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QUANTITATIVE GENETICS OF RESISTANCE  
TO NET BLOTCH OF BARLEY (*Hordeum vulgare* L.)

A thesis  
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## ABSTRACT

A half diallel cross was made amongst five barley (*Hordeum vulgare* L.) genotypes (Zephyr, Hassan, Mata, Kaniere and Manchuria) which exhibited differential resistance to net blotch disease, incited by the fungus, *Drechslera teres*.

The 15 genotypes (5 parents and 10  $F_1$ 's) were grown to physiological maturity under two nitrogen regimes ( $20 \text{ mg } 10^{-3} \text{ m}^{-3}$  and  $105 \text{ mg } 10^{-3} \text{ m}^{-3} \text{ N}$ ), 14 hour photoperiod, light intensity of  $170 \text{ Wm}^{-2} \text{ PAR}$ ,  $22^\circ\text{C}$  (day)/ $16^\circ\text{C}$  (night) and relative humidities of 70% (first seven weeks) and 95 to 100%.

All genotypes were first inoculated ( $9,000 \text{ conidia } 10^{-6} \text{ m}^{-3}$ ) at 14 days and thereafter ten times at approximately weekly intervals. Infection occurred only in the high nitrogen environment. Absence of net blotch at the lower nitrogen level was probably due to insufficient plant nitrogen concentrations for growth of the fungus.

The procedures of Mather and Jinks were used to analyse the half diallel cross for net blotch resistance of the following intact plant parts: (1) top canopy (2) bottom canopy (3) flag leaf and (4) second leaf. Resistance of leaf sections in petri dishes was also analysed.

Biometrical analyses for all net blotch data sets showed that additivity was of much greater importance than dominance in controlling resistance. Epistasis and/or correlated gene distributions were trivial. Resistance was conditioned by a partially dominant, single effective factor which was suggested as one gene. Heritability estimates were moderate.

In the high nitrogen environments, net blotch had no significant effect on plant height, tillers per plant, spikelet and grain numbers per ear, grain yield, 100 kernel weight and intensity of physiological brown

spot. However heading date of diseased plants was significantly earlier than for fungicide treated plants.

Nitrogen concentration had a marked effect on most characters. Significant genotype-nitrogen (G x E) interactions occurred for plant height, spikelet number per ear, grain yield, 100 kernel weight and physiological brown spot.

Further biometrical analyses showed that additivity was most important for plant height, tillers per plant, spikelet and grain numbers per ear, and physiological brown spot. Dominance was noteworthy for heading date, grain yield and 100 kernel weight. Epistasis was relatively unimportant.

Keywords: biometrics, barley, diallel analysis, disease resistance, growth stages, inoculation, intensity, leaf area diagrams, net blotch, pathogen, quantitative genetics, susceptibility.

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## INTRODUCTION

Barley (*Hordeum vulgare* L.) is susceptible to many diseases (Nyvall, 1979) which may severely affect the quantity and quality of the harvested product. A disease which has received considerable attention, particularly in the last decade, is net blotch (Shipton *et al.*, 1973; Arnst *et al.*, 1978). This is a seed-borne (Neergaard, 1977), foliage disease caused by the fungus *Drechslera teres* (Sacc.) Shoem. (syn. *Helminthosporium teres*), conidial state of *Pyrenophora teres* (Died.) Drechsler. Net blotch is widespread, occurring wherever barley is grown in the humid, temperate regions of the world (Dickson, 1956). In New Zealand, its presence was first recorded by Dingley (1969).

Grain yield losses due to net blotch have been of economic significance in many barley growing regions of the world and yield reductions of up to 50 per cent are documented (Shipton *et al.*, 1973). The most important adverse effect of the disease is grain shrivelling as measured by reduction in 1,000 grain weight (Buchannon and Wallace, 1962; Hampton and Arnst, 1978). Other agronomic features which may be affected adversely by *D. teres* attack include tillering (Hampton, 1977), straw stiffness (Hampton, 1977) and carbohydrate content of grain (Shipton, 1966).

The dramatic yield losses attributable to net blotch have prompted extensive investigation into effective methods of disease control. Numerous studies have been conducted overseas to identify genetic stocks which exhibit a high degree of resistance to net blotch (Shipton *et al.*, 1973), with a view to their possible inclusion in breeding programmes. Qualitative inheritance of resistance has been studied

(Schaller, 1955; Mode and Schaller, 1958; Khan and Boyd, 1969a). However, the findings are conflicting (Bockelman *et al.*, 1977) and further investigation on inheritance of net blotch resistance is warranted. Results from qualitative studies are discussed shortly.

Environmental conditions may markedly affect the incidence and severity of net blotch (Shipton *et al.*, 1973). Studies on the effect of nitrogen nutrition on host resistance to *D. teres* are very limited and demonstrate both an increase (Singh, 1963; Piening, 1967) and decrease (Piening, 1967) in resistance with increasing nitrogen levels.

The main objective of the present study is to estimate quantitative genetic statistics for resistance to net blotch under controlled environmental conditions, using a diallel analysis (Griffing, 1956a, b; Mather and Jinks, 1971, 1977). These statistics cannot be estimated from qualitative studies, as conducted previously.

Further objectives are:

- 1) Examination of change in host resistance to net blotch with variation in nitrogen concentration, including estimation of genotype - nitrogen interaction (G x E), and
- 2) Investigation of the adverse effects of net blotch and formulation of a relationship between net blotch intensity and grain yield loss (disease - yield loss model).

CHAPTER 1

## CHAPTER 1.

### LITERATURE REVIEW

#### 1.1 NET BLOTCH

##### 1.1.1 SYMPTOMS

Infection is first evident as pin-point, brown lesions which enlarge longitudinally to produce brown blotches at or near the tip of the blade, surrounded by a narrow chlorotic zone (Drechsler, 1923; Shipton *et al.*, 1973; Hampton, 1977). As blotches develop, irregular dark brown lines appear, orientated both longitudinally and transversely to the axis of the leaf, to produce the characteristic netted appearance. With age, blotch centres become uniformly brown and the net effect is often no longer visible. With heavy infection, lesions may coalesce or elongate to form dark, brown, limited, stripes with irregular margins. When lesions are so numerous that most of the leaf is involved, the leaf withers from tip to base and the lesions fade to dull brown or black.

Lesions may develop on leaves, culms and leaf sheaths, awns, glumes and sterile spikelets. On resistant genetic stocks, lesions remain small and restricted with little or no chlorosis (Buchannon and McDonald, 1965; Kahn and Boyd, 1969a, b, c; Keeling and Bantari, 1975).

##### 1.1.2 EFFECTS ON BARLEY GROWTH AND YIELD

Grain yield reductions attributable to net blotch vary from 5 to 50 per cent (Shipton *et al.*, 1973), although commonly losses are less than 30 per cent. For example, yield reductions of 9 to 11 per cent in Denmark (Smedegard-Peterson, 1974), 11 per cent in Canada (McDonald and Buchannon, 1964) and 17 per cent in Western Australia

(Shipton, 1966) have been reported. In New Zealand, studies have shown yield losses of up to 25 to 30 per cent (Hampton and Arnst, 1978). With such losses, the disease may be of great economic significance.

The most significant adverse effect of net blotch is reduction in grain size, as measured by 1,000 grain weight (Buchannon and Wallace, 1962; Shipton, 1966; Hampton, 1977; Hampton and Arnst, 1978). In fungicide spray trials, Shipton (1966) found highly significant ( $P \leq 0.001$ ) differences in 1,000 grain weight between fungicide treated (means of 47.93g (Zineb) and 51.65g (Maneb)) and untreated (44.23g) plots of Beecher barley. Hampton and Arnst (1978) also used fungicides in Manawatu trials and showed yield losses for Zephyr barley of 20 per cent in 1975/76 and 25 to 30 per cent in 1976/77. In some trials, most of this loss was due to reduction in 1,000 grain weight.

Reduction in grain weight is particularly pronounced when net blotch infection causes a large loss in the photosynthetic area of the flag leaf, which is a major contributor to yield (Thorne, 1966, 1973; Evans and Wardlaw, 1976). However, significant decreases in grain weight may not always occur as compensation for reduced photosynthetic leaf area is possible. In physiological studies, Nosberger and Thorne (1965) showed that, when photosynthesis by barley leaves was prevented by excluding light from them, ear photosynthesis compensated for much of the loss in photosynthesis that resulted. Capacity for yield component compensation is acknowledged widely for many cereals (Evans and Wardlaw, 1976).

Several less important effects of net blotch are documented. The effect of *Drechslera teres* on seed germination is not well understood and there is lack of agreement between authorities regarding its importance (Shipton et al., 1973; Hampton, 1977). In New Zealand, Hampton (1977) reported that germination was not affected by *D. teres*

infection. However, statistics, including level of seed infection and germination percentage, were not presented. Seedling emergence is unaffected by *D. teres* (Hampton, 1977) but no supporting data were given. Tillering may be adversely affected depending on the severity of *D. teres* attack and timing of the epiphytotic in relation to plant growth stage (Rintelen, 1969; Hampton, 1977). Rintelen (1969) found that tillering was not significantly affected by early infection. However, information on the source of inoculum, levels of net blotch infection, and development of the disease prior to tillering, was unavailable.

Grain number per ear is often not affected by net blotch (Hampton, 1977; Hampton and Arnst, 1978). However, this may not always be true as Hampton (in preparation) found a low, negative, correlation (- 0.21) between net blotch intensity on the flag leaf and grain number per ear. The correlation was significantly ( $P \leq 0.01$ ) different from zero. Grain number per ear, in the absence of net blotch, was 29.8 while at a mean net blotch intensity of 21.3 per cent it was 27.2 grains per ear. This represented an 8.7 per cent loss in grain number per ear.

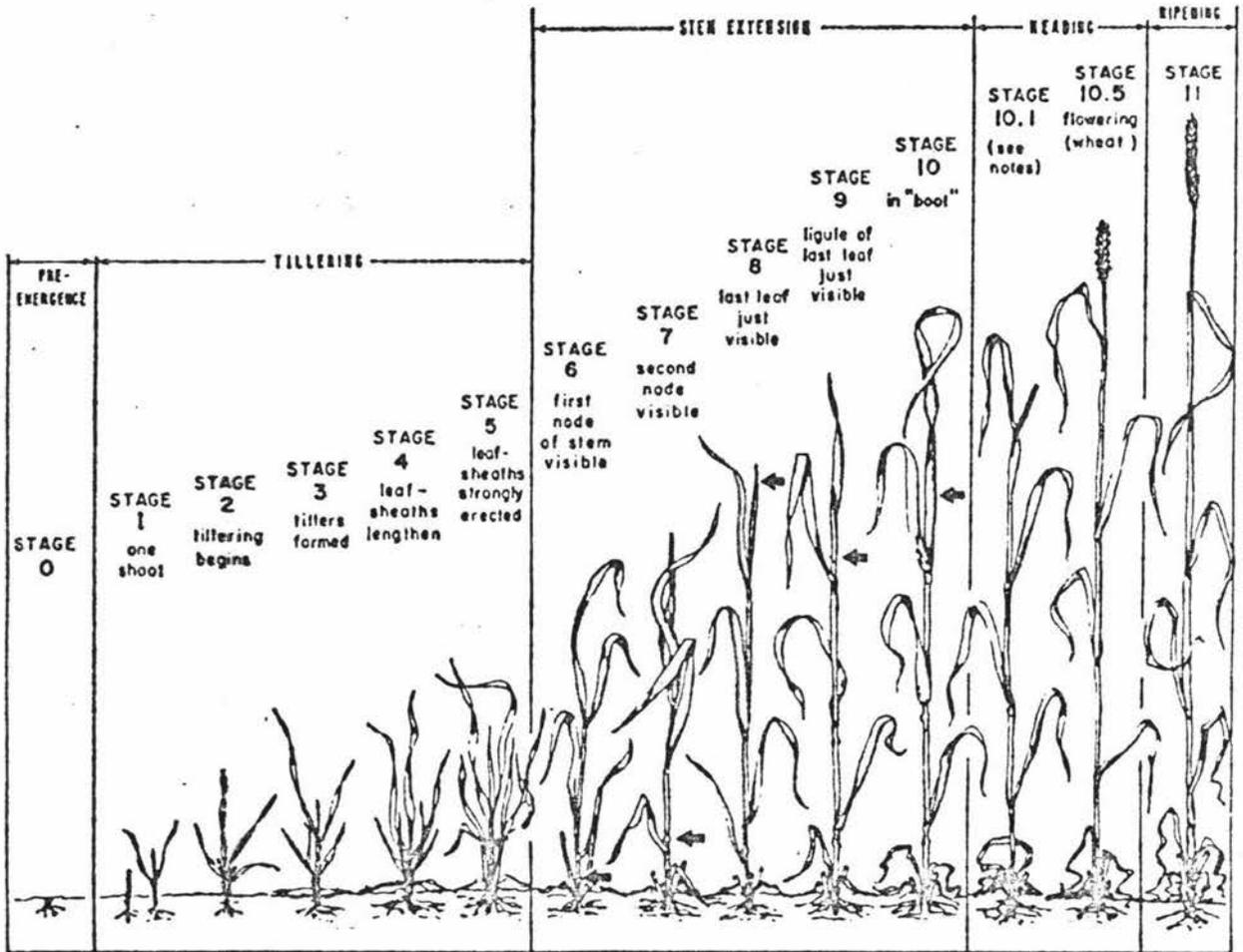
Stems may be weakened in severely infected crops, resulting in lodging. However, stem length is unaltered (Hampton, 1977). Malt yield (g) and malt extract yield (%) may be reduced significantly by net blotch epiphytotics. Shipton (1966) found a mean malt yield of 51.46g for grain collected from plots receiving no fungicide spray treatment and this was significantly ( $P \leq 0.001$ ) less than estimates from plots receiving either Maneb (52.40g) or Zineb (52.30g) sprays. Material receiving no fungicide treatment also had a significantly ( $P \leq 0.05$ ) lower estimate of malt extract yield (%) than treated material.

