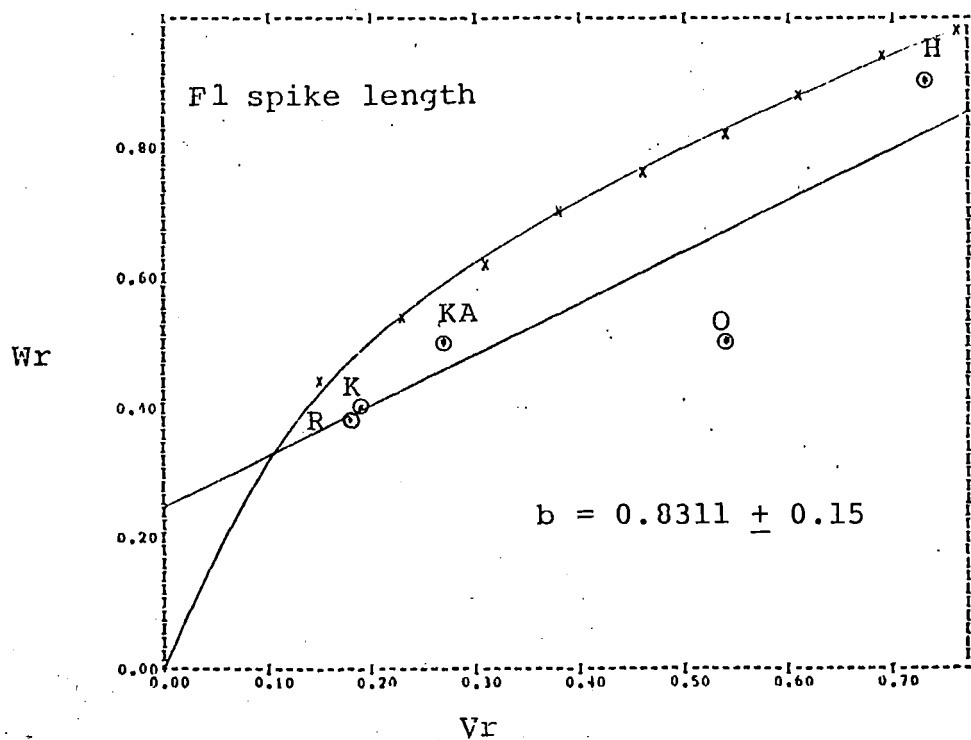


FIG. 5.3 The relationship between W_r and V_r for the F1 and F2 generations for spike length.

as given by $\sqrt{(H1/d)}$ and is 0.6997. This partial dominance is again illustrated by the graph of W_r-V_r of Figure 5.3 when the regression line cuts the Y axis above the origin. $H1$ is equal to $H2$, hence there are equal allele frequencies of $u = v = 0.5$. uv as given by $\frac{1}{4}(H1/H2)$ is 0.2266. The equal allele frequencies provided in this estimate is contrary to the significance of the 'b2' item which implied asymmetry of gene distribution, i.e. $H1 > H2$. This apparent contradiction can be due to the small block interaction used for testing the significance of 'b2'. Narrow sense heritability estimate was 0.7485 indicative of a good potential response to selection for this trait.

5.3.5 F2 Half Diallel Analysis of Spike Length

The F2 measurements on spike lengths (shown in Table 5.14) confirmed the F1 observations (Table 5.14). Cultivar Ruru maintained the lead with the longest spike length and cultivar Hilgendorf had the shortest. The other three cultivars showed intermediate values. The analysis of variance of the Half Diallel Table 5.15 confirmed the highly significant 'a' or additive effect as detected in the F1 studies. However, the highly significant dominant 'b' and its component items, recorded in the F1 studies, did not approach significance in the F2 studies. This difficulty in obtaining significance for the dominance items was partly due to the halving of the frequency of the heterozygotes in advancing from F1 to the F2 generation and partly due to the high block interaction recorded in this F2 study (Table 5.15).

The test of the adequacy of the additive and dominance

model by the joint regression analysis showed the joint regression highly significant and the heterogeneity of regression was not significant. The joint regression coefficient of 0.9396 ± 0.0857 was not significantly different from one but significantly different from zero (Table 5.16). The additive and dominance model was therefore acceptable. This confirmed the conclusion of the F1 analysis. The difficulty of detecting the dominance component discussed earlier was reflected in the distribution of the parents in the Wr-Vr graph of Figure 5.3. Such random distribution was contrary to that of the F1 studies as shown in Figure 5.3. While conclusive evidence was made available by the Wr-Vr graph of the F1 generation on the parents carrying the most dominant genes, no such support can be obtained from the F2 analysis. The difficulty in confirming the direction of dominance as being towards long spike, was shown by the correlation coefficient between $(Wri + Vri)$ with Pi . The correlation coefficient was -0.4426 ± 0.5177 and was not significant. In the F1 studies, the correlation coefficient was 0.9698 ± 0.1600 and was significant. This poor tally of the F2 results with the F1's can be attributed to the halving of the dominance in the F2 which has also resulted directly in the quartering of the variance and covariance terms. These observations highlight the difficulty of using a segregating F2 population for a Diallel study, when the sample size has to be restricted to a manageable one. It is obvious that the sample size of sixty F2 plants per family sampled here cannot adequately provide a representative estimate of the family mean, while the twenty plants per family in the F1 studies was satisfactory for spike length.

Table 5.18 Mean spike length of the parents (Pi), common tester (Pc), F1, F2, and backcrosses (Bc1, Bc2) - Experiment III.

Parents Generation	Hilgendorf	Kopara	Oroua	Ruru	Atlas 66
Pi	8.4	9.6	10.4	10.4	10.3
Pc	10.5	10.7	10.9	10.7	10.5
Bc1	8.9	10.5	10.2	10.7	10.4
Bc2	10.3	10.9	10.4	11.4	10.7
F1	9.8	11.3	10.3	10.9	11.0
F2	9.4	10.1	10.2	10.6	10.4

Standard Error of Mean = 0.12

Coefficient of Variation = 8.06%

Table 5.19 Sums of squares of comparisons for detecting epistasis on spike length.

Parent (Pi)	Ai Comparison Sum of Squares	Bi Comparison Sum of Squares
Hilgendorf	0.6294	0.4624
Kopara	0.0822	0.3600
Oroua	0.4182	0.9474
Ruru	0.2055	12.8164
Atlas 66	3.0044	0.0576
Total	4.3397	14.6438
Mean square	0.0964	0.3254
Pooled variance	0.1000	0.0873
F ratio	0.9640 ns	3.7276 **

ns non significance

* $P < 0.05$

** $P < 0.01$

Table 5.20 Scaling Tests and estimates of parameters for spike length.

Parent (Pi)		Hilgendorf	Kopara	Oroua	Ruru	Atlas 66
Test	A	-0.2644	0.0956	-0.2156	0.1511	-0.5778
	B	0.2267	-0.2000	-0.3244	1.1933***	-0.0800
	C	-0.7095	-2.3686***	-0.9832*	-0.3375	-1.2489
	S	-0.1679	-0.5660	-0.1108	-0.4205**	-0.1478
Estimates						
	m	8.8116***	7.8970***	10.1900***	8.8703***	9.8156***
	d	-1.0656***	-0.5344***	-0.2722***	-0.1256	-0.0911
	h	1.5890	5.5716***	-0.0259	5.0183***	1.1356
	i	0.6717	2.2641	0.4432	1.6819**	0.5911
	j	-0.4911	0.2956	0.1089	-1.0422	-0.4978
	l	0.6340	-2.1597**	0.0968	-3.0263	0.0667
Joint Test						
	m	9.47 + 0.06	10.06 + 0.07	10.57 + 0.07	10.58 + 0.07	10.34 + 0.07
	d	-1.09 ± 0.06	-0.52 ± 0.07	-0.27 ± 0.07	-0.20 ± 0.07	-0.13 ± 0.08
	h	0.22 ± 0.12	1.09 ± 0.11	-0.42 ± 0.12	0.36 ± 0.13	0.57 ± 0.13
	χ^2 (3)	4.5645	28.71***	5.7095 ns	18.06***	5.35 ns

* P < 0.05

** P < 0.01

*** P < 0.001

As the additive and dominance model has been accepted as adequate, perfect fit estimates of genetic parameters can be made. The estimate for the additive component, D , was 0.8650 and $H1$, the dominance component was 0.3616. The value of $(H1/D)^{\frac{1}{2}}$ was 0.6465. This indicated partial dominance for this trait and confirmed the observation made in the $F1$ study (Table 5.17).

5.3.6 *New Triple Test Cross and Scaling Test*

Analyses on Spike Length

The additional cultivar, Atlas 66, used in this study, possessed similar spike length as cultivars Oroua, Ruru and Karamu (Table 5.18). The New Triple Test Cross analysis shown in Table 5.19 provided contradictory evidence for the presence of epistasis. The A_i comparison was not significant but the B_i comparison was significant. The A and B individual Scaling Tests supported the conclusions of the New Triple Test Cross analysis. Moreover, the C , S and the Cavalli's Joint Tests were significant in some family arrays (Table 5.20). The C test was significant in the Kopara and Oroua arrays while the B and S tests were significant in the Ruru array. Furthermore, the Cavalli's Joint Test was significant for the Kopara and the Ruru arrays. The contradictory evidence provided by the C , S and Cavalli's Joint Tests to those of the A test could be explained by the difficulty of obtaining reliable estimates of the $F2$ and back-cross family means under the present sampling techniques.

Our conclusion on the absence of epistasis for spike length shall therefore be based on the A_i comparison of the

Table 5.21 Analysis of variance for the additive value for spike length.

Source of Variation	df	SS	MS	F	Prob.
Mean	1	0.2441	0.2441	5.0446	0.0549*
Additive	5	1.0694	0.2139	4.4210	0.0315*
Deviation	4	0.8254	0.2063	4.2651	0.0387*
Block	2	0.4517	0.2258	4.6679	0.0454*
Error	8	0.3879	0.0484		
Total	15	1.9081			

The Estimate of the Additive Value = 0.2207

Table 5.22 Analysis of variance for the dominance value for spike length.

Source of Variation	df	SS	MS	F	Prob.
Mean	1	0.5894	0.5894	2.9876	0.1222
Dominance	5	2.0453	0.4091	2.0735	0.1715
Deviation	4	1.4559	0.3641	1.8450	0.2137
Block	2	0.0554	0.0277	0.1403	0.8712
Error	8	1.5782	0.1973		
Total	15	3.6789			

The Estimate of the Dominance Value = 0.2824.

New Triple Test Cross, the A Scaling Test and F1 and F2 Half Diallel analyses.

As one of the epistatic comparison (A_i) of the New Triple Test Cross analysis has provided no evidence for the presence of epistasis, attempt can be made to estimate the value of the additive and dominance components by the analysis of variance of the additive and dominance comparisons. These analyses are shown in Tables 5.21 and 5.22. The dominance comparison, however, failed to reach significance in the analysis on spike length. Difficulty in obtaining significance for the dominance component has also been encountered by Chahal and Jinks (1978). They attributed the absence of significance to their inefficient experimental designs. However, the generally high standard error accompanying the estimate for the dominance component as discussed by Kearsey and Jinks (1968) could also explain the difficulty of achieving significance for the dominance component. On the other hand, the analysis of variance for the additive component was highly significant (Table 5.21). In spite of this absence of significance for the dominance component, estimates of the additive (D) and dominance (H) components is attempted (Tables 5.21, 5.22).

5.3.7 F1 Half Diallel Analysis of Flag Leaf Length

The flag leaf lengths of the five parents used in this study can be divided into three classes. Cultivar Karamu had the longest flag leaf, while cultivars Kopara, Hilgendorf and Ruru were of intermediate leaf lengths. The cultivar Oroua had the shortest flag leaf (Table 5.23). The Half

Table 5.23 Mean flag leaf length of parent and F1 plants (cm) .

	1	2	3	4	5
1. Hilgendorf	18.4	19.5	17.0	19.6	18.2
2. Kopara		18.7	17.5	19.2	16.6
3. Oroua			15.3	18.6	17.5
4. Ruru				18.7	18.8
5. Karamu					20.3

Standard Error of Mean = 0.48

Coefficient of Variation = 11.74%

Table 5.24 Mean flag leaf length of parent and F2 families (cm) .

	1	2	3	4	5
1. Hilgendorf	18.3	20.4	18.0	20.1	20.5
2. Kopara		19.4	17.9	20.9	19.7
3. Oroua			17.0	19.1	20.2
4. Ruru				19.9	21.3
5. Karamu					18.9

Standard Error of Mean = 0.38

Coefficient of Variation = 15.05%

Table 5.25 Half Diallel analysis of variance on flag leaf length.

Source of Variation	df	MS	df	MS
		5 x 5 Half Diallel		4 x 4 Half Diallel (without Karamu array)
a	4	6.9995***	3	8.9657**
b1	1	0.0000	1	3.2275
b2	4	3.5258*	3	0.1798
b3	5	1.2068	2	0.8855
b	10	2.0137	6	0.9230
Block X a	4	0.2234	3	0.2172
Block X b1	1	0.4950	1	0.2940
Block X b2	4	1.9515	3	2.8089
Block X b3	5	0.1965	2	0.0452
Block X b	10	0.9284	6	1.4685
Block Interaction	14	0.7269	9	1.0574

*** P < 0.001

** P < 0.01

* P < 0.05

Table 5.26 Joint regression analysis of W_r on V_r for estimates from two blocks for F1 and F2 flag leaf length.

Item	df	Mean Square			
		With Karamu		Without Karamu	
		F1	F2	F1	F2
Joint regression	1	1.6030 ns	1.015*	6.0846**	1.0521
Heterogeneity of regression	1	1.0527	0.0214	0.0875	0.0731
Remainder	6	0.9562	0.1152	0.1718	0.0568
Joint regression coefficient		0.7998	0.7563	1.1728	0.729
Standard error		0.6177	0.2446	0.1971	0.1694
Probability of significance from 1.0		0.8074	0.6397	0.7169	0.5462
Probability of significance from 0.0		0.3481	0.1770	0.0575	0.1512

* $P < 0.05$ ** $P < 0.01$ *** $P < 0.001$

Diallel analysis of variance (Table 5.25) showed a highly significant additive effect for this trait and significant dominance. The 'b1' item was, however, not significant, showing an absence of any directional dominance. The 'b2' item was significant and therefore the dominant alleles were not equally distributed among the parents. The 'b3' item was not significant, although significance was expected because of the presence of specific combining ability in the cross involving Kopara and Karamu. The F1 of this cross had a shorter flag leaf than either of the parents. This lack of significance was due to the high pooled block interaction mean square used for testing its significance. The lack of fit for the additive and dominance model which could be attributed to the specific combining ability of the above cross was shown by the joint regression analysis of Table 5.26. The joint regression analysis was not significant and the joint regression coefficient was not significantly different from one nor zero. A re-analysis, after the deletion of the Karamu array, thereby eliminating the cross showing specific combining ability, was carried out. The new analysis showed presence of only additive effect (Table 5.25). The adequacy of the additive and dominance model was shown by the significance of the joint regression analysis and the joint regression coefficient which was significantly different from zero but not from one. The adequacy of the additive and dominance model for the four parents Half Diallel after removing the Karamu array and the presence of the largely additive effect suggested early response to selection in crosses without Karamu. The lack of fit in this restrictive model in the five parents diallel could be due to epistatic effect in the cross

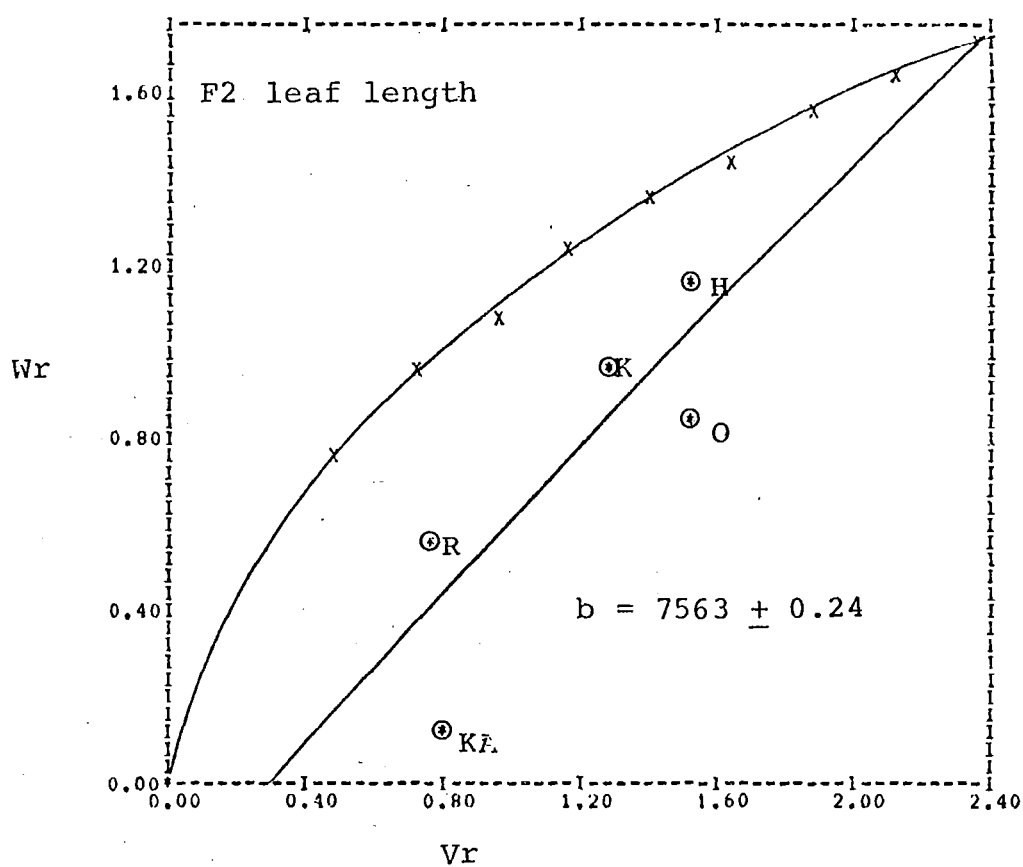
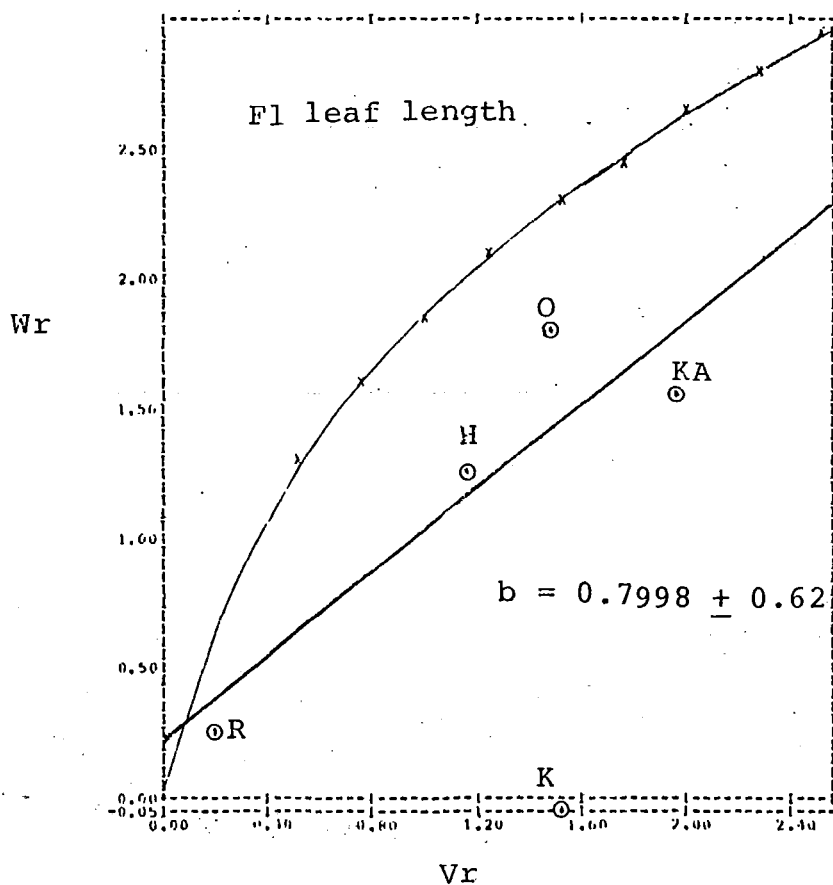


FIG. 5.4 The relationship between W_r and V_r for the F1 and F2 generations for leaf length.

Table 5.27 F2 Half Diallel analysis of variance on flag leaf length.

Source of Variation	df	MS	df	MS
		5 x 5 Half Diallel		4 x 4 Half Diallel (without Karamu array)
a	4	6.12***	3	17.11
b1	1	8.47***	1	6.47*
b2	4	0.87	3	1.67
b3	5	1.17*	2	1.20
b	10	1.78*	6	2.31
B x a	4	0.74	3	0.69
B x b1	1	2.07**	1	1.72
B x b2	4	0.12	3	0.06
B x b3	5	0.05	2	0.04
B x b	10	0.28	6	0.33
Block Interaction	14	0.41	9	0.45

* $P < 0.05$ ** $P < 0.01$ *** $P < 0.001$

between Karamu and Kopara and unless this epistatic effect is of the additive x additive type, selection will be ineffective. The perfect fit estimates were not included for this trait because of this absence of fit for the additive and dominance model in the five parents Half Diallel.

5.3.8 *F2 Half Diallel Analysis of Flag Leaf Length*

Cultivar Oroua continued to exhibit the shortest leaf length (Table 5.24) amongst the parental cultivars studied. However, cultivar Karamu which produced the longest leaf length in the previous season, showed equal leaf length to cultivars Kopara and Ruru in the present study. Cultivar Hilgendorf maintained its intermediate position in the leaf length ranking.

The Half Diallel analysis of variance of the F2 generation (Table 5.27) indicated agreement on the significance of the 'a' item with that in the F1 analysis. However, the 'b' and its component items of the F2 generation showed considerable departure from the F1 analysis. In the F1 generation, only the 'b2' item of all the 'b' components was significant whereas the reverse was true in the F2 generation.

The joint regression coefficient (Table 5.26) of the W_r-V_r graph (Figure 5.4) was not significantly different from one or zero. This suggested an absence of fit for the simple additive and dominance model and confirmed the F1 results. However, the removal of the Karamu array which was responsible for the absence of fit for the additive and dominance model in the F1 generation (Table 5.26) failed to repeat the F1 results. This is suggestive of a more general expression of absence of

Table 5.28 Mean flag leaf lengths of the parents (Pi), common tester (Pc), F1, F2 and backcrosses (Bc1, Bc2) - Experiment III.

Parents	Hilgendorf	Kopara	Oroua	Ruru	Atlas 66
Generation					
Pi	20.5	21.4	17.1	19.3	20.8
Pc	21.1	20.1	21.0	20.9	21.8
Bc1	19.8	20.5	17.9	20.4	21.3
Bc2	20.2	21.6	20.4	20.6	21.0
F1	18.6	20.5	17.5	20.4	20.6
F2	20.3	19.6	19.9	20.6	21.1

Standard Error of Mean = 0.43

Coefficient of Variation = 14.22%

Table 5.29 Sums of squares of comparisons for detecting epistasis on flag leaf length.

Parent (Pi)	Ai Comparison Sum of Squares	Bi Comparison Sum of Squares
Hilgendorf	1.2544	2.8448
Kopara	8.0278	63.2555
Oroua	12.7687	47.0596
Ruru	9.5687	0.0374
Atlas 66	11.0224	2.2700
Total	42.6420	115.4674
Mean Square	0.9476	2.5660
Pooled Variance	1.1565	1.0233
F ratio	0.8094 ns	2.5074*

ns non significance

* $P < 0.05$

Table 5.30 Scaling Tests and estimates of parameters for flag leaf length.

Parent (Pi)	Hilgendorf	Kopara	Oroua	Ruru	Atlas 66
Test					
A	0.3733	-0.9444	1.1911	1.0311	1.1067
B	0.5622	2.6511**	2.2867*	-0.0644	-0.5022
C	2.3178	-3.9867	6.4498***	1.3635	0.2444
S	0.3456	-1.4233	0.7430	0.0992	0.0900
Estimates					
m	22.2278***	15.0622***	22.0576***	20.5224***	20.9889***
d	-0.3033	0.6222*	-1.9611***	-0.7944*	-0.5000*
h	-4.0656	12.8156**	-4.0452	0.4630	0.6111
i	-1.3822	5.6933**	-2.9721	-0.3968	0.3600
j	-0.1889	-3.5956*	-1.0956	1.0956	1.6089
l	0.4467	-7.4000*	-0.5057	-0.5698	-0.9644
Joint Test					
m	20.97 + 0.23	20.59 + 0.25	19.39 + 0.24	20.23 + 0.29	21.38 + 0.24
d	-0.32 + 0.23	0.34 + 0.25	-1.97 + 0.24	-0.71 + 0.29	-0.38 + 0.24
h	-1.93 + 0.47	-0.39 + 0.45	-1.39 + 0.41	0.36 + 0.51	-0.64 + 0.46
χ^2 (3)	2.44 ns	19.83***	21.665***	1.19 ns	1.7566 ns
* P < 0.05 ** P < 0.01 *** P < 0.001					

fit in the arrays studied. The difficulty of obtaining agreement in both the F1 and F2 analyses may be indicative of possible genotype environmental interaction. However, what appeared to be a more plausible explanation in the present study is the difficulty in obtaining sufficiently accurate family means due to high sampling variations. These means have been used for both the Diallel analyses of variance and the Wr-Vr analysis. As the Wr-Vr analysis used the variance and covariance of the observed family means, any inaccuracies will be seriously compounded because their squared values were utilised in such analysis.

5.3.9 *The New Triple Test Cross and Scaling Test Analyses of Flag Leaf Length*

The difficulty of obtaining concurrent results for flag leaf length was further shown by the results of the New Triple Test Cross and those of the Scaling Tests analyses. The New Triple Test Cross analysis of Table 5.29 provided contradictory evidence for the presence of epistasis. The Bi comparison was significant while the Ai was not. The Scaling Test analysis also provided contradictory evidence as those of the New Triple Test Cross analysis. The B and C Scaling Tests and the Cavalli's Test were found significant in both the Kopara and Oroua arrays (Table 5.30). Such contradictions provided further evidence of the difficulty of using inaccurate estimates of the family means owing to high sampling variations (Table 5.29).

Genotype x Env. interaction

5.4 DISCUSSION

5.4.1 *Plant Height*

Our results on the nature of gene action on plant height, are in agreement with earlier work. The semidwarfness in the cultivars Karamu, Oroua and Ruru is shown mainly to be controlled by recessive genes. Such observation is in line with the understanding of the genetics of the Norin 10 - Brevor 14 based semidwarfism (Allan and Vogel, 1963; Allan, Vogel and Patterson, 1968). The semidwarfness in the Norin 10 derivatives has been attributed to two major dwarfing genes, *Rht1/Gail* and *Rht2/Gai2* (Gale and Law, 1978). Our conclusions on the adequacy of the additive and dominance model for plant height for all the cultivars used, with the exception of the parent Atlas 66, is in accord with records on other common wheat cultivars (Johnson *et al.*, 1966; Bitzer *et al.*, 1971; Bhatt, 1972; Fick and Qualset, 1973; Gill *et al.*, 1973). Moreover, the dominance of tallness recorded here is also in line with earlier work by Merkle and Allans (1964) and Bhatt (1972).