### 1.1.3 METHODS OF YIELD LOSS ASSESSMENT

Techniques to provide quantitative yield loss relationships for cereal foliar pathogens commonly involve standard regression analysis (Draper and Smith, 1966; Snedecor and Cochran, 1967; Steel and Torrie, 1980) of grain yield loss (%) against one or more values of disease intensity (James and Shih, 1973; Teng and Gaunt, 1980). Intensity is defined as infected area of plant tissue expressed as a percentage of total area.

Assessment of intensity requires decisions on plant parts assessed and the number of assessments to be made during the cereal growing season. As regards the former, it is routine practice to record intensity on the flag and/or second (Evans, 1969; James, 1974; Richardson *et al.*, 1975, 1976; Melville *et al.*, 1976; King, 1977) leaves (flag = leaf 1), although additional leaves have been assessed (James *et al.*, 1968; Habgood, 1974). Assessments may be made at a single growth stage (critical-point model) when the plant is particularly sensitive to the effects of disease; or at many consecutive stages during crop development (multiple-point model) (James, 1974). Multiple-point models are the most accurate and are preferred for disease-yield loss research. However, critical-point models have been highly successful when applied to cereals in the presence of late epiphytotics occurring near the time of grain filling (James, 1974; Teng and Gaunt, 1980). Grain filling occurs between Feekes-Large Growth Stages 10.5 and 11.3 (Large, 1954; Zadoks *et al.*, 1974). These and other Growth Stages (G.S.) are illustrated in Figure 1.1.

Both simple and multiple regression analyses (Steel and Torrie, 1980) have been used to formulate yield loss relationships. For net blotch, Hampton and Arnst (1978) found that the percentage flag leaf area infected at G.S. 11.1 gave the best indication of yield loss. This was



- Stage 10.1. Firstspikes just visible (awns just showing in barley, spike escaping through split of sheath in wheat or oats)
- 10.2. Quarter of heading process completed
- 10.3. Half of heading process completed
- 10.4. Three-quarters of heading process completed
- 10.5. All spikes out of sheath
- 10.5.1. Beginning of flowering (wheat)
- 10.5.2. Flowering complete to top of spike
- 10.5.3. Flowering over at base of spike
- 10.5.4. Flowering over, kernel watery ripe
- 11.1. Milky ripe
- 11.2. Mealy ripe, contents of kernel soft but dry
- 11.3. Kernel hard (difficult to divide by thumb-nail)
- 11.4. Ripe for cutting. Straw dead

Figure 1.1 Growth stages in cereals (after E.C. Large, 1954).

a linear relationship and showed that for a 10 per cent increase in flag leaf area infected, there was a corresponding 10 per cent reduction in grain yield. Estimates of coefficient of determination (R-square) and standard error of regression, were omitted. The study indicated that a critical-point model was applicable for net blotch.

Hampton (in preparation) later conducted yield loss studies in two consecutive seasons. Regression relationships derived between yield and percentage flag leaf area infected, for each season, were:

$$1977/78 \quad \text{Yield} = 1.43 - (0.01 \times \text{net blotch intensity}) \dots\dots (1)$$

$$1978/79 \quad \text{Yield} = 1.52 - (0.01 \times \text{net blotch intensity}) \dots\dots (2)$$

Equations (1) and (2) had coefficients of determination of 0.10 and 0.097 respectively. These low estimates indicated that yield was influenced by many factors apart from net blotch. It is noted that tillers were the unit of measurement in this study and one would expect a high level of variation between separate tillers, much more so than between plots where the variation is evened out by the size of the sample. Standard errors of regression were not stated. Estimates of percentage yield losses calculated from equations (1) and (2) were 0.70 per cent and 0.66 per cent respectively, giving a mean for the two seasons of 0.68 per cent. That is, over both seasons a one per cent increment of net blotch infection on the flag leaf resulted in a 0.68 per cent loss in grain yield. Net blotch intensity on the second leaf was also recorded. However, there was no significant ( $P \leq 0.05$ ) effect due to second leaf infection over and above that already explained by flag leaf infection.

Results of the above two studies provide some justification for use of a critical-point model in formulation of a net blotch-yield loss model in the present study. As far as the writer is aware, these are the only

studies which have quantified the relationship between grain yield loss and net blotch intensity. Multiple regression approaches relating yield loss with several measures of net blotch intensity, such as on flag and second leaves, have not been conducted.

Examples of yield loss relationships for other cereal diseases follow. James *et al.* (1968) showed for barley that yield loss due to leaf blotch (scald) (*Rhynchosporium secalis* (Oud.) Davis) was equivalent to approximately two-thirds and one-half of disease intensity on flag and second leaves respectively, at milky ripe stage of growth. Predicted loss was an average of the two estimates.

Richardson *et al.* (1975, 1976) developed yield loss models for several cereal diseases using many single tillers. Seed weight (mg), seeds per ear and yield per ear (mg) were measured at varying levels of disease intensity. Yield component data sets were then regressed individually against the corresponding disease intensity data. For mildew on spring barley, Richardson *et al.* (1975) estimated mean regression coefficients of 0.26mg loss in seed weight, 0.29 decrease in seeds per ear and 0.50mg yield loss per ear, all for one per cent increases in mildew intensity. Percentage of variation accounted for by each of the several regression analyses was low and ranged from 7 to 16 per cent. Attempts to fit curves rather than straight lines to the data did not improve the results. It was concluded that there was no evidence to suggest that the relationships were non-linear.

An excellent example of use of multiple regression is the study of Burleigh *et al.* (1972) on wheat leaf-rust (*Puccinia recondita* f.sp. *tritici*). These workers derived the following relationship:

$$\% \text{ Yield Loss} = 5.3788 + 5.5260X_2 - 0.3308X_5 + 0.5019X_7$$

The independent variables ( $X_2$ ,  $X_5$ ,  $X_7$ ) are percentage leaf-rust severity

at the boot, early berry and early dough growth stages, respectively. The equation explained 79 per cent of the variation with a standard error of  $\hat{Y}$  of 9 per cent. Predictive ability of the model was reliable when tested on data other than that used for developing the model.

#### 1.1.4 SCREENING AND GENETICS OF HOST

##### RESISTANCE TO NET BLOTCH

The growing of resistant cultivars is often the most practical and effective means of controlling diseases of extensively grown crops such as barley (Shipton *et al.*, 1973; Dalmacio, 1979). Development of these cultivars involves comprehensive field and/or controlled environment screening trials to identify genetic stocks exhibiting a desirable degree of resistance. Analysis of the underlying genetic resistance mechanism(s) and a subsequent breeding programme, may follow. With regards to net blotch, numerous screening studies and genetic analyses have been conducted.

Working under Californian field conditions, Schaller and Wiebe (1952) identified highly resistant germplasm which was predominantly of Manchurian origin. Schaller (1955) later identified a single incompletely dominant gene, Pt (referring to the perfect stage of the causal agent of net blotch, *Pyrenophora teres*), as the gene conditioning resistance in the Manchurian genetic stock, Tifang. In a subsequent study, Mode and Schaller (1958) identified two additional incompletely dominant genes, Pt<sub>2</sub> and Pt<sub>3</sub>. Gene Pt was redesignated as Pt<sub>1</sub> and was linked closely to Pt<sub>2</sub>. Recombination frequency was 2.57 per cent. Gene Pt<sub>3</sub> was inherited independently.

Buchannon and McDonald (1965) in a Canadian controlled environment study, conducted screening trials but no genetic analyses, on germplasm similar to that investigated by Schaller and Wiebe (1952). In contrast

however, these later workers found that the greatest number of resistant genetic stocks originated from Ethiopia (seventeen of forty resistant stocks), rather than Manchuria. It was concluded that this lack of agreement between the two studies indicated that *D. teres* isolates from different regions, or even within the same region, may differ in pathogenicity. Differences between pathogenicities of isolates, as detected by reactions on a series of differential hosts (physiological specialisation), are now well established for *D. teres* (Khan and Boyd, 1969a; Shipton *et al.*, 1973; Tekauz and Buchannon, 1977; Khan and Portmann, 1979).

Environment and genotype-environment interaction effects in both host and pathogen were not discussed by Buchannon and McDonald (1965). However, it is suggested that these effects were at least as likely to cause lack of agreement between results of the two studies mentioned previously, as possible differences in pathogenicity of *D. teres* isolates. For example, Khan and Boyd (1969c) found that genetic stocks originating from Ethiopia generally exhibited stable resistant reactions when tested against a single *D. teres* isolate under a range of environmental conditions. However, variable host reactions were exhibited by stocks of Manchurian origin. This indicates that genotype-environment interaction may be very important amongst net blotch resistant genetic stocks (also see Section 1.2.3).

In Western Australia, reactions of parents and several generations of progeny of a 6-parent half diallel were recorded against a known physiological race of *D. teres* (Khan and Boyd, 1969a). Five parents were resistant to net blotch. The relatively susceptible parent was a commercially grown local cultivar (Dampier). Tests showed that resistance was conditioned by a single gene,  $Pt_a$ , in each of three

genetic stocks of Manchurian origin: Manchuria (C.I. 2330), Ming (C.I. 4797) and Tifang (C.I. 4407-1). Resistance in the remaining two stocks, which were of Ethiopian origin (C.I. 5791 and C.I. 9819), was conditioned by duplicate genes, one of which was allelic to  $Pt_a$ . Symbols were not given. The relationship of the additional gene could not be determined. Results suggested that  $Pt_a$  differed from  $Pt_1$ ,  $Pt_2$  and  $Pt_3$  previously identified. Quantitative analyses were not conducted.

Omar *et al.* (1970) in Middle East studies, recorded mature plant reactions to net blotch of  $F_1$ ,  $F_2$  and  $F_3$  generations of thirteen crosses. These were derived from various pairwise combinations of ten genetic stocks. Material consisted of three susceptible stocks (Baladi 16, Giza 117 and C.I. 6713) and seven resistant and semi-resistant introduced stocks (Japan 3, C.I. 8566, C.I. 8570, C.I. 8698, C.I. 8760, Bonus and C.I. 2225). Semi-resistant stocks were defined as those which produced "small to medium" lesions in response to *D. teres* attack and gave a net blotch intensity per plant ranging from 16 to 45 per cent. Crossing groups were resistant x susceptible, resistant x resistant, susceptible x susceptible, susceptible x semi-resistant and semi-resistant x semi-resistant. Resistance was conditioned by three completely dominant genes (I,  $Pt_1$ ,  $Pt_2$ ). Gene I conditioned highly resistant reactions while  $Pt_1$  and  $Pt_2$  controlled semi-resistance. The latter two genes demonstrated a complementary effect to give resistance. Three genes controlling resistance were also reported by Mode and Schaller (1958) in crosses involving eight genetic stocks, as discussed previously. However, relationships between the resistance genes found by Omar *et al.* (1970) and those designated by Mode and Schaller (1958) cannot be established.

Selim *et al.* (1973), cited by Bockelman *et al.* (1977), identified

duplicate genes in C.I. 11458, C.I. 11460 and Estate and a single gene in C.I. 4929, C.I. 5822 and C.I. 5791. Further information on this study was not provided. The relationship of these genes to one another and to those identified in previous studies cannot therefore be determined.

A more recent inheritance study is that by Bockelman *et al.* (1977). These workers crossed resistant genetic stocks of Tifang (C.I. 14373), C.I. 9819 and C.I. 7584 with primary trisomics in the cultivar Betzes. Chromosomal location of resistance genes was determined, a feature which had not been examined previously. Resistance in Tifang was conditioned by a single, incompletely dominant gene which was designated Rpt1a and occurred on chromosome 3. This finding verified those of Schaller (1955) and Khan and Boyd (1969a) with respect to number of genes conditioning net blotch resistance in Tifang and degree of dominance. C.I. 7584 contained a single resistance gene, Rpt3d, on chromosome 2. Degree of dominance could not be determined from the data. The writer was unable to locate previous work on inheritance of net blotch resistance in this genetic stock. Resistance in C.I. 9819 was conditioned by two dominant genes, Rpt1b and Rpt2c, located on chromosomes 3 and 5, respectively. Khan and Boyd (1969a) also identified two dominant genes ( $Pt_a$  and one undesignated) in the same genetic stock. Furthermore, both studies showed that the resistance gene in Tifang and one of the duplicate genes in C.I. 9819, were allelic. The relationship between  $Pt_1$  (Mode and Schaller, 1958) and  $Pt_a$  was not clarified by Bockelman *et al.* (1977).

It is noted that data in the above inheritance studies were analysed by examining segregation ratios of crosses, often up to  $F_3$  generation, and subsequently conducting chi-square ( $\chi^2$ ) tests for goodness of fit of observed with expected segregation ratios (Le Clerg

et al., 1962; Steel and Torrie, 1980). Such analysis cannot be conducted on a diallel if it consists of parental and  $F_1$  generations only. However, if the diallel is continued to later generations, the above procedure is appropriate. The diallel referred to earlier (Khan and Boyd, 1969a) was developed to the  $F_3$  generation and was therefore amenable to analysis by segregation ratios.

In summary, the above studies indicate that resistance to net blotch is conditioned by genes at several loci and there may be numerous alleles at each locus. It is suggested that testing of a wider range of net blotch resistant material against more *D. teres* isolates, may identify other loci and/or additional alleles at loci designated presently. Indeed, in any inheritance study of disease resistance, host resistance genes can be identified only in terms of available cultures of the pathogen (Hooker and Saxena, 1971). Furthermore, it should be realized that different sources of resistance appearing to have the same gene (allele) may subsequently be differentiated by use of more biotypes of the pathogen.