However, depending on the genetical background of the parents used, epistasis can be of relevance, as shown by such expression in the crosses involving cultivar Atlas 66. This observation emphasizes the need to conduct preliminary genetical analysis with each parent before selection and breeding programmes. Epistasis in height has been recorded by Ketata *et al.* (1976b), and Gill *et al.* (1977). Chapman and McNeal (1971) recorded epistasis of the dominance x dominance type whereas Fick and Qualset (1973) observed additive x additive

and dominance x dominance epistasis. Our Scaling Test analysis has provided evidence for the presence of additive x additive (i), additive x dominance (j), and dominance x dominance (l) types of epistasis in the families with Atlas 66 as a parent. The understanding of the inheritance of plant height is of immense relevance to the future of wheat breeding. This is because of the presence of a strong relationship between height and yield and increasing yield problems encountered in semidwarf wheats (Law, 1978; Gale and Law, 1976; Stoskopf and Fairey, 1975). These observations have prompted workers at the Plant Breeding Institute at Cambridge to propose the breeding of "tall dwarf" wheats. A model for integrating the desirable major genes for semidwarfism and the yield related genes for height as proposed by Gale and Law (1976) is shown in Figure 5.1. The rationale behind this model is based on the need to preserve many of the genes which increase both height and yield in the presence of the Rht/Gai genes, which have the reverse effects, i.e. decrease height and increase yield. The best course of action to achieve higher yield is therefore to fix the semidwarfing factors early in the breeding programme while maintaining other variations in the population. Thereafter, selection should be for increased height rather than for shorter and shorter versions of the dwarf ideotype. It is therefore, not unduly speculative for us to argue that such an ideal selection could be achieved in the context of breeding among the three semidwarfs and two standard height wheats used in this study. This is because our study has indicated large narrow sense heritability emphasizing the presence of mainly additive genes in the parents studied here. The tall dwarf breeding strategy of

fixing the dwarfing genes in early generations and selecting for increasing height at later generations appears feasible. However, the presence of epistasis in the families involving Atlas 66 must be identified as a major constraint to the general application of the "tall dwarf" breeding strategy. The complexity of epistasis as suggested by our results and those of Fick and Qualset (1973) and Gill *et al.* (1976) will conceivably result in the inability to fix the observed tallness. This is particularly true in the case of the Atlas 66 crosses because of the existence of duplicate type epistasis.

In New Zealand, a parallel breeding and selection strategy has been proposed by McEwan (1973). He suggested that further increases in grain yield could be achieved if the total dry matter productivity of some cultivars of conventional stature could be combined with the high harvest index of the Norin 10 based semidwarfs. To put theory into practice, McEwan (1978) has crossed Raven, a standard height wheat with high biological yield, to two unnamed Mexican semidwarfs of high harvest indexes. The result from these efforts is the recent release of the cultivar Rongotea. This cultivar was shown to possess high harvest index of the Mexican derivative and to transgress the cultivar Raven in biological yield. The height of this new cultivar, however, approximated the semidwarf cultivar, Karamu. It is therefore strictly not a "tall dwarf" selection but a 'high biological yielding dwarf'.

5.4.2 *Spike and Flag Leaf Length*

Our conclusion on the absence of epistasis in spike length conforms with previous works by Johnson *et al.* (1966); Hsu and Walton (1970a); Gill *et al.* (1973) and Gill *et al.* (1977). The significance of this trait was discussed by Hsu and Walton (1970a) who were of the opinion that photosynthetic areas above the flag leaf could be complementary to conventional yield components, for yield selection response. Moreover, Lupton (1975) has observed that the high yielding semidwarf, Hobbit, possessed larger spikes, thereby providing a larger 'sink' for carbohydrates than those of taller cultivars. The large spike ideotype is apparently attractive. This is particularly relevant with the evidence provided above on the absence of complex gene interactions for this trait. This provides prospects for selection response. However, this evidence should not warrant over-enthusiasm on yield increase through spike length selection. A large spike may be able to enhance the 'source' through increased photosynthetic areas as proposed by Hsu and Walton (1970a), but may not possess the large sink described by Lupton (1975). The size of the 'sink' within a spike is determined by other constraints such as the spikelet density (spikelet per unit length of spike) and the inherent floret fertility of each spikelet. Little work has been carried out for these two traits. The need for genetical studies into floret survival has been emphasized by Gallagher (1978). Halloran (1974) in his genetic analysis of hexaploid wheat, has estimated four genes controlling spikelet density and five genes for floret fertility of the spike. Fertility of the glume has been recorded by Wright (1968). This trait was found under multifactorial

control.

Our genetical analyses of flag leaf length are inconclusive because of the high sampling variability. Record of direct yield increase through selection for high photosynthetic area of the flag leaf is not available up to now. The prospects of yield increase through flag leaf area selection, as proposed by Hsu and Walton (1970a, b), appear to be poor. Moreover, the indirect evidence provided by Watson *et al.* (1963) and the observations provided by McNeal and Berg (1977) discussed in the literature review, indicated little prospects of a yield response through flag leaf area increase.

CHAPTER 6

DIALLEL ANALYSIS ON PROTEIN CONTENT OF WHEAT

6.0 REVIEW OF LITERATURE

Protein content of flour is positively related to baking quality (O'Brien and Oath, 1977). To improve the protein content of wheat (*Triticum aestivum* L. em Thell), an understanding of the physiological and genetical basis of its production is essential. The scope for breeding high protein wheat is considerable. This was emphasized by Konzak (1977) in his extensive review of the genetic control of protein in wheat. He remarked that no wheat cultivar known today has a protein composition approaching the level desired. However, considerable progress has been made towards this important challenge. Plant breeders at the University of Nebraska have released a high protein wheat called Lancota with grain yield as high as commercial cultivars such as Centurk and Scout 66. The merit of Lancota was shown in the 1972-73 international winter wheat trial when it showed a protein content of 15.5 per cent, an advantage of 1.1 to 2.3 per cent over cultivars with comparable yield (Johnson et al., 1978).

While practical plant breeders are striving for better cultivars, geneticists are working on the genetical basis of protein inheritance. Work in this area was pioneered by Clark (1926) whose genetical studies on high protein wheat dates back to the 1920's. Ausemus et al. (1946) in their

review established the acceptance of polygene control of grain protein. Stuber *et al.* (1962), using frequency distribution also concluded that inheritance of grain protein was under polygenic control. Haunold *et al.* (1962), while acknowledging polygene control, concluded that in the crosses Atlas 66 x Wichita and Atlas 66 x Comanche a relatively small number of genes was involved. Further evidence of the polygenic nature of this important trait was provided by workers dealing with whole chromosome substitution lines. Law *et al.* (1978) showed that at least two genes, Pro 1 and Pro 2, affecting grain protein, could be identified on chromosome 5 D. Chromosomes of homologous Group 2 were also found to affect grain protein. Halloran (1976) using the 21 intervarietal chromosome substitution lines of the cultivar Hope, found that only chromosome 5 D significantly influenced grain protein content. He postulated the control of grain protein to be most likely due to many genes, each with small effect.

With the acceptance of polygene control of grain protein content, recent workers have been concerned with studying the relationship of the various genes controlling grain protein. An understanding of this relationship is essential before any efficient breeding and selection can take place. For this purpose, quantitative genetical analysis has been most useful. Davis *et al.* (1961), in studies involving four crosses, obtained heritability estimates of 54% to 69%. They concluded that considerable genetic variability was present in all four populations. Genetic progress through selection was indicated in all the populations. Haunold *et al.* (1962) using the parent offspring regression method, pro-

vided heritability estimates of 65% in the crosses involving Atlas 66 x Wichita and Atlas 66 x Comanche. Stuber *et al.* (1962) in their studies on the cross between Atlas 66 and Wichita, obtained heritability values from 68% to 83%. A high heritability estimate was also found by Jain *et al.* (1975) in studies based on the absolute amount of protein in a fixed number of seeds. These studies are indicative of the importance of the large genetic effects on protein content. Low and more variable heritability estimates have also been reported. Lebsock *et al.* (1964) recorded variable heritability estimates from 37 to 70%. Sunderman *et al.* (1965) in studies on the cross Atlas 66 x Itana, provided heritability estimates of between 15% to 26%. Ali (1976) estimated heritability of 41% to 45%. The presence of substantial and variable environmental effects on this trait has also been recorded. Halloran (1975) while confirming the additive nature of protein content inheritance, also reported that the control of protein content, whether by dominant or recessive genes, appeared to be influenced by the environment. The need to conduct protein inheritance studies under environment similar to those for which high protein genotypes are required was emphasized by him.

The genetic value can be partitioned into its components of additive, dominance and epistatic effects. The presence and magnitude of each of these components vary from cross to cross. For example, Chapman and McNeal (1970), using Hayman's analysis of six generations (P1, P2, F1, BC1 and BC2) found only two of the five crosses to have significant dominance effect on this character. Additive genetic effect was found to be highly significant in all five crosses and no epistatic

interaction was detected. Crosses showing differing effects were also reported by Bains *et al.* (1972). In their studies involving an eight parents diallel, they found the dominance effect significant in only three of the eight arrays. On removal of these three arrays and on re-analysis of the remaining five arrays, only the additive effect was found to be significant. Much of the dominance was attributed to the presence of epistasis in the three arrays. Halloran (1975) also recorded epistasis in his eight parents diallel analysis. The strong epistatic effect detected was attributed to one of the parents, Argentine IX. Removal of this array removed the epistatic effect. Other evidence suggesting the presence of epistasis was provided by Diehl (1978). Their results failed to fit the Hayman's three parameters model of mean, additive and dominance effects.

It is therefore evident that grain protein inheritance in wheat varies between crosses. While only additive effect is present in some, other crosses are capable of exhibiting dominance and epistasis.

Studies of flour protein are fewer than those of grain protein. This was pointed out by Kaul and Solsuski (1965). The general lack of research in this direction is because of the difficulty of extracting the flour in early generations. The earliest recorded work on flour protein inheritance was by Thompson and Whitehouse (1962), who carried out a diallel analysis of this trait and found evidence of interaction of epistasis with environment. This was followed by Kaul and Solsuski (1965) who worked on the six generations, P₁, P₂, F₁, F₂, BC₁, and BC₂, derived from the cross Selkirk x Gabo.

Heritability estimates were also calculated from the F3, F4 and F5 generations. Several methods were used to compute the heritability estimates which were generally very high. The lowest value was 66% for narrow sense heritability. Lofgren et al. (1968) in their work involving Atlas 50, Atlas 66, Triumph and Kaw, recorded flour protein inheritability of between 30% to 70%. Sharma et al. (1973) using the dye binding capacity method to measure flour protein, found heritability estimates of only 26.7% to 27.9%. Heritability estimates of flour protein are therefore as variable as those of grain protein.

Plant breeders in New Zealand have been conscious of the need to improve the protein content of New Zealand wheats. As early as the 1920's, they have been attempting to raise the quality of New Zealand bread wheat by breeding. These efforts resulted in Frankel and Hullet (1947) releasing the cultivar Hilgendorf. They ranked Hilgendorf as of outstanding baking quality. Cultivar Hilgendorf was further improved by backcrossing to incorporate mildew resistance. This resulted in the release of Hilgendorf 61 (Copp, 1967), which is still leading as a high protein wheat.

However, with the introduction of the semidwarf wheat, Karamu, New Zealand was to face a wheat of extremely variable quality (Langer, 1977). The importance of this variability was highlighted by Malcolm (1977) who ^{reported} obtained grain N of 1.45% following July N application. When the same amount of N was applied in September, the grain N was 2.55%. This extreme variability has led plant breeders to seek alternative cultivars. Two cultivars, Oroua and Rongotea with good grain

protein content and baking quality, superior to Karamu, have been released for commercial production (McEwan, 1978).

6.1 INTRODUCTION

Prospects for breeding a cultivar with high yield and protein content as high as Hilgendorf are good. This project was initiated to study the nature of gene action on flour protein content of New Zealand wheat cultivars. A better understanding of the inheritance of protein content can help in a systematic approach for breeding a cultivar with improved protein content.

6.2 MATERIAL AND METHODS

Grains harvested in Experiments I and II, A, B, C and D were used in this study. Grains were cleaned with a 2.0 mm sieve and 30 grams of grain from each family of each block were equilibrated to 14% moisture. The grains were then milled with a Brabender experimental mill. Approximately two grams of the flour were used for protein determination by the Technicon Near Infra Red Reflectance Analyser. The two grams of flour were fed into the analyser via a disk with a transparent glass cover. The flour protein content was computed and printed out by the analyser soon after the sample was scanned. The computation of the flour protein percentage by the analyser was based on the reflectance property of the flour sample. The analyser was programmed to compute wheat flour protein. The efficiency of the Near Infra Red Analyser for protein determination has been recorded by Klepper and Wilhelm (1979).

Hayman's (1954) analysis was used to study the genetic variation of the Full Diallel Experiments A, B, C and D. The Half Diallel Experiments I and II were analysed by Morley Jones' (1965) method. The covariance-variance graphical analysis of Jinks (1954) and Mather and Jinks (1971) was applied to work out the adequacy of the additive and dominance model and the dominance relationship of the parents. These analyses were executed with the computer programmes Binhalf and Bindial (Appendices I and II).

6.3 RESULTS

6.3.1 *F1 Generation Half and Full Diallel Analyses*

The flour protein contents of the parents varied significantly. The cultivar with the highest flour protein content was Hilgendorf. Cultivars Ruru and Karamu showed low protein contents, while cultivar Kopara and Oroua had intermediate protein contents. The percentages of flour protein in the parents and in the F1 progenies are shown in Tables 6.1, 6.2 and 6.3

The presence of highly significant genetic differences in flour protein content is demonstrated by the highly significant additive and dominant effects, that is, items 'a' and 'b' of the Hayman's analysis shown in Table 6.4. Moreover, the additive effect 'a' is of a greater magnitude than the dominant 'b' effect. Of the components of the 'b' item, only the 'b2' item showed significance in all three experiments. This level of significance suggests that the mean dominance deviation of the F1 from the mid parental values within each

Table 6.1 Parent and Fl mean flour percentage - Experiment A.

	1	2	3	4
1. Hilgendorf	15.63	14.31	15.76	12.74
2. Kopara	14.06	14.04	14.56	12.49
3. Oroua	16.57	14.80	14.63	13.76
4. Ruru	12.97	11.97	13.84	11.82

Standard Error of Mean = 0.4395

Coefficient of Variation = 4.44%

Table 6.2 Parent and Fl mean flour protein percentage - Experiment B

	1	2	3	4
1. Hilgendorf	16.30	13.95	15.63	12.64
2. Kopara	14.48	13.60	14.61	11.96
3. Oroua	16.22	14.86	14.89	13.54
4. Ruru	13.16	12.35	13.49	11.98

Standard Error of Mean = 0.2055

Coefficient of Variation = 2.08%.

Table 6.3 Parent and F1 mean flour protein percentage -
Experiment I.

	1	2	3	4	5
1. Hilgendorf	16.61	14.11	15.88	13.70	14.51
2. Kopara		13.91	15.13	11.72	14.51
3. Oroua			15.27	13.79	13.93
4. Ruru				11.77	14.26
5. Karamu					12.30

Standard Error of Mean = 0.4172

Coefficient of Variation = 8.50%

Table 6.4 Diallel analysis of variance, after Hayman (1954), and Half Diallel analysis, after Morley Jones (1965), of flour protein content.

Experiment Item	df	Full Diallel		df	Half Diallel
		I MS	II MS		III MS
a	3	17.3299***	15.8791***	4	9.7093***
b	6	0.8781***	1.1211*	10	1.5367***
b1	1	0.4931*	0.0115	1	0.2470
b2	3	1.5195***	1.9887**	4	1.6120**
b3	2	0.1085	0.3745	5	1.7344***
c	3	0.3549*	0.1358	-	-
d	3	0.0192	0.2350	-	-
B x a	3	0.0111	0.5575	4	0.3516
B x b	6	0.1427	0.5918	10	0.3467
B x b1	1	0.0793	1.2083	1	0.2522
B x b2	3	0.2502	0.5659	4	0.3774
B x b3	2	0.0131	0.3324	5	0.3409
B x c	3	0.1286	0.1063	-	-
B x d	3	0.0060	0.1726	-	-
Block Interaction	15	0.0862	0.4040	14	0.3481

* P < 0.05 ** P < 0.01 ***P < 0.001

array differs over arrays. This is because some parents contain more dominant alleles than others. The 'b3' item is not significant in the Full Diallel (Experiments A and B). It is, however, highly significant in the Half Diallel (Experiment I). The significance of 'b3' implies the presence of specific combining ability. This significance can be attributed to the crosses Karamu x Ruru and Karamu x Kopara. Both the F1 flour protein means exceeded their respective mid parental values. In only one experiment, that is the Full Diallel of Experiment A, were the items 'b1' and 'c' significant. The detection of significant 'b1' and 'c' is due to the extremely low block interaction item used for testing their significance (Table 6.4). Therefore, significant directional dominance ('b1') and maternal effect ('c') are not general.

In the test of the adequacy of the additive and dominance model (Jinks, 1954, and Mather and Jinks, 1971), the Half Diallel (Experiment I) failed to meet this restrictive model. This was demonstrated by the joint regression analysis of covariance (W_r) on variance (V_r) for the two blocks. The joint regression was found to be non-significant ($P = 0.1459$). The joint linear regression coefficient was 0.5448 with a standard error of ± 0.3262 (Table 6.5). This coefficient was found to be not significantly different from 1.0 nor 0.0 ($P = 0.4558$ and $P = 0.3770$ respectively). This, together with the high standard error, is indicative of the lack of fit for the additive dominance model. Epistasis could be implicated in this absence of fit. The inter-acting array, that is, the array contributing to this lack of fit, was found to be Karamu. When the Karamu array was removed, the joint

Table 6.5 Joint regression coefficient of Wr-Vr graph testing the adequacy of the additive-dominance model (Mather and Jinks, 1971).

Experiment	Joint Regression coefficient	SE ±	Probability from 1.0	Significance from 0.0
A	0.8066	0.0971	0.1172	0.0011
B	0.9110	0.0949	0.4016	0.0007
I	0.5448	0.3262	0.4558	0.3770
Without Karamu array	0.8013	0.1187	0.5950	0.0807
C	0.8370	0.0996	0.1771	0.0011
D	0.8883	0.1154	0.3878	0.0015
II	0.8826	0.0595	0.6473	0.0111

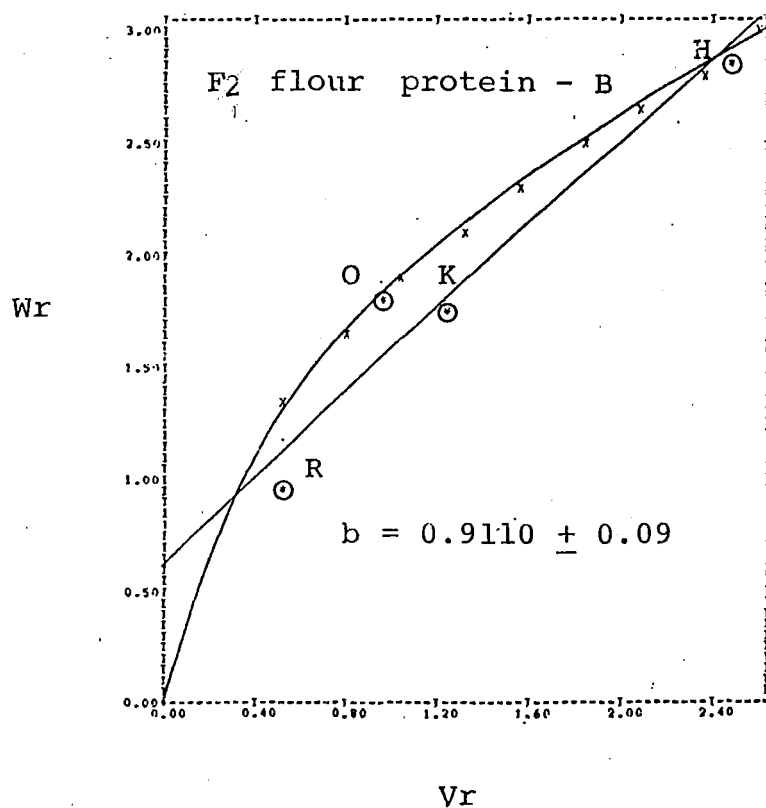
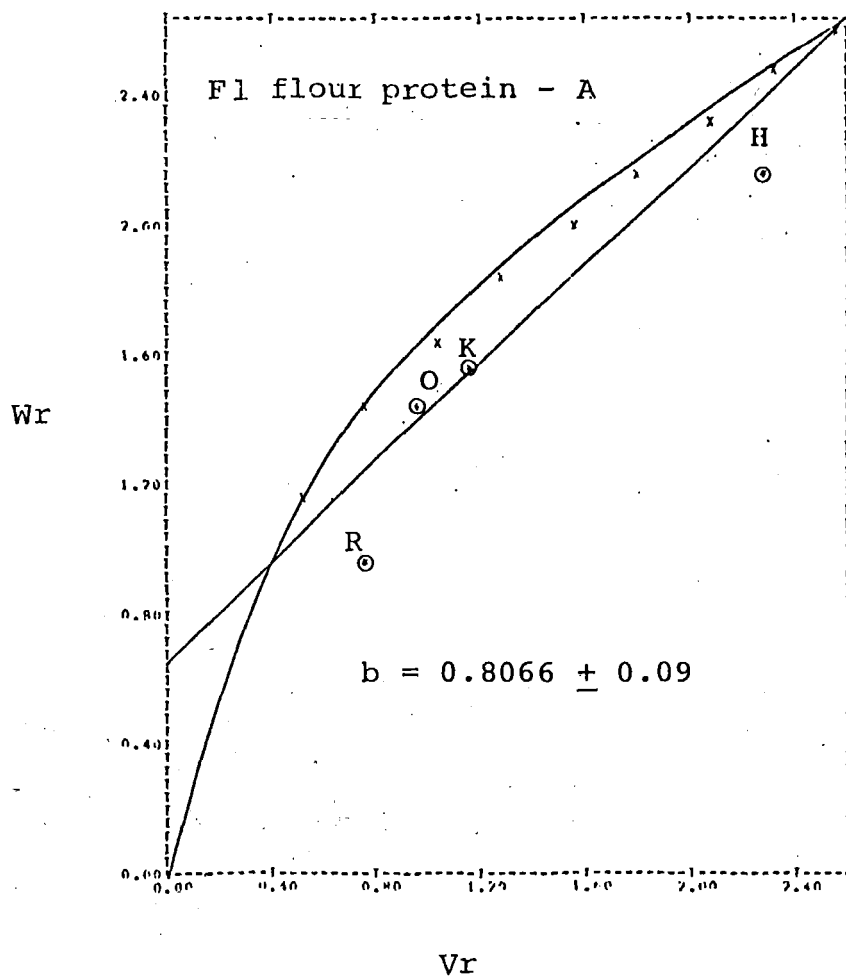


FIG. 6.1 The relationship between W_r and V_r for the F1 and F2 generations for flour protein.

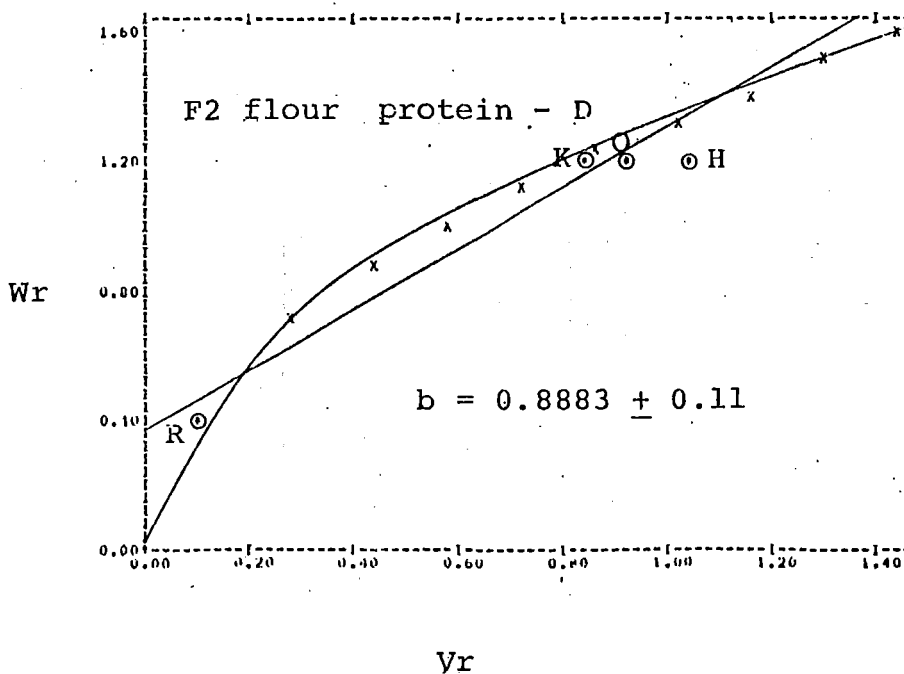
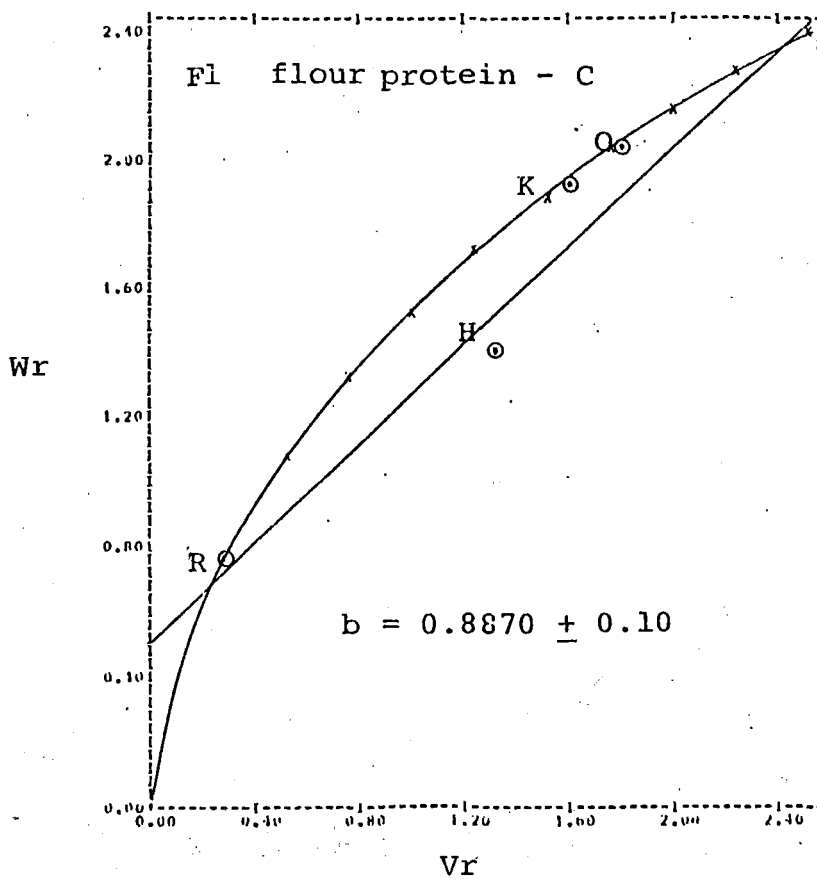


FIG. 6.2 The relationship between W_r and V_r for the F1 and F2 generations for flour protein.

regression analysis of W_r on V_r for the two blocks was found to be highly significant ($P = 0.0025$). Moreover, the joint linear regression coefficient changed from 0.5448 to 0.8013 with a standard error of 0.1187. This coefficient was not significant from 1.0 and approaching significance from 0.0 ($P = 0.5950$, $P = 0.0807$ respectively). The removal of the Karamu array therefore provided an improved fit for the additive and dominance model.