The single gene inheritance pattern demonstrated in genetic stocks such as Manchuria (C.I. 2330), Ming (C.I. 4797), Tifang (C.I. 4407-1) and C.L7584, is reported for resistance to many diseases of a very wide host range (Hooker and Saxena, 1971). From a plant breeding viewpoint, incorporation of a single net blotch resistance gene into an agronomically acceptable barley is relatively straightforward and rewarding (Nelson, 1973; Tekauz and Mills, 1974; Tekauz and Buchannon, 1977; Khan and Portmann, 1979). Backcross, pedigree and bulk population breeding methods are the most commonly employed for incorporation of resistance genes (Allard, 1960; Simmonds, 1979). If the resistance gene donor is of otherwise poor agronomic value, the backcross method is the logical choice as a breeding procedure. Agronomic value of net blotch

resistant genetic stocks has received little attention, although Buchannon and McDonald (1965) indicated that some stocks had desirable combinations of net blotch resistance and agronomic and quality characteristics.

#### 1.1.5 NITROGEN AND NET BLOTCH SYMPTOM EXPRESSION

Nutritional status of the host affects net blotch intensity.

For example, reductions in applied nitrogen have resulted in decreased net blotch intensity (Böning and Wallner, 1934; Singh, 1963; Piening, 1967). The findings indicate that decreased nitrogen application may improve resistance to net blotch.

In a series of pot experiments, Singh (1963) found that net blotch intensity on barleys irrigated with a complete nutrient solution, including 97 p.p.m. N ( $0.7\text{mg KNO}_3 \cdot 10^{-3} \text{m}^{-3}$ ) was markedly higher than on barleys receiving nutrient solution deficient solely in nitrogen. A similar result occurred when net blotch intensities were compared on barleys fed with nutrient solutions containing 46 p.p.m. N ( $0.13\text{mg NH}_4\text{NO}_3 \cdot 10^{-3} \text{m}^{-3}$ ) and 182 p.p.m. N ( $0.52\text{mg NH}_4\text{NO}_3 \cdot 10^{-3} \text{m}^{-3}$ ). Singh (1963) concluded that the results were due directly to the effect of nitrogen on host vigour.

Piening (1967) grew Gateway barley in potted soil samples in an investigation on the effect of nutrition on net blotch intensity. This was in contrast to the quartz sand potting medium used by Singh (1963). Soil samples were collected from three widely separated sites and differed considerably in available nitrogen, phosphorus and potassium. Ammonium nitrate, superphosphate (20%) and potassium chloride were added to the soils in all combinations to raise each nutrient "to that of the highest naturally occurring level".

Net blotch was measured on a scale of 0 to 5, the latter value

indicating greater than 50 per cent of total leaf area diseased. Individual leaves were rated and means estimated for all plants in a pot. Net blotch symptoms increased when nitrogen was added to Cooking Lake and Hanna soils, in the presence of constant levels of phosphorus and potassium. NPK ratios, in p.p.m. available nutrient, and associated disease ratings (in parentheses) before and after addition of nitrogen for Cooking Lake and Hanna soils, were 8:0:6 (1.75) to 60:0:6 (2.40) and 45:1:30 (1.83) to 60:5:30 (1.98), respectively. Only the increase in disease rating for Cooking Lake soil was significant ( $P \leq 0.01$ ). Nitrogen was not added to Edmonton soil samples. However, additions of phosphorus and/or potassium to this soil always decreased the disease rating. Results indicated that net blotch intensity was influenced by all three nutrients, with nitrogen having the greatest effect. Balance between the three nutrients may have also been important.

Increased disease intensities resulting from elevated nitrogen levels may also occur for other barley diseases. Examples include leaf scald (*Rhynchosporium secalis*) and powdery mildew (*Erysiphe graminis f.sp. hordei* Marchal) (Jenkyn and Griffiths, 1978; Walter and Ayres, 1980).

## 1.2 QUANTITATIVE GENETICS AND PLANT BREEDING

Quantitative genetics investigates the inheritance of those differences among individuals that are expressed in terms of degree rather than kind (Moll and Stuber, 1974; Falconer, 1981). Most economically important plant characters exhibit variability of a quantitative type.

Genetic variability is essential for success of a plant breeding programme. Quantitative genetic research measures this variability and delves into the action, interaction and linkage relationships of genes.

Phenotypic statistics (means, variances, covariances and measures of skewness and kurtosis) may be employed for this purpose (Breese, 1971; Mather and Jinks, 1971; Jinks, 1979).

#### 1.2.1 ANALYSIS OF VARIANCE

Experimental observations may be described by linear models which consist of several components (variables of classification), a mean and a random element (Steel and Torrie, 1980). The analysis of variance technique estimates variability due to each component in terms of sums of squares of deviation about an overall mean. Subtraction of all defined sources of variation from the total sum of squares leaves a residual "error".

Analysis of variance may be applied appropriately only when the data conform to the underlying assumptions. When one or more of the assumptions is not fulfilled, confounding of error variance occurs and significance levels and sensitivities of the F-test and t-test are affected.

Assumptions underlying the analysis of variance are:

1. Additivity of effects,
2. Independence of effects,
3. Normal distribution of effects, especially the error,
4. Homogeneity of effects across subsets of the data, especially the error.

Each has been described in detail including discussion of the consequences when it is not satisfied (Cochran, 1947; Eisenhart, 1947; Le Clerg *et al.*, 1962). Several assumptions may fail to hold in practice (Cochran, 1947) and indeed, it is unlikely that all assumptions are ever exactly realized. Data omission or data modification, such as transformation (logarithm, square root, arcsin), may enable some

assumptions to be fulfilled (Bartlett, 1947; Cochran, 1947; Eisenhart, 1947).

However, for most biological data, common disturbances resulting from failure of data to satisfy the above requirements are negligible (Steel and Torrie, 1980). The consequences are often acceptable too. For example, although nonadditivity in data results in confounding of pooled error variance, significance level of the F-test for all treatment means may be affected only slightly (Cochran, 1947; Steel and Torrie, 1980). Furthermore, the pooled error variance may estimate false significance levels for only certain, specific, comparisons of treatment means.

#### 1.2.2 PARTITIONING GENETIC VARIANCE

Measurements made on phenotypes may be split into genetic, environmental and interaction effects (Falconer, 1981). Fisher (1918) recognized that total genetic variance could be further subdivided into portions due to average effects of genes (additivity), interactions of allelic gene effects (dominance), and interactions of non-allelic gene effects (epistasis).

Estimation of these genetic variances requires use of appropriate mating and environmental designs. A mating design is a system of mating used to develop particular sets of progenies. Cockerham (1963) classified these designs as one, two, three, or four factor designs depending upon the number of ancestors per progeny over which control is exercised. The most extensively used mating systems are designs I, II, and III proposed by Comstock and Robinson (1948, 1952a) and the diallel cross first analysed with statistical genetic techniques by Sprague and Tatum (1942). All are two factor designs (Cockerham, 1963) and are appropriate for separation of additive and dominance variance (assuming epistasis is absent).

As regards self-fertilized plants, two mating designs have been employed primarily for genetic studies. These are:

1. experiments involving various generations of material arising from the cross of two pure lines where gene frequency is approximately 0.5, and
2. diallel mating system.

Both designs are widely documented (Mather, 1949; Hayman, 1954a, b, 1958a; Jinks, 1954, 1979; Griffing, 1956a, b; Whitehouse *et al.*, 1958; Matzinger, 1963).

Resemblance between relatives derived from a mating system is reflected in similarities of expression of quantitative traits (Falconer, 1981). This provides the basis for estimating genetic parameters, which are obtained from analysis of variance and regression applied to data collected on various parent/ progeny combinations. Resulting variance and covariance component estimates and parent-offspring regression coefficients are interpreted in view of their genetic expectations based on the particular genetic model assumed (Comstock and Robinson, 1952b; Kempthorne, 1957; Cockerham, 1963; Jinks, 1979; Falconer, 1981).

The diallel mating design has been utilized extensively to obtain genetic variance component estimates (Moll and Stuber, 1974). Recent developments in the design relating to ultimate genetic material under investigation, estimation methods and postulated underlying genetic mechanisms, are detailed by Hayman (1954a, b), Jinks (1954, 1979) and Griffing (1956a, b). The Hayman and Jinks genetic analyses deal with differences within a set of inbred lines and are generally regarded collectively as one method of diallel analysis (Hayman, 1960; Mather and Jinks, 1971, 1977).

This diallel analysis model operates under the following genetic assumptions (Hayman, 1954a; Mather and Jinks, 1971):

1. parental homozygosity,
2. normal diploid segregation,
3. no differences between reciprocal crosses,
4. independent action of non-allelic genes,
5. no multiple alleles,
6. uncorrelated gene distributions.

Assumptions 1 to 4 are general to all biometrical analyses (Hill, 1964) and at least the first three may be safely assumed in appropriate circumstances (Johnson, 1963). As regards assumptions 1 and 2, barley is a diploid species and four of the five genotypes studied presently are standard New Zealand cultivars which were taken from carefully monitored stocks of this strongly inbreeding species. Satisfaction of assumption 4 is particularly difficult as epistasis occurs widely and maybe as important as additivity or dominance in genetic variation (Hayman, 1958b). However, depending on the type of epistasis, there may be very little adverse effect on estimates of genetic parameters (Hayman, 1954a). Assumptions 5 and 6 are "justified on the basis of probable unimportance" (Johnson, 1963). The effects of epistasis and correlated gene distributions on the presently adopted analysis technique are noted shortly.

A diallel mating system involves the crossing of  $p$  parent genotypes in various combinations (Hayman, 1954a; Griffing, 1956a, b; Hinkelmann, 1977). Measurements on a full diallel of  $p^2$  combinations may be represented in a diallel table and divided into three portions:

1.  $p$  parents,
2. one set of  $p(p-1)/2$   $F_1$ 's,
3. reciprocal set of  $F_1$ 's.

Hence, there are four possible diallel combinations:

1. parents,  $F_1$ 's and reciprocals ( $p^2$ ),
2. parents and  $F_1$ 's ( $p(p + 1)/2$  genotypes) (half diallel),
3.  $F_1$ 's and reciprocals ( $p(p - 1)$  genotypes),
4. one set of  $F_1$ 's only ( $p(p - 1)/2$  genotypes) (Griffing, 1956a).

There are two well known and widely used approaches to analysis of diallel cross data - the method proposed by Hayman (1954a, 1960) and Mather and Jinks (1971); and the combining ability analysis elucidated by Griffing (1956a, b). The analysis of Kempthorne (1956) is less widely documented and is related to Griffing's combining abilities analysis (Hayman, 1960).

Hayman and Jinks, in a series of papers since 1953 and more recently (Mather and Jinks, 1971, 1977), developed an analysis of diallel cross data based on variance and covariance estimates of a sample of parents and their  $F_1$ 's. The most important feature of their analysis is regression of  $W_r$  on  $V_r$  (covariances and variances of parental arrays respectively) which provides information on:

1. average degree of dominance and hence dominance type (partial, complete, or overdominance),
2. relative proportion of dominant and recessive genes in the parents,
3. genetical diversity among the parents.

These features may be interpreted from the  $W_r/V_r$  graph only when a simple additive-dominance model of gene action provides an adequate description of the data (Mather and Jinks, 1971). When this is so, the regression line is linear and its slope does not depart significantly from one. However, in the presence of non-allelic interactions (epistasis) and/or correlated gene distributions, this linear relationship is lost. Certain types of disturbance cause graphical distortion in

characteristic ways which may thus permit their detection (Mather, 1967; Coughtrey and Mather, 1970). For example, complementary interaction bends the  $W_r/V_r$  line concavely upwards, while duplicate interaction has the reverse effect (Mather and Jinks, 1971). Remedial procedures for disturbances are presented in Materials and Methods.

Diallel cross data, conforming to the additive-dominance model of Mather and Jinks (1971), may be analysed further to estimate four genetic components:

$$\begin{aligned} D &= \sum 4uvd^2 & H_1 &= \sum 4uvh^2 \\ H_2 &= \sum 16u^2v^2h^2 & F &= \sum 8uv(u-v)dh. \end{aligned}$$

D is the additive genetic component of variation;  $H_1$  and  $H_2$  measure dominance components of variation; and F accounts for non-independent contributions of additive (d) and dominance (h) effects when gene frequencies (u and v) are unequal.  $\Sigma$  signifies a sum over all segregating loci.

Equations which estimate D,  $H_1$ ,  $H_2$  and F from one or more combinations of  $V_p$  (variance of parents entering the diallel),  $\bar{V}_r$  (mean variance of parental arrays),  $\bar{W}_r$  (mean of the covariances between the family means within each array and the phenotypes of their respective non-recurrent parents) and  $V_r^-$  (variance of parental array means), are presented in Section 2.8 and Table 2.3.

Additive genetic variance ( $\sigma_A^2$ ) and dominance genetic variance ( $\sigma_D^2$ ) may be estimated from the above genetic parameters when it is assumed that the parameters apply to a random mating base population of which the parents used in the diallel are a sample. With the assumption,  $u = v = 0.5$  (and assuming no epistasis), D and  $H_2$  become  $D_R$  and  $H_R$  where R equals random mating. Equating these with Falconer's (1981) equations for additive and dominance variance, we obtain

$$\sigma_A^2 = \frac{1}{2}D \quad \text{and} \quad \sigma_D^2 = \frac{1}{4}H_2 (H_R)$$

These estimates show the proportion of genetic variation attributable to additive and dominance genetic variances. Heritabilities (see Section 1.2.4) may be estimated, although equations for estimating heritabilities in terms of  $D$ ,  $H_1$ ,  $H_2$  and  $F$  already exist (Mather and Jinks, 1971).

In Griffing's (1956a, b) method, genetic variance between crosses is partitioned into two components. The first is variance of general combining abilities ( $\sigma_{gca}^2$ ) which contains only additive genetic variance and epistatic interactions of additive x additive, ... types. The second component is variance of specific combining abilities ( $\sigma_{sca}^2$ ) which involves dominance genetic variance and all other types of epistatic interaction. Sprague and Tatum (1942) first defined general combining ability as "average performance of a line in hybrid combination". Specific combining ability was "those cases in which certain combinations do relatively better or worse than would be expected on the basis of the average performance of the lines involved." Estimates of  $\sigma_{gca}^2$  and  $\sigma_{sca}^2$  are derived from covariances of full- and half-sibs (Kempthorne, 1957). Details follow.

Formulae for estimation of covariances of full- and half-sibs are:

$$\begin{aligned} \text{cov (full-sibs)} &= \left(\frac{1+F}{2}\right)\sigma_A^2 + \left(\frac{1+F}{2}\right)\sigma_D^2 + \left(\frac{1+F}{2}\right)^2\sigma_{AA}^2 \\ &\quad + \left(\frac{1+F}{2}\right)^3\sigma_{AD}^2 + \left(\frac{1+F}{2}\right)^4\sigma_{AAA}^2 + \dots \\ \text{cov (half-sibs)} &= \left(\frac{1+F}{4}\right)\sigma_A^2 + \left(\frac{1+F}{4}\right)^2\sigma_{AA}^2 + \left(\frac{1+F}{4}\right)^3\sigma_{AAA}^2 + \dots \end{aligned}$$

where  $\sigma_A^2$  = additive genetic variance of a random mating inferential population.

$\sigma_D^2$  = dominance genetic variance of a random mating inferential population.

$\sigma_{AA}^2$  = epistatic interaction of additive x additive type.

F = inbreeding coefficient from a random mating  
inferential population.

Variances of g.c.a. ( $\sigma_{gca}^2$ ) and s.c.a. ( $\sigma_{sca}^2$ ) may then be calculated as (Kempthorne, 1957):

$\sigma_{gca}^2$  = cov (half-sibs) (because half-sib sets are being compared)

$\sigma_{sca}^2$  = cov (full-sibs) - 2 cov (half-sibs).

Diallels have been used extensively in barley breeding investigations (Johnson and Aksel, 1959, 1964; Aksel and Johnson, 1961; Upadhyaya and Rasmusson, 1967; Gulati *et al.*, 1969; Riggs and Hayter, 1972, 1973, 1975; Virk and Verma, 1973; Briggs, 1974; Chaudhary *et al.*, 1974, 1977a, b; Grafius and Okoli, 1974; Habgood, 1974; Hayes and Paroda, 1974; Hayter and Giles, 1974; Jana, 1975, 1976; Nasr and Khayrallah, 1976; Greenberg, 1977; Nikitenko *et al.*, 1977, 1979; Surma, 1978; Fejer and Fedak, 1979; Khalifa, 1979).

Examination of these studies shows no consistent trends in type of genetic variation identified for specific characters. However, as a generalization additive, dominance, and epistatic genetic variances are reported for plant height, ear length, harvest index, grain number per ear, 1,000-kernel weight and grain yield. Epistasis occurs most often for plant height and 1,000-kernel weight. Solely additive and dominance genetic variances are documented for heading date, reproductive tillering and spikelet number per ear. Although dominance is reported often, there is little classification of its degree (partial, complete, or overdominance).

Diallel analysis has been used very little in studies on resistance to diseases of barley. As regards net blotch, the only study located was that by Khan and Boyd (1969a) in Western Australia. These workers

crossed six genetic stocks (five net blotch resistant and one susceptible) in half-diallel fashion. Segregation ratios based on net blotch reaction were examined to  $F_3$  generation and used to estimate number of genes conditioning net blotch resistance in the parents. A Mather and Jinks (1971) analysis was not conducted. Further details and results of the study were presented in Section 1.1.4.

A detailed study on inheritance of resistance to another disease - leaf scald (*Rhynchosporium secalis*) - was conducted in the United Kingdom by Habgood (1974) using a Mather and Jinks (1971) analysis. A half-diallel set of crosses was made from six spring barley cultivars (Proctor, Ruby, Zephyr, Cambrinus, Inis and Old Cornish) which exhibited a range of resistance to *R. secalis*.  $F_2$  populations were raised as well as  $F_3$  populations from the diallel subset involving crosses between Zephyr, Proctor and Inis. Intensity of leaf scald was assessed on  $F_2$  and  $F_3$  populations in field trials. Results of  $F_2$  studies indicated that a simple additive-dominance genetic model could account for resistance to *R. secalis*. Significant additive gene effects occurred and non-additive genetic variation was attributed to dominance effects. Examination of genetic components of variation (D,  $H_1$ ,  $H_2$ , F) showed that  $H_2$  was less than  $H_1$  which indicated unequal allele frequencies between parents. Parents contained more dominant than recessive alleles (F positive) and average degree of dominance ( $\sqrt{H_1/D}$ ) was 0.72, indicating partial dominance. In  $F_3$  material, transgressive segregation occurred in all cross combinations. Genetic components were unable to be estimated. Habgood (1974) concluded that resistance to *R. secalis* was complex in inheritance.

In summary then, diallel studies on barley show a preponderance of additive and dominance genetic variance but their relative importance is sometimes difficult to ascertain. Epistasis, although often identified

as a cause of distortion of the  $F_1 W_r/V_r$  graph (Jana, 1975; Riggs and Hayter, 1973; Surma, 1978) has been apparently less important.

### 1.2.3 GENOTYPE - ENVIRONMENT INTERACTION

When conducting evaluation trials over several environments, performance of genotypes relative to each other can vary according to environment. Such differential ranking of genotypes between environments is termed genotype-environment interaction (Breese, 1971; Moll and Stuber, 1974; Hill, 1975).

These interactions are important since they can cause considerable upward bias in estimates of genetic components (Comstock and Moll, 1963; Matzinger, 1963), which may lead to erroneous choices of breeding methods. In considering this aspect, Gardner (1963) stressed the need to estimate relative magnitudes of additive genetic and dominance genetic  $\times$  environment interaction variances before choosing which component of genetic variation could be exploited in a breeding programme.

Genotype - environment interactions may be estimated by two main approaches, as follows:

1. purely statistical (Yates and Cochran, 1938; Finlay and Wilkinson, 1963; Eberhart and Russell, 1966),
2. biometrical genetics (Mather and Jones, 1958; Bucio Alanis, 1966; Perkins and Jinks, 1968a, b; Freeman and Perkins, 1971; Mather and Jinks, 1971, 1977; Jinks, 1979).

The most comprehensive barley diallel analyses conducted across environments are those of Paroda and Hayes (1971) and Surma (1978). In their study of ear emergence, Paroda and Hayes (1971) identified significant additive and dominance components in eight environments. Additive components interacted more with environment than dominance components and both interaction types operated in parental and  $F_1$

generations. Surma (1978) investigated seven characters (tillering, ears per plant, ear length, spikelet number per ear, 1,000-kernel weight, grain yield and total protein content in the dry kernel weight) in six environments and found significant interactions of environment with additive and dominance components for all characters.

Other studies have also shown significant genotype - environment interactions (Rasmusson and Lambert, 1961; Finlay and Wilkinson, 1963; Yap and Harvey, 1972; Riggs and Hayter, 1973; Hayes and Paroda, 1974; Habgood, 1977; Johnson and Whittington, 1977; Hadjichristodoulou and Della, 1978; McGuire *et al.*, 1979).

Little information is available on genotype - environment interactions for diseases of barley. Studies involving statistical analyses for this character were not located. As regards net blotch of barley, several findings indicate that genotype and/or pathogen - environment interactions may be important. These are:

1. Differential reactions of genetic stocks in environments throughout the world. In California, Schaller and Wiebe (1952) found that most of their highly resistant material originated from Manchuria. In contrast, Canadian studies by Buchannon and McDonald (1965) identified resistant stocks which originated most frequently from Ethiopia. Similar germplasm was used in the two investigations. These studies have been discussed elsewhere (see Section 1.1.4) and do not need expounding here. Net blotch screening studies were also conducted in Morocco (Caddel and Wilcoxson, 1975) and Western Australia (Khan and Boyd, 1969b; Khan, 1971) and provided further indications of genotype - environment interaction.
2. Variability in host reaction to one isolate under controlled environmental changes. Khan and Boyd (1969c) examined host

reactions of two Ethiopian (C.I. 5791 and C.I. 9819) and four Manchurian (Harbin, Manchuria, Tifang and Ming) resistant genetic stocks and one susceptible stock (Dampier W.45) to a single *D. teres* isolate under a range of environmental conditions. Effects on host resistance of changes in temperature (degree and timing around inoculation), light intensity and duration, age of host and pathogen and conidial concentrations, were studied. The study was also mentioned in Section 1.1.4. Ethiopian stocks consistently exhibited resistant reactions whereas Manchurian stocks gave variable reactions under different conditions tested. A genotype - environment interaction was clearly shown. Manchuria was most sensitive to environmental change. Variations in pre-inoculation temperature and light intensity during incubation period had the greatest effect on Manchuria's reaction. Resistance in this stock was enhanced by a high pre-inoculation temperature ( $36^{\circ}\text{C}$ ) or high light intensity ( $38.75 \times 10^4$  lux for 12 hours per day) during the incubation period. Expression of resistance increased with age for the four Manchurian hosts. Manchuria gave more resistant reactions to inoculum prepared from 10-day-old cultures than inoculum obtained from older cultures. Host resistance decreased as Manchuria was inoculated with increasing concentrations of conidial suspension.

#### 1.2.4 HERITABILITY

Heritability is defined as the proportion of phenotypic variability which is consistently due to heredity, the remainder being due to environmental influence (Allard, 1960).

Estimates may be determined by three main techniques: parent-offspring regressions; variance components from phenotypic analysis of

variance; and use of genetically uniform populations (Warner, 1952; Hanson, 1963; Falconer, 1981). As regards variance components, several types of heritability may be estimated depending on the genetic variances comprising numerator and denominator of the heritability ratio. Narrow and broadsense heritabilities are estimated using additive and total genetic variances respectively, as numerators. Modifications to measurement of phenotypic variation (denominator) are described by Gordon *et al.* (1972).

Equations estimating narrow and broadsense heritabilities using genetical parameters -  $D$ ,  $H_1$ ,  $H_2$ ,  $F$  (Mather and Jinks, 1971) - have been employed in several barley diallel studies (Riggs and Hayter, 1975; Greenberg, 1977; Khalifa, 1979).

Heritability estimates for barley (narrow and/or broadsense) reported in these studies plus those previously cited, include heading date (0.83), plant height (0.69 to 0.92), 1,000-kernel weight (0.43 to 0.79), ears per plant (0.36 to 0.91), grain number per ear (0.31), grain yield (0.10 to 0.49) and harvest index (0.62 to 0.81). Heritability estimates given by Habgood (1974) for resistance to *R. secalis* were 0.37 (narrow sense) and 0.58 (broad sense) calculated on a plot basis. Estimates on a single plant basis were 0.16 (narrow sense) and 0.26 (broad sense).

Comparison of estimates for a particular character obtained in different studies is of "doubtful utility" (Robinson, 1963). This is because heritability depends so much on plot size, plant density, replication and the genetic and environmental diversity sampled in the experiment.

### 1.2.5 HETEROSIS

Heterosis is the superiority of an  $F_1$  hybrid over its better parent (Jinks, 1979) or mid-parent value (Moll and Stuber, 1974; Sinha and Khanna, 1975). The former definition has also been termed heterobeltiosis (Virk and Verma, 1973). Superiority may be expressed as a magnitude or percentage. The reverse phenomenon is inbreeding depression (Moll and Stuber, 1974; Falconer, 1981).

Formulae for heterosis and heterobeltiosis are presented here in genetic terms (Falconer, 1981). Designations for genotypic frequencies and values are those of Mather and Jinks (1971). Parental means of two random mating populations,  $\bar{P}_1$  and  $\bar{P}_2$ , may be represented as:

$$\begin{aligned}\bar{P}_1 &= d(u - v) + 2uvh \\ \bar{P}_2 &= d(u - v - 2y) + 2h(uv + y(u - v) - y^2)\end{aligned}$$

where  $u$  and  $v$  are allele frequencies at a single locus in one parental population;  $d$  is departure of one of a pair of corresponding homozygotes from their mid-point;  $h$  measures the departure of the heterozygote from the mid-point; and  $y$  is difference in gene frequency between the two populations such that  $y = u - u' = v' - v$  ( $u'$  and  $v'$  are corresponding allele frequencies in the second parental population).

Mid-parent value (MP) is

$$\begin{aligned}MP &= \frac{1}{2}(\bar{P}_1 + \bar{P}_2) \\ &= d(u - v - 2y) + 2h(uv + y(u - v) - y^2)\end{aligned}$$

Mean genotypic value of  $F_1$  from intercrossing randomly selected individuals of the parental populations is

$$\begin{aligned}F_1 &= d(u - v - y) + h(2uv + y(u - v)) \\ \text{Heterosis} &= \bar{F}_1 - MP = hy^2 \\ \text{Heterobeltiosis} &= \bar{F}_1 - \bar{P}_1 = -y(d + h(u - v))\end{aligned}$$

From above, heterosis depends for its occurrence on dominance (h) while heterobeltiosis depends on additivity (d) in addition to dominance. For heterosis, the square of the difference in gene frequency (y) between the two populations (or lines) determines the amount of heterosis expected from a cross involving these populations.

In barley, heterosis (and/or heterobeltiosis) is well documented (Grafius, 1959; Carleton and Foote, 1968; Virk and Verma, 1973; Tseng and Poehlman, 1974; Gymer, 1976, 1977; Johnson and Whittington, 1977; Riggs and Kirby, 1978; Spunar, 1978; Done and Macer, 1979; Khalifa, 1979). Estimates have been reported for grain yield (- 17.0 to + 29.8 per cent, although mostly positive), 1,000-kernel weight (+ 4.7 to + 13.0 per cent), tillers per plant (- 2.0 to + 21.0 per cent), grain number per ear (+ 11.1 per cent) and harvest index (+ 7.22 per cent). Of particular interest to many breeders is heterosis of hybrids between two-row and six-row ear types (examined in most of the above studies) as each has different desirable grain characteristics (Riggs and Kirby, 1978).