The adequacy of additive and dominance model for the crosses without Karamu is shown by the analysis of Full Diallel (Experiments A and B). In both experiments, joint regression analysis showed high significance ($P = 0.0011$ and $P = 0.0007$ respectively). The joint linear regression coefficients were also found to be not significant from 1.0 ($P = 0.1172$ and $P = 0.4016$ respectively), but highly significant from 0.0 ($P = 0.0011$ and $P = 0.0007$ respectively).

The covariance (W_r) - variance (V_r) graphical analysis also enable the dominance relationship of the parents and their average degree of dominance to be worked out. The graphs of Figures 6.1 and 6.2 showed the regression lines lying close to the limiting parabolas. It can therefore be inferred that the average dominance level is incomplete or partial. It can also be concluded that the dominance is towards low protein content. This conclusion is drawn from the fact that cultivar Ruru the lowest protein parent, had W_r - V_r array values nearest to the origin, whereas the cultivar Hilgendorf had its values furthest away from the origin (Figures 6.1, 6.2). Therefore, cultivar Ruru carries the most dominant genes whereas cultivar Hilgendorf has the most recessive genes

controlling this trait. Moreover, the dominance relationship and the average partial dominance shown by the parents remained unchanged for all three experiments. Therefore, time of sowing has not affected these genetic effects. Similarly, an absence of effect of sowing time on the magnitude of the 'a' and 'b' items is also evident (Table 6.4).

6.3.2 *F1 and F2 Generations Full Diallel and F2 Generation Half Diallel Analyses*

The mean flour protein contents of the families of the second season (Tables 6.6, 6.7 and 6.8) showed lower absolute values than those of the first season (Tables 6.1, 6.2 and 6.3). However, the relative values of the parents remained in the same order in all six experiments. Cultivar Hilgendorf still maintained its high flour protein content, while cultivars Kopara and Oroua had intermediate protein contents. Cultivars Ruru and Karamu again recorded low flour protein content. The differential effects of the environments on the flour protein status, as shown by the differences in the absolute protein percentages between the two seasons, are further emphasized by results in Experiments II, C and D. Higher mean flour protein percentages were recorded in Experiment II than Experiments C and D, although these three Experiments were conducted in the same field. This difference was due to the fertility gradient present in the field and was observed as nitrogen deficient symptoms during early vegetative growth.

The Full Diallel and the Half Diallel analyses of variance, shown in Table 6.9, confirmed the high significance of

Table 6.6 Parents and F₁ generation mean flour protein percentages - Experiment C.

	1	2	3	4
1. Hilgendorf	13.40	12.92	13.44	10.91
2. Kopara	12.77	10.89	10.82	9.91
3. Oroua	12.62	10.82	11.06	9.82
4. Ruru	10.76	9.71	9.80	9.76

Standard Error of Mean = 0.3439

Coefficient of Variation = 4.34%

Table 6.7 Parents and F₂ generation mean flour protein percentages - Experiment D

	1	2	3	4
1. Hilgendorf	12.95	12.00	11.97	10.67
2. Kopara	12.52	11.50	10.52	10.09
3. Oroua	12.52	10.65	10.67	9.99
4. Ruru	10.42	10.36	9.88	9.86

Standard Error of Mean = 0.3121

Coefficient of Variation = 4.00%

Table 6.8 Parents and F2 generation mean flour protein percentages - Experiment II

	1	2	3	4	5
1. Hilgendorf	15.31	13.60	14.26	12.19	12.04
2. Kopara		11.55	12.25	10.99	11.56
3. Oroua			11.70	10.37	11.20
4. Ruru				10.37	10.30
5. Karamu					10.31

Standard Error of Mean = 0.2814

Coefficient of Variation = 3.35%

Table 6.9 Diallel analysis of variance, after Hayman (1954) and Half Diallel analysis, after Morley Jones (1965) of flour protein content.

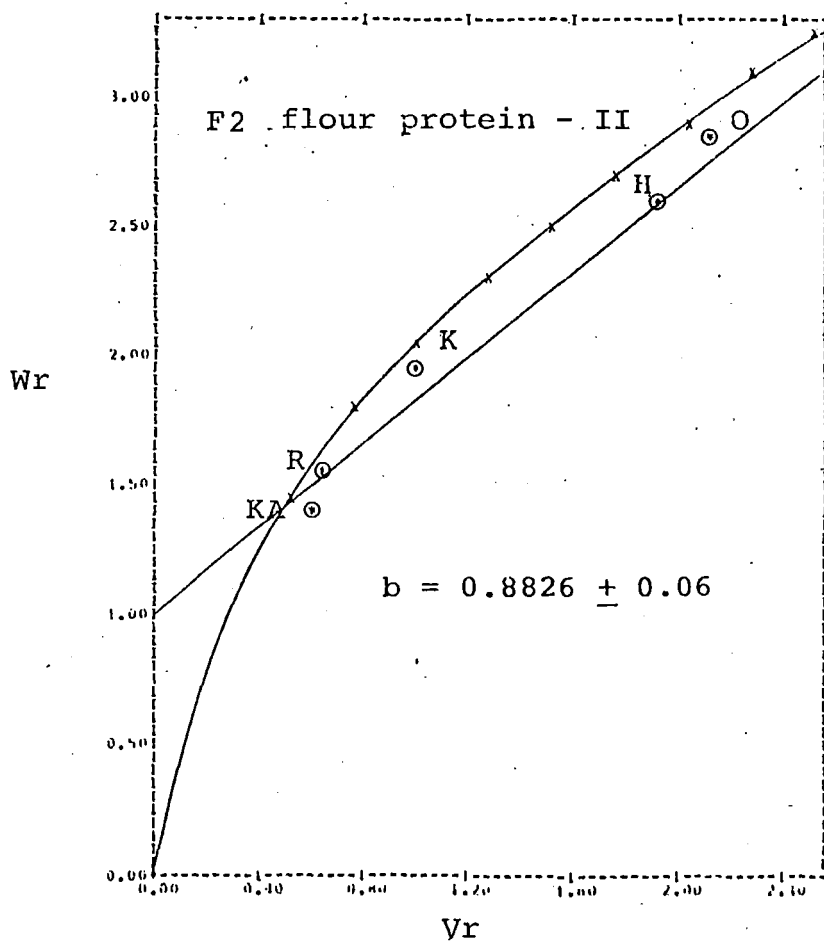
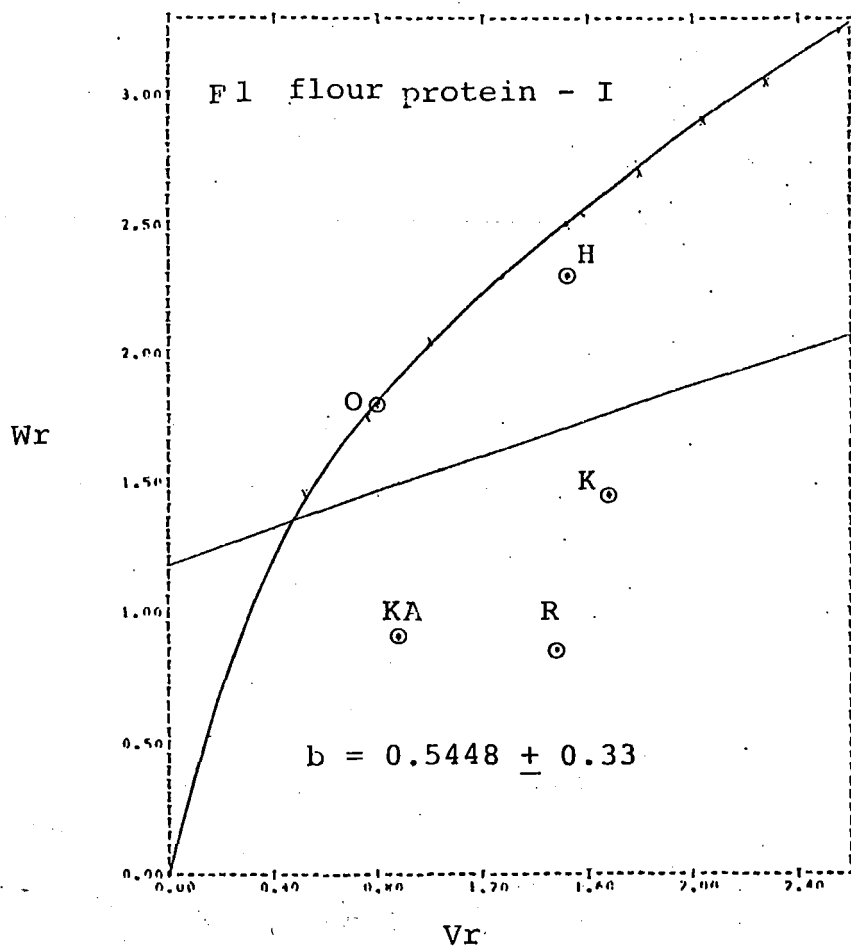
Experiment	df	Full Diallel		df	Half Diallel	
		C MS	D MS		II MS	
a	3	16.4420***	9.3349***	4	14.7617***	
b	6	0.9106*	0.5620*	10	0.4357*	
b1	1	0.0446	0.4988	1	0.0056	
b2	3	1.2580*	0.3236	4	0.4655	
b3	2	0.8224*	0.9511*	5	0.4978*	
c	3	0.1706	0.1379	-	-	
d	3	0.0842	0.1465	-	-	
B x a	3	0.1284	0.2121	4	0.2321	
B x b	6	0.2154	0.1060	10	0.1289	
B x b1	1	0.8559	0.0000	1	0.5568*	
B x b2	3	0.0683	0.0153	4	0.1467	
B x b3	2	0.1154	0.2951	5	0.0290	
B x c	3	0.4438	0.4447	-	-	
B x d	3	0.1798	0.1054	-	-	
Block Interaction	15	0.2366	0.1949	14	0.1584	

* $P < 0.05$ ** $P < 0.01$ *** $P < 0.001$

the 'a' or additive effect and the presence of the dominance or 'b' component. However, of the components of the 'b' item, the 'b2' item was significant only in Experiment C. This contradicts the results of Experiments I, A and B. An additional difference was the presence of a significant 'b3' item in all three Experiments (II, C and D) as compared to the significance of 'b3' in only one experiment for the first season (Experiment I). However, the 'b1', 'c' and 'd' items were all not significant thereby confirming the previous conclusions on absence of directional dominance, maternal effect and reciprocal differences.

In the test for the adequacy of the additive and dominance model, using the joint regression analysis shown in Table 6.5, all three experiments (II, C and D) showed complete agreement in confirming the adequacy of the model. The conclusions of the 4 x 4 Full Diallel Experiments (C and D) involving the F1 and F2 generations are similar to that of the 4 x 4 Full Diallel Experiments (A and B) involving the F1 generation. However, the results of the Half Diallel Experiment II involving the F2 generation contradict those of the Half Diallel Experiment I involving only the F1 generation. While the results of Experiment I showed the inadequacy of the additive and dominance model due to the Karamu array, there is no such evidence in the F2 generation of the Half Diallel in Experiment II. The inability in this season to detect the expression of epistasis recorded in the previous season underlined the importance of the environment on the inheritance of protein content.

The covariance (Wr) - variance (Vr) graphical analysis



F.G. 6.3 The relationship between W_r and V_r for the F1 and F2 generations for flour protein.

confirmed the dominance relationship of the five parents (Figures 6.1, 6.2, 6.3). The low protein cultivars Ruru and Karamu occupied the positions near the origin indicating that the low protein parents carried most dominant alleles. The high protein parents were positioned away from the origin suggesting the presence of recessive alleles in these parents. The Wr-Vr line consistently cut the intercept above the origin and approximately the limiting parabola. This confirmed the average partial dominance recorded in the previous season for flour protein content.

6.4 DISCUSSION

The ultimate objective of a quantitative genetic analysis, such as this one, is to predict the outcome of a cross. This information could enable a breeder to plan his crossing and selection strategies. It is evident, at this stage, that for high protein selections, parents such as Hilgendorf, Oroua and Kopara would be the most useful. The generally high additive effect of this high protein trait is emphasized by the high narrow sense heritability estimates of 52%, 83.4%, 63.9%, 71.2%, 60.6% and 79.5% in our six experiments (I, A, B, II, C and D) respectively. Progress in early generation selection is therefore possible. A further point of interest that can be drawn from this study is the interactions of the environment with the expression of the genetic components. The inconsistency in the expression of significance of the 'b3' and 'b2' items in all six experiments emphasized the role of the environment in the genetic expression of this trait. Moreover, the highly significant combin-

ing abilities in the crosses Karamu x Kopara and Karamu x Ruru recorded in Experiment I could not be duplicated in the F2 studies of Experiment II. It must, therefore, be cautioned that conclusions based on isolated experiments on this trait could be too simplistic. Studies should be conducted over seasons and locations to arrive at a consensus conclusion. The difficulties of studies of this nature have been highlighted by Halloran (1975). Thompson and Whitehouse (1962) have found similar inconsistencies in their genetical studies on flour N. While they found the additive and dominance model adequate in two locations, they also recorded epistasis in two other locations.

CHAPTER 7

A CORRELATION AND PATH COEFFICIENT ANALYSIS
OF YIELD COMPONENTS AND RELATED TRAITS
IN SOME NEW ZEALAND WHEAT CULTIVARS

7.1 INTRODUCTION

An adequate understanding of the interrelationship among various traits is vital in the practice of crop breeding. In wheat simple correlations of morphological and yield related characters have been provided by various workers (Fonseca and Patterson, 1968; Hsu and Walton, 1970; Paroda and Joshi, 1970; Dougherty *et al.*, 1975; Scott *et al.*, 1977). The correlation coefficient of spike number with yield has been shown to be high by these workers. They also recorded moderately high correlation coefficients for grains per spike and average grain weight with yield. Bhatt (1973), however, cautioned that, although these correlation coefficient estimates provided some understanding of the relationship between yield and its components, they did not provide an exact picture of the relative importance of the direct contributions of each of the traits towards yield. The path coefficient analysis can be used effectively to provide measures of the direct and indirect effects of the components of yield and related traits on yield.

The path coefficient analysis was first proposed by Wright (1921) and further described by Li (1955, 1956). The earliest application of path coefficient analysis to a plant

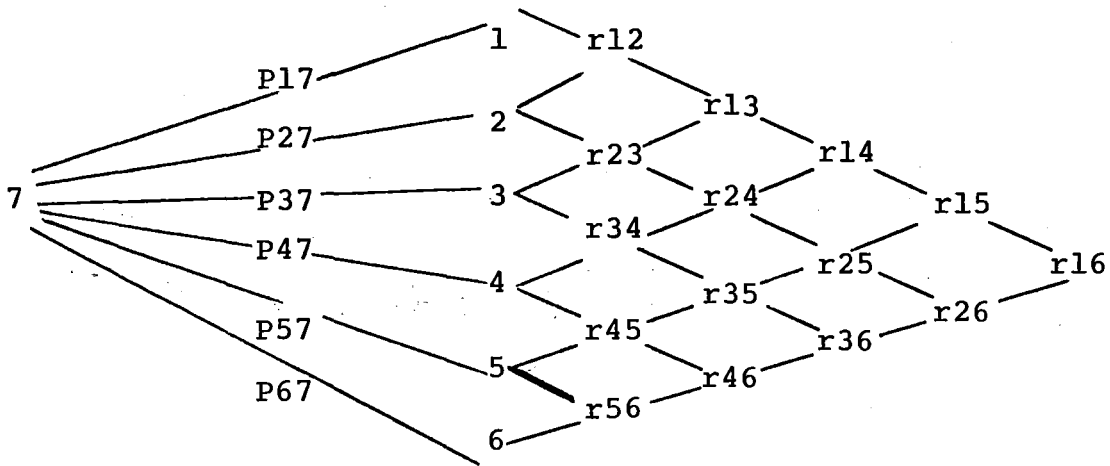
breeding study was discussed by Dewey and Lu (1959).

Workers like Fonseca and Patterson (1968), Paroda and Joshi (1970) and Bhatt (1973) had found the application of path coefficient analysis useful in elucidating the direct and indirect effects of various yield component traits on wheat yield. Fonseca and Patterson (1968) showed the number of spikes per plant to have a high and positive direct effect on yield per plant. Similar conclusions on the importance of the direct effect of spikes per plant were made by Paroda and Joshi (1970) and Bhatt (1973). Bhatt (1973) also found grain weight to have a strong direct effect on yield. Grain number per spike also exerted significant direct contribution to grain yield per plant (Fonseca and Patterson, 1968).

7.2 MATERIALS AND METHODS

The simple correlation coefficient can be partitioned by the path coefficient analysis into components of direct effect and indirect effects. The direct effect component is termed the path coefficient and is simply the standardized partial regression coefficient, and it measures the direct effect of one variate on another. Furthermore, because of the standardization, the standardized partial regression coefficients or the path coefficients can be used to provide an estimation of the relative importance of different independent variates on a dependent variate. The use of the path coefficient analysis demands a cause and effect situation and is ideally suited for the analysis of the effects of yield components and related traits on yield. In this study, four yield components and two yield related traits form the causal system and the yield (weight of grain per

FIG. 7.1 Path Diagram of Yield and Yield Related Traits.



Path Coefficients

Correlation Coefficients

1. Height of Main Stem (cm)
2. Length of Spike of Main Stem (cm)
3. Number of Spikelets of Main Stem
4. Average Weight of Grain (g)
5. Average Grain Number per Spike
6. Number of Spikes
7. Yield Per Plant (g)

plant) represents the effect in this model.

In the path diagram (Figure 7.1), mutual associations are represented by the correlation of coefficients, r_{ij} , and direct effects are measured by the path coefficients, P_{ij} . The path system is governed by the following relationship between correlation and path coefficients.

$$r_{17} = P_{17} + r_{12} * P_{27} + r_{13} * P_{37} + r_{14} * P_{47} + r_{15} * P_{57} + r_{16} * P_{67}$$

$$r_{27} = P_{27} + r_{12} * P_{17} + r_{23} * P_{37} + r_{24} * P_{47} + r_{25} * P_{57} + r_{26} * P_{67}$$

$$r_{37} = P_{37} + r_{23} * P_{27} + r_{13} * P_{17} + r_{34} * P_{47} + r_{35} * P_{57} + r_{36} * P_{67}$$

$$r_{47} = P_{47} + r_{34} * P_{37} + r_{24} * P_{27} + r_{14} * P_{17} + r_{45} * P_{57} + r_{46} * P_{67}$$

$$r_{57} = P_{57} + r_{45} * P_{47} + r_{35} * P_{37} + r_{25} * P_{27} + r_{15} * P_{17} + r_{56} * P_{67}$$

$$r_{67} = P_{67} + r_{56} * P_{57} + r_{46} * P_{47} + r_{36} * P_{37} + r_{26} * P_{27} + r_{16} * P_{17}$$

The path coefficients in this system can be obtained by solving the above simultaneous equations. In this study, the measurements were made on 300 individual plants from 15 genotypes (Experiment I) and were analysed by the computer programme BASIS. The output from BASIS includes the simple correlation matrix and the beta coefficients which are the standardised partial regression coefficients or the path coefficients. The indirect effects of each trait on yield, were obtained by multiplying the respective path coefficients with the correlation coefficients as in the equations above. The respective path coefficients and the indirect components were summed to tally the correlation coefficients of the respective traits with yield.

Table 7.1 Path-coefficient analysis of traits contributing to grain yield in five parent diallel in wheat.

Pathways of Association	Direct Effect path coefficient (P)	Indirect effect (P x r)	Correlation coefficient (r)
<u>Spike length vs yield</u>			
Direct effect	-0.01963		
Indirect effect via height		0.00054	
Indirect effect via spikelet no.		-0.00307	
Indirect effect via average grain wt.		-0.05040	
Indirect effect via grain/spike		0.32565	
Indirect effect via no. of spike/plant		0.08796	
Total			0.3397
<u>Number of spikelet/spike vs yield</u>			
Direct effect	-0.00421		
Indirect effect via spike length		0.00076	
Indirect effect via height		-0.01429	
Indirect effect via average grain wt.		-0.07915	
Indirect effect via grains/spike		0.37023	
Indirect effect via no. of spike/plant		0.16381	
Total			0.4276
<u>Average grain weight vs yield</u>			
Direct effect	0.53771		
Indirect effect via grain/spike		-0.1080	
Indirect effect via no. of spike		-0.07367	
Indirect effect via spikelet/spike		0.00062	
Indirect effect via spike length		0.00184	
Indirect effect via height		0.00289	
Total			0.3546

Table 7.1 (cont'd....)

Pathways of Association	Direct Effect path coefficient (P)	Indirect effect (P x r)	Correlation coefficient (r)
<u>Grains per spike vs yield</u>			
Direct effect	0.73146		
Indirect effect via no. of spike		-0.14291	
Indirect effect via height		-0.00175	
Indirect effect via spike length		-0.00870	
Indirect effect via spikelet/spike		-0.00207	
Indirect effect via average grain wt.		0.080121	
Total			0.4959
<u>Number of spikes vs yield</u>			
Direct effect	0.75098		
Indirect effect via grain/spike		-0.13918	
Indirect effect via height		0.00064	
Indirect effect via spike length		-0.00230	
Indirect effect via spikelet no.		0.00093	
Indirect effect via average grain wt.		-0.05270	
Total			0.5565
<u>Height vs yield</u>			
Direct effect	-0.01194		
Indirect effect via spike length		0.00054	
Indirect effect via grain/spike		-0.00027	
Indirect effect via spikelet no.		0.13034	
Indirect effect via average grain wt.		+0.10708	
Indirect effect via. no. of spike/plant		-0.04025	
Total			0.1864

7.3 RESULTS AND DISCUSSION

Details of the path coefficients, indirect effect components and correlation coefficients are shown in Table 7.1.

Effect of Spike Length and Spikelet Number Per Spike On Yield

Spike length and spikelet number were found to have correlation coefficients of 0.3397 and 0.4276 with grain weight per plant respectively. However, the path analysis revealed the negligible direct effects of both these traits on yield. Their path coefficients were -0.0196 and -0.0042 respectively. The moderate correlation coefficients between these two traits and yield were mainly attributed to their effects via grain number per spike. The indirect effects via grain number per spike were 0.3255 and 0.3702 respectively.

Effect of Average Grain Weight, Grains Per Spike and Number of Spikes on Yield

Average grain weight, grains per spike and number of spike expressed moderate to high association with grain yield. Their correlation coefficients with grain yield were 0.3546, 0.4959 and 0.5565 respectively. Strong direct effects were registered by all three traits on yield per plant. Average grain weight had a direct effect or path coefficient of 0.53771, whereas for grain number per spike and number of spikes per plant, the path coefficient were 0.7315 and 0.7510. This underlined the relative importance of these three on yield per plant. Grain number per spike and number of spikes

were the two major and equal components of yield with average grain weight of grain followed in third order of importance. A further point of interest that can be deduced from this analysis is the compensatory nature of these three major yield components. For example, the strong direct effect of average grain weight was substantially reduced by its indirect path via grains per spike. This indirect effect was of the order -0.108 . More evidence on the compensatory effects of these components was provided by the other two traits, grain number per spike and number of spikes. Grain number per spike had a negative indirect effect, via number of spikes, of -0.1429 , whereas number of spikes had an indirect effect via grains per spike of -0.1392 .

Effect of Plant Height on Yield

The correlation coefficient of plant height with yield was 0.1864 . The path coefficient analysis showed height to have a very small direct effect on yield of 0.0119 . However, this observation is contrary to the high positive correlation recorded between height and yield (Law et al., 1978). This apparent anomaly can be explained on the basis of the genotypes used in this study. The semidwarf cultivars used in this study had been highly selected for yield and lodging resistance. It is therefore to be expected that the association between height and yield would be low. The high correlation between height and yield recorded by Law et al. (1978) was on segregating populations of F₃, F₄ and F₅ generations in a cross between Cappela-Desprez and Besostaya I. This observation has led them to propose the breeding of "tall dwarfs". This involves a strategy of maintaining the genetic

variation of tallness and therefore the correlated effect of height and yield and a parallel introduction of dwarfing genes to prevent lodging.

The path coefficient analysis is thus useful in identifying the traits that contribute directly to yield. It gave a somewhat different picture from that of the simple correlation coefficient. For example, the simple correlation coefficients between spike length, spikelet number and yield indicated a moderate association. The path analysis, however, help to expose the absence of any direct effect of these two traits on yield. On the other hand, the path analysis reinforced the association between average grain weight, grains per spike and number of spikes per plant with plant yield. The importance of grain number per plant and spikes per plant had been demonstrated by Scott *et al.* (1977). They showed that the most obvious method of producing a large number of grain per unit area was by improving grain number per spikelet and maintaining a high spike density through reduction in tiller mortality. Dougherty *et al.* (1975) in fitting yield components against grain yield by stepwise linear regression technique, found spikelets per spike, spikes per m^2 , grains per spikelet and average grain weight ranked in decreasing order of importance. The role of spike number and grain number per spike as major yield determinants has been emphasized by Langer (1976, 1978) when he proposed that the critical time to influence yield was when the growth processes involved in determining these components were in progress.

With the identification of the major yield determining traits, the agronomist can improve yield by strategically timing

his fertiliser and irrigation applications to influence the main determinants of yield. The plant breeder can do equally well by concentrating in his breeding programmes on these important traits.

CHAPTER 8

EPILOGUE

8.1 CRITICAL EVALUATION OF THE BIOMETRICAL METHODS

The application of quantitative genetical methods to understanding the inheritance of wheat has been considerable. Interest in the application of quantitative genetical analysis has developed since the publications of Jinks and Hayman (1953), Jinks (1954). These papers discussed the application of genetic algebra to plant breeding and have opened new directions to plant breeders seeking methods for evaluating their crosses. The need for such quantitative methods was shown by their rapid and widespread acceptance by plant breeders ^{Cofp & Wright} (Whitehouse, 1958; Lupton, 1961; Crum- ^{Fisher (1918)} packer and Allard, 1962; Johnson, 1963). More recently, other biometrical methods allowing for more refined understanding, including the detection of epistasis, have been utilised (Ketata et al., 1976b; Singh and Singh, 1976; Snape et al., 1977). Conclusions derived are as diverse as the available methods. The contradictions are so widespread that conclusions derived from the same method are often conflicting. This absence of agreement has often been attributed, sometimes rightly so, to differences in the genetical background of the parents and genotype environmental interactions.

It is the intention of the following passage to highlight and evaluate some aspects of our limited experience with the three biometrical methods discussed in the previous chapter.

8.1.1 *High Sampling Variances*

Genetical studies are often conducted with small sample sizes when availability of crossed seeds and the need to restrict family replicates to manageable proportions are major constraints. This handicap often results in high within family variances for complex trait such as yield. These high within family variances, often unreported, could explain some of the conflicting reports. The problem can be illustrated by our study of grain yield per plant. This trait has consistently showed a high coefficient of variation of between 20 to 30 per cent. The family means derived from these experiments therefore are likely to be biased. In the Covariance (W_r) and Variance (V_r) graphical analysis of the Half Diallel, the use of such bias means (simulating a genetic effect) will greatly inflate the variance and covariance terms leading to an absence of fit to the additive and dominance model. On the other hand, the high within family variances, which are pooled to test for the presence of epistasis, in both the Scaling Tests and the New Triple Test Cross analyses, will enhance the difficulty of obtaining a significant epistasis. Contradictory and misleading results can therefore be produced by different biometrical methods under high sampling variances.

8.1.2 *The New Triple Test Cross Analysis*

Perhaps one of the major criticism that can be directed at the method of Chahal and Jinks (1978), is the use of two statistical approaches for testing the significance of the additive, dominance and epistatic comparisons. The signifi-

cance of the additive and dominance comparisons is tested by the rigorous analysis of variance (Method I) while the significance of the epistatic comparison is tested against the pooled variance derived from the variances of the back-cross, F1 and parental family means (Method II). Method II is a less stringent test compared to Method I and often a significant epistatic effect detected by Method II cannot be reproduced by Method I.

In practice, the need to resort to the less stringent statistical approach of Method II is necessary and may be explained by the inherently low magnitude of epistatic effects. However, to ensure comparable conclusions it may be more satisfactory to test all three comparisons, additive, dominance and epistatic, by the less stringent Method II. This may possibly avoid the situation where detection of significant epistasis by the less stringent test is accompanied by absence of significant dominance or additive effect.

The less stringent Method II for testing epistasis has, however, provided realistic and confirmatory conclusions to those results obtained by the Half Diallel, the Scaling Tests and the Curilinear Regression analyses. For instance, the Half Diallel analysis on plant height on the five parents (except Atlas 66), has provided evidence for the adequacy of the additive and dominance model. The New Triple Test Cross analysis on plant height on the parents without Atlas 66 has also shown no evidence of epistasis. This concurrent evidence suggests the use of the liberal Method II is sufficiently conservative to detect an absence of epistasis. Furthermore, when the Atlas 66 and Karamu cross is included

in the analysis, Method II was able to detect the presence of epistasis. Unfortunately, no Half Diallel analysis involving the Atlas 66 family was available to confirm the significance of epistasis. A separate method, a Curilinear Regression analysis (Li, 1964) (not reported in this thesis), was used to verify the absence of fit for the additive and dominance model for the Atlas 66 cross. Significant linear and quadratic regressions confirm the lack of fit for the additive and dominance model for plant height in the cross involving Atlas 66 and Karamu. Further confirmation of the presence of epistatic effect for plant height in the Atlas 66 and Karamu cross was provided by the significant *i*, *j*, and *l* estimates. Significance of these terms indicates epistasis (Mather and Jinks (1971)).

The tests by Method II of the New Triple Test Cross have also shown satisfactory agreement with the Half Diallel analyses for traits such as spikelets per spike, grains at P10, grains per spike, spike per plant, 1000 grain weight and spike length.

8.2 SUMMARY

8.2.1 *Multiplicative Epistasis*

Three principal components of yield, spikes per plant, 1000 grain weight and grains per spikes, have complex genetical control involving epistasis. The yield component approach has therefore not entirely resolved epistasis. However, grains per spike can further be resolved into grains at individual spikelet position and number of spikelets per spike.

This partition has resolved the multiplicative epistasis for grains per spike. Grain number at a particular spikelet position, as exemplified by spikelet position ten (Pl0), is controlled by mainly additive genes, whereas spikelet per spike is under additive and dominance control. This evidence of multiplicative epistasis can possibly explain the failure to fix the large spikes in early generations. A strategy, based on the selection of the subcomponent of grains per spike, is proposed. Early generation selection should be concentrated on fixing the additive genes controlling grain number at each spikelet position. This method could possibly lead to a yield increase if component compensation is not complete.

8.2.2 *Yield Per Plant*

As discussed in the previous section 8.1.1, different biometrical analyses of this highly variable trait have given conflicting conclusions. Therefore, no understanding of the nature of gene action can be reached by the present study. Ample evidence has, however, been presented on the difficulty of obtaining a unified conclusion under the present sampling technique. It is suggested that future work for this trait should be carried out with a greater sample size to overcome difficulties brought about by high sampling variances. As the increase in sample size will rapidly increase the work load to an unmanageable level, experiments should be designed solely for the study of this single important trait.

8.2.3 *Plant Height*

Plant height is under the control of additive and dominance genes for all the cultivars studied except Atlas 66 which expresses duplicate type epistasis. This type of epistasis is particularly unsuitable for selection of inbred line. This suggests the impracticality of the 'tall dwarf' breeding strategy for the cross involving Atlas 66 and Karamu. The 'tall dwarf' model can, however, be gainfully utilised in crosses among the semidwarf and standard height cultivars studied here. This is because the semidwarf cultivars possess the major dwarfing genes and height is under mainly additive gene control. The major dwarfing genes can be fixed in early generation, while the selection for the tall genes can be delayed until later generation, thereby meeting the requirements of the 'tall dwarf' selection strategy.

8.2.4 *Spike and Flag Leaf Length*

Spike length has been shown to be under additive and dominance gene control, with considerable prospect for response to selection. There is scope for indirect increase in photosynthetic area above the flag leaf through increase of spike length. However, the direct contribution to yield improvement of increased spike length is doubted because of the over-riding importance of spikelet density and floret fertility.

8.2.5 *Flour Protein*

Flour protein content is controlled by mainly additive effect as shown by the high narrow sense heritability recorded

in this study. This suggests good scope for early response to selection for this trait. Cultivars such as Hilgendorf, Kopara and Oroua should be most useful for high protein selection. However, the detection of epistasis in one of the two seasons in the crosses involving Karamu emphasized the existence of genotype environmental interaction for flour protein. This highlights the need to conduct such studies over a range of environments.

8.2.6 *Path Analysis of Yield and Related Traits*

In this spaced planted study, spikes per plant and grains per spike have been shown to have strong direct influence on yield per plant. The other yield component, 1000 grain weight, was found to have a moderate influence whereas spikelet per spike was found to have no direct effect on yield. The moderate correlation between spikelet per spike and yield was due mainly to the indirect effect via grains per spike. The two morpho-physiological traits, plant height and spike length, also recorded negligible direct effects on yield.

APPENDIX I

H6700 FORT RAN C O M P I L A T I O N M A R K 3

A
=

```

C*****
C*****THIS PROGRAMME IS BASED ON THE *****
C*****HALF DIALLEL ANALYSIS *****
C*****R. MORLEY JONES HEREDITY(1965) 20:117-121*****
C*****

```

```

      DIMENSION E(9,9),F(9,9),G(9,9),BI(9),ND(9),TITLE(6),CSS(9),VRA(9),
1 VPT(9),P(9),PP(9),A(9,9),B(9,9),CAPT(6),DERVR(9),TWRVR(9),
2 VR(9),WR(9),DERVR1(9),TWRVR1(9),LT(9),CP(9),CP1(9),VR1(9),WR1(9),
3 PAR(9),DERVR2(9),TWRVR2(9),VR2(9),CP2(9),FMT(16)
4 BSS(7),ALOGCV(7),VALOG(7),RENDR(7)
      DATA TITLE/'A','B1','B2','B3','B','T'//
      DATA CAPT/'BA','BB1','BB2','BB3','BB','BT'//
      DATA LT/'D','H1','H2','F','E','SRH1/D','UV','HEPAR','HERBD'/
1919 READ(5,1,END=2929)(FMT(K),K=1,16)

```

```

      WRITE(6,2)(FMT(K),K=1,16)

```

```

1  FORMAT(16A5)

```

```

2  FORMAT(1X,/,T30,16A5,/)

```

```

C*****
C*****N IS NOS. OF FAMILIES,IBLK IS NOS OF BLOCKS*****
C*****

```

```

      READ(5,9,END=2929)N,IBLK

```

```

9  FORMAT(2I4)

```

```

      WRITE(6,100)

```

```

100 FORMAT(1,1X,T35,'INPUT VALUES OF HALF DIALLEL',//)

```

```

C*****
C*****E(I,J) AND F(I,J) ARE ARRAYS WITH MEAN VALUES FOR EACH*****
C*****FOR EACH OF THE TWO BLOCKS*****
C*****G(I,J) IS THE APRAY CONTAINING THE MEAN OF THE TWO BLOCKS*****
C*****

```

```

      DO 1001 I=1,N

```

```

      READ(5,11,END=2929)(E(I,J),J=1,N)

```

```

11  FORMAT(9F8.4)

```

```

      WRITE(6,12)I,(N+1)-I,(E(I,J),J=1,N)

```

```

12  FORMAT(0,*(10X),*(2X,F8.4))

```

```

1001 CONTINUE

```

```

      CALL DIAL(E,N,A1,BB1,BB2,BB3,T1SSQ,NDA,NDB1,NDB2,NDB3,NDT)

```

```

      WRITE(6,100)

```

```

      DO 101 I=1,N

```

```

      READ(5,11,END=2929)(F(I,J),J=1,N)

```

```

      WRITE(6,12)I,(N+1)-I,(F(I,J),J=1,N)

```

```

101  CONTINUE

```

```

      CALL DIAL(F,N,A2,BB1,BB2,BB3,T2SSQ,NFA,NFB1,NFB2,NFB3,NFT)

```

```

      WRITE(6,702)

```

```

702  FORMAT(1,1X,T35,'THE MEAN VALUES OF THE HALF DIALLEL INPUT',//)

```

```

      DO 102 I=1,N

```

```

      DO 102 J=1,N

```

```

      G(I,J)=(E(I,J)+F(I,J))/2

```

```

102  CONTINUE

```

```

      DO 103 I=1,N

```

```

      WRITE(6,12)I,(N+1)-I,(G(I,J),J=1,N)

```

```

103  CONTINUE

```

```

      CALL DIAL(G,N,A3,BF1,BF2,BF3,T3SSQ,NA,NB1,NB2,NB3,NT)

```

```

      WRITE(6,28)

```

```

28  FORMAT(1,0,*,T2,'SOURCE OF VARIATION',2X,'DF',13X,'MEAN SQUARE',

```

```

113X,'VRA',15X,'PROB',17X,'VRT',15X,'PROB',//)

```

```

      DO 750 J=1,N

```

```

750  PAR(J)=G(J,J)

```

```

C*****ARRAY ND CONTAINS THE DEGREES OF FREEDOM*****
C*****
ND(1)=EDA
ND(2)=EDB1
ND(3)=EDB2
ND(4)=EDB3
ND(5)=ND(2)+ND(3)+ND(4)
ND(6)=EDT
C*****
C*****ARRAY CSS CONTAINS THE CORRECTED SUM OF SQUARES*****
C*****
CSS(1)=(2*A3)/ND(1)
CSS(2)=(2*BF1)/ND(2)
CSS(3)=(2*BF2)/ND(3)
CSS(4)=(2*BF3)/ND(4)
CSS(5)=2*(BF1+BF2+BF3)/ND(5)
C*****ARRAY BI CONTAINS THE BLOCK INTERACTION SUM OF SQUARES*****
BI(1)=ABS((2*A3-A2-A1)/ND(1))
BI(2)=ABS((2*BF1-SBB1-BB1)/ND(2))
BI(3)=ABS((2*BF2-SBB2-BB2)/ND(3))
BI(4)=ABS((2*BF3-SBB3-BB3)/ND(4))
BI(5)=(BI(2)+ND(2)+BI(3)+ND(3)+BI(4)*ND(4))/ND(5)
BI(6)=ABS((2*T3SSQ-T2SSQ-T1SSQ)/ND(6))
DO 201 I=1,5
VRA(I)=CSS(I)/BI(I)
VRT(I)=CSS(I)/BI(6)
P(I)=FISHER(ND(I),ND(1),VRA(I))
PP(I)=FISHER(ND(I),ND(6),VRT(I))
WRITE(6,29) TITLE(I),ND(I),CSS(I),VRA(I),P(I),VRT(I),PP(I)
201 CONTINUE
29 FORMAT('0',T5,A6,10X,I4,10X,F12.4,10X,F12.4,10X,F8.4,10X,F12.4,
110X,F8.4)
WRITE(6,33)
33 FORMAT(1X,T35,'THE BLOCK INTERACTION MEAN SQUARE',/)
WRITE(6,32)
32 FORMAT(1X,T40,'OF',20X,'INTERACTION MEANSQUARE',/)
WRITE(6,30)(CAPT(I),ND(I),BI(I),I=1,6)
30 FORMAT('0',T30,A6,10X,I4,10X,F12.4)
CALL DATEST(BI,ND)
WRITE(6,701)
701 FORMAT('1',1X,'THE VARIANCE COVARIANCE ANALYSIS',/)
CALL WRVR(E,N,DWRVR,TWRVR,WR,VR,VP,VRM,WRM,VARM,CP,BI)
DO 204 I=1,N
A(1,I)=DWRVR(I)
B(1,I)=TWRVR(I)
204 CONTINUE
CALL WRVR(E,N,DWRVR1,TWRVR1,WR1,VR1,VP1,VRM1,WRM1,VARM1,CP1,BI)
CALL WRVR(G,N,DWRVR2,TWRVR2,WR2,VR2,VP2,VRM2,WRM2,VARM2,CP2,BI)
DO 205 I=1,N
A(2,I)=DWRVR1(I)
B(2,I)=TWRVR1(I)
205 CONTINUE
WRITE(6,89)
89 FORMAT('1',1X,/,T30,'THE REGRESSION ANALYSIS OF WRVR',/)
CALL REG(H,VR,WP,SSX1,SSY1,SP1,COC1,SECOC1,PX)
CALL REG(N,VR1,WR1,SSX2,SSY2,SP2,COC2,SECOC2,PY)
CALL REG(H,VR2,WR2,SSX3,SSY3,SP3,COC3,SECOC3,PZ)
CALL REG(N,TWRVR2,PAR,SD2X,SD2Y,SDXS0Y,COC,SECOC,P2)
MDB1B2=2
NDJR=1
NDTR=(N-1)*IBLK
NDRE=NDTR-MDB1B2
XJB=(SP1+SP2)/(SSX1+SSX2)
XJRSS=(SP1+SP2)*(SP1+SP2)/(SSX1+SSX2)
SSB1B2=SP1**2/SSX1+SP2**2/SSX2
TSSY=SSY1+SSY2
REMS=SSB1B2-XJRSS
HRSS=SSB1B2-XJRSS
FJR=XJRSS/(REMS/NDRE)
PJR=FISHER(NDJR,NDRE,FJR)
SEJB=SQRT((REMS/NDRE)/(SSX1+SSX2))
WRITE(6,329)
WRITE(6,330)XJBSS,NDJR,FJR,PJR
WRITE(6,331)REMS,NDJR
WRITE(6,332)REMS/NDRE,NDRE

```

```

329 FORMAT(1X, //T20, 'ITEM', 30X, 'NS', 15X, 'DF', 15X, 'VR', 15X, 'PROB', //)
330 FORMAT('0', T20, 'JOINT REGRESSION', 7X, F15.4, 10X, 14, 10X, F12.4, 10X,
1F8.4, //)
331 FORMAT('0', T20, 'HETEROGENEITY OF REGRESSION', F15.4, 10X, 14, //)
332 FORMAT('0', T20, 'REMAINDER', 17X, F15.4, 10X, 14, //)
      TJB0=(XJB-6.0)/SORT(SEJB)
      TJB1=(XJB-1.0)/SORT(SEJB)
      PTJB0=FISHER(EDJR, NDRE, TJB0**2)
      PTJB1=FISHER(EDJR, NDRE, TJB1**2)
      WRITE(6, 333) XJB, SEJB, PTJB1, PTJB0
333 FORMAT(1X, 'JOINT REGRESSION COEFFICIENT IS', F8.4, 5X, 'SE', F8.4, 5X,
* 'SIGNIFICANT FROM 1.0', F8.4, 5X, 'SIGNIFICANT FROM 0.0', F8.4, //)
      WRITE(6, 1010) CDC, SECOC, P2
1010 FORMAT('0', 'THE CORRELATION COEF. OF WR+VR AND PI IS', F8.4, 'SE',
*F8.4, 'IT IS', F8.4, //)
      WRITE(6, 802)
802 FORMAT('1', ///, 1X, T20, 'THE ANALYSIS OF VARIANCE OF WRVR', //)
      CALL ANOVA(A, IBLK, N)
      CALL ANOVA(B, IBLK, N)
      WRITE(6, 607)
607 FORMAT('1', ///, 1X, T35, 'ESTIMATES OF COMPONENTS OF VARIATION', //)
      WRITE(6, 609)
608 FORMAT(1X, F15, 'STATISTICS', 5X, 'BLK1', 20X, 'BLK2', 18X, 'MEAN', 22X
1, 'MODEL', //)
      WRITE(6, 622) VP, VP1, (VP+VP1)/2
      WRITE(6, 603) VR1, VR11, (VR1+VR11)/2
      WRITE(6, 604) WR1, WR11, (WR1+WR11)/2
      WRITE(6, 605) VAR1, VAR11, (VAR1+VAR11)/2
      WRITE(6, 606) B1(6), B1(6), B1(6)
622 FORMAT('0', T20, 'VP', 3(F15.4, 10X, ), 10X, 'D+E', //)
603 FORMAT('0', T20, 'VR1', 3(F15.4, 10X, ), 10X, '1/4D+1/4H1-1/4F+5/9E', //)
604 FORMAT('0', T20, 'WR1', 3(F15.4, 10X, ), 10X, '1/2D-1/4F+1/9E', //)
605 FORMAT(1X, T10, 'VAR1', 3(F15.4, 10X, ), 10X, '1/4D+1/4H1-1/4H2-1/4F
1+5/81E', //)
606 FORMAT('0', T20, 'E', 10X, 3(F15.4, 10X, ), 10X, 'E', //)
      WRITE(6, 611)
611 FORMAT(1X, //, T35, 'PERFECT FIT ESTIMATES OF THE COMPONENTS', //)
      WRITE(6, 609)
609 FORMAT(1X, T15, 'COMPONENT', 12X, 'BLK1', 27X, 'BLK2', 30X, 'MEAN', //)
      DO 601 L=1, 9
      WRITE(6, 602) LT(L), CP(L), CP1(L), (CP(L)+CP1(L))/2
601 CONTINUE
602 FORMAT('0', T20, A6, 2X, F15.4, 20X, F15.4, 20X, F15.4, //)
      GO TO 1919
2929 STOP
      END
002:0233:1 IS THE LOCATION FOR EXCEPTIONAL ACTION ON THE I/O STAT
002:0236:2 IS THE LOCATION FOR EXCEPTIONAL ACTION ON THE I/O STAT
002:0239:3 IS THE LOCATION FOR EXCEPTIONAL ACTION ON THE I/O STAT
002:023C:4 IS THE LOCATION FOR EXCEPTIONAL ACTION ON THE I/O STAT

```

```

SUBROUTINE DIAL(D,N,A,B1,B2,B3,TSSQ,NQFA,NQFB1,NQFB2,NQFB3,NQFT)
DIMENSION D(9,9),SUMR(9),SUMUR(9),SUMC(9),SUMTR(9)
WRITE(6,14)N,(D(I,J),J=1,N)
DO 30 J=2,N
DO 40 I=1,J-1
D(J,I)=D(I,J)
40 CONTINUE
WRITE(6,14)N,(D(J,L),L=1,N)
14 FORMAT(1X,*(F10.3,2X),/)
30 CONTINUE
DO 50 J=1,N
SUMR(J)=0.0
SUMC(J)=0.0
SUMUR(J)=0.0
DO 60 L=1,N
SUMR(J)=SUMR(J)+D(J,L)
SUMC(J)=SUMC(J)+D(L,J)
60 CONTINUE
SUMUR(J)=SUMR(J)+D(J,J)
SUMTR(J)=2*SUMUR(J)-N*D(J,J)
50 CONTINUE
SUMSQ=0.0
SUMPA=0.0
DO 20 I=1,N
DO 201 J=1,N
SUM=SUM+D(I,J)
SUMSQ=SUMSQ+D(I,J)*D(I,J)
201 CONTINUE
SUMPA=SUMPA+D(I,1)
20 CONTINUE
WRITE(6,15)N,(SUMR(L),L=1,N)
15 FORMAT(1X,'SUM OF ROW',T20,*F12.4//)
WRITE(6,16)N,(SUMC(L),L=1,N)
16 FORMAT(1X,'SUM OF COLUMN',T20,*F12.4//)
WRITE(6,17)N,(SUMUR(L),L=1,N)
17 FORMAT(1X,'SUM OF ROW+PARABOL',T20,*F12.4,/)
WRITE(6,19)N,(SUMTR(J),J=1,N)
19 FORMAT(1X,'SUM OF TR',T20,*F12.4//)
WRITE(6,18)SUM,SUMPA,SUMSQ
18 FORMAT(1X,3F15.4)
SSQR=0.0
TOTUR=0.0
SSQTR=0.0
TOTTR=0.0
DO 70 L=1,N
SSQR=SSQR+SUMUR(L)*SUMUR(L)
TOTUR=TOTUR+SUMUR(L)
TOTTR=TOTTR+SUMTR(L)
SSQTR=SSQTR+SUMTR(L)*SUMTR(L)
70 CONTINUE
TSSQ=SUMSQ-(2*SUM*SUM)/(N*(N+1))
A=((SSQR-(TOTUR*TOTUR)/N)/(N+2))
B1=((2*SUM-(N+1)*SUMPA)**2/(N*(N**2-1)))
B2=((SSQTR-(TOTTR*TOTTR)/N)/(N*N-4))
B3=TSSQ-A-B1-B2
NQFA=N-1
NQFB1=1
NQFB2=N-1
NQFT=(N*(N+1)/2)-1
NQFB3=NQFT-NQFA-NQFB1-NQFB2
WRITE(6,22)
22 FORMAT(1X,10X,'SUM OF SQUARE',10X,'DF',10X,'MEAN SQUARE',/)
WRITE(6,21)A,NQFA,A/NQFA
WRITE(6,21)B1,NQFB1,B1/NQFB1
WRITE(6,21)B2,NQFB2,B2/NQFB2
WRITE(6,21)B3,NQFB3,B3/NQFB3
WRITE(6,21)TSSQ,NQFT
21 FORMAT(1X,10X,F12.4,10X,I4,10X,F12.4,/)
RETURN
END

```

```

SUBROUTINE ANOVA(A,IBLK,IM)
DIMENSION A(9,9)
SUM=0.0
SSQ=0.0
SSQR=0.0
DO 20 J=1,IBLK
SUMR=0.0
DO 30 J=1,IM
SUM=SUM+A(I,J)
SSQ=SSQ+A(I,J)*A(I,J)
SUMR=SUMR+A(I,J)
30 CONTINUE
SSQR=SSQR+SUMR*SUMR
20 CONTINUE
SSQT=0.0
DO 40 J=1,IM
SUMT=0.0
DO 50 I=1,IBLK
SUMT=SUMT+A(I,J)
50 CONTINUE
SSQT=SSQT+SUMT*SUMT
40 CONTINUE
CF=SUM*SUM/(IM*IBLK)
TOTSSQ=SSQ-CF
TOTTSQ=SSQT/IBLK-CF
TOTRSQ=SSQR/IM-CF
ERRSQ=TOTSSQ-TOTTSQ-TOTRSQ
NDFT=IM-1
NDFR=IBLK-1
NEDF=IM*IBLK-1
NEDF=NEDF-NDFT-NDFR
AMSQ=TOTTSQ/NDFT
AMSQR=TOTRSQ/NDFR
AMSE=ERRSQ/NEDF
FR=AMSQ/AMSE
PR=FISHER(NDFT,NEDF,FR)
WRITE(6,12)
WRITE(6,13)NDFR,TOTRSQ,AMSQR,FR,PR
WRITE(6,14)NDFT,TOTTSQ,AMSQ
WRITE(6,15)NEDF,ERRSQ,AMSE
12 FORMAT(1X,5X,'SOURCE OF VARIATION',5X,'DEGREES OF FREEDOM',5X,
1 'SUM OF SQUARES',5X,'MEAN SQUARE',5X,'F',5X,'PROBABILITY',//)
13 FORMAT(1X,10X,'REPLICATION',14X,14,10X,F15.4,4X,F15.4,2X,F8.4,2X,
1 F8.4,//)
14 FORMAT(1X,10X,'TREATMENT',16X,14,10X,F15.4,2X,F15.4,//)
15 FORMAT(1X,10X,'RESIDUALS',16X,14,12X,F15.4,2X,F15.4,//)
RETURN
END

```

```

SUBROUTINE REG(N,X,Y,SD2X,SD2Y,SDXSDY,COC,SECOC,P2)
DIMENSION X(9),Y(9)
SX=0.0
SY=0.0
SXY=0.0
SXX=0.0
SYY=0.0
DO101=1,N
SX=SX+X(1)
SY=SY+Y(1)
SXY=SXY+X(1)*Y(1)
SXX=SXX+X(1)*X(1)
SYY=SYY+Y(1)*Y(1)
10 CONTINUE
NF=N-1
SD2X=SXX-SX*SX/N
SD2Y=SYY-SY*SY/N
SDXSDY=SXY-SX*SY/N
COC=SDXSDY/(SQRT(SD2X*SD2Y))
REGC=SDXSDY/SD2X
REGSS=(SDXSDY)*(SDXSDY)/SD2X
RESOSS=SD2Y-REGSS
VAR=RESOSS/(N-2)
FT=REGSS/VAR
NDREG=1
NDRES=N-2
PPP=FISHER(NDREG,NDRES,FT)
SEB=VAR/SD2X
59 FORMAT('0',T20,'THE REGR COEF IS',F8.4,5X,'SE',F8.4,/)
WRITE(6,49)
49 FORMAT(1X,T45,'DE',15X,'F',18X,'PROB')
WRITE(6,69)REGSS,NDREG,FT,PPP
69 FORMAT(1X,T10,'REGSS',2X,F15.4,10X,I4,10X,F12.4,10X,F12.4,/)
WRITE(6,79)VAR,NDRES
79 FORMAT('0',T10,'RESID SS/(N-2)',7X,F15.4,10X,I4,/)
T=(REGC-1)/SQRT(VAR/SD2X)
P1=FISHER(NDREG,NDRES,T*T)
WRITE(6,89)P1
89 FORMAT(1X,/,/, 'THE PROBABILITY THAT THE REGR COEF=1.0 IS',F8.4,/)
XB=SX/N
YB=SY/N
YCEPT=YB-REGC*XB
WRITE(6,99)YCEPT
99 FORMAT('0',T30,'THE Y INTERCEPT IS ',F8.4,/)
SECOC=SQRT((1.0-COC*COC)/(N-2))
TOC=COC/SECOC
P2=FISHER(1,NDRES,TOC*TOC)
RETURN
END

```

```

SUBROUTINE WRVR(D,H,DWRVR,TWRVR,VR,VR,VP,VRM,VRM,VARM,CP,BI)
DIMENSION D(9,9),RSSQ(9),SRSQ(9),VE(9),SPPR(9),SUMR(9),
1 SQPPR(9),PPRSQ(9),VRPPR(9),WR(9),E(9,9),VPVR(9),DWRVR(9),TWRVR(9)
2,CP(9),ARM(9),BI(9)
DO 30 J=2,N
DO 40 I=1,J-1
D(J,I)=D(I,J)
40 CONTINUE
30 CONTINUE
SSQPA=0.0
SUMPA=0.0
DO 50 J=1,N
VRPPR(J)=0.0
PPRSQ(J)=0.0
SPPR(J)=0.0
SQPPR(J)=0.0
SRSQ(J)=0.0
SUMR(J)=0.0
RSSQ(J)=0.0
DO 60 L=1,N
E(J,L)=D(J,L)+D(L,L)
SPPR(J)=SPPR(J)+E(J,L)
SQPPR(J)=SQPPR(J)+E(J,L)*E(J,L)
SUMR(J)=SUMR(J)+D(J,L)
RSSQ(J)=RSSQ(J)+D(J,L)*D(J,L)
60 CONTINUE
PPRSQ(J)=PPRSQ(J)+SPPR(J)*SPPR(J)
VRPPR(J)=(SQPPR(J)-PPRSQ(J)/H)/(N-1)
SRSQ(J)=SRSQ(J)+SUMR(J)*SUMR(J)
SUMPA=SUMPA+D(J,J)
SSQPA=SSQPA+D(J,J)*D(J,J)
50 CONTINUE
PASQ=0.0
VP=0.0
PASQ=PASQ+SUMPA*SUMPA
VP=(SSQPA-PASQ/N)/(N-1)
DO 44 J=1,N
VR(J)=0.0
WR(J)=0.0
DWRVR(J)=0.0
TWRVR(J)=0.0
VPVR(J)=0.0
44 CONTINUE
DO 100 J=1,N
VR(J)=(RSSQ(J)-SRSQ(J)/N)/(N-1)
WR(J)=(VRPPR(J)-VR(J)-VP)/2
DWRVR(J)=WR(J)-VR(J)
TWRVR(J)=WR(J)+VR(J)
VPVR(J)=SQRT(VR(J)*VP)
100 CONTINUE
VARM=0.0
WRM=0.0
VRM=0.0
SARM=0.0
WRSUM=0.0
VRSUM=0.0
TARMSQ=0.0
DO 301 K=1,N
ARM(K)=0.0
WRSUM=WRSUM+WR(K)
VRSUM=VRSUM+VR(K)
ARM(K)=SUMR(K)/N
SARM=SARM+ARM(K)
TARMSQ=TARMSQ+ARM(K)*ARM(K)
VARM=(TARMSQ-(SARM*SARM)/N)/(N-1)
301 CONTINUE
WRM=WRSUM/N
VRM=VRSUM/N