## CHAPTER 2

## CHAPTER 2.

### MATERIALS AND METHODS

#### 2.1 GENOTYPES

A diallel mating system consisting of five parents and one set of  $F_1$ 's was used in the investigation (giving 15 genotypes). Parents were a random sample from the collection of barley cultivars selected on the basis of their degree of resistance to net blotch under Manawatu field and controlled environmental conditions (McEwan, pers. comm.; Douglas, 1979). Material ranged from low to high net blotch resistance. Genotypes, approximately in increasing order of resistance were: Zephyr, Hassan, Mata, Kaniere and Manchuria. Apart from Manchuria, all cultivars are 2-row and grown commercially in New Zealand. Manchuria is a 6-row type.

All  $F_1$  seed was produced by hand crossing under glasshouse conditions.

In addition to the diallel material, all parents were grown as control (disease-free) plants. Their management during the study is described in Section 2.4.

#### 2.2 EXPERIMENTAL DESIGN

Two environments were used, each in a controlled room.

A randomised complete block design with four blocks per room was employed. Both diallel and control plant material were randomised within blocks. Single experimental units (plots) consisted of two plants in individual pots giving a total of 160 experimental plants per room. Two guard rows were established in the direction of blocking (front to back of room). All pots were placed on steel trolleys to

facilitate easy movement.

A random-effects model was assumed for all tests of significance (Eisenhart, 1947; Crump, 1951). The population, from which parents were assumed to be a random sample, was defined in Section 2.1. Environments were considered random samples from two strata of environments, differing on the basis of large nitrogen concentrations in the nutrient solution.

### 2.3 CONTROLLED ROOM ENVIRONMENT

A light intensity of  $170 \text{ Wm}^{-2}$  Photosynthetically Active Radiation ( $0.4 - 0.7 \times 10^{-6} \text{ m}$  waveband from Sylvania "Metal-arc" and Phillips tungsten iodide lamps) was used in both rooms. Light duration was 14 hours and there was an instantaneous light/dark conversion.

An optimum temperature regime, favouring both pathogen (*Drechslera teres*) and host development, was selected following an extensive literature review (Khan and Boyd, 1969d; Shipton et al., 1973; Thorne, 1973). Maximum and minimum temperatures were  $22^{\circ}\text{C}$  (day) and  $16^{\circ}\text{C}$  (night) respectively, with a variation of less than  $\pm 0.5^{\circ}\text{C}$ . A 120 minute changeover time was used.

Relative humidity (R.H.) was maintained initially at  $70\% \pm 5\%$ . At seven weeks, humidity in both rooms was elevated permanently to  $95\% \pm 5\%$  to encourage more favourable net blotch symptom expression.

Velocity of air currents within the rooms ( $0.3 - 0.5 \text{ m sec}^{-1}$  at top of plant canopy) was expected to be sufficient for conidial spread throughout the plant populations (Hampton, pers. comm.).

### 2.4 HANDLING OF PLANT MATERIAL AND NUTRIENT SUPPLY

All seed intended for sowing was placed in glass petri dishes containing filter paper moistened with  $0.25\% \text{ KNO}_3$ . Dishes were then

placed in an incubator at 15°C/dark (16 hr.) and 20°C/light (8 hr.) for 50 hours to break any dormancy. Parent seed for control plants had been treated beforehand with a systemic fungicide ("Vitaflo 200" - a mixture of carboxin/thiram) mixed at the rate of 1 part "Vitaflo 200" : six parts water.

Plants were grown in  $1.2 \times 10^{-3} \text{ m}^3$  plastic pots filled with a potting mix composed of 70 per cent gravel ( $2 \times 10^{-3} \text{ m}^3$ ), 15 per cent peat and 15 per cent vermiculite. This is a standard North Carolina State University preparation. Six days after sowing, seedlings were thinned to one per pot, using vigour and evenness of establishment as criteria.

Two levels of applied nitrogen ( $20 \text{ mg } 10^{-3} \text{ m}^{-3}$  (20 p.p.m.) and  $105 \text{ mg } 10^{-3} \text{ m}^{-3}$ ) of a modified, half-strength, Hoagland's nutrient solution (Brooking, 1972) were used (Table 2.1). These are henceforth termed low and high nitrogen, respectively. One level was used per room.

In the low nitrogen environment, each pot received  $50 \times 10^{-6} \text{ m}^3$  of the appropriate nutrient solution, twice daily. For high nitrogen environment, pots were given the same rate as the low nitrogen environment until Day 56 (approximately heading date for most genotypes), but thereafter received  $50 \times 10^{-6} \text{ m}^3$  applications four times per day. High nitrogen plants were much larger at heading than those in the low nitrogen environment and therefore application of nutrient solution was increased to prevent water stress.

All pots were flushed fortnightly with  $100 \times 10^{-6} \text{ m}^3$  water to remove excess ions from the potting medium.

Control plants received a weekly foliar application of mancozeb (a fungicide protectant) commencing at five weeks. This was necessary since the seed treatment chemical, "Vitaflo 200", often loses its disease protection properties within the first six weeks of plant development (de Borst, pers. comm.; Hampton, pers. comm.). The treatment was

Table 2.1 High and low nitrogen nutrient solutions ( $\text{mg } 10^{-3} \text{ m}^{-3}$ )<sup>†</sup>

Nutrient	High Nitrogen	Low Nitrogen
$\text{Ca}(\text{NO}_3)_2 \cdot 4\text{H}_2\text{O}$	590.38	126.36
$\text{CaSO}_4 \cdot 2\text{H}_2\text{O}$	-	251.48
Sequestrene 330 (10% DTPA NaFe)	20.80	41.60
$\text{KH}_2\text{PO}_4$	68.04	136.08
KCl	3.15	166.40
$\text{KNO}_3$	252.78	36.08
$\text{MgSO}_4$	246.48	492.96
MICRO $\text{H}_3\text{BO}_3$	1.430	2.860
$\text{MnCl}_2 \cdot 4\text{H}_2\text{O}$	0.905	1.810
$\text{ZnSO}_4 \cdot 7\text{H}_2\text{O}$	0.110	0.220
$\text{CuSO}_4 \cdot 5\text{H}_2\text{O}$	0.040	0.080
$\text{NaMoO}_4 \cdot 2\text{H}_2\text{O}$	0.013	0.027

<sup>†</sup> Stock solutions (in  $\text{g } 10^{-3} \text{ m}^{-3}$ ), together with dilution ratios when made up into usable solution, are presented in Appendix 1.

continued until approximately a fortnight after heading. Mancozeb was mixed at the rate of  $2\text{g } 10^{-3}\text{ m}^{-3}$  water and applied until runoff.

An outbreak of powdery mildew (*Erysiphe graminis* f.sp. *hordei* Marchal) in the thirteenth week of the study was controlled using "Benlate" (a.i. = 50% w/w benomyl) mixed at the rate of 3g wettable powder per  $10^{-3}\text{ m}^3$  water.

Drying-off was commenced when most plants had reached the physiological state of at least 75 per cent senescent tissue. Nutrient solution was applied at half the previous rates for three days and then applications ceased. Plants in the low nitrogen environment were harvested on Day 112 and those in the high nitrogen environment on Day 123.

## 2.5 DISEASE ESTABLISHMENT AND MANAGEMENT

### 2.5.1 INOCULUM PREPARATION

An isolate of *Drechslera teres*, from a Manawatu sample of infected Zephyr barley, was grown and subcultured on 10% V-8 agar at  $20^{\circ}\text{C}$  under continuous light, such conditions promoting abundant sporulation (Douglas, 1979). At seven days, inoculum (9,000 conidia per  $10^{-6}\text{ m}^3$ ) was prepared and a sticker ("Tween 80") added at the rate of one drop per  $50 \times 10^{-6}\text{ m}^3$ . Volumes of  $200 \times 10^{-6}\text{ m}^3$  were prepared for each inoculation run per room. Average conidial germination on Water Agar (WA) after 48 hours was 60 per cent.

### 2.5.2 INOCULATION PROCEDURE

Seedlings were first inoculated at 14 days, when the second leaf had partially emerged. A small sprayer attached to an air pump (adjusted to deliver 138 kPa air pressure) was used and seedlings were sprayed uniformly until runoff. Control plants were removed just before inoculation and then returned to their original trolley positions.

Individual plants were enclosed in plastic polythene bags (270 x 600mm) containing saturated paper towels for 48 hours to provide 95 to 100% relative humidity and encourage a net blotch outbreak on relatively susceptible genetic stocks. This was successful in the high nitrogen environment only. Individually bagged plants are shown in Plate 2.1.

From the primary net blotch lesions, it was endeavoured to develop secondary cycles of the disease by provision of 72 hour periods of high humidity (Wenham, pers. comm.). Due to rapid increases in plant height and vegetative mass, plastic bags could not be used again and high humidity was therefore provided by overhead misting. Discontinued use of bags also enabled inoculum transfer between plants. The longer humidity period allowed for the additional process involved in secondary infection of conidial production from primary lesions. Initial 72 hour periods consisted of ten second mistings every 25 minutes. This combination ensured adequate leaf moisture.

Secondary cycles of infection were not detected, as evidenced by no noticeable increases in net blotch prevalence and intensity. Inoculum was therefore introduced artificially (as originally) into both environments. Ten inoculations were conducted at approximately weekly intervals until three weeks after heading. The misting period after each inoculation was reduced to 48 hours as this was adequate for only conidial germination and germ tube penetration (Douglas, 1979).

With the 48 hour humidity periods, involving ten second mists every 25 minutes, net blotch development was relatively slow. Several new frequency and duration combinations of misting were therefore used in an attempt to improve development of the disease. There were five second mists every two minutes/30 minutes and 20 second mists every five minutes. It was suspected that low disease development resulted from removal of conidia and/or drying of leaf surfaces with a consequent



Plate 2.1

Individually bagged plants in high nitrogen environment, following first inoculation.

reduction in conidial germination. However, these hypotheses were not examined. Maintenance of a constantly moist leaf surface with no runoff was the expected optimum humidity level for conidial germination and the above mentioned misting combinations were used in an attempt to identify this level. Presumably at such a level, disease development would progress favourably. However, a suitable combination was not found as marked increases in net blotch still did not occur.

For the final two inoculations, single large plastic bags were placed over all plants on each trolley. Misting was not used. Increase in expression of net blotch was superior to that found using misting. However, differences between the two methods were not quantified.

### 2.5.3 PETRI DISH STUDIES

In view of the disappointing development of net blotch in the high nitrogen environment, particularly in the early to middle stages of the study, an alternative experiment incorporating use of leaf sections, was conducted. The technique was similar to that used to investigate *Marssonina* species on poplar leaf discs (Spiers, 1978).

Three leaf sections, approximately  $2 \times 10^{-2}$  m long, were removed from the second and/or third healthy leaves of selected ear-producing tillers within each plot and placed in wells formed in 2% Water Agar (WA). Sections were inoculated individually with  $0.3 - 0.4 \times 10^{-6}$  m<sup>3</sup> inoculum, which had a concentration of 100 conidia per  $10^{-6}$  m<sup>3</sup> (10,000 conidia per  $10^{-6}$  m<sup>3</sup> diluted 100 fold). Following inoculation, all dishes were incubated at 20°C under continuous light (2 x 15W cool, white fluorescent tubes) for five days. The above procedure was repeated for both low and high nitrogen environments.

## 2.6 MEASUREMENTS

### 2.6.1 NET BLOTCH SEVERITY AND INFECTION TYPE

Two methods of assessment of intensity (severity) on intact plant tissue were used:

1. Intensity was assessed on the top and bottom halves of each plant canopy using a scale of 1 (least intensity) to 10. Scores corresponded approximately to increments of 10 per cent infection.

Plant canopies were halved for assessment as initially net blotch expression on relatively susceptible genetic stocks was higher, or occurred only on the lower foliage. This was perhaps due to higher humidity towards the base of plants and longer plant exposure to *D.teres* as a consequence of age. When assessments were conducted (Day 89), intensity on higher foliage had improved and it was therefore decided to assess net blotch on both plant canopy halves.

2. Intensity based on percentage leaf area infected, was assessed on the flag leaf (leaf number = 1) and second leaf of five randomly selected tillers per plant. Standard leaf area diagrams (Hampton and Arnst, 1978) were used (Figure 2.1). All percentages between 0 and 100% may be assessed using appropriate combinations of the diagrams.

Another assessment made on intact plant tissue was infection type predominating on the foliage of individual plants. The following scale (Khan and Boyd, 1969b) was used:

- no observable infection (0);
- pin-point lesions with no chlorosis (1);
- slightly elongated dark brown lesions without chlorosis (2);
- restricted longitudinal dark brown lesions with slight chlorosis of adjacent areas (3);

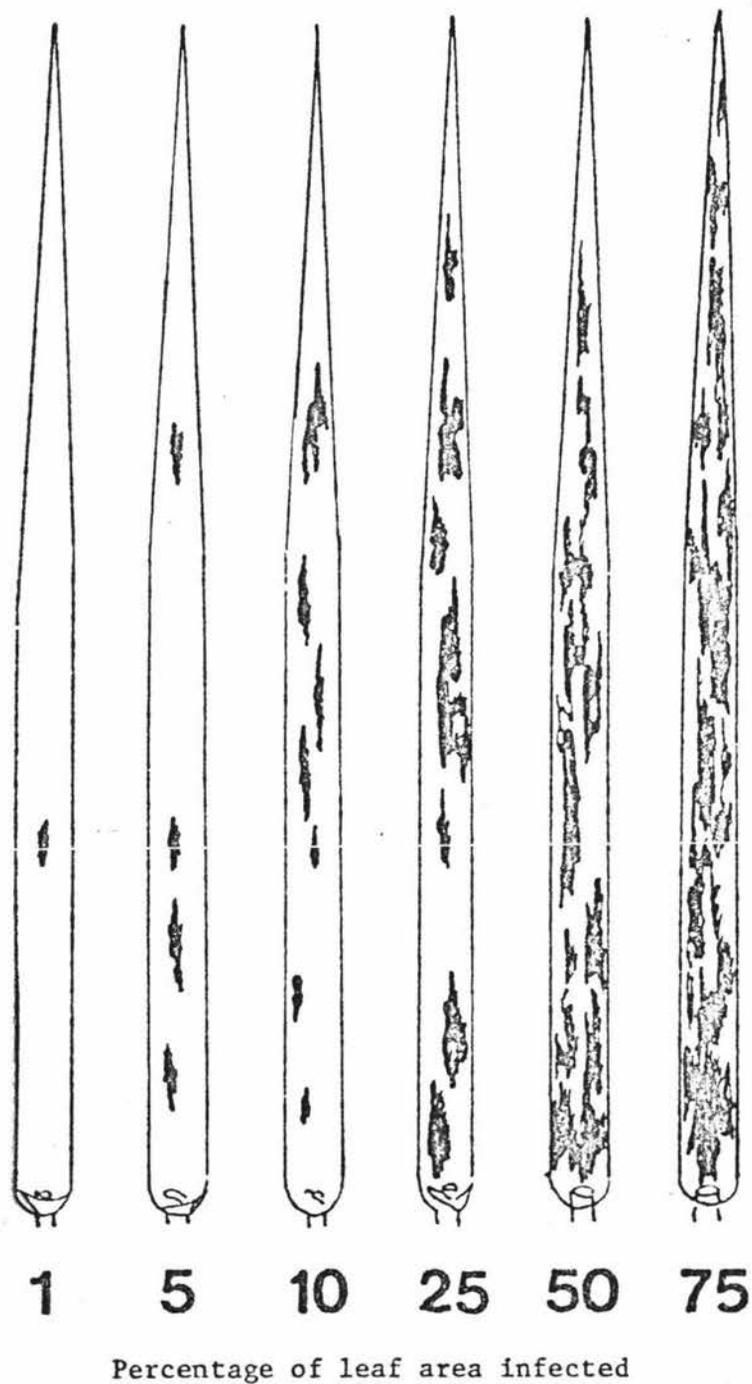


Figure 2.1 Standard leaf area diagrams used to assess net blotch intensity (after Hampton and Arnst, 1978).

- brown elongated lesions, criss-crossed with dark-coloured, net-like venation, surrounded by marked chlorotic areas(4).

All net blotch data on intact plant tissue were collected at Day 89 of experiment.

In the petri-dish studies, intensity was assessed in multiples of 5 per cent leaf section area infected. Leaf area diagrams were not used as available diagrams pertained to whole leaves only.

#### 2.6.2 OTHER CHARACTERS

Data were also collected on the following characters:

1. Heading Date: This was number of days from sowing until emergence of first awns (< 1.5cm above the flag leaf).

2. Plant Height: Distance in centimetres from potting mix surface to tip of highest head, excluding awns, was measured.

3. Fertile Tillers: All tillers with grain were counted for each plant.

4. Spikelet Number per Ear: Three randomly selected, well-developed ears per plant were used for assessment.

5. Grain Number per Ear: In low nitrogen environment, counts were made on three to five randomly selected, well-developed ears per plant. Measurements were conducted on four ears per plant in high nitrogen environment. Ears used for spikelet number determinations were not necessarily used here due to random sampling of ears.

6. Grain Yield: All grain producing ears per plant were threshed using a machine thresher. Yields were recorded on a per plant basis.

7. Kernel Weight: Weight in grams of a random sample of 100 kernels was measured. Where insufficient kernels were available, weight of the reduced kernel sample was multiplied by 100 and divided by kernel number in the sample, to determine the equivalent kernel weight.

8. Moisture Content: A random sample of 20 to 30 kernels was oven dried at 105°C for 24 hours (van Wyk, 1978). Moisture fraction (%) was expressed on a wet-weight basis.

9. Physiological Brown Spot: The intensity of a physiological brown spot, similar to that reported by Clark *et al.* (1979), was assessed on the second or third leaves of three (low nitrogen)/four (high nitrogen) randomly selected tillers per plot. Assessments were made at Days 70 (high nitrogen) and 72 (low nitrogen) of the study using standard leaf area diagrams (Figure 2.2). Although the diagrams were prepared originally for powdery mildew on cereals (James, 1971), they were appropriate here as they showed symptom shapes and leaf distributions very similar to those of the spotting phenomenon.

## 2.7 DATA HANDLING AND PRELIMINARY ANALYSIS

In an initial examination of data, SPSS (Statistical Package for the Social Sciences) was used (Nie *et al.*, 1975). Scattergrams of grain yield and 100-kernel weight against moisture content (%) were computed. These were used to decide whether adjustment of the former two variables to a constant moisture content was necessary.

Regression coefficients of determination for grain yield and 100-kernel weight against moisture content, in low and high nitrogen environments, are given in Table 2.2a (diallel material) and 2.2b (material receiving fungicide treatment). There was no evidence to suggest that there was a linear relationship between the variables. Therefore, adjustments of grain yield and 100-kernel weight measurements to constant moisture contents were not conducted.

All data were subsequently converted to plot means using Fortran programmes written by the author. Net blotch infection type data were not analysed further as they were measured on an ordinal scale

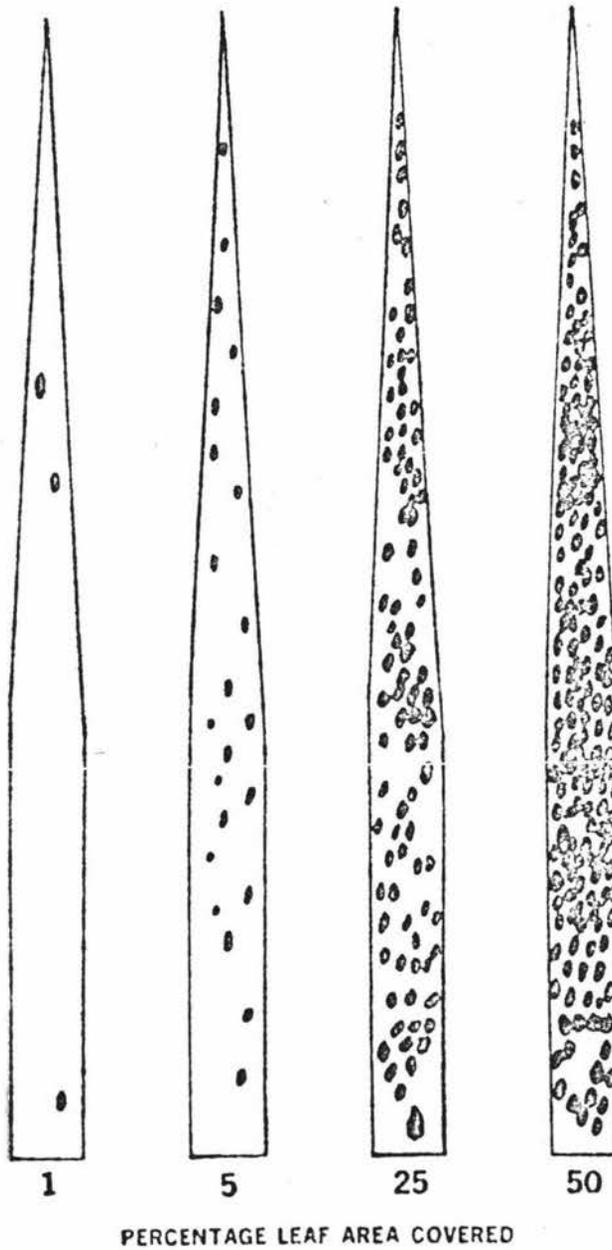


Figure 2.2 Standard leaf area diagrams used to assess coverage of physiological brown spot (adapted from James, 1971).

Table 2.2 R-square estimates for grain yield and 100-kernel weight against moisture content, in low and high nitrogen environments. Estimates are presented separately for diallel material (a) and material receiving fungicide treatment (b).

(a) Diallel Material

Variables against Moisture Content	Low Nitrogen	High Nitrogen
Grain Yield	0.08	0.00
100-Kernel Weight	0.23	0.19

(b) Material receiving fungicide treatment

Variables against Moisture Content	Low Nitrogen	High Nitrogen
Grain Yield	0.09	0.00
100-Kernel Weight	0.05	0.00

(Siegel, 1956; Anderberg, 1973). Parametric statistics and procedures, such as those of diallel analysis, are unsuitable for data measured on this type of scale. Nevertheless, net blotch infection types are presented in Chapter 3 as similar data have been reported in numerous studies (Buchannon and McDonald, 1965; Khan and Boyd, 1969a,b,c; Khan, 1971) and it was therefore worthwhile to compare results.

Top and bottom canopy net blotch scores, flag and second leaf areas infected and leaf area infected in the petri dish study, were plotted individually against age factors (time after heading when assessments were made) using Scattergrams. R-square estimates for dependent variables against their respective age factors, for diallel material, were 0.09 (top and bottom canopies and flag leaf), 0.10 (second leaf) and 0.13 (petri dish). It was concluded in all cases that adjustment to constant age after heading was unnecessary. The assumption was made that linear relationships between the variables were also poor for material treated with fungicides. Scattergrams of leaf area occupied by brown spot against leaf position divided by leaf number per tiller, also gave no justification for measurement modification. R-square estimates for high and low nitrogen environments were 0.03 and 0.02, respectively.

All disease percentage measurements and percentage leaf area with brown spot, were transformed using the angular (arcsin) transformation. This is standard practice for percentages which have a binomial basis (Snedecor and Cochran, 1967; Steel and Torrie, 1980). Grain number per ear data were transformed using  $10 \log_{10} X$  which gave a homogeneous set of error variances across environments ( $\chi^2 = 0.11$  with probability of 0.74). Logged data were multiplied by 10 to give two digits to the left of the decimal point, as was so for the original data.

Analysis of variance (Steel and Torrie, 1980) was conducted on all

characters for single and pooled environments using a computer programme, PHANIE (Phenotypic Analysis In Environments) (Gordon, unpubl.).

Analysis of variance for single environments was based on the following model:-

$$X_{ij} = \mu + \gamma_i + \beta_j + E_{ij}$$

where  $X_{ij}$  is the  $ij$ -th phenotypic variate,  $i = 1, \dots, g$ , where  $g$  is the number of genotypes,  $j = 1, \dots, b$ , where  $b$  is the block number,  $\mu$  is the grand mean,  $\gamma_i$  is the  $i$ -th genotype effect,  $\beta_j$  is the  $j$ -th block effect, and  $E_{ij}$  is the residual variation associated with the  $ij$ -th observation. The more complex model assumed for the pooled analysis of variance over environments was:

$$X_{ijk} = \mu + \gamma_i + \beta_{j(k)} + \eta_k + \gamma_{\eta_{ik}} + E_{ijk}$$

where  $X_{ijk}$  is the  $ijk$ -th phenotypic variate,  $i = 1, \dots, g$ , where  $g$  is the number of genotypes,  $j = 1, \dots, b$ , where  $b$  is the block number,  $k = 1, \dots, e$ , where  $e$  is the number of environments,  $\mu$  and  $\gamma_i$  are defined as in the single environment model,  $\beta_{j(k)}$  is the  $j$ -th block effect in the  $k$ -th environment,  $\eta_k$  is the  $k$ -th environment effect,  $\gamma_{\eta_{ik}}$  is the interaction between the  $i$ -th genotype and  $k$ -th environment, and  $E_{ijk}$  is the residual variation associated with the  $ijk$ -th observation.

PHANIE also estimated variance components together with their associated standard error estimates (Crump, 1946; Comstock and Robinson, 1951; Comstock and Moll, 1963) and tested homogeneity of error variances using Bartlett's (1947) chi-square test. In addition, Least Significant Difference was computed (Snedecor and Cochran, 1967; Steel and Torrie, 1980). Duncan's New Multiple Ranges were used where genotype number was greater than ten (Balaam, 1963). Ratios of estimated mean squares were tested (F-test) for significance in the usual manner (Crump, 1951; Le Clerg *et al.*, 1962). In the pooled analysis of variance, PHANIE

estimated a complex F-ratio ( $F'$ ) for source of variation due to environment using linear combinations of mean squares (Crump, 1951). Degrees of freedom for this test were estimated using the formula of Satterthwaite (1946).

Analysis of variance of net blotch data was conducted only in the high nitrogen environment as infection was absent in the other environment. Analyses were performed separately on all genotypes ( $g = n(n + 1)/2$ ), parents ( $g = n$ ) and hybrids ( $g = n(n - 1)/2$ ). The latter two analyses provided error mean squares for estimation of genetical components (Mather and Jinks, 1971) and standard errors of heterosis and heterobeltiosis (Section 2.10). This was also the procedure for all other characters in each environment. A pooled analysis of variance over environments (nitrogen levels) was done for all characters, apart from net blotch, with every genotype included. Parents and hybrids were not analysed separately as two-tailed F-tests (Steel and Torrie, 1980), examining whether  $E_P$  and  $E_F$  were significantly different in single environments, showed that only three of twenty-one variance ratios were significant at the 5 per cent level (Appendix 2).