```

```

CP(1)=VP-BI(6)
CP(2)=4*VRM+VP-4*WRM-((3*N-2)*BI(6))/N
CP(3)=4*VRM-4*VARB-((2*(N*N-1)*BI(6))/(N*N))
CP(4)=2*VP-4*WRM-((2*(N-2)*BI(6))/N)
CP(5)=BI(6)
CP(6)=SQRT(ABS(CP(2)/CP(1)))
CP(7)=0.25*(CP(3)/CP(2))
DEM=0.5*(CP(1)+CP(2)-0.5*CP(3)-CP(4)+2*CP(5))
CP(8)=0.5*(CP(1)+CP(2)-CP(3)-CP(4))/DEM
CP(9)=0.5*(CP(1)+CP(2)-0.5*CP(3)-CP(4))/DEM
WRITE(6,20)
20 FORMAT(1X,T17,'WR',18X,'VR',25X,'VPVR',16X,'DWRVR',20X,'TWRVR',/)
WRITE(6,15)(WR(J),VR(J),VPVR(J),DWRVR(J),TWRVR(J),J=1,N)
15 FORMAT(1X,5(10X,F12.4),/)
WRITE(6,55)
55 FORMAT(1X,T10,100('*'))
WRITE(6,16)VP
16 FORMAT(1X,'THE PARENTAL VARIANCE=',F12.4,/)
RETURN
END

```

```

SUBROUTINE BATEST(CV,NDR)
DIMENSION BSS(7),CV(9),NDR(9),ALOGCV(7),VALOG(7),RENDR(7)
NBDR=0.0
TBSS=0.0
SVALOG=0.0
SRENDR=0.0
DO 556 K=1,4
BSS(K)=CV(K)*NDR(K)
TBSS=TBSS+BSS(K)
NBDR=NBDR+NDR(K)
ALOGCV(K)=ALOG(CV(K))
VALOG(K)=ALOGCV(K)*NDR(K)
SVALOG=SVALOG+VALOG(K)
RENDR(K)=1.0/NDR(K)
SRENDR=SRENDR+RENDR(K)
556 CONTINUE
SPVAR=TBSS/NBDR
ALOGPV=ALOG(SPVAR)
BART=(ALOGPV*NBDR)-SVALOG
CART=1.0+(1.0/(3.0*(1.0-1.0)))*(SRENDR-(1.0/NBDR))
BARTET=BART/CART
BARCHI=FISHER(3,10000,BARTET/3.0)
WRITE(6,10)BARCHI
10 FORMAT(1X,T20,'THE HOMOGENEITY OF VARIANCE BY BARTLETT IS',F12.4
RETURN
END

```

FUNCTION FISHER(M,N,X)

SUBROUTINE TO CALCULATE PROBABILITY THAT F-RATIO GREATER THAN X
 ARGUMENTS : M IS D.F. FOR TREATMENTS, N IS D.F. FOR ERROR AND X IS
 CALCULATED F-RATIO

```

IF(X.LE.0.0) GO TO 110
INTEGER A,B
A=2*(M/2)-M+2
B=2*(N/2)-N+2
W=X*M/FLOAT(N)
Z=1.0/(1.0+W)
IF(A.EQ.1.AND.B.EQ.1) P=SQRT(W)
IF(A.EQ.1.AND.B.EQ.1) D=0.3183098862 *Z/P
IF(A.EQ.1.AND.B.EQ.1) P=0.6365197724 *ATAN(P)
IF(A.EQ.1.AND.B.NE.1) P=SQRT(W*Z)
IF(A.EQ.1.AND.B.NE.1) D=0.5 * P * Z/W
IF(A.NE.1.AND.B.EQ.1) P=SQRT(Z)
IF(A.NE.1.AND.B.EQ.1) D=0.5 * Z * P
IF(A.NE.1.AND.B.NE.1) P=1-P
IF(A.NE.1.AND.B.NE.1) D=Z+Z
IF(A.NE.1.AND.B.NE.1) P=W*Z
Y=2.0*P/Z
IF(A.NE.1) GO TO 90
IF(B+2.GT.M) GO TO 95
DO 80 J = B+2,M,2
  D=(1.0 + A/FLOAT(J-2))*D*Z
  P = P + D * Y/(J-1)
80 CONTINUE
GO TO 95
90 ZK = Z*((M-1)/2)
  D = D * ZK * M/B
  P = P * ZK + W * Z * (ZK -1)/(Z -1)
95 Y = W * Z
  Z = 2.0/Z
  B = N -2
IF(A+2.GT.M) GO TO 105
DO 100 I = A+2,M,2
  J = I + B
  D = Y + D * J/FLOAT(I-2)
  P = P - Z * D/J
100 CONTINUE
105 IF(P.GT.1.0) P = 1.0
  IF(P.LT.0.0) P = 0.0
  FISHER = 1 - P
RETURN
110 FISHER=1.0
RETURN
END

```

APPENDIX II

E6700 FORTRAN COMPILATION MARK 3.

 Δ

INDIAN
INDIAN
THIS PROGRAMME IS BASED ON THE
FULL DIALLAGE ANALYSIS OF HAYMA
BIOMETRICAL GENETICS K. MATHUR, J. L. JENKS (1971)
PAGE 249-271

```

1 DIMENSION E(8,8),F(8,8),G(8,8),T(8,8),A(8,8),B(8,8),DERVR(8)
2 ,TWRVR(8),BI(8),VRA(3),VRT(3),P(8),PP(8),RDR(9)
3 ,AE(8,8),AF(8,8),TITLE(8),CAPT(8),CV(8),LT(9)
4 ,THFVR1(8),DRKVR1(3),CP(9),VR(8),WR(8),AX(3,8),BX(8,8)
5 ,VR1(8),BE1(8),CP1(9),PAR(8),FMT(16),TWRVR2(8),DRVR2(8)
6 ,VR2(8),WR2(8),CP2(9)
7 ,BSS(7),ALOGCV(7),VALOG(7),REFDR(7)
8 DATA TITLE/BB',BA',BC',BD',BE',BF',BG',BH',BI'/
9 DATA CAP1/BB',BA',BC',BD',BE',BF',BG',BH',BI'/
10 DATA LT/BB',BA',BC',BD',BE',BF',BG',BH',BI'/
11 READ(5,1,END=1920)(FMT(K),K=1,16)

```

```
1 FORMAT(16A5)
   WRITE(6,2)(FMT(K),K=1,16)
```

2 FORMAT(1X,/,T30,16A5,/)

C *****
C *****
C *****

```
READ(5,9,FEND=1920)IN,N,IBLK
```

```

9  FORMAT(314)
   WRITE(6,65)

```

```
66 FORMAT(//,1X,T40,'THE DIALLEL ANALYSIS FOR BLOCK 1',/)
```

```

**86** **C** **E** **F** **T** **I** **S** **A** **B** **O** **U** **T** **T** **H** **E** **D** **I** **S** **T** **R** **I** **B** **U** **T** **I** **O** **N** **S** **O** **F** **T** **H** **E** **P** **O** **P** **U** **L** **A** **T** **I** **O** **N** **S** **O** **F** **T** **H** **E** **U** **N** **I** **T** **E** **D** **S** **T** **A** **T** **E** **S** **O** **F** **A** **M** **E** **R** **I** **C** **A** **S** **O** **F** **T** **H** **E** **P** **O** **P** **U** **L** **A** **T** **I** **O** **N** **S** **O** **F** **T** **H** **E** **U** **N** **I** **T** **E** **D** **S** **T** **A** **T** **E** **S** **O** **F** **A** **M** **E** **R** **I** **C** **A** **S** **O** **F** **T** **H** **E** **P** **O** **P** **U** **L** **A** **T** **I** **O** **N** **S** **O** **F** **T** **H** **E** **U** **N** **I** **T** **E** **D** **S** **T** **A** **T** **E** **S** **O** **F** **A** **M** **E** **R** **I** **C** **A** **S** **O** **F** **T** **H** **E** **P** **O** **P** **U** **L** **A** **T** **I** **O** **N** **S** **O** **F** **T** **H** **E** **U** **N** **I** **T** **E** **D** **S** **T** **A** **T** **E** **S** **O** **F** **A** **M** **E** **R** **I** **C** **A** **S** **O** **F** **T** **H** **E** **P** **O** **P** **U** **L** **A** **T** **I** **O** **N
```

```
DO 10 I=1,IM  
READ(5,11,END=1920)IM,(E(I,J),J=1,IM)
```

```
11  FORMAT(*F8.4)
    WRITE(6,12)IH,(E(I,J),J=1,IH)
```

```
12  FORMAT(1X,*(4X,F12.4),/)
```

10 CONTINUE

CALL DIALF(E,IM,NDA,NDB,NDC,NDD,NDB1,NDB2,NDB3,NDT,
1AVA1,AV1B,AV1C,AV1D,AV1B1,AV1B2,AV1B3,AV1F)

WRITE(6,77)

```
77 FORMAT('1',///,IX,T40,'THE DIALLEL ANALYSIS FOR BLOCK 2')
```

DD 101 1=1, 111

```
READ(5,11,END=1920)IM,(F(I,J),J=1,IM)
```

```
WRITE(6,12) IP, (F(I,J), J=1, 1N)
```

101 CONTINUED

CALL DIALF(F,IM,NFA,NFB,NFC,NFD,NFB1,NFB2,NFB3,NFT,
1,VA2,AV2B,AV2C,AV2D,AV2B1,AV2B2,AV2B3,AV2T)

$$00\ 1001\ \vdash 1, 1, 1$$
$$G(I, J) = (E(I, J) + F(I, J)) / 2$$

```
*****
*****ARRAY AE, AF, AND AG ARE AVERAGES FOR E, F, AND G*****
*****
```

$$\begin{aligned} AE(I, J) &= (E(I, J) + E(J, I)) / 2 \\ AF(I, J) &= (F(I, J) + F(J, I)) / 2 \end{aligned}$$

1001 CONTINUE

```

88 FORMAT('(',///,1X,T40,'THE DIALLED ANALYSIS FOR BLOCK 1+2')

```

```
DO 1002 I=1,1M  
WRITE(6,12)1M,(G(I,J),J=1,1M)
```

1002 CONTINUE

CALL DTALF(G,IA,NA,HB,HC,HD,HB1,HB2,HB3,NT,
1AVA3,AV3B,AV3C,AV3D,AV3B1,AV3B2,AV3B3,AV3T)

DO 1003 1=1, 1/1

$$T(I, J) = (G(I, J) + G(J, I)) / 2$$

1003 CONTINUE -

```

WRITE(6,99)
99  FORMAT('1',///,1X,T40,'THE MEAN VALUES OF THE RECIPROCALES')
DO 51 J=1,IM
51  PAR(J)=C(J,J)
DO 1004 I=1,IM
1004 WRITE(6,20)I,(IM+1)-1,(T(I,J),J=1,IM)
CONTINUE
20  FORMAT('0',*(10X),*(2X,F8.4))
WRITE(6,21)IM,(T(I,J),J=1,IM)
DO 30 J=2,IM
DO 40 I=1,J-1
T(J,I)=T(I,J)
40  CONTINUE
21  FORMAT(1X,*(F12.4,2X),///)
30  CONTINUE
WRITE(6,999)
999  FORMAT('1',///,1X,T40,'CORRECTED VALUES FOR DIALECT ANALYSIS OF'
1  BLOCK 1+2')
CV(1)=2*AV3B
CV(2)=2*AVA3
CV(3)=2*AV3C
CV(4)=2*AV3D
CV(5)=2*AV3B1
CV(6)=2*AV3B2
CV(7)=2*AV3B3
CV(8)=2*AV3T
22  FORMAT('0',T30,A6,5X,F15.4,10X,I4,///)
NDR(1)=NB
NDR(2)=NA
NDR(3)=NC
NDR(4)=ND
NDR(5)=NB1
NDR(6)=NB2
NDR(7)=NB3
NDR(8)=NT
NDR(9)=NDR(1)+NDR(2)+NDR(3)+NDR(4)
DO 23 I=1,8
WRITE(6,22)CAPT(I),CV(I),NDR(I)
23  CONTINUE
BI(1)=ABS(2*AV3B-AV2B-AV1B)
BI(2)=ABS(2*AVA3-AVA2-AVA1)
BI(3)=ABS(2*AV3C-AV2C-AV1C)
BI(4)=ABS(2*AV3D-AV2D-AV1D)
BI(5)=ABS(2*AV3B1-AV2B1-AV1B1)
BI(6)=ABS(2*AV3B2-AV2B2-AV1B2)
BI(7)=ABS(2*AV3B3-AV2B3-AV1B3)
BI(8)=(BI(1)+NDR(1)+BI(2)+NDR(2)+BI(3)+NDR(3)+BI(4)+NDR(4))/NDR(9)
DO 109 I=1,7
VRA(I)=CV(I)/BI(I)
VRT(I)=CV(I)/BI(8)
P(I)=FISHER(NDR(I),NDR(8),VRA(I))
PP(I)=FISHER(NDR(I),NDR(8),VRT(I))
109  CONTINUE
WRITE(6,1000)
1000  FORMAT(1X,///,T40,'THE BLOCK INTERACTION MEAN SQUARES')
DO 80 I=1,8
WRITE(6,801)(TITLE(I),BI(I),NDR(I))
80  CONTINUE
CALL HATEST(BI,NDR)
WRITE(6,111)
801  FORMAT(1X,T30,A6,5X,F15.4,10X,I4,///)
111  FORMAT(1X,///,T33,'VRA',15X,'PROB',17X,'VRT',17X,'PROB')
DO 110 I=1,7
WRITE(6,38)VRA(I),P(I),VRT(I),PP(I)
110  CONTINUE
38  FORMAT('0',T17,4(5X,F15.4))
WRITE(6,1005)
1005  FORMAT('1',///,1X,T40,'THE VARIANCE/COVARIANCE ANALYSIS')
CALL WRVR(AE,H,DWRVR,TWRVR,VR,VP,WP,VRM,WRM,VARE,BI,CP)
CALL WRVR(AF,H,DWRVR1,TWRVR1,VR1,VP1,WR1,VRM1,WRM1,VARN1,BI,CP1)
CALL WRVR(T,H,DWRVR2,TWRVR2,VR2,VP2,WR2,VRM2,WRM2,VARN2,BI,CP2)
DO 50 I=1,IM
A(1,I)=DWRVR(I)
B(1,I)=TWRVR(I)
A(2,I)=DWRVR1(I)
B(2,I)=TWRVR1(I)
50  CONTINUE
WRITE(6,89)

```

```

89  FORMAT('1',1X,/,T30,'THE REGRESSION ANALYSIS OF WRVR',/)
    CALL REG(N,VR,WR,SSX1,SSY1,SP1,COC,SECOC,P2)
    CALL REG(N,VR1,WR1,SSX2,SSY2,SP2,COC,SECOC,P2)
    CALL REG(N,VR2,WR2,SSX3,SSY3,SP3,COC,SECOC,P2)
    CALL REG(N,TRVR2,PAR,SD2X,SD2Y,SDXSDY,COC,SECOC,P2)
    NDB1B2=2
    NDJP=1
    NDTR=(N-1)*IBLK
    NDRE=NDTR-NDB1B2
    XJB=(SP1+SP2)/(SSX1+SSX2)
    XJRSS=(SP1+SP2)*(SP1+SP2)/(SSX1+SSX2)
    SSB1B2=SP1**2/SSX1+SP2**2/SSX2
    TSSY=SSY1+SSY2
    RESS=TSSY-SSB1B2
    HRSS=SSB1B2-XJRSS
    FJR=XJRSS/(RESS/NDRE)
    PJR=FISHER(NDJR,NDRE,FJR)
    SEJB=SQRT((RESS/NDRE)/(SSX1+SSX2))
    TJB0=(XJB-0.0)/SEJB
    TJB1=(XJB-1.0)/SEJB
    PTJB0=FISHER(NDJR,NDRE,TJB0**2)
    PTJB1=FISHER(NDJR,NDRE,TJB1**2)
    WRITE(6,329)
    WRITE(6,330)XJRSS,NDJR,FJR,PJR
    WRITE(6,331)HRSS,NDJR
    WRITE(6,332)RESS/NDRE,NDRE
329  FORMAT(1X,/,T20,'ITEM',30X,'MS',15X,'DF',15X,'VR',15X,'PROB',/)
330  FORMAT('0',T20,'JOINT REGRESSION',7X,F15.4,10X,I4,10X,F12.4,10X,
    1F8.4,/)
331  FORMAT('0',T20,'HETEROGENEITY OF REGRESSION',F15.4,10X,I4,/)
332  FORMAT('0',T20,'REMAINDER',17X,F15.4,10X,I4,/)
    WRITE(6,333)XJB,SEJB,PTJB1,PTJB0
333  FORMAT(1X,'JOINT REGRESSION COEFFICIENT IS',F8.4,5X,'SE',F8.4,5X,
    *'SIGNIFALCE FROM 1.0',F8.4,5X,'SIGNIFICANCE FROM 0.0',F8.4,/)
    WRITE(6,1010)COC,SECOC,P2
1010  FORMAT('0',T20,'THE CORRELATION COEF. OF WR+VR AND PI IS',F8.4,'SE',
    *F8.4,'IT IS',F8.4,/)
    WRITE(6,802)
802  FORMAT('1',/,1X,T20,'THE ANALYSIS OF VARIANCE OF WRVR',/)
    CALL ANOVA(A,IBLK,IM)
    CALL ANOVA(B,IBLK,IM)
    WRITE(6,607)
607  FORMAT('1',/,1X,T35,'ESTIMATES OF COMPONENTS OF VARIATION',/)
    WRITE(6,608)
608  FORMAT(1X,T15,'STATISTICS',5X,'BLK1',20X,'BLK2',10X,'MEAN',22X
    1,'MODEL',/)
    WRITE(6,622)VP,VP1,(VP+VP1)/2
    WRITE(6,603)VRH,VRH1,(VRH+VRH1)/2
    WRITE(6,604)WRH,WRH1,(WRH+WRH1)/2
    WRITE(6,605)VARH,VARH1,(VARH+VARH1)/2
    WRITE(6,606)BI(3),BI(8),BI(8)
622  FORMAT('0',T20,'VP',3(F15.4,10X),10X,'D+E',/)
603  FORMAT('0',T20,'VRH',3(F15.4,10X),10X,'1/4D+1/4H1-1/4F+5/9E',/)
604  FORMAT('0',T20,'WRH',3(F15.4,10X),10X,'1/2D-1/4F+1/9E',/)
605  FORMAT(1X,T10,'VARH',3(F15.4,10X),10X,'1/4D+1/4H1-1/4H2-1/4F
    1+5/81E',/)
606  FORMAT('0',T20,'E',10X,3(F15.4,10X),10X,'E',/)
    WRITE(6,611)
611  FORMAT(1X,/,T35,'PERFECT FIT ESTIMATES OF THE COMPONENTS',/)
    WRITE(6,609)
609  FORMAT(1X,T15,'COMPONENT',12X,'BLK1',27X,'BLK2',30X,'MEAN',/)
    DO 601 L=1,9
    WRITE(6,602)LT(L),CP(L),CP1(L),(CP(L)+CP1(L))/2
601  CONTINUE
602  FORMAT('0',T20,A6,2X,F15.4,20X,F15.4,20X,F15.4,/)
    GO TO 1919
1920  STOP
    END
002:02A7:1  IS THE LOCATION FOR EXCEPTIONAL ACTION ON THE I/O STATE
002:02AA:2  IS THE LOCATION FOR EXCEPTIONAL ACTION ON THE I/O STATE
002:02AD:3  IS THE LOCATION FOR EXCEPTIONAL ACTION ON THE I/O STATE
002:02B0:4  IS THE LOCATION FOR EXCEPTIONAL ACTION ON THE I/O STATE

```

```

SUBROUTINE DIALF(A,IM,DFA,DFB,DFC,DFD,DFB1,DFB2,DFB3,DFT,
1AVA,AVB,AVC,AVD,B1,AVB2,AVB3,AVT)
DIMENSION A(8,8),SUMRF(8),SUMCF(8),SUMMF(8),DIFMF(8),
1DEVPA(8),D(8,8),DSUMCF(8),T(8,8)
SDEVSSQ=0.0
SRCDSQ=0.0
SRCTSQ=0.0
SUM=0.0
SYRSSQ=0.0
DO 20 I=1,IM
SUMRF(I)=0.0
SUMCF(I)=0.0
DO 30 J=1,IM
SUMRF(I)=SUMRF(I)+A(I,J)
SUMCF(I)=SUMCF(I)+A(J,I)
SYRSSQ=SYRSSQ+A(I,J)*A(I,J)
30 CONTINUE
SUM=SUM+SUMRF(I)+SUMCF(I)
SUMMF(I)=SUMRF(I)+SUMCF(I)
DIFMF(I)=SUMRF(I)-SUMCF(I)
SUMPA=SUMPA+A(I,I)
DEVPA(I)=SUMMF(I)-IM*A(I,I)
SRCTSQ=SRCTSQ+SUMMF(I)*SUMMF(I)
SRCDSQ=SRCDSQ+DIFMF(I)*DIFMF(I)
SDEVSSQ=SDEVSSQ+DEVPA(I)*DEVPA(I)
20 CONTINUE
SREDSQ=0.0
DO 140 I=1,IM-1
DO 150 J=I+1,IM
D(I,J)=A(I,J)-A(J,I)
SREDSQ=SREDSQ+D(I,J)*D(I,J)
150 CONTINUE
140 CONTINUE
WRITE(6,13)(SUMRF(K),K=1,IM)
13 FORMAT(1X,'SUM OF MALE ROW=',T32,8F12.4//)
WRITE(6,14)(SUMCF(I),I=1,IM)
14 FORMAT(1X,'SUM OF FEMALE COLUMN=',T32,8F12.4//)
WRITE(6,15)(SUMMF(K),K=1,IM)
15 FORMAT(1X,'SUM OF MALE ROW FEMALE COLUMN=',T32,8F12.4//)
WRITE(6,16)(DIFMF(K),K=1,IM)
16 FORMAT(1X,'DIF OF MALE ROW FEMALE COLUMN=',T32,8F12.4//)
WRITE(6,17)(DEVPA(I),I=1,IM)
17 FORMAT(1X,'PARENTAL DEVIATION=',T32,8F12.4//)
DO 110 I=1,IM-1
WRITE(6,18)I,IM-1,(D(I,J),J=I+1,IM)
110 CONTINUE
18 FORMAT('0',*(14X),*(2X,F12.4))
AA=SRCTSQ/(2*IM)-2*(SUM/2)**2/(IM*IM)
B=SYRSSQ/((SUM/2)**2)/(IM*IM)
1-SREDSQ/2-SRCTSQ/(2*IM)
C=SRCDSQ/(2*IM)
DD=SREDSQ/2-C
TOTSSQ=SYRSSQ-(SUM/2*SUM/2)/(IM*IM)
B1=(SUM/2-IM*SUMPA)**2/(IM**2*(IM-1))
B2=SDEVSSQ/(IM*(IM-2))-((SUM-(IM*SUMPA))**2)/((IM*IM)*(IM-2))
B3=B-B1-B2
DFA=IM-1
DFB=0.5*(IM)*(IM-1)
DFC=IM-1
DFD=0.5*(IM-1)*(IM-2)
DFT=(IM*IM)-1
DFB1=1.00
DFB2=IM-1
DFB3=0.5*IM*(IM-3)
AVA=AA/DFA
AVB=B/DFB
AVC=C/DFC
AVD=DD/DFD
AVB1=B1/1.00
AVB2=B2/DFB2
AVB3=B3/DFB3
AVT=TOTSSQ/DFT

```

```

WRITE(6,201)
WRITE(6,21)AA,DFA,AVA
WRITE(6,22)B,DFB,AVB
WRITE(6,23)C,DFC,AVC
WRITE(6,24)DD,DFD,AVD
WRITE(6,25)B1,DFB1,AVB1
WRITE(6,26)B2,DFB2,AVB2
WRITE(6,27)B3,DFB3,AVB3
WRITE(6,28)TOTSSQ,DFT,AVT
201 FORMAT(1X,25X,'SUM OF SQUARE',14X,'DF',15X,'MEAN SQUARE',/)
21  FORMAT(1X,10X,'A',10X,F15.4,10X,14,10X,F15.4,/)
22  FORMAT(1X,10X,'B',10X,F15.4,10X,14,10X,F15.4,/)
23  FORMAT(1X,10X,'C',10X,F15.4,10X,14,10X,F15.4,/)
24  FORMAT(1X,10X,'D',10X,F15.4,10X,14,10X,F15.4,/)
25  FORMAT(1X,10X,'B1',10X,F15.4,10X,14,10X,F15.4,/)
26  FORMAT(1X,10X,'B2',10X,F15.4,10X,14,10X,F15.4,/)
27  FORMAT(1X,10X,'B3',10X,F15.4,10X,14,10X,F15.4,/)
28  FORMAT(1X,10X,'T',10X,F15.4,10X,14,10X,F15.4,/)
RETURN
END