## 2.8 DIALLEL ANALYSIS

### 2.8.1 BASIC ARRAY STATISTICS

The following statistics were estimated for each block (Mather and Jinks, 1971, 1977):

$V_{r_i}$  and  $\bar{V}_r$  - variance of  $i$ -th array ( $r_i$ ) and mean variance across arrays respectively,

$$V_{r_i} = \left( \sum_j X_{ij}^2 - \frac{X_{i.}^2}{m} \right) / m - 1, \text{ for each } i,$$

where  $i = 1, \dots, n$ , and  $n$  is the number of parents,  $j = 1, \dots, m$ , where  $m$  is progeny number and  $X_{i.}$  is the summation across all  $j$  observations for each  $i$  ( $X_{i.} = \sum_j X_{ij}$ ).

$$\bar{V}_r = \left( \sum_i V_{r_i} \right) / n$$

$W_{r_i}$  and  $\bar{W}_r$  - covariance of  $i$ -th array with nonrecurrent parent and mean covariance across arrays, respectively.

$$W_{r_i} = \left( \sum_{jk} X_{ij} Y_{jk} - \frac{X_{i.} Y_{.j}}{m} \right) / m - 1$$

where  $i$  and  $j$  are defined as before and  $k = 1, \dots, p$ , where  $p$  is the number of leading diagonal entries (parents)

$$\bar{W}_r = \left( \sum_i X_{r_i} \right) / n$$

$\bar{r}_i$  - mean of the  $i$ -th array.

$$\bar{r}_i = \left( \sum_j X_{ij} \right) / m$$

$V_p$  and  $V_r$  - variance of parents (leading diagonal of diallel table) and variance of array means, respectively.

$$V_p = \left( \sum_i X_i^2 - \frac{X.^2}{n} \right) / n - 1$$

where  $X_i$  is the  $i$ -th phenotypic variate,  $i = 1, \dots, n$ , and  $n$  is the number of parents, and  $X.$  is the sum of the entries in the leading diagonal of the diallel table.

$$V_r = \left( \sum_l X_l^2 - \frac{X.^2}{n} \right) / n - 1$$

where  $X_l$  is the  $l$ -th phenotypic variate,  $l = 1, \dots, a$ , where  $a$  is the number of array means,  $n$  is the number of parents, and  $X.$  is the sum of the parental array means.

### 2.8.2 ANALYSIS OF VARIANCE OF ARRAY STATISTICS

Analysis of variance was conducted on  $(W_r + V_r)$  and  $(W_r - V_r)$  data using computer programme GENSTAT (Alvey et al., 1977), to determine the applicability of an additive-dominance model in accounting for the data. The analysis partitioned the variance into amongst arrays and residual mean squares. Amongst array mean squares were compared with their respective error mean squares (F-tests). A non-significant F-test for  $(W_r - V_r)$  indicated that an additive-dominance model could not be

rejected. Significance of this source of variation showed that the data did not conform adequately to the model due to epistasis and/or correlated gene distributions (Mather and Jinks, 1971). Significance and non-significance of  $(W_r + V_r)$  indicated presence and absence of dominance, respectively. As an example, when both  $(W_r + V_r)$  and  $(W_r - V_r)$  were non-significant, it was concluded that the simple genetic model was still applicable but that dominance was trivial.

### 2.8.3 REGRESSION OF ARRAY STATISTICS

Further confirmation of the adequacy of an additive-dominance model was obtained from regression of  $W_r$  against  $V_r$ . For each character, 20 pairs of  $(W_r, V_r)$  statistics (from five arrays, four blocks) were used. Regression equations were computed by SPSS and printouts included estimates of R-square, standard errors of regression and slope, and F-tests (Draper and Smith, 1966; Steel and Torrie, 1980). Adequacy of the simple model was indicated when a significant regression slope (with respect to the null hypothesis that  $\hat{\beta} = 0$ ) did not differ significantly from unity. Significance was tested using a standard t-test (Draper and Smith, 1966).

When a significant regression slope for a character deviated significantly from one, each array (parent plus progeny) was omitted singly and regression analyses repeated on the smaller diallel data sets. Regression equations from these analyses were then compared for slope, standard error and R-square value. This was to decide which array deleted enabled remaining diallel data to conform most adequately to an additive-dominance model. The most appropriate regression equations generally had slopes closest to unity, relatively low standard errors for regression and slope and R-square estimates of greater than 0.5 - 0.6 (Sections 3.1.3 to 3.1.7). Analysis ceased where single

Table 2.3 Equations to estimate genetic components for a half diallel in a single environment.

Genetic Component	Basic Array Statistics	Using $E_F$ and $E_p$ (original data sets)	Using $E_{POOL}$ (reanalysed data sets)
D	$= V_p$	$= E_p$	$= E_{POOL}$
$H_1$	$= 4\bar{V}_r + V_p - 4\bar{W}_r$	$= \left(\frac{4(n-1)}{n} E_F + E_p\right)$	$= \frac{(5n-4)}{n} E_{POOL}$
$H_2$	$= 4\bar{V}_r - 4V_F$	$= \frac{4(n-1)^2}{n^2} E_F - \frac{4(n-1)}{n^2} E_p$	$= 4 E_{POOL} \left(1 - \frac{1}{n}\right)$
F	$= 2V_p - 4\bar{W}_r$	$= \frac{2(n-2)}{n} E_p$	$= \frac{2(n-2)}{n} E_{POOL}$

where n = number of parents

$E_F$  =  $F_1$  error mean square

$E_p$  = parent error mean square

$E_{POOL}$  = error mean square (parents and  $F_1$ 's in analysis of variance).

array deletion failed to achieve the expected linear relationship.

The limiting parabola,  $W_{ri}^2 = V_{ri} \cdot V_p$ , was plotted for all characters. All points ( $W_r$ ,  $V_r$ ) on lines of slope one must lie within this parabola (Hayman, 1954b; Mather and Jinks, 1971).

The regression line was used to obtain a ranking of parents with respect to dominance. This was possible since relative dominance decreases along the  $W_r/V_r$  graph as distance from origin increases (Mather and Jinks, 1971).

#### 2.8.4 GENETICAL COMPONENT STATISTICS

Genetic components - D,  $H_1$ ,  $H_2$  and F (defined in Review Section 1.2.2) - were estimated for all characters in both environments. Estimates were also calculated for reanalysed characters (Section 2.8.3) which conformed to an additive-dominance model.

The Mather and Jinks (1971) equations to estimate genetic components were modified (Appendix 3) to suit a half-diallel. These equations, consisting of basic array statistics and different error mean square combinations, are presented in Table 2.3. Basic array statistics were averaged over blocks (Jinks, 1954; Mather and Jinks, 1977) before estimating genetic components.

Parental and  $F_1$  error mean squares ( $E_p$  and  $E_F$  respectively) were used in initial analysis of all characters in each environment. For reanalysed characters, only "full" genotype (parents and hybrids) error mean squares were used as two-tailed F-tests showed that for most original data sets,  $E_p$  and  $E_F$  were not significantly different (Section 2.7). This was assumed for the reanalysed data sets.

Standard errors of genetical components were not estimated as "no worthwhile estimate of the errors of these components is available" (Mather and Jinks, 1971).

2.8.5 SECONDARY GENETICAL STATISTICS

The following statistics were also estimated (Mather and Jinks, 1971):

1.  $\sqrt{H_1}/D$  measures average degree of dominance over all segregating loci. The statistic may describe three dominance types, namely, partial ( $< 1$ ), complete ( $= 1$ ) and overdominance ( $> 1$ );
2.  $0.5F/\sqrt{D(H_1 - H_2)}$  measures the extent to which dominance level varies from one locus to another. The absolute value of the statistic varies from 0 to 1, where 1 indicates a constant dominance level over all loci;
3.  $uv$  is the product of the frequencies of increasing and decreasing alleles, respectively, over all loci and is estimated by  $H_2/4H_1$ . The maximum value of 0.25 occurs when gene frequencies are equal, that is,  $u = v = 0.5$ . Gene asymmetry is indicated when  $H_2/4H_1 < 0.25$ .
4.  $(\sqrt{4DH_1} + F) / (\sqrt{4DH_1} - F)$  measures the proportion of dominant to recessive alleles over all parents;
5. broadsense heritability ( $h_B^2$ ) and narrowsense heritability ( $h_N^2$ ) estimates were calculated from

$$h_B^2 = \frac{\frac{1}{2}D + \frac{1}{2}H_1 - \frac{1}{2}H_2 - \frac{1}{2}F}{\frac{1}{2}D + \frac{1}{2}H_1 - \frac{1}{2}H_2 - \frac{1}{2}F + E} \quad \text{and}$$

$$h_N^2 = \frac{\frac{1}{2}D + \frac{1}{2}H_1 - \frac{1}{2}H_2 - \frac{1}{2}F}{\frac{1}{2}D + \frac{1}{2}H_1 - \frac{1}{2}H_2 - \frac{1}{2}F + E}.$$

$E$  is the pooled error mean square from analysis of variance of  $(n(n+1)/2)$  genotypes.

6.  $r(\bar{P}_R, (W_R + V_R))$  is correlation between  $(W_R + V_R)$  for each array and mean of common parent. The statistic identifies whether the distribution of dominant to recessive alleles is correlated with common parent phenotype.

7.  $\sigma_{AR}^2 / \sigma_{DR}^2$  ( $= \frac{1}{2}D / \frac{1}{4}H_2$ ) shows the relative importance of these two genetic variances in a random mating (R) population where it is assumed  $u = v = 0.5$  and epistasis is absent.

8. Number of effective factors (K) was estimated using the formula:

$$K = (M_{F_1} - M_p)^2 / \frac{1}{4}H_2$$

where  $M_{F_1}$  and  $M_p$  are the overall means for  $F_1$  progeny and parents, respectively. This is based on the formula given by Jinks (1954) for  $F_1$  diallels.

## 2.9 ANALYSIS OF CONTROL/DISEASED PARENTS

A PHANIE analysis of variance was conducted for all characters measured on plants receiving fungicide treatment (controls). Least Significant Differences (L.S.D.) were estimated where F-ratio for genotypes was significant. Control and diseased diallel parents were regarded as two treatments. A pooled analysis of variance over treatments was therefore conducted for all characters and note taken of source of variation due to treatment.

Yield loss (%) and 100-kernel weight loss (%) were estimated using appropriate control and diseased parent values. The formula used for estimating both percentage losses was:

$$\text{Loss (\%)} = \frac{\text{control value} - \text{disease value}}{\text{control value}} \times 100$$

Stepwise multiple regression (Draper and Smith, 1966) of grain yield (Y) against flag and second leaf areas infected with net blotch ( $X_1$ ,  $X_2$  respectively), was conducted for the diseased, diallel, parents. SPSS output included the R-square increment attributable to inclusion of  $X_2$ , together with standardized partial regression coefficients (Steel and Torrie, 1980) for  $X_1$  and  $X_2$ . Relative importance of  $X_1$  and  $X_2$  in determining yield was ascertained by comparing magnitudes of the respective

partial regression coefficients and examining the R-square change resulting from inclusion of  $X_2$  in the regression equation. A relatively large partial regression coefficient indicates a greater influence on yield while a low R-square change shows that the contribution of the additional independent variable ( $X_2$ ) to yield, is negligible.

## 2.10 HETEROSIS AND HETEROBELTIOSIS

Heterosis was estimated as  $(\bar{F}_1 - \text{mid parent mean (MP)})$  and heterobeltiosis as  $(\bar{F}_1 - \text{better parent mean } (\bar{P}_1))$  (Virk and Verma, 1973). For all characters, better parent was defined as the one with greater expression.

Standard errors were estimated for heterosis and heterobeltiosis.

For single environments, formulae used were:

$$\begin{aligned} \text{standard error of heterosis} &= \sqrt{\frac{\sigma_{F_1}^2}{b} + \frac{\sigma_p^2}{2b}} && \text{(single environments)} \\ \text{(seed for } \bar{x}_{F_1} - \text{MP)} &&& \\ \text{standard error of heterobeltiosis} &= \sqrt{\frac{\sigma_{F_1}^2}{b} + \frac{\sigma_p^2}{b}} && \\ \text{(seed for } \bar{x}_{F_1} - \bar{x}_{P_{\text{better}}}) &&& \end{aligned}$$

where  $b$  = block number and  $\sigma_{F_1}^2$  and  $\sigma_p^2$  are error mean squares of  $F_1$ 's and parents, respectively. For nearly all characters,  $\sigma_{F_1}^2$  was not significantly different from  $\sigma_p^2$  (Section 2.7). Therefore, for the pooled analysis over environments ( $e$ ), an error mean square obtained from analysis of variance conducted on all genotypes ( $\sigma_{\text{POOL}}^2$ ) was used to estimate the two standard errors. Hence,

$$\begin{aligned} \text{standard error of heterosis} &= \sqrt{\frac{3 \sigma_{\text{POOL}}^2}{2be}} && \text{(pooled analysis over environments)} \\ \text{(seed for } \bar{x}_{F_1} - \text{MP)} &&& \\ \text{standard error of heterobeltiosis} &= \sqrt{\frac{2 \sigma_{\text{POOL}}^2}{be}} && \\ \text{(seed for } \bar{x}_{F_1} - \bar{x}_{P_{\text{better}}}) &&& \end{aligned}$$

Derivations of the above formulae are presented in Appendix 4.

## 2.11 SIGNIFICANCE LEVELS

The following classification was used for all significance tests in this study -

NS =  $P > 0.10$

(NS) =  $0.10 \geq P > 0.05$

\* =  $0.05 \geq P > 0.01$

\*\* =  $0.01 \geq P > 0.001$

\*\*\* =  $P \leq 0.001$ .

### CHAPTER 3

## CHAPTER 3.

### RESULTS AND DISCUSSION

#### 3.1 NET BLOTCH

Net blotch symptoms occurred only in the high nitrogen environment. At Growth Stage 11.1 (Hampton and Arnst, 1978), prevalence and intensity of net blotch were insufficient for assessment of infection type and leaf area infected. Consequently, these assessments were conducted later (Day 89 of trial) when net blotch prevalence and intensity had improved. Explanations for initial poor disease development are suggested in Chapter 4.

##### 3.1.1 INFECTION TYPE

Infection types (Section 2.6.1) predominating on the foliage of each genotype (parents and hybrids) at Day 89, are presented in Table 3.1 for individual blocks. The most frequent (modal) infection type for each genotype across blocks, is also presented. A dash indicates that there was no modal infection type and therefore a median is given.

The most resistant infection types were exhibited by Manchuria and ranged from no observable infection to slightly elongated dark brown lesions with no chlorosis. Crosses between Manchuria and relatively susceptible genetic stocks generally displayed less resistant infection types. Susceptibility of the other four parents and their progeny was relatively high. Zephyr, Kaniere and Mata exhibited highly susceptible infection types in all blocks while Hassan gave slightly more resistant reactions.

Diallel analysis was not conducted on these data as they were measured on an ordinal scale (Siegel, 1956). Data of this type are unsuitable for analysis by parametrical procedures such as diallel analysis.