```

APPENDIX III

B6700 FORTRAN COMPILATION MARK

A
=

```

C *****
C
C      BBBB      IIII      H   H      TTTT      RRRR      IIII
C      B   B      I       NN  N      T       R   R      I
C      BBBB      I       N  N  N      T       R  R      I
C      B   B      I       N   N      T       R   R      I
C      BBBB      IIII      H   H      T       R   R      IIII
C
C *****
C
C      DIETRI IS A PROGRAMME BASED ON THE MATING DESIGN
C      PROPOSED BY CHANAL AND JINKS HEREDITY(1978), 40(1), 117-125
C      THIS PROGRAMME CAN TEST AND ESTIMATE THE ADDITIVE AND
C      DOMINANCE VARIATION. IT CAN ALSO PROVIDE AN UNBIASED
C      TEST FOR THE PRESENCE OF EPISTASIS
C *****
C
C      DIMENSION PI(4,20,50),PC(4,20,50),BCI(4,20,50),BII(4,20,50),
C      1FII(4,20,50),AI(4,20),BI(4,20),ADD(4,20),SUMP(4,20),
C      2SUMP(4,20),SUMBCI(4,20),SUMFII(4,20),SUMBII(4,20),AVPI(4,20),
C      3AVPC(4,20),AVBCI(4,20),AVFII(4,20),AVBII(4,20),ND(6),TIT(15)
C *****
C      *****READS IN THE NOS. OF BLOCKS AS IBLK, NOS. OF FAMILY AS IF, *****
C      *****NOS OF SAMPLE AS IS *****
C *****
C      1110 READ(5,29)IBLK,IF,IS
C
C      29 FORMAT(3I4)
C      WRITE(6,700)
C
C      700 FORMAT(1X,T40,50('*')/1X,T40,'*',T89'*/1X,T40,'*',T89'*/
C      *1X,T40,'*',8X,'THE BEN TRIPLE TEST CROSS ANALYSIS',6X,'*/
C      *1X,T40,'*',T89'*/1X,T40,'*',T89'*/1X,T40,50('*'),//)
C *****
C      *****RAW DATA FOR THE FAMILIES IN EACH GENERATION READ IN *****
C *****PI ARE PARENTS,PC THE COMMON TESTER, FII AND BCI ARE THE *****
C *****BACKCROSSES AND FII ARE THE F1 FAMILIES *****
C *****
C      WRITE(6,500)
C      500 FORMAT(1X,T40,'INPUT VALUE OF RAW DATA',//)
C      DO 10 I=1,IBLK
C      WRITE(6,411)I
C      DO 20 J=1,IF
C      WRITE(6,412)J
C      WRITE(6,417)
C      READ(5,9) (PI(I,J,K),K=1,IS)
C      READ(5,9) (PC(I,J,K),K=1,IS)
C      READ(5,9) (BII(1,J,K),K=1,IS)
C      READ(5,9) (BCI(1,J,K),K=1,IS)
C      READ(5,9) (FII(1,J,K),K=1,IS)
C      WRITE(6,410)
C      WRITE(6,19)IS,(PI(I,J,K),K=1,IS)
C      WRITE(6,413)
C      WRITE(6,19)IS,(PC(I,J,K),K=1,IS)
C      WRITE(6,416)
C      WRITE(6,19)IS,(BII(1,J,K),K=1,IS)
C      WRITE(6,415)
C      WRITE(6,19)IS,(BCI(1,J,K),K=1,IS)
C      WRITE(6,414)
C      WRITE(6,19)IS,(FII(1,J,K),K=1,IS)
C
C      20 CONTINUE
C      10 CONTINUE
C      410 FORMAT(1X,T52,'PI')
C      411 FORMAT(1X,T10,'BLOCK',I2,//)
C      412 FORMAT(1X,T20,'PARENT PI',I2,//)
C      413 FORMAT(1X,T52,'PC')
C      414 FORMAT(1X,T52,'FII')
C      415 FORMAT(1X,T52,'BCI')
C      416 FORMAT(1X,T52,'BII')
C      19 FORMAT('0',T5,'*(F5.1,I1X),//)
C      9 FORMAT(10F8.4)
C      417 FORMAT(1X,T5,125('*'))

```

```

C*****
C***CALCULATION OF THE SUM OF EACH OF EACH FAMILY IN EACH GENERATION***
C*****
DO 40 J=1,IBLK
DO 50 J=1,IF
SUMPI(I,J)=0.0
SUMPC(I,J)=0.0
SUMB1I(I,J)=0.0
SUMBCI(I,J)=0.0
SUMF1I(I,J)=0.0
DO 60 K=1,IS
SUMPI(I,J)=SUMPI(I,J)+PI(I,J,K)
SUMPC(I,J)=SUMPC(I,J)+PC(I,J,K)
SUMBCI(I,J)=SUMBCI(I,J)+BCI(I,J,K)
SUMB1I(I,J)=SUMB1I(I,J)+B1I(I,J,K)
SUMF1I(I,J)=SUMF1I(I,J)+F1I(I,J,K)
60 CONTINUE
C*****
C***CALCULATION OF THE MEAN OF EACH BLOCK OF EACH FAMILY IN*****
C***EACH GENERATION*****
C*****
AVPI(I,J)=SUMPI(I,J)/IS
AVPC(I,J)=SUMPC(I,J)/IS
AVB1I(I,J)=SUMB1I(I,J)/IS
AVBCI(I,J)=SUMBCI(I,J)/IS
AVF1I(I,J)=SUMF1I(I,J)/IS
C*****
C***CALCULATION OF THE RESPECTIVE COMPARISON*****
C*****
AI(I,J)=2*AVB1I(I,J)-AVF1I(I,J)-AVPI(I,J)
BI(I,J)=2*AVBCI(I,J)-AVF1I(I,J)-AVPC(I,J)
ADD(I,J)=AVBCI(I,J)-AVB1I(I,J)-AVPC(I,J)+AVPI(I,J)
DOM(I,J)=AVBCI(I,J)+AVB1I(I,J)-AVPC(I,J)-AVPI(I,J)
50 CONTINUE
40 CONTINUE
DO 514 I=1,IBLK
WRITE(6,505)I
WRITE(6,530)
WRITE(6,506)IF,(AVPI(I,J),J=1,IF)
WRITE(6,531)
WRITE(6,506)IF,(AVPC(I,J),J=1,IF)
WRITE(6,532)
WRITE(6,506)IF,(AVB1I(I,J),J=1,IF)
WRITE(6,533)
WRITE(6,506)IF,(AVBCI(I,J),J=1,IF)
WRITE(6,534)
WRITE(6,506)IF,(AVF1I(I,J),J=1,IF)
WRITE(6,535)
WRITE(6,506)IF,(AI(I,J),J=1,IF)
WRITE(6,536)
WRITE(6,506)IF,(BI(I,J),J=1,IF)
WRITE(6,537)
WRITE(6,506)IF,(ADD(I,J),J=1,IF)
WRITE(6,538)
WRITE(6,506)IF,(DOM(I,J),J=1,IF)
514 CONTINUE
506 FORMAT('0',T40,*(F8.4),//)
505 FORMAT('1',1X,T50,'BLOCK',12,/)
WRITE(6,600)
600 FORMAT('1',1X,T40,'THE ANALYSIS OF VARIANCE FOR B1I FAMILY',/)
CALL ANOVA(B1I,B1AMSE,IBLK,IF,IS,NDFI)
WRITE(6,601)
601 FORMAT('1',1X,T40,'THE ANALYSIS OF VARIANCE FOR BCI FAMILY',/)
530 FORMAT(1X,T40,'THE MEAN VALUE FOR PARENTS PI',/)
531 FORMAT(1X,T40,'THE MEAN VALUE FOR THE PARENT PC',/)
532 FORMAT(1X,T40,'THE MEAN VALUES FOR B1I',/)
533 FORMAT(1X,T40,'THE MEAN VALUES FOR BCI',/)
534 FORMAT(1X,T40,'THE MEAN VALUES FOR F1I',/)
535 FORMAT(1X,T40,'THE VALUE FOR THE EPISTASIS CONTRAST AI',/)
536 FORMAT(1X,T40,'THE VALUE FOR THE EPISTASIS CONTRAST BI',/)
537 FORMAT(1X,T40,'THE VALUE FOR THE ADDITIVE CONTRAST',/)
538 FORMAT(1X,T40,'THE VALUE FOR THE DOMINANCE CONTRAST',/)
CALL ANOVA(BCI,BCAMSE,IBLK,IF,IS,NDFI)
WRITE(6,602)
602 FORMAT('1',1X,T40,'THE ANALYSIS OF VARIANCE FOR PC FAMILY',/)
CALL ANOVA(PC,PCAMSE,IBLK,IF,IS,NDFI)
WRITE(6,603)
603 FORMAT('1',1X,T40,'THE ANALYSIS FOR PI FAMILY',/)
CALL ANOVA(PI,PIAMSE,IBLK,IF,IS,NDFI)
WRITE(6,604)
604 FORMAT('1',1X,T40,'THE ANALYSIS OF VARIANCE FOR F1I FAMILY',/)
CALL ANOVA(F1I,F1AMSE,IBLK,IF,IS,NDFI)

```

ISAM=IBLK*IS

```

C*****
C*****CALCULATION OF THE VARIANCES OF THE FAMILY MEANS*****
C*****
VPCM=PCANSE/ISAM
VPJF=PJANSE/ISAM
VFIM=FIANSE/ISAM
VBIF=BIANSE/ISAM
VBCM=BCANSE/ISAM
C*****
C*****CALCULATION OF THE POOLED VARIANCE OF COMPARISONS*****
C*****
PVAL=4*VBIM+VFIM+VDIM
PVB1=4*VBCM+VFIM+VPCM
WRITE(6,504)
504 FORMAT(1,1X,T40,'THE MEAN SQUARED DEVIATION FROM ZERO',//)
CALL PVAR(B1,BISQM,IBLK,IF)
CALL PVAR(A1,AISQM,IBLK,IF)
WRITE(6,701)PVAL,PVB1
701 FORMAT(1X,T20,'THE POOLED VARIANCE OF A1 CONTRAST IS',F15.4//1X,
* T20,'THE POOLED VARIANCE OF B1 CONTRAST IS',F15.4,//)
FRA1=AI SQM/PVAL
FRB1=BI SQM/PVB1
ND A1=IF
ND B1=IF
ND PVB1=ND F1
ND PVAL=ND F1
PRA1=FISHER(ND B1,ND PVB1,FRB1)
PRA1=FISHER(ND A1,ND PVAL,FRA1)
WRITE(6,39)FRA1,PRA1,FRB1,PRA1
39 FORMAT(1X,T20,'THE F RATIO FOR A1 CONTRAST IS',F15.4,'PROB',F5.4,
* T20,'THE F RATIO FOR THE B1 CONTRAST IS',F15.4,'PROB',F5.4,//)
IC=4
C*****
C*****THE ANOVA FOR THE ADDITIVE AND DOMINANCE VALUE*****
C*****
C*****THE ANOVA FOR THE EPISTASIS CONTRAST A1 AND B1*****
C*****
WRITE(6,526)
526 FORMAT(1,1X,T40,'THE ANALYSIS OF VARIANCE FOR DOMINANCE',//)
CALL CANOVA(DOM,IBLK,IF,IC,SSQT,ND,H,TD,XMD)
WRITE(6,203)H
203 FORMAT(0,'THE DOMINANCEVALUE IS',F15.4,//)
WRITE(6,205)TD
205 FORMAT(0,'THE TOTAL DOMINANCE VALUE IS',F15.4,//)
206 FORMAT(0,'THE AVERAGE DOMINANCE VALUE IS',F15.4,//)
WRITE(6,516)
516 FORMAT(1,1X,T40,'THE ANALYSIS OF VARIANCE FOR THE ADDITIVE',//)
CALL CANOVA(ADD,IBLK,IF,IC,SSQT,ND,D,TA,XMA)
WRITE(6,204)D
204 FORMAT(0,'THE ADDITIVE VALUE IS',F15.4,//)
WRITE(6,207)TA
207 FORMAT(0,'THE TOTAL ADDITIVE VALUE IS',F15.4,//)
208 FORMAT(0,'THE AVERAGE ADDITIVE VALUE IS',F15.4,//)
WRITE(6,556)
556 FORMAT(1,1X,T40,'THE ANALYSIS OF VARIANCE OF A1 EPISTASIS',//)
CALL CANOVA(A1,IBLK,IF,IC,SSQT,ND,DE,TE,XME)
WRITE(6,546)
546 FORMAT(1,1X,T40,'THE ANALYSIS OF VARIANCE OF B1 EPISTASIS',//)
CALL CANOVA(B1,IBLK,IF,IC,SSQT,ND,DE,TE,XMF)
GO TO 1110
STOP
END

```

```

SUBROUTINE PVAR(AI,AISQ,IBLK,IF)
DIMENSION AIBSQ(20),AIBSSQ(20),AI(4,20)
DO 300 J=1,IF
  AIBSQ(J)=0.0
  DO 301 I=1,IBLK
    AIBSQ(J)=AIBSQ(J)+AI(I,J)
301 CONTINUE
300 CONTINUE
  SAISQ=0.0
  DO 302 J=1,IF
    AIBSSQ(J)=0.0
    AIBSSQ(J)=AIBSQ(J)+AIBSQ(J)**2
    SAISQ=SAISQ+AIBSSQ(J)
302 CONTINUE
  IO=6
  AISQ=SAISQ/(IBLK*IBLK*IF)
  WRITE(6,307)
307 FORMAT(1X,T40,'THE BLOCK SUM OF THE CONTRAST',//)
  WRITE(6,303)IF,(AIBSQ(J),J=1,IF)
  WRITE(6,308)
308 FORMAT(1X,T40,'THE BLOCK SUM SQUARED OF THE CONTRAST',//)
  WRITE(6,303)IF,(AIBSSQ(J),J=1,IF)
303 FORMAT(1X,T40,'(F15.4)')
  WRITE(6,305)SAISQ,AISQ
305 FORMAT(1X,T10,'THE VALUE OF SUM OF EPISTSTATIC CONTRAST IS',F15.4
1////1X,T10,'THE VALUE OF THE CONTRAST MEAN IS',F15.4,//)
  RETURN
END

```

```

SUBROUTINE CANOVA(A,IBLK,IF,IC,SSOT,ND,HD,TD,XMD)
DIMENSION SUMR(4),SUMT(30),SS(6),ND(6),TITLE(6),P(6),FR(6),XMS(6)
1, A(4,30)
DATA TITLE/'MEAN','CTRAST','DEV','BLOCK','ERROR','TOTAL'/
COMMON SUM3L(4,30),DIF2L(4,30),C(4,30)
DO 505 I=1,6
XMS(I)=
FR(I)=
505 P(I)=
SUM=0.0
SSQ=0.0
SSQR=0.0
DO 20 I=1,IBLK
SUMR(I)=0.0
DO 30 J=1,IF
SUM=SUM+A(I,J)
SSQ=SSQ+A(I,J)*A(I,J)
SUMR(I)=SUMR(I)+A(I,J)
30 CONTINUE
SSQR=SSQR+SUMR(I)*SUMR(I)
20 CONTINUE
SSQT=0.0
DO 40 J=1,IF
SUMT(J)=0.0
DO 50 I=1,IBLK
SUMT(J)=SUMT(J)+A(I,J)
50 CONTINUE
SSQT=SSQT+SUMT(J)*SUMT(J)
40 CONTINUE
SS(1)=SUM*SUM/(IF*IBLK*IC)
SS(2)=SSOT/(IBLK*IC)
SS(3)=SSQT/(IBLK*IC)-SS(1)
SS(4)=SSQR/(IF*IC)-SS(1)
SS(6)=SSQ/IC
SS(5)=SS(6)-SS(3)-SS(4)-SS(1)
ND(1)=1
ND(2)=IF
ND(3)=IF-1
ND(4)=IBLK-1
ND(6)=IF*IBLK
ND(5)=(IF-1)*(IBLK-1)
DO 101 I=1,5
XMS(I)=SS(I)/ND(I)
101 CONTINUE
DO 1001 I=1,4
FR(I)=XMS(I)/XMS(5)
P(I)=FISHER(ND(I),ND(5),FR(I))
1001 CONTINUE
SIGMA=(XMS(3)-XMS(5))/(4*IBLK)
HD=64*SIGMA
WRITE(6,202)HD
202 FORMAT('0',F15.4,/)
TD=((XMS(2)-XMS(5))*((IF*4)))/IBLK
XMD=TD/IF
WRITE(6,100)
100 FORMAT('0','SOURCE OF VARIATION',10X,'DF',14X,'SS',
118X,'MS',23X,'F',15X,'PROB')
DO 1002 I=1,6
WRITE(6,110)TITLE(I),ND(I),SS(I),XMS(I),FR(I),P(I)
1002 CONTINUE
110 FORMAT('0',T10,A6,10X,I4,10X,F12.4,10X,F12.4,10X,F15.4,10X,
1F8.4,/)
RETURN
END

```

```

SUBROUTINE ANOVA(A,AMSSAE,IR,IT,IQ,NDFSAE)
C ANALYSIS OF VARIANCE THREE WAY CLASSIFICATION
DIMENSION A(4,20,50),SUMSA(20,50),SAM(20,50)
SUM=0.0
SSQ=0.0
SSQR=0.0
SSQSA=0.0
DO 30 I=1,IR
  SUMR=0.0
  DO 40 J=1,IT
    SUMSA(I,J)=0.0
    DO 50 K=1,IQ
      SUMSA(I,J)=SUMSA(I,J)+A(I,J,K)
      SUM=SUM+A(I,J,K)
      SUMR=SUMR+A(I,J,K)
      SSQ=SSQ+A(I,J,K)*A(I,J,K)
50 CONTINUE
    SSQSA=SSQSA+SUMSA(I,J)*SUMSA(I,J)
40 CONTINUE
    SSQR=SSQR+SUMR*SUMR
30 CONTINUE
  SSQT=0.0
  DO 60 J=1,IT
    SUMT=0.0
    DO 70 I=1,IR
      DO 80 K=1,IQ
        SUMT=SUMT+A(I,J,K)
80 CONTINUE
70 CONTINUE
    SSQT=SSQT+SUMT*SUMT
60 CONTINUE
  CF=SUM*SUM/(IT*IR*IQ)
  TOTSSQ=SSQ-CF
  TOTTSQ=SSQT/(IR*IQ)-CF
  TOTRSQ=SSQR/(IT*IQ)-CF
C TOTAL SAMPLE SUM OF SQUARES/INTERACTION
  TOTSSA=SSQSA/IQ-CF
C SAMPLING ERROR SUM OF SQUARES
  SAESSQ=TOTSSQ-TOTSSA
C EXPERIMENTAL ERROR SUM OF SQUARES
  ERRSQ=TOTSSQ-TOTRSQ-TOTTSQ-SAESSQ
  NDTF=IT*IR*(IQ-1)
  NDFI=(IT-1)*(IR-1)
  NDFE=IT-1
  NDFJ=IR-1
  NDFSAE=IT*IR*(IQ-1)
  AMSQT=TOTTSQ/NDFE
  AMSQR=TOTRSQ/NDFI
  AMSE=ERRSQ/NDFI
  IF(NDFSAE.GT.0)AMSSAE=SAESSQ/NDFSAE
  F=AMSQT/AMSE
  PRO=FISHER(NDFE,NDFI,F)
  WRITE(6,12)
  WRITE(6,13)NDFE,TOTRSQ,AMSQR,F,PRO
  WRITE(6,14)NDFE,TOTTSQ,AMSQT
  WRITE(6,15)NDFSAE,SAESSQ,AMSSAE
  WRITE(6,16)NDFI,ERRSQ,AMSE
12 FORMAT(1X,5X,'SOURCE OF VARIATION',5X,'DEGREES OF FREEDOM',5X,
1 'SUM OF SQUARES',10X,'MEAN SQUARE',22X,'F',13X,'PROB',//)
13 FORMAT(1X,10X,'REPLICATION',15X,14,10X,F15.4,10X,F15.4,
*10X,F15.4,8X,F5.4,//)
14 FORMAT(1X,10X,'FAMILY',18X,14,10X,F15.4,10X,F15.4,//)
15 FORMAT(1X,10X,'EXPERIMENTAL ERROR',10X,14,10X,F15.4,10X,F15.4,//)
16 FORMAT(1X,10X,'SAMPLING ERROR',12X,14,10X,F15.4,10X,F15.4,//)
  DO 500 I=1,IR
    DO 501 J=1,IT
      SAM(I,J)=0.0
      SAM(I,J)=SAM(I,J)+SUMSA(I,J)/IQ
501 CONTINUE
    WRITE(6,503)
    WRITE(6,502)IT,(SUMSA(I,J),J=1,IT)
    WRITE(6,504)
    WRITE(6,502)IT,(SAM(I,J),J=1,IT)
500 CONTINUE
502 FORMAT('0',T10,*(F15.4,2X),/)
503 FORMAT(1X,T30,'THE SUM OF SAMPLE FOR EACH FAMILY',//)
504 FORMAT(1X,T30,'THE MEAN OF EACH FAMILY',//)
  RETURN
END

```

```

FUNCTION FISHER(M,N,X)
SUBROUTINE TO CALCULATE PROBABILITY THAT F-RATIO GREATER THAN X
ARGUMENTS : M IS D.F. FOR TREATMENTS, N IS D.F. FOR ERROR AND X IS
CALCULATED F-RATIO
IF(X.LE.0.0) GO TO 110
INTEGER A,B
A=2*(M/2)-M+2
B=2*(N/2)-N+2
W=X*M/FLOAT(N)
Z=1.0/(1.0+W)
IF(A.EQ.1.AND.B.EQ.1) P=SQRT(W)
IF(A.EQ.1.AND.B.EQ.1) D=0.3183092862 *Z/P
IF(A.EQ.1.AND.B.EQ.1) P=0.6366197724 *ATAN(P)
IF(A.EQ.1.AND.B.EQ.1) P=SQRT(W*Z)
IF(A.EQ.1.AND.B.EQ.1) D=0.5 * P * Z/W
IF(A.EQ.1.AND.B.EQ.1) P=SQRT(Z)
IF(A.EQ.1.AND.B.EQ.1) D=0.5 * Z * P
IF(A.EQ.1.AND.B.EQ.1) P=1-P
IF(A.EQ.1.AND.B.EQ.1) D=Z*Z
IF(A.EQ.1.AND.B.EQ.1) P=W*Z
Y=2.0*W/Z
IF(A.EQ.1) GO TO 90
IF( B+2 .GT. N) GO TO 95
DO 80 J = B+2,N,2
  D=(1.0 + A/FLOAT(J-2))*D*Z
  P = P + D * Y/(J-1)
80 CONTINUE
GO TO 95
90 ZK = Z*((N-1)/2)
  D = D * ZK * N/B
  P = P * ZK + W * Z * (ZK -1)/(Z -1)
95 Y = W * Z
  Z = 2.0/Z
  B = N -2
  IF( A+2 .GT. M) GO TO 105
  DO 100 I = A+2,M,2
    J = I + B
    D = Y * D * J/FLOAT(I-2)
    P = P - Z * D/J
100 CONTINUE
105 IF( P .GT. 1.0) P = 1.0
  IF( P .LT. 0.0) P = 0.0
  FISHER = 1 - P
  RETURN
110 FISHER=1.0
  RETURN
END

```

APPENDIX IV

B6700 FORTRAN COMPILATION MARK 3.

A
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*****
BBBBB I I I N N N T T T T E E E E S S S T T T T *
B B I N N N T E S S T *
BBBBB I N N N T E E E S S S T *
B B I N N T E S S T *
BBBBB I I I N N T E E E E S S S T *
*****
PROGRAMME FOR THE SCALING TEST ANALYSIS BASED ON THE
METHODS OF MATHER AND JINKS (1971)-BIOMETRICAL GENETICS
*****

      DIMENSION P1(5,50),P2(5,50),F1(5,50),B1(5,50),F2(5,50),F2(5,50),
      *PIXMS(3),P2XMS(3),F1XMS(3),B1XMS(3),B2XMS(3),F2XMS(3),BD(3),
      *TJTL(16),SCT(10),V(10),SE(10),P(10),CAPT(10),T(10),COEF(6,3)
      *,HDF(10),PHDF(10),BD1(3),BD2(3),BD3(3),BD4(3),BD5(3),BD6(3)
      DATA CAPT/'A','B','C','D','H','I','J','L','S'/
C*****
C*****IBLK IS NOS OF BLOCKS IS SAMPLE SIZE OF P1,P2,F1,IQ,IR SAMPLE
C*****FOR BC1,BC2,F2,N SIZE OF MATRIX TO BE INVERTED *****
C*****
1110 READ(5,9)IBLK,IS,IQ,IR,N
      9 FORMAT(5I4)
      READ(5,39)(TITLE(I),I=1,16)
39 FORMAT(16A5)
      WRITE(6,39)(TITLE(I),I=1,16)

C*****
C*****MATRIX COEF IS THE COEFFICIENT OF M,D,H FOR THE SIX *****
C*****GENERATIONS P1,B1,F1,F2,B2, AND P2*****
C*****
      DO 200 I=1,6
      READ(5,91)(COEF(I,J),J=1,3)
200 CONTINUE
91 FORMAT(3F4.2)
C*****
C*****RAW DATA FOR THE GENERATION S ARE READ IN AND WRITTEN OUT*****
C*****
      DO 10 I=1,IBLK
      READ(5,19) (P1(I,J),J=1,IS)
      READ(5,19) (P2(I,J),J=1,IS)
      READ(5,19) (B1(I,J),J=1,IQ)
      READ(5,19) (B2(I,J),J=1,IQ)
      READ(5,19) (F1(I,J),J=1,IS)
      READ(5,19) (F2(I,J),J=1,IR)
      WRITE(6,93)I
      WRITE(6,92)
      WRITE(6,29) (P1(I,J),J=1,IS)
      WRITE(6,29) (B1(I,J),J=1,IQ)
      WRITE(6,29) (F1(I,J),J=1,IS)
      WRITE(6,29) (F2(I,J),J=1,IR)
      WRITE(6,29) (B2(I,J),J=1,IQ)
      WRITE(6,29) (P2(I,J),J=1,IS)
10 CONTINUE
93 FORMAT('0',T30,'BLOCK',I3,/)
92 FORMAT(1X,T30,'THE INPUT VALUES FOR GENERATION',/)
19 FORMAT(10F8.4)
29 FORMAT(1X,20(F5.1,1X),/,20(F5.1,1X),/,10(F5.1,1X))
      SUMP1=0.0
      SUMP2=0.0
      SUMF1=0.0
      DO 20 I=1,IBLK
      DO 30 J=1,IS
      SUMP1=SUMP1+P1(I,J)
      SUMP2=SUMP2+P2(I,J)
      SUMF1=SUMF1+F1(I,J)
30 CONTINUE
20 CONTINUE
      SUMB1=0.0
      SUMB2=0.0
      DO 120 I=1,IBLK
      DO 130 J=1,IQ
      SUMB1=SUMB1+B1(I,J)

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      SUMB2=SUMB2+B2(I,J)
130 CONTINUE
120 CONTINUE
      SUMF2=0.0
      DO 220 I=1,IBLK
      DO 230 J=1,IR
      SUMF2=SUMF2+F2(I,J)
230 CONTINUE
220 CONTINUE
      ISAM=IBLK*(IS-1)
      ISAQ=IBLK*(IQ-1)
      ISAR=IBLK*(IR-1)

C*****
C*****THE GENERATION MEANS ARE BEING CALCULATED*****
C*****
      ISAM=IBLK*IS
      ISAMQ=IBLK*ISQ
      ISAMR=IBLK*IR
      P1M=SUMP1/ISAM
      P2M=SUMP2/ISAM
      F1M=SUMF1/ISAM
      B1M=SUMB1/ISAMQ
      B2M=SUMB2/ISAMQ
      F2M=SUMF2/ISAMR
      WRITE(6,11)
      WRITE(6,12)P1M,P2M,F1M,B1M,B2M,F2M
C*****
C*****ARRAY SCT CONTAINS THE VALUES OF THE SCALING TEST*****
C*****
      SCT(1)=2*B1M-P1M-F1M
      SCT(2)=2*B2M-P2M-F1M
      SCT(3)=4*F2M-2*F1M-P1M-P2M
      SCT(4)=0.5*P1M-0.5*P2M
      SCT(5)=6*B1M+6*B2M-8*F2M-F1M-1.5*P1M-1.5*P2M
      SCT(6)=2*B1M+2*B2M-4*F2M
      SCT(7)=2*B1M-P1M-2*B2M+P2M
      SCT(8)=P1M+P2M+2*F1M+4*F2M-4*B1M-4*B2M
      SCT(9)=0.5*P1M+0.5*P2M+4*F2M-2*B1M-2*B2M
      SCT(10)=F2M-0.5*B1M-0.5*B2M
      WRITE(6,94)
      WRITE(6,95)
C*****
C*****CALCULATION OF VARIANCES OF THE GENERATION MEANS*****
C*****
      CALL ANOVA(F1,IBLK,IS,P1XMS,ND1)
      WRITE(6,96)
      CALL ANOVA(B1,IBLK,IQ,B1XMS,ND2)
      WRITE(6,97)
      CALL ANOVA(F1,IBLK,IS,F1XMS,ND3)
      WRITE(6,98)
      CALL ANOVA(F2,IBLK,IR,F2XMS,ND4)
      WRITE(6,99)
      CALL ANOVA(B2,IBLK,IQ,B2XMS,ND5)
      WRITE(6,81)
      CALL ANOVA(P2,IBLK,IS,P2XMS,ND6)
81  FORMAT(1X,T30,'THE ANALYSIS OF VARIANCE FOR THE P2 GENERATION',/)
94  FORMAT(1X,T30,'THE ANALYSIS OF VARIANCE FOR EACH GENERATION',/)
95  FORMAT(1X,T30,'THE ANALYSIS OF VARIANCE FOR THE P1 GENERATION',/)
96  FORMAT(1X,T30,'THE ANALYSIS OF VARIANCE FOR THE B1 GENERATION',/)
97  FORMAT(1X,T30,'THE ANALYSIS OF VARIANCE FOR THE F1 GENERATION',/)
98  FORMAT(1X,T30,'THE ANALYSIS OF VARIANCE FOR THE P2 GENERATION',/)
99  FORMAT(1X,T30,'THE ANALYSIS OF VARIANCE FOR THE B2 GENERATION',/)
      VP1M=P1XMS(3)/ISAM
      VP2M=P2XMS(3)/ISAM
      VF1M=F1XMS(3)/ISAM
      VF2M=F2XMS(3)/ISAMR
      VB1M=B1XMS(3)/ISAMQ
      VB2M=B2XMS(3)/ISAMQ
C*****
C*****ARRAY V CONTAINS THE VARIANCES OF SCT*****
C*****
      V(1)=4*VB1M+VP1M+VF1M
      V(2)=4*VB2M+VP2M+VF1M
      V(3)=16*VF2M+4*VF1M+VP1M+VP2M
      V(4)=0.25*VP1M+0.25*VP2M
      V(5)=36*VB1M+36*VB2M+64*VF2M+VF1M+2.25*VP1M+2.25*VP2M
      V(6)=4*VB1M+4*VB2M+16*VF2M
      V(7)=4*VB1M+VP1M+4*VB2M+VP2M
      V(8)=VP1M+VP2M+4*VF1M+16*VF2M+16*VB1M+16*VB2M
      V(9)=0.25*VP1M+0.25*VP2M+16*VF2M+4*VB1M+4*VB2M
      V(10)=VF2M+0.25*VB1M+0.25*VB2M

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C*****
C*****ARRAY NDF CONTAINS THE POOLED DE OF THE VARIANCE OF MEANS
C*****
NDF(1)=ND2(3)+ND1(3)+ND3(3)
NDF(2)=ND5(3)+ND6(3)+ND3(3)
NDF(3)=ND4(3)+ND3(3)+ND1(3)+ND6(3)
NDF(4)=ND1(3)+ND6(3)
NDF(5)=ND2(3)+ND5(3)+ND4(3)+ND3(3)+ND1(3)+ND6(3)
NDF(6)=ND2(3)+ND5(3)+ND4(3)
NDF(7)=ND2(3)+ND1(3)+ND5(3)+ND6(3)
NDF(8)=ND1(3)+ND6(3)+ND3(3)+ND4(3)+ND2(3)+ND5(3)
NDF(9)=ND1(3)+ND6(3)+ND5(3)+ND4(3)+ND2(3)
NDF(10)=ND4(3)+ND5(3)+ND2(3)
C*****
C*****ARRAY SE CONTAINS THE STANDARD ERRORS OF SCT*****
C*****
DO 202 I=1,10
  SE(I)=SQR(V(I))
  T(I)=SCT(I)/SE(I)
  PNDF(I)=FISHER(1,NDF(I),T(I)*T(I))
202 CONTINUE

  WRITE(6,82)
82  FORMAT(1,'1X,T30,'THE SCALING TEST ANALYSIS ON THE GENERATION',/)
  WRITE(6,49)
11  FORMAT(1X,T25,'P1 MEAN',10X,'P2 MEAN',10X,'F1 MEAN',10X,
  *'B1 MEAN',10X,'B2 MEAN',10X,'F2 MEAN',/)
12  FORMAT(0,T15,6(5X,F12.4))
49  FORMAT(1X,T10,'SCALING TEST',10X,'VALUE',10X,'STANDARD ERROR',18X,
  *'T',10X,'SIGNIFICANCE',/)
  DO 201 K=1,10
    WRITE(6,59)CAPT(K),SCT(K),SE(K),T(K),PNDF(K)
201 CONTINUE
59  FORMAT(0,T10,A6,F15.4,10X,F15.4,10X,F15.4,10X,F5.4,/)
  WRITE(6,83)
83  FORMAT(1,'1X,T30,'THE CAVALLI SCALING TEST ANALYSIS',/)
  CALL CAVALLI(0,COEF,VP1M,VB1M,VF1M,VF2M,VB2M,VP2M,P1M,B1M,
  *F1M,F2M,B2M,P2M)
  HER0=0.5*((2.0*VF2M)-(VB1M+VB2M))/VF2M
  WRITE(6,111)HER0
111 FORMAT(0,T10,'THE HERITABILITY ESTIMATE BY WARNER METHOD'F8.4,/)
  GO TO 1110
  STOP
  END

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```

SUBROUTINE ABOVA(A,IBLK,IS,XMS,ND)
DIMENSION A(5,50),SS(3),ND(3),TITLE(3),XMS(3),FR(3),P(3)
DATA TITLE/'TOTAL','BLOCK','ERROR'/
DO 505 I=1,3
XMS(I)=.
FR(I)=.
P(I)=.
505 CONTINUE
SUM=0.0
SSQ=0.0
SSQR=0.0
DO 20 I=1,IBLK
SUMR=0.0
DO 30 J=1,IS
SUM=SUM+A(I,J)
SSQ=SSQ+A(I,J)*A(I,J)
SUMR=SUMR*A(I,J)
30 CONTINUE
SSQR=SSQR+SUMR*SUMR
20 CONTINUE
CF=SUM*SUM/(IBLK*IS)
SS(1)=SSQ-CF
SS(2)=SSQR/IS-CF
SS(3)=SS(1)-SS(2)
ND(1)=IBLK*IS-1
ND(2)=IBLK-1
ND(3)=ND(1)-ND(2)
DO 10 I=1,3
XMS(I)=SS(I)/ND(I)
10 CONTINUE
FR(2)=XMS(2)/XMS(3)
P(2)=FISHER(ND(2),ND(3),FR(2))
WRITE(6,100)
100 FORMAT('0','SOURCE OF VARIATION',10X,'DF',14X,'SS',
118X,'MS',23X,'F',15X,'PROB')
DO 101 I=1,3
WRITE(6,110)TITLE(I),ND(I),SS(I),XMS(I),FR(I),P(I)
101 CONTINUE
110 FORMAT('0',T10,A6,10X,I4,3(10X,F12.4),10X,F5.4,/)
RETURN
END

```

```

SUBROUTINE CAVALI(N,COEF,VP1M,VB1M,VF1M,VF2M,VB2M,VP2M,P1M,B1M,
*F1M,F2M,B2M,P2M)
DIMENSION WT(6),XMEAN(6),COEF(6,3),WTH(6),WTCF(6,3),R(6,3)
*,UEIT(3,3),TEMP(3),XM(3,3),EXPT(6),ITITLE(6),DEV(6),
*RWTH(6,3),R1(6,3),R2(6,3),A(3,3),S(3)
DATA TITLE/'P1','B1','F1','F2','B2','P2'/
WT(1)=1.0/VP1M
WT(2)=1.0/VB1M
WT(3)=1.0/VF1M
WT(4)=1.0/VF2M
WT(5)=1.0/VB2M
WT(6)=1.0/VP2M
XMEAN(1)=P1M
XMEAN(2)=B1M
XMEAN(3)=F1M
XMEAN(4)=F2M
XMEAN(5)=B2M
XMEAN(6)=P2M
DO 201 K=1,6
WTH(K)=0.0
WTH(K)=WT(K)*XMEAN(K)
201 CONTINUE
DO 202 I=1,6
DO 203 J=1,3
WTCF(I,J)=0.0
WTCF(I,J)=WT(I)*COEF(I,J)
203 CONTINUE
202 CONTINUE
DO 204 J=1,3
DO 205 I=1,6
R(I,J)=0.0
R1(I,J)=0.0
R2(I,J)=0.0
RWTH(I,J)=0.0
R(I,J)=COEF(I,J)*WTCF(I,1)
R1(I,J)=COEF(I,J)*WTCF(I,2)
R2(I,J)=COEF(I,J)*WTCF(I,3)
RWTH(I,J)=COEF(I,J)*WTH(I)
205 CONTINUE
204 CONTINUE
C*****APPRAY A(I,J) IS THE INFORMATION MATRIX AFTER CALLING MINV ****
C*****APPRAY A(I,J) CONTAINS THE INVERSE OF THE INFORMATION MATRIX****
DO 303 J=1,3
A(1,J)=0.0
A(2,J)=0.0
A(3,J)=0.0
303 CONTINUE
DO 302 I=1,6
DO 330 J=1,3
A(1,J)=A(1,J)+R(I,J)
A(2,J)=A(2,J)+R1(I,J)
A(3,J)=A(3,J)+R2(I,J)
330 CONTINUE
302 CONTINUE
WRITE(6,503)
WRITE(6,300)(WT(L),L=1,6)
WRITE(6,504)
WRITE(6,300)(WTH(K),K=1,6)
WRITE(6,505)
WRITE(6,300)((WTCF(I,J),J=1,3),I=1,6)
WRITE(6,506)
WRITE(6,301)((R(I,J),J=1,3),I=1,6)
WRITE(6,507)
WRITE(6,301)((R1(I,J),J=1,3),I=1,6)
WRITE(6,508)
WRITE(6,301)((R2(I,J),J=1,3),I=1,6)
WRITE(6,509)
WRITE(6,300)((RWTH(I,J),I=1,6),J=1,3)

```

```

300 FORMAT('0',6(F15.4,2X))
301 FORMAT('0',3(F15.4,4X))
503 FORMAT('0',T30,'THE WEIGHTS-1/RECIPROCAL OF GENERATION MEAN',/)
504 FORMAT('0',T30,'THE WEIGHTED MEANS',/)
505 FORMAT('0',T30,'THE PRODUCT OF WT. AND COEF. OF EQUATION',/)
508 FORMAT('0',T20,'THE WT. COEF.*COEF OF H',/)
507 FORMAT('0',T20,'THE WT. COEF.*COEF OF D',/)
506 FORMAT('0',T20,'THE WT.COEF.*COEF. OF M',/)
509 FORMAT('0',T30,'THE COEF*WT.MEAN',/)
WRITE(6,400)
400 FORMAT(1X,T30,'THE INFORMATION MATRIX',/)
WRITE(6,301)((A(I,J),J=1,3),I=1,3)
DO 305 I=1,3
S(I)=0.0
DO 306 K=1,6
S(I)=S(I)+RWTM(K,I)
306 CONTINUE
305 CONTINUE
WRITE(6,401)
401 FORMAT(1X,T30,'THE S MATRIX',/)
WRITE(6,301)(S(I),I=1,3)
CALL MIXV(A,N,UNIT,TEMP)
DO 500 I=1,3
DO 501 J=1,3
XM(I,J)=A(I,J)*S(J)
501 CONTINUE
500 CONTINUE
WRITE(6,402)
402 FORMAT(1X,T30,'THE INVERSE OF THE INFORMATION MATRIX-',/)
WRITE(6,301)((A(I,J),J=1,3),I=1,3)
XXM=XM(1,1)+XM(1,2)+XM(1,3)
DD=XM(2,1)+XM(2,2)+XM(2,3)
HH=XM(3,1)+XM(3,2)+XM(3,3)
SEXMM=SQRT(A(1,1))
SEDD=SQRT(A(2,2))
SEHH=SQRT(A(3,3))
403 FORMAT('1',1X,'THE ESTIMATE OF M D H AND TEST OF GOODNESS OF FIT',
*)
WRITE(6,403)
WRITE(6,310)XXM,SEXMM
WRITE(6,311)DD,SEDD
WRITE(6,312)HH,SEHH
310 FORMAT(1X,T15,'THE ESTIMATE OF M IS',F12.4,'IT S SE IS',F8.6,/)
311 FORMAT(1X,T15,'THE ESTIMATE OF D IS',F12.4,'IT S SE IS',F8.6,/)
312 FORMAT(1X,T15,'THE ESTIMATE OF H IS',F12.4,'IT S SE IS',F8.6,/)
DO 321 I=1,6
EXPT(I)=COEF(I,1)*XXM+COEF(I,2)*DD+COEF(I,3)*HH
321 CONTINUE
CHISQ=0.0
DO 323 I=1,6
DEV(I)=((EXPT(I)-XMEAN(I))**2)*WT(I)
323 CHISQ=CHISQ+DEV(I)
PCHISQ=FISHER(3,10000,CHISQ/3)
WRITE(6,315)
315 FORMAT('0',T12,'GENERATION',18X,'EXPECTED',18X,'OBSERVED',14X,'
*DEVIATION',/)
DO 322 I=1,6
322 WRITE(6,313)TITLE(I),EXPT(I),XMEAN(I),DEV(I)
313 FORMAT('0',T15,A6,10X,F15.4,10X,F15.4,10X,F15.4,/)
WRITE(6,314)CHISQ,PCHISQ
314 FORMAT('0',T15,'THE CHI SQUARE VALUE IS',F15.4,'PROBABILITY',F5.4)
RETURN
END

```

```

SUBROUTINE MINV(X,N,UNIT,TEMP)
REAL X(N,N),UNIT(N,N),TEMP(N)
DO 15 I=1,N
DO 15 J=1,N
IF(I-J)5,10,5
5 UNIT(I,J)=0.0
GO TO 15
10 UNIT(I,J)=1.0
15 CONTINUE
DO 70 K=1,N
IF(X(K,K))50,20,50
20 J=K+1
IF(J-N)25,25,35
25 DO 30 I=J,N
IF(X(I,K))40,30,40
30 CONTINUE
35 WRITE(6,323)
323 FORMAT(IX,'MATRIX IS SINGULAR',50X,'***ERROR***')
STOP
40 DO 45 J=1,N
TEMP(J)=X(K,J)
X(K,J)=X(I,J)
X(I,J)=TEMP(J)
TEMP(J)=UNIT(K,J)
UNIT(K,J)=UNIT(I,J)
UNIT(I,J)=TEMP(J)
45 CONTINUE
50 DIV=X(K,K)
DO 55 J=1,N
X(K,J)=X(K,J)/DIV
UNIT(K,J)=UNIT(K,J)/DIV
55 CONTINUE
DO 65 I=1,N
FACT=X(I,K)
IF(I-K)60,65,60
60 DO 65 J=1,N
X(I,J)=X(I,J)-FACT*X(K,J)
UNIT(I,J)=UNIT(I,J)-FACT*UNIT(K,J)
65 CONTINUE
70 CONTINUE
DO 80 I=1,N
DO 80 J=1,N
X(I,J)=UNIT(I,J)
80 CONTINUE
RETURN
END

```

C
C
C

FUNCTION FISHER(N,H,X)
 SUBROUTINE TO CALCULATE PROBABILITY THAT F-RATIO GREATER THAN X
 ARGUMENTS : H IS D.F. FOR TREATMENTS, N IS D.F. FOR ERROR AND X IS
 CALCULATED F-RATIO

```

IF(X.LE.0.0) GO TO 110
INTEGER A,B
A=2*(H/2)-H+2
B=2*(N/2)-H+2
W=X*N/FLOAT(H)
Z=1.0/(1.0+W)
IF(A.EQ.1.AND.B.EQ.1) P=SQRT(W)
IF(A.EQ.1.AND.B.EQ.1) D=0.3183098862 *Z/P
IF(A.EQ.1.AND.B.EQ.1) P=0.5366197724 *ATAN(P)
IF(A.EQ.1.AND.B.NE.1) P=SQRT(W*Z)
IF(A.EQ.1.AND.B.NE.1) D=0.5 * P * Z/W
IF(A.NE.1.AND.B.EQ.1) P=SQRT(Z)
IF(A.NE.1.AND.B.EQ.1) D=0.5 * Z * P
IF(A.NE.1.AND.B.EQ.1) P=1-P
IF(A.NE.1.AND.B.NE.1) D=Z*Z
IF(A.NE.1.AND.B.NE.1) P=W*Z
Y=2.0*E/Z
IF(A.NE.1) GO TO 90
IF( B+2 .GT. H) GO TO 95
DO 80 J = B+2,H,2
  D = (1.0 + A/FLOAT(J-2))*D*Z
  P = P + D * Y/(J-1)
80 CONTINUE
GO TO 95
90 ZK = Z*((N-1)/2)
  D = D * ZK * N/H
  P = P * ZK + W * Z * (ZK -1)/(Z -1)
95 Y = W * Z
  Z = 2.0/Z
  B = N -2
  IF( A+2 .GT. H) GO TO 105
  DO 100 I = A+2,H,2
    J = I + B
    D = Y * D * J/FLOAT(I-2)
    P = P - Z * D/J
100 CONTINUE
105 IF( P .GT. 1.0) P = 1.0
  IF(P .LT. 0.0) P = 0.0
  FISHER = 1 - P
  RETURN
110 FISHER=1.0
  RETURN
END

```

BINGRAPH

Bingraph is a programme for plotting the W_r - V_r graphs. The subroutine graph is not included here. It is available at the Lincoln College Computer Centre.

```

1  DIMENSION Y(54,9),X(54,9),WR(9),VR(9),TITLE(16)
1  READ(5,90,END=3)IF
90  FORMAT(14)
   READ(5,9,END=2)IF,(WR(1),I=1,IF)
2  CONTINUE
9  FORMAT(*16.4)
   READ(5,12,END=39)(TITLE(K),K=1,16)
39  CONTINUE
12  FORMAT(16A5)
   WRITE(6,13)(TITLE(K),K=1,16)