Table 3.1 Infection types for all genotypes in each block in high nitrogen environment.

Cross †	Infection type in blocks (front to back of room)	Modal Infection Type	Cross †	Infection type in blocks (front to back of room)	Modal Infection Type
1 x 1	4, 4, 4, 4	4	4 x 3	4, 4, 4, 4	4
1 x 2	4, 4, 4, 4	4	4 x 4	4, 4, 4, 4	4
2 x 2	4, 4, 4, 4	4	5 x 1	2, 2, 3, 2	2
3 x 1	3, 4, 4, 4	4	5 x 2	4, 2, 3, 0	2.5
3 x 2	4, 4, 4, 4	4	5 x 3	2, 3, 3, 3	3
3 x 3	3, 3, 4, 3	3	5 x 4	3, 3, 4, 2	3
4 x 1	4, 4, 4, 4	4	5 x 5	2, 2, 0, 0	1
4 x 2	3, 4, 4, 4	4			

† 1 - Kaniere; 2 - Zephyr; 3 - Hassan; 4 - Mata; 5 - Manchuria; (female parents first).

All other data in this study were analysed parametrically since they were measured on interval or ratio scales (Siegel, 1956). Infection types are presented as similar features have been recorded in numerous other studies on net blotch resistance (Khan and Boyd, 1969a, b, c). Comparisons were therefore possible.

### 3.1.2 INITIAL ANALYSIS OF VARIANCE

Analysis of variance of top and bottom canopy scores (1 to 10 scoring system where scores corresponded approximately to increments of 10 per cent infection) and flag leaf, second leaf and leaf section (petri dish study) areas infected (all measured on a percentage basis), are presented in Appendix 5.1. Standard deviation, coefficient of variation and standard error (S.E.) of genotype means are also given. In all cases, source of variation due to genotypes (whether parents, hybrids or parents plus hybrids) was significant ( $P \leq 0.05$ ), indicating that at least one type of genetic variation was present. Coefficients of variation were highest for leaf section area infected. Overall, characters ranged from 27 to 69 per cent. From analysis of variance with parents and hybrids included, blocks were significantly different for bottom canopy scores ( $P \leq 0.05$ ) and flag leaf area infected ( $P \leq 0.001$ ). This suggested, particularly in the latter instance, large variation in degree of net blotch development within the climate room environment.

### 3.1.3 TOP CANOPY SCORE

Genotype means and estimates of heterosis and heterobeltiosis, expressed as positive and negative deviations and percentages, are presented in Table 3.2. Also included are the Least Significant Difference (L.S.D.) for genotype means and standard errors and L.S.D.'s for heterosis and heterobeltiosis (all at 5 per cent level of significance), plus overall means for parents, hybrids and total genotypes. Below each

**Table 3.2**

Genotype means (upper triangle) and estimates of heterosis and heterobeltilosis (bottom triangle) for top canopy scores (transformed to  $\sin^{-1} \sqrt{X/10}$ )

Male/Female	1	2	3	4	5
1	21.7 bcde #	32.1 abc	35.0 a	28.2 abcd	19.5 cde
2	7.5 30.4 (4.5)†(16.3)	27.6 abcd	28.2 abcd	33.5 ab	16.4 de
3	13.3 61.3 (13.3) (6.1)	3.6 14.6 (0.6) (2.2)	21.7 bcde	28.1 abcd	19.5 cde
4	5.0 21.6 (3.5) (14.2)	7.4 28.3 (5.9) (21.4)	4.9 21.1 (3.4) (13.8)	24.7 abcd	19.1 cde
5	4.1 26.5 (-2.2)(-10.1)	-2.0 -10.9 (-11.2)(-40.6)	4.1 26.5 (-2.2)(-10.1)	2.2 13.0 (-5.6)(-22.7)	9.2 e

$\bar{F}_1 = 26.0$     $\bar{P} = 21.0$    Grand mean = 24.3   L.S.D.(5%) = 11.3   L.S.D.(1%) = 15.1

Heterosis: S.E. = 4.8   L.S.D.(5%) = 9.7   Heterobeltilosis: S.E. = 5.6   L.S.D.(5%) = 11.3

† Estimates of heterosis and heterobeltilosis (latter in brackets) expressed as deviations and percentages (left and right sets of figures for each cross, respectively).

# Figures underscored by different letters differ at the 5% level of significance.

genotype mean are one or more letters indicating to which significance group(s) the mean belongs, as estimated by Duncan's Multiple Ranges test (Le Clerg *et al.*, 1962; Steel and Torrie, 1980). Genotype means underscored by the same letter(s) are in a common significance group and are declared as indistinguishable (Snedecor and Cochran, 1967).

The most striking feature shown in Table 3.2 is that Manchuria (5) was significantly ( $P \leq 0.05$ ) more resistant to net blotch than Zephyr (2) and Mata (4). Furthermore, all crosses involving Zephyr and Mata, apart from those with Manchuria, were significantly less resistant than Manchuria. In total, eight genotypes were significantly ( $P \leq 0.05$ ) more susceptible to net blotch than Manchuria.

Estimates of  $V_r$ ,  $W_r$ ,  $(W_r + V_r)$  and  $(W_r - V_r)$  are presented in Tables 3.3 and 3.4. Basic array statistics and the  $W_r/V_r$  regression equation plus associated statistics are shown, together with the appropriate statistics for other net blotch characters, in Tables 3.5 and 3.6 respectively. For each character, the regression slope ( $\hat{\beta}_1$ ) was significantly ( $P \leq 0.01$ ) different from zero (Table 3.6). It was therefore appropriate to conduct t-tests on the departure of each  $\hat{\beta}_1$  from unity (Mather and Jinks, 1971). The results are presented in Table 3.7.

The  $W_r/V_r$  regression plot for top canopy score is presented in Figure 3.1(a). R-square and regression slope are included. Numbers one to five correspond to parents and have been listed before (Table 3.1). Analysis of variance of  $(W_r + V_r)/(W_r - V_r)$  for arrays, is presented in Appendix 5.2. Significance of the major sources of variation are presented in Table 3.7.

Analysis of variance of  $(W_r + V_r)$  and  $(W_r - V_r)$  showed no significant differences between parental arrays (Table 3.7). Nonsignificance of  $(W_r - V_r)$  indicated that data may be adequately accounted for by an

Table 3.3

Estimates of variances ( $V_r$ ) and covariances ( $W_r$ ) of arrays for top canopy score  
(transformed to  $\sin^{-1} \sqrt{X/10}$ )

Array	Block 1		Block 2		Block 3		Block 4	
	$V_r$	$W_r$	$V_r$	$W_r$	$V_r$	$W_r$	$V_r$	$W_r$
1	62.53	7.36	3.80	3.68	57.06	67.19	128.61	73.06
2	69.79	-12.82	19.48	8.32	69.41	84.41	387.64	195.85
3	22.39	0.66	11.86	11.86	62.28	47.70	141.19	83.93
4	22.39	0.66	11.38	4.99	52.61	69.87	185.70	133.56
5	26.77	-5.04	6.07	-1.36	110.86	122.43	109.93	44.15

Table 3.4

Estimates of  $(W_r + V_r)$  and  $(W_r - V_r)$  for top canopy score  
 (transformed to  $\sin^{-1} \sqrt{x/10}$ )

Array	Block 1		Block 2		Block 3		Block 4	
	$W_r + V_r$	$W_r - V_r$						
1	69.89	-55.18	7.48	-0.13	124.25	10.13	201.67	-55.54
2	56.96	-82.61	27.81	-11.16	153.82	14.99	583.49	-191.79
3	23.05	-21.73	23.71	0.00	109.98	-14.58	225.12	-57.27
4	23.05	-21.73	16.38	-6.39	122.48	17.26	319.25	-52.14
5	21.73	-31.82	4.71	-7.43	233.29	11.56	154.09	-65.78

Table 3.5 Basic array statistics (means across blocks) for biometrical analyses of net blotch resistance.

Character	$V_p$	$V_r$	$W_r$	$V_r^-$
Top canopy score (arcsin score)	88.10	76.44	47.53	34.62
Bottom canopy score (arcsin score)	103.03	67.76	48.96	29.65
Flag leaf area (arcsin percentage)	138.86	67.23	62.20	38.44
Second leaf area (arcsin percentage)	184.89	96.90	76.34	45.18
Leaf section area (arcsin percentage)	133.15	139.36	77.10	64.17

**Table 3.6**  $W_r/V_r$  regression equations and associated statistics for biometrical analyses of net blotch resistance.

Character	Regression equation	F-test	S.E. regression	S.E. ( $\hat{\beta}_1$ )
Top canopy score (arcsin score)	$W_r = 0.55V_r + 5.37$	64.74***	26.82	0.07
Bottom canopy score (arcsin score)	$W_r = 0.61V_r + 7.54$	75.70***	24.19	0.07
Flag leaf area (arcsin percentage)	$W_r = 0.48V_r + 30.22$	24.39***	28.15	0.10
Second leaf area (arcsin percentage)	$W_r = 0.65V_r + 12.97$	15.35**	42.63	0.17
Leaf section area (arcsin percentage)	$W_r = 0.48V_r + 9.74$	12.89**	57.02	0.13

\*\* =  $0.01 \geq P > 0.001$

\*\*\* =  $P \leq 0.001$ .

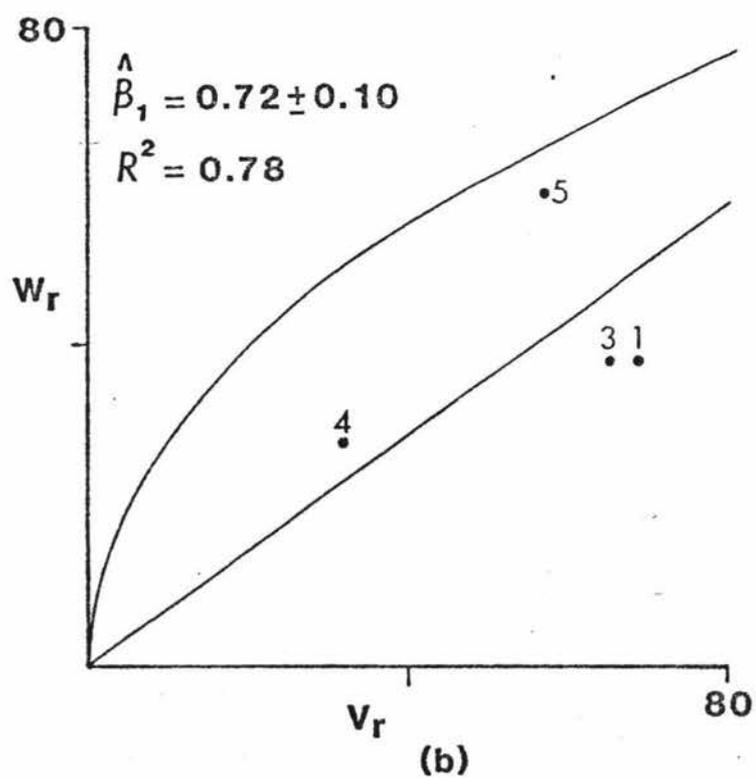
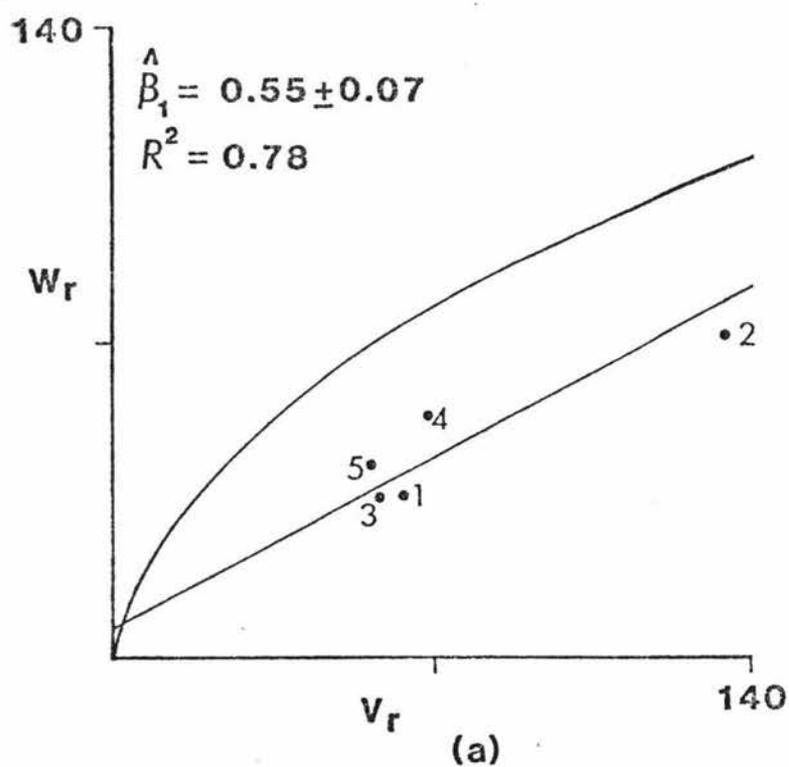
Table 3.7

Genetic statistics for five biometrical analyses of net blotch resistance.

Statistic	Top canopy score (transformed to $\sin^{-1} \sqrt{X/10}$ )		Bottom canopy score (transformed to $\sin^{-1} \sqrt{X/10}$ )		Flag leaf area (transformed to $\sin^{-1} \sqrt{X/100}$ )		Second leaf area (transformed to $\sin^{-1} \sqrt{X/100}$ )	Leaf section area (transformed to $\sin^{-1} \sqrt{X/100}$ )	
	original	reanalysed	original	reanalysed	original	reanalysed	original	original	reanalysed
$W_R + V_R$ (sig.)	NS	NS	NS	NS	(NS)	NS	*	NS	NS
$W_R - V_R$ (sig.)	NS	NS	NS	NS	NS	NS	NS	*	NS
t-test ( $\hat{\beta}_1 - 1$ )