13  FORMAT(*1*,1X,T15,16A5,/)
   READ(5,9,END=29)IF,(VR(1),I=1,IF)
29  CONTINUE
   READ(5,19,END=79)VP,YCEPT,REGC,SEPEGC
79  CONTINUE
19  FORMAT(4F8.4)
   WRITE(6,69)VP,YCEPT,REGC,SEPEGC
69  FORMAT('0', 'THE PARENTAL VARIANCE IS',F15.4, '// 'THE Y INTERCEPT IS'
* ,F15.4, '// 'THE REGRESSION COEFFICIENT IS',F15.4, 'SE',F15.4, //)
   VRSUM=0.0
   DO 10 I=1,IF
   VRSUM=VRSUM+VR(I)
   Y(I,1)=WR(1)
   X(I,1)=VR(1)
10  CONTINUE
   VRM=VRSUM/IF
   DO 20 J=1,10
   XJJ=(VRM/5.0)*((J-1)+1)
   Y(J,2)=SQRT((VP*XJJ))
   X(J,2)=XJJ
20  CONTINUE
   CALL GRAPH(Y,X,2,9,10...025,0,1,1,1,1)
   GO TO 1
3  STOP
   END

```

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REFERENCES

- Adams, M.W. 1967. Basis of yield components compensation in crop plants with special reference to field beans, *Phaseolus vulgaris*. *Crop Science* 7: 505-510.
- Ali, M.I. 1976. Inheritance of protein content and protein yield and their relationships to other characters in common wheat. *Scientific Report of the Agricultural University of Norway* 55: 1-17.
- Allan, R.E.; Vogel, O.A. 1963. F2 Monosomic analysis of culm length in wheat crosses involving semidwarf Norin 10 - Brevor 14 and the Chinese Spring series. *Crop Science* 3: 538-540.
- Allan, R.E.; Vogel, O.A.; Peterson, J.C. 1968. Inheritance and differentiation of semidwarf culm length of wheat. *Crop Science* 8: 701-704.
- Anwar, A.R.; Chowdhry, A.R. 1969. Heritability and inheritance of plant height heading date, and grain yield in four spring wheat crosses. *Crop Science* 9: 760-761.
- Ausemus, E.R.; Harrington, J.B.; Rertz, L.P.; Worzella, W.W. 1946. A summary of genetic studies in hexaploid and tetraploid wheat. *Agronomy Journal* 38: 1082-1099.
- Bains, K.S.; Gill, K.S.; Sehgal, K.L. 1972. Epistatic bias in additive and dominance variation and genetic association between two quality traits in bread wheat. *Canadian Journal Genetics and Cytology* 14: 49-55.
- Baker, R.J. 1978. Issues in diallel analysis. *Crop Science* 18: 533-536.

- Balalic, M.K. 1973. Inheritance of leaf area in vulgare wheat. *Proceedings of 4th International Wheat Genetics Symposium* 821-825.
- Bhatt, G.M. 1971. Heterotic performance and combining ability in a diallel cross among spring wheat. *Australia Journal of Agriculture Research* 22: 359-68.
- Bhatt, G.M. 1972. Inheritance of heading date, plant height, and kernel weight in two spring wheat crosses. *Crop Science* 12: 95-98.
- Bhatt, G.M. 1973. Significance of path coefficient analysis in determining the nature of character association. *Euphytica* 22: 338-343.
- Bitzer, M.J.; Patterson, F.L.; Nyquist, W.E. 1971. Hybrid vigour and gene action in a six parent and diallel cross of soft winter wheat. *Canadian Journal of Genetics and Cytology* 13: 131-137.
- Briggle, L.W.; Vogel, O.A. 1968. Breeding short statured, disease resistant wheats in the United States. *Supplementary Euphytica*: 107-130.
- Briggs, K.G.; Shebeski, L.H. 1970. Visual selection for yielding ability of F3 lines in hard red spring wheat breeding programme. *Crop Science* 10: 400-402.
- Chahal, G.S.; Jinks, J.L. 1978. A general method of detecting the additive, dominance and epistatic variation that inbred lines can generate using a single tester. *Heredity* 40(1): 117-125.
- Chapman, S.R.; McNeal, F.H. 1970. Gene effects for grain protein in five spring wheat crosses. *Crop Science* 10: 45-46.
- Chapman, S.R.; McNeal, F.H. 1971. Gene action for yield components and plant height in a spring wheat cross. *Crop Science* 11: 384-386.

- Chandhanamutta, P.; Frey, K.J. 1973. Indirect mass selection for grain yield in oat population. *Crop Science* 13: 470-473.
- Clark, J.A. 1926. Breeding wheat for high protein content. *Agronomy Journal* 18: 648-661.
- Coles, G.D.; Wrigley, C.W. 1976. Laboratory methods for identifying New Zealand wheat cultivars. *New Zealand Journal of Agriculture Research* 19: 499-503.
- Comstock, A.E.; Robinson, H.F. 1952. Estimation of average dominance of genes. *Heterosis* Iowa State College Press, Iowa. 522 p.
- Cooley, W.W.; Lohnes, P.R. 1971. *Multivariate data analysis*. New York. Wiley. 364 p.
- Copp, G.L. 1965-67. Wheat breeding in New Zealand. *New Zealand Wheat Review* 10: 78-86.
- Copp, L.G.L.; Cawley, R.W. 1971-1973. Kopara 73 an improved line of Kopara. *New Zealand Wheat Review* 12: 35-37.
- Cross, R.J.; Haslemore, R.M. 1979. Nitrogen uptake and redistribution in Karamu and other spring wheats. *New Zealand Journal of Agriculture Research* 22: 547-552.
- Crumpacker, D.W.; Allard, R.W. 1962. A diallel cross analysis of heading date in wheat. *Hilgardia* 32: 275-318.
- Davis, W.H.; Middleton, G.K.; Herbert, T.T. 1961. Inheritance of protein, texture and yield in wheat. *Crop Science* 1: 235-238.
- Depauw, R.M.; Shebeski, L.H. 1973. An evaluation of an early generation yield testing procedure in *Triticum aestivum*. *Canadian Journal of Plant Science* 53: 465-470.

- Dewey, D.R.; Lu, K.H. 1959. A correlation and path coefficient analysis of components of crested wheatgrass seed production. *Agronomy Journal* 5: 515-518.
- Diehl, A.L.; Johnson, V.A.; Mattern, P.J. 1978. Inheritance of protein and lysine in three wheat crosses. *Crop Science* 18: 391-395.
- Dougherty, C.T.; Scott, W.R.; Langer, R.H.M. 1975. Effects of sowing rate, irrigation, and nitrogen on the components of yield of spring-sown semidwarf and standard New Zealand wheats. *New Zealand Journal of Agriculture Research* 18: 197-207.
- Dougherty, C.T.; Love, B.G.; Mountier, N.S. 1978. *New Zealand Journal of Agricultural Research* 21: 655-663. X
- Dunn, O.J.; Clark, V.A. 1974. *Applied Statistics*. New York. Wiley. 387 p. X
- Edwards, L.H.; Ketata, H.; Smith, E.L. 1976. Gene action of heading date, plant height and other characters in two winter wheat crosses. *Crop Science* 16: 275-277.
- England, F. 1974. A general approximate method for fitting additive and specific combining abilities to the diallel cross with unequal numbers of observations in the cells. *Theoretical and Applied Genetics* 44: 378-380.
- Engledow, F.L.; Wadham, S.M. 1923. Investigations of yield in cereals. *Journal Agriculture Science Cambridge* 13: 390-439.
- Evans, L.T.; Wardlaw, I.F. 1976. Aspects of the comparative physiology of grain yield in cereals. *Advances in Agronomy* 28: 301-350.
- Falconer, D.S. 1960. *Introduction to quantitative genetics*. London: Longmans. 365 p.

- Fick, G.N.; Qualset, C.O. 1973. Genes for dwarfness in wheat, *Triticum aestivum* L. *Genetics* 75: 531-539.
- Fonseca, S.; Patterson, F.L. 1968. Yield components, heritabilities and interrelationships in winter wheat (*Triticum aestivum* L.). *Crop Science* 8: 614-617.
- Frankel, O.H.; Hullet, E.W. 1947. Hilgendorf - A new wheat of outstanding baking quality. *New Zealand Wheat Review* 3: 29-33.
- Fraser, J.; Dougherty, C.T. 1977. Effects of sowing rate and nitrogen fertilizer on tillering of Karamu and Kopara wheats. *Proceedings of Agronomy Society of New Zealand* 7: 81-87.
- Gale, J.G.; Mather, K.; Jinks, J.L. 1977. Joint scaling tests. *Heredity* 38: 47-51.
- Gale, M.D.; Law, C.N.; Worland, A.J. 1975. The chromosomal location of a major dwarfing gene for Norin 10 in new British semi dwarf wheats. *Heredity* 35: 417-421.
- Gale, M.D.; Marshall, G.A. 1976. The chromosomal location of Gail and Rht1, genes for gibberellin insensitivity and semidwarfism, in a derivative of Norin 10 wheat. *Heredity* 37: 283-289.
- Gale, M.D.; Law, C.N. 1976. Norin-10-based semidwarfism. In: *Genetic Diversity in Plants, Volume 8*. New York: Plenum Press.
- Gallagher, J.N. 1978. Ear development: processes and prospects. In *Crop Physiology and Cereal Breeding. Proceeding of a Eurcapia Workshop*.
- Gaunt, R. 1979. (Personal communication).

- Gibori, A.; Hillel, J.; Cahaner, A.; Ashri, A. 1978. A 9 x 9 diallel analysis in peanuts (*A. hypogaea* L.): Flowering time, tops weight, pod yield per plant and pod weight. *Theoretical and Applied Genetics* 53: 169-179.
- Gilbert, N.E.G. 1958. Diallel cross in plant breeding. *Heredity* 12: 477-492.
- Gill, K.S.; Bains, S.S.; Singh, G.; Bains, K.S. 1973. Partial diallel test crossing for yield and its components in *Triticum aestivum* L. *Proceedings 4th International Wheat Genetics Symposium* 29-33.
- Gill, K.S.; Nanda, G.S.; Singh, G. 1977. Inheritance of plant height, tiller number, ear length and number of spikelets in two spring X winter crosses in wheat. *Genetic Agriculture* 31: 227-237.
- G/rafius, J.E. 1964. A geometry of plant breeding. *Crop Science* 4: 241-246.
- Griffing, B. 1956. A generalised treatment of the use of diallel crosses in quantitative inheritance. *Heredity* 10: 31-50.
- Halloran, G.M. 1974. Genetic analysis of hexaploid wheat, *Triticum aestivum* using intervarietal chromosome substitution lines. I culm length, ear density, spikelet number and fertility. *Canadian Journal of Genetics and Cytology* 10: 449-456.
- Halloran, G.M. 1975. Genetic analysis of grain protein in wheat. *Theoretical and Applied Genetics* 46: 79-86.
- Halloran, G.M. 1976. Genetic analysis of hexaploid wheat, *T. aestivum*, using intervarietal chromosome substitution lines: Protein content and grain weight. *Euphytica* 25: 65-71.

- Haunold, A.; Johnson, V.A.; Schmidt, J.W. 1962.
Genetic measurement of protein in the grain of
Triticum aestivum L. *Agronomy Journal* 54: 203-
206.
- Hayman, B.I. 1954a. The analysis of variance of diallel
tables. *Biometrics* 10: 235-244.
- Hayman, B.I. 1954b. The analysis of continuous variation
in a diallel cross of *Nicotiana rustica* varieties.
Genetics 39: 794-829.
- Hayman, B.I. 1960. Heterosis and quantitative inherit-
ance. *Heredity* 15: 327-328.
- Hayman, B.I. 1963. Notes on diallel-cross theory. In:
Statistical Genetics and plant breeding. National
Academy Science and National Research. Council
Publication 982: 571-575.
- Hermansen, J.G.Th. 1963. The localization of two genes
for dwarfing in the wheat variety Timstein by means
of substitution lines. *Euphytica* 12: 126-129.
- Hsu, P.; Walton, P.D. 1970a. The quantitative inherit-
ance in spring wheat of morphological structures and
above the flag leaf node. *Canadian Journal of
Genetics and Cytology* 12: 738-742.
- Hsu, P.; Walton, P.D. 1970b. The inheritance of morpho-
logical and agronomic characters in spring wheat.
Euphytica 19: 55-60.
- Jain, H.K.; Singhal, N.C.; Singh, M.P.; Austin, A. 1975.
An approach to breeding for higher protein content in
bread wheat. *Breeding for seed protein improvement
using nuclear technique*. Vienna: IAEA.
- Janossy, A.; Lupton, F.G.H. 1974. *Heterosis in plant
breeding*. New York - Elsevier. 364 p.
- Jinks, J.L. The analysis of continuous variation
a diallel cross of *Nicotiana rustica* varieties.
Genetics 39: 767-788.

- Jinks, J.L.; Hayman, B. 1953. Analysis of diallel crosses. *Maize Genetics Cooperation Newsletter* 27: 48-54.
- Jinks, J.L.; Jones, R.M. 1958. Estimation of the components of heterosis. *Genetics* 43: 223-234.
- Jinks, J.L.; Kearsey, M.J.; Breese, E.L. 1969. A general method of detecting additive, dominance and epistatic variation for metrical traits. II. Application to inbred lines. *Heredity* 24: 45-57.
- Jinks, J.L.; Perkins, Jean M.; Breese, E.L. 1969. A general method of detecting additive, dominance and epistatic variation for metrical traits. II. Application to inbred lines. *Heredity* 24: 115-127.
- Jinks, J.L.; Perkins, J.M. 1970. A general method for the detection of additive, dominance and epistatic components of variation. III. F₂ and Backcross Populations. *Heredity* 25: 419-429.
- Jinks, J.L.; Virk, D.S. 1977. A modified triple test cross analysis to test and allow for inadequate testers. *Heredity* 39: 165-170.
- Johnson, L.P.V. 1963. Applications of the diallel cross technique to plant breeding. *National Academy Science and National Research Council Publication* 982: 561-570.
- Johnson, V.A.; Biever, K.J.; Haunold, A.; Schmidt, J.W. 1966. Inheritance of plant height, yield of grain, and other plant and seed characteristics in a cross of hard red winter wheat, *Triticum aestivum* L. *Crop Science* 6: 336-338.
- Johnson, V.A.; Mattern, P.J.; Schmidt, J.W. 1967. Nitrogen relations during spring growth in varieties of *Triticum aestivum* L. differing in grain protein content. *Crop Science* 7: 664-667.

- Johnson, V.A.; Mattern, P.J.; Wilhelmi, K.D.; Kuhr, S.L.
1978. Seed protein improvement in common wheat
(*T. aestivum* L.), opportunities and constraints.
In: Seed protein improvements by nuclear techniques.
Vienna: IAEA.
- Jones, M.R. 1965. Analysis of variance of the half diallel
table. *Heredity* 20: 117-121.
- Kaul, A.K.; Sosulski, F.W. 1965. Inheritance of flour
protein content in a Selkirk x Gabo cross.
Canadian Journal of Genetics and Cytology 7: 12-17.
- Kearsey, M.J. 1965. Biometrical analysis of a random
mating population: A comparison of five experimental
designs. *Heredity* 20: 205-236.
- Kearsey, M.J.; Jinks, J.L. 1968. A general method of
detecting additive, dominance and epistatic variation
for metrical traits. I. Theory. *Heredity* 23:
403-309.
- Kempthorne, O. 1969. *An introduction to genetic statistics.*
Ames: Iowa State University. 545 p.
- Ketata, H.; Edwards, L.H.; Smith, E.L. 1976a. Inherit-
ance of eight agronomic characters in a winter wheat
cross. *Crop Science* 16: 19-22.
- Ketata, H.; Smith, E.L.; Edwards, L.H.; McNew, R.W. 1976b.
Detection of epistatic, additive and dominance variat-
ion in winter wheat. *Crop Science* 16: 1-4.
- Keuls, M; Garretsen, F. 1977. A general method for the
analysis of genetic variation in complete and incom-
plete diallels and North Carolina II designs. Part
I: Procedures and general formulas for random models.
Euphytica 26: 537-551.
- Klepper, L.; Wilhelm, K. 1979. Comparison between Kjeldahl
and Near Infra Red protein analyses on vegetative and
head samples of wheat. *Crop Science* 19: 922-924.

- Knott, D.R. 1972. Effects of selection for F₂ plant yield on subsequent generations in wheat. *Canadian Journal of Plant Science* 52: 721-726.
- Knott, D.R. 1979. Selection for yield in wheat breeding. *Euphytica* 28: 37-40.
- Knott, D.R.; Kumar, J. 1975. Comparison of early generation yield testing and a single seed descent procedure in wheat breeding. *Crop Science* 15: 295-299.
- Knott, D.R.; Talukdar, B. 1971. Increasing seed weight in wheat and its effect on yield, yield components and quality. *Crop Science* 11: 280-283.
- Konzak, C.F. 1977. Genetic control of the content, amino acid composition and processing properties of proteins in wheat. *Advances in Genetics* 19: 407-582.
- Kronstad, W.E.; Foote, W.H. 1964. General and specific combining ability estimates in winter wheat. *Crop Science* 4: 616-619.
- Kuspira, J.; Unrau, J. 1957. Genetic analysis of certain characters in common wheat using whole chromosome substitution lines. *Canadian Journal of Plant Science* 37: 300-326.
- Langer, R.H.M. 1965. A study of New Zealand wheats. I. *New Zealand Journal of Agriculture Research* 8: 10-14.
- Langer, R.H.M. 1974. Control of tiller bud growth in Gramineae. *Proceedings of the 12th International Grassland Congress*: 179-190.
- Langer, R.H.M. 1976. Yield determination in wheat: The Physiologist point of view. *New Zealand Wheat Review* 13: 54-55.

- Langer, R.H.M. 1977. Controversial Karamu. *DSIR Cereal News* 4: 1-2.
- Langer, R.H.M. 1978. The dynamics of wheat yield. *Proceedings of the 28th Lincoln College Farmers Conference*: 115-127.
- Langer, R.H.M.; Dougherty, C.T. 1976. Physiology of grain yield in wheat, p. 59-67. In: Sunderland, N. *Perspectives in Experimental Biology*, Volume 2, Botany. New York: Pergamon Press.
- Law, C.N.; Snape, J.W.; Worland, A.J. 1978. The genetical relationship between height and yield in wheat. *Heredity* 40: 135-151.
- Law, C.N.; Young, C.F.; Brown, J.W.S.; Snape, J.W.; Worland, A.J. 1978. The study of grain protein control in wheat using whole chromosome substitution lines. In *Seed Protein Improvement by Nuclear Technique*. Vienna: IAEA.
- Lebsock, K.L.; Fifield, C.C.; Gurney, G.M.; Greenaway, W.T. 1965. Variation and evaluation of mixing tolerance, protein content and sedimentation value in early generations of spring wheat, *Triticum aestivum* L. *Crop Science* 4: 171-174.
- Leng, E.R. 1963. Component analysis in inheritance studies of grain yield in maize. *Crop Science* 3: 187-190.
- Li, C.C. 1955. An introduction to population genetics. Chicago: *University of Chicago Press*.
- Li, C.C. 1956. The concept of path coefficient and its impact on population genetics. *Biometrics* 12: 190-210.
- Li, C.R. 1964. Statistical Inference. II. Michigan: Edwards Brothers, Inc. 575 p.

- Lofgren, I.R.; Finney, K.F.; Heyne, E.G.; Bolte, L.C.; Hoseney, R.C.; Shogren, M.D. 1968. Heritability estimates of protein content and certain quality and agronomic properties in breed wheats. *Crop Science* 8: 563-567.
- Lupton, F.G.H. 1961. Further studies in cross prediction. *Euphytica* 10: 209-224.
- Lupton, F.G.H. 1975. Dwarf wheats with big ears. *Spectrum* 135: 9-11.
- Lupton, F.G.H. 1979. *Annual Report Plant Breeding Institute*, Cambridge: Plant Breeding Institute.
- Malcolm, J.P. 1977. Karamu quality. *DSIR Cereal News* 4: 6-7.
- Mather, K. 1967. Complementary and duplicate gene interactions in biometrical genetics. *Heredity* 22: 97-103.
- Mather, K.; Jinks, J.L. 1971. *Biometrical Genetics*. 2nd ed. London: Chapman and Hall. 382 p.
- McCracken, D.D. 1965. *A guide to Fortran IV programming*. 2nd ed. New York: Wiley. 288 p.
- McEwan, J.C. 1973. The performance of semi dwarf wheats in New Zealand : Implication for New Zealand Wheat Breeding. *Proceeding 4th International Wheat Genetics Symposium*. 557-559.
- McEwan, J.M.; Cross, R.J. 1978. Evolutionary changes in New Zealand wheat cultivars. *Proceeding 5th International Wheat Genetics Symposium* : 198-203.
- McEwan, J.M.; Hadfield, P.D. 1978. Rongotea and Oroua wheats promising replacements for Karamu. *DSIR Cereal News* 6: 15-17.
- McEwan, J.M.; Vizer, K.J. 1972. Karamu - a new spring wheat. *New Zealand Journal of Agriculture Research* 125: 50-51.

- McEwan, J.M.; Vizer, K.J.; Douglas, J.A. 1979. New spring wheats: Rongotea and Oroua. *New Zealand Journal of Agriculture Research* 139: 18-19.
- McGinnis, R.C.; Shebeski, L.H. 1968. The reliability of single plant selection for yield in F₂. *Proceeding 3rd International Wheat Genetics Symposium*: 109-114.
- McNeal, F.H. 1960. Yield components in a Lehnis x Thatcher wheat cross. *Agronomy Journal* 52: 348-349.
- McNeal, F.H.; Qualset, C.O.; Baldrige, D.E.; Stewart, V.R. 1978. Selection for yield and yield components in wheat. *Crop Science* 18: 795-799.
- McNeal, F.H.; Berg, M.A. 1977. Flag leaf area in five spring wheat crosses and the relationship to grain yield. *Euphytica* 26: 739-744.
- McVitte, J.A.; Gale, M.D.; Marshall, A.G.; Westcott, B. 1978. The intrachromosomal mapping of Norin 10 and Tom Thumb dwarfing genes. *Heredity* 40: 67-70.
- Meredith, P. 1970. The baking quality of New Zealand wheat. *New Zealand Wheat Review* 11: 20-28.
- Merkle, O.C.; Atkins, I.M. 1964. Inheritance of plant height and stem rust resistance in wheat, *Triticum aestivum* L. *Crop Science* 4: 453-454.
- Middleton, G.K.; Bode, C.E.; Boyles, B.B. 1954. A comparison of the quantity and quality of protein in certain varieties of soft wheat. *Agronomy Journal* 46: 500-502.
- Moll, R.H.; Kojima, K.; Robinson, H.F. 1962. Components of yield and overdominance in corn. *Crop Science* 2: 78-79.

- Morris, M.R.; Schmidt, J.W.; Mattern, P.J.; Johnson, V.A. 1973. Chromosomal location of genes for high protein in the wheat cultivar Atlas 66. *Proceedings 4th International Wheat Genetics Symposium*: 715-718.
- Nassar, R.F. 1965. Effect of correlated gene distribution due to sampling on the diallel analysis. *Genetics* 52: 9-20.
- Novoa, R.V. 1973. Inheritance of height and other characters under condition of the coast of Peru. *Proceedings 4th International Wheat Genetics Symposium*: 611-615.
- O'Brien, L.; Baker, R.J.; Evans, L.E. 1978. Response to selection for yield in F3 of four wheat crosses. *Crop Science* 18: 1029-1033.
- O'Brien, L.O.; Orth, R.A. 1977. Effect of geographic location of growth on wheat milling yield, farinograph properties, flour protein and residue protein. *Australian Journal of Agricultural Research* 28: 5-9.
- Panigrahi, T. 1962. Estimates of heritability, dominance and genetic advances in a wheat hybrid with semidwarf parent. From: *Plant Breeding Abstract* 33: 164.
- Paroda, R.S.; Joshi, A.B. 1970. Genetic architecture of yield and components of yield in wheat. *Indian Journal of Genetics and Plant Breeding* 30: 298-314.
- Paroda, R.S.; Joshi, A.B. 1970. Correlations, path coefficients and the implications of discriminant function for selection in wheat (*Triticum aestivum* L.). *Heredity* 25: 383-392.
- Pearson, R.G.; McArthur, A.T.G. 1975. *Subroutine Graph*. Canterbury: Lincoln College Computer Centre.
- Perkins, J.M.; Jinks, J.L. 1971. Analysis of genotype environment interaction in triple test cross data. *Heredity* 26: 203-209.

- Rahman, M.S.; Halloran, G.M.; Wilson, J.W. 1977. Genetic control of spikelet number in hexaploid wheat. *Crop Science* 17: 296-299.
- Reddi, M.V.; Heyne, E.G. 1970. Inheritance of plant height and kernel weight in two wheat crosses. *Indian Journal of Genetics and Plant Breeding* 30: 109-115.
- Rowe, K.E.; Alexander, W.L. 1980. Computations for estimating the genetic parameters in Joint Scaling Tests. *Crop Science* 20: 109-110.
- Scott, W.R.; Dougherty, C.T.; Langer, R.H.M. 1977. Development and yield components of high yielding wheat crops. *New Zealand Journal of Agriculture Research* 20: 205-212.
- Seth, J.; Herbert, T.T.; Middleton, G.K. 1960. Nitrogen utilization in high and low protein wheat varieties. *Agronomy Journal* 52: 207-209.
- Sharma, T.R.; Gupta, V.P.; Gassi, S.; Kaul, A.K. 1973. Estimation of genetic parameter of some quality characters in wheat (*Triticum aestivum* L.) and Bengal Grain (*Cicer arietinum* L.).
- Sharma, T.R.; Knott, D.R. 1964. The inheritance of seed weight in a wheat cross. *Canadian Journal of Genetics Cytology* 6: 419-425.
- Singh, S.; Singh, R.B. 1976. Triple test cross analysis in two wheat crosses. *Heredity* 37: 173-177.
- Singh, S.; Singh, R.B. 1978. Triple test cross analysis in first backcross populations of four wheat crosses. *Journal of Agriculture Science, Cambridge* 91: 505-508.
- Singh, J.; Arnand, S.C. 1971. Inheritance of spike number in wheat. *Indian Journal Genetics* 31: 212-217.

- Smith, H.C. 1974. Recommended wheat varieties. *New Zealand Journal of Agriculture Research* 128: 50-55.
- Snape, J.W.; Law, C.N.; Worland, A.J. 1977. Whole chromosome analysis of height in wheat. *Heredity* 38: 25-26.
- Sneep, J. 1977. Selection for yield in early generation of self fertilizing crops. *Euphytica* 26: 27-30.
- Sokol, M.J.; Baker, R.J. 1977. Evaluation of the assumptions required for the genetic interpretation of diallel experiments in self pollinating crops. *Canadian Journal Plant Science* 57: 1185-1191.
- Stoskoff, N.C.; Fairey, D.T. 1975. A synchronous tiller maturity - a potential problem in the development of dwarf winter wheat. *Plant Breeding Abstracts* 45: 467-472.
- Stubber, C.W.; Johnson, V.A.; Schmidt, J.W. 1962. Grain protein content and its relationship to other plant and seed characters in the parents and progeny of a cross of *Triticum aestivum* L. *Crop Science* 2: 505-508.
- Sunderman, D.W.; Wize, M.; Sneed, E.M. 1965. Inter-relationships of wheat protein content, flour sedimentation value, farinograph peak time, dough mixing and baking characteristics in F2 and F3 generation of winter wheat, *T. aestivum* L. *Crop Science* 5: 537-540.
- Syme, J.R. 1970. A high yielding Mexican semidwarf wheat and the relationship of yield to harvest index and other varietal characteristics. *Australian Journal Experimental Animal Husbandry* 10: 350-353.
- Syme, J.R. 1972. Features of high yielding wheats grown at two seed rates and two nitrogen levels. *Australian Journal Experimental Animal Husbandry* 12: 165-170.
- Tan, W.Y. 1974. The approximate overall test for epistatic effects in biometrical genetics. *Biometrics* 30: 697-703.

- Thompson, J.B.; Whitehouse, R.N.H. 1962. Environment and the inheritance of quality in spring wheat. *Euphytica* 11: 181-196.
- Thorne, G.N. 1965. Photosynthesis of ears and flag leaves of wheat and barley. *Annals of Botany N.S.* 29: 317-329.
- Thorne, G.N. 1966. Physiological aspects of grain yield in cereals. In Milthorpe, F.L.; Ivins, J.D. *The growth of cereals and grasses*. London: Butterworth.
- Virk, D.S.; Jinks, J.L. 1977. The consequences of using inadequate testers in the simplified triple test cross. *Heredity* 38: 237-257.
- Vogel, O.A.; Allan, R.E.; Peterson, C.J. 1963. Plant performance characteristics of semidwarf winter wheats producing most efficiently in Eastern Washington. *Agronomy Journal* 55: 397-398.
- Vogel, O.A.; Craddock, J.C.; Muir, C.E.; Everson, E.H.; Rohde, C.R. 1956. Semidwarf growth habit in winter wheat improvement in the Pacific North West. *Agronomy Journal* 48: 76-78.
- Walton, P.D. 1969. Inheritance of morphological characters associated with yield in spring wheat. *Canadian Journal of Plant Science* 49: 587-596.
- Watson, D.J.; Thorne, G.N.; French, S.A.W. 1963. Analysis of growth and yield of winter and spring wheats. *Annals Botany N.S.* 27: 1-22.
- Whitehouse, R.N.H.; Thompson, J.B.; Do Valle Ribeiro, M.A.M. 1958. The use of a diallel in yield prediction. *Euphytica* 7: 147-169.
- Wilson, J.B. 1979. *Teddybear Programme*. Canterbury: Lincoln College Computer Centre.

- Wright, G.M. 1968. A developmentally unstable character in wheat glume fertility. *Proceedings of 3rd International Wheat Genetics Symposium*: 294.
- Wright, G.M. 1977. (Personal communication).
- Wright, G.M. 1978. Wheat breeding. *DSIR Cereal News* 5: 16-21.
- Wright, G.M. 1980. New cultivars and their contribution to maximum wheat production. *Proceedings of the 30th Lincoln College Rural Conference*. In Press.
- Wright, G.M.; Thomas, G.A. 1976. An evaluation of the single seed descent method of breeding. *New Zealand Wheat Review* 13: 46-49.
- Wright, S. 1921. Correlation and causation. *Journal of Agriculture Research* 20: 556-587.
- Yates, F. 1947. Analysis of data from all possible reciprocal crosses between a set of parental lines. *Heredity* 1: 287-301.
- Zar, J.H. 1974. *Biostatistical analysis*. New Jersey: Prentice Hall. 619 p.

TABLE A Mean plant height of the parents (Pi), common tester (Pc),
F1, F2 and backcrosses (B1, B2) - Experiment III

Parents	Hilgendorf	Kopara	Oroua	Ruru	Atlas 66
Pi	106.6	107.6	100.7	87.2	143.8
Pc	89.8	89.9	89.0	88.9	89.5
B1	104.0	106.0	96.1	93.2	138.7
B2	100.2	100.3	93.3	91.6	111.6
F1	105.6	111.7	99.3	94.0	129.2
F2	100.2	103.8	93.5	88.5	117.2

Standard Error of Mean = 1.57

Coefficient of Variation = 10.11%

TABLE B Sums of squares of comparisons for detecting epistasis on
plant height

Parent (Pi)	Ai Comparison Sum of Squares	Bi Comparison Sum of Squares
Hilgendorf	155.75	217.46
Kopara	481.66	7.04
Oroua	557.89	26.76
Ruru	251.12	0.29
Atlas 66	175.47	175.83
Total	1621.59	427.39
Mean Square	36.04	9.50
Pooled Variance	13.64	15.44
F Ratio	2.64*	0.62

*** $p < 0.001$

** $p < 0.01$

* $p < 0.05$

TABLE C Scaling tests and estimates of parameters

Trait	1000 Grain weight	Plant	Height
Parent (Pi)	Ruru	Atlas 66	Oroua
Test			
A	2.13	4.41	-7.87
B	-4.79	4.42	-1.72
C	-13.79	-22.81*	-14.37**
S	-1.72	-7.91**	-1.19
Estimates			
m	33.75	84.98***	90.05***
d	0.06	27.14***	5.82***
h	18.26	84.74***	4.45
i	6.87	31.64**	4.78
j	2.66	-0.00	-6.15
l	0.06	-40.48**	4.82
Joint Test			
m	40.30±0.36	116.94±0.67	94.45±0.49
d	0.150±0.36	27.42±0.67	5.61±0.49
h	10.78±0.73	12.51±0.01	4.68±0.62
χ^2 (3)	12.41**	9.46*	9.84*

*p<0.05

**p<0.01

*** p<0.001

TABLE D The analysis of variance for the dominance value for plant height

Source of variation	df	ss	ms	F	Prob
Mean	1	265.7492	265.7492	51.8108	0.0001
Additive	5	379.0764	75.8153	14.7810	0.0007
Deviation	4	113.3272	28.3318	5.5236	0.0197
Block	2	0.2312	0.1156	0.0225	0.9778
Error	8	41.0338	5.1292	-	-
Total	15	420.3415	-	-	-

The estimate of the dominance value is 94.2481

TABLE E The analysis of variance for the additive value for plant height

Source of variation	df	ss	ms	F	Prob
Mean	1	498.0097	498.0097	83.6441	0.0000
Dominance	5	855.2921	171.0584	28.7304	0.0001
Deviation	4	357.2824	89.3206	15.0020	0.0009
Block	2	14.9825	7.4912	1.2582	0.3349
Error	8	47.6313	5.9539	-	-
Total	15	917.9059	-	-	-

The estimate of the additive value is 220.1393

TABLE F Mean 1000 grain weight of mainstem of parents (Pi), common tester (Pi), F1, F2, B1 and B2 families - Experiment III

Parents Generation	Hilgendorf	Kopara	Oroua	Ruru	Atlas 66
Pi	46.3	39.6	38.4	40.7	37.6
Pc	40.5	41.8	42.3	40.6	40.3
B1	48.3	41.8	41.7	45.3	42.3
B2	45.5	39.3	39.3	43.9	40.7
F1	48.4	41.3	42.8	52.0	44.8
F2	45.2	40.7	41.1	42.9	41.2

Standard Error of Mean = 0.9021

Coefficient of Variation = 14.17%

TABLE G Sums of squares of comparisons for detecting epistasis on 1000 grain weight of the mainstem.

Parent (Pi)	Ai Comparison sums of squares	Bi comparison sums of squares
Hilgendorf	31.88	40.34
Kopara	68.36	173.59
Oroua	40.88	372.28
Ruru	40.84	206.69
Atlas 66	36.61	120.68
Total	218.57	913.58
Mean Square	4.86	20.30
Pooled Variance	3.03	4.69
F Ratio	1.61	4.32***

*p<0.05

**p<0.01

***p<0.001

5.3.3 The New Triple Test Cross and Scaling Test Analyses of Plant Height.

In the above analyses, the Block I values for the Atlas 66 BC2 family have been read for computation as the BC1 values and vice versa. The interpretation and discussion in Chapter 5 have been based on these analyses and should be read with this in mind. A re analysis with the backcross families in their right order is presented in tables A, B and C. A significant deviation from the conclusion of the previous analyses is the absence of epistasis in the Atlas 66 family. This is shown by the non significant A and B Scaling Tests and the low contribution of the Atlas 66 array to the A_i and B_i sums of squares.

However, of the two epistatic comparisons, the A_i comparison is still significant suggesting presence of non allelic interaction. This can be attributed mainly to the Kopara, Ruru and Oroua arrays. This significant test for epistasis is, however, not supported by the A and B Scaling Tests which were not significant except for the A test of the Kopara array. Moreover, the estimates of the (i), (j) and (l) epistatic terms were all not significant. The additive and dominance model is therefore acceptable and the additive and dominance values can be estimated as in tables D and E.

4.3.3. The New Triple Test Cross and Scaling Test Analyses of 1000 Grain Weight.

The high mean value for the B2 family of the Ruru Cross (table 46) has been traced to a tabulation and card punching error. The corrected analyses are presented in tables C, F and G. In contrast to the original analyses of Chapter 4, the re analysis showed signi-

fificance only for the Bi comparison. The significance for this comparison can be traced to the Oroua and Ruru families. The Scaling Test Analysis also indicated significant B test for these two families.

ERRATA

page 3, line 18

Pioneering studies by Palmer (1952) showed selection for grain weight to be successful but selection for yield and other yield components was not.

Palmer, T.P. 1952 Population and selection studies in a Triticum cross Heredity 6 : 171 - 185

Page 220, line 10

by plant breeders (Copp and Wright, 1952)

Copp, L.G.L. and Wright, G.M. 1952. The inheritance of kernel weight in a Triticum vulgare Cross. Heredity 6 : 187-199

Page 10 Materials and Methods

Experiment A, B, C and D were four experiments involving the 4 x 4 full diallel crossing of cultivars Hilgendorf, Kopara, Oroua and Ruru. Experiment A and B were F1 families planted in June and August 1978 respectively. Experiment C involved the F1 families while Experiment D consisted of the F2 families and they were raised in August 1979. The plants were all spaced planted as in Experiments I, II and III and two replicate blocks were grown per family. The grains harvested for each family in each replicate block were used for the diallel analysis on protein content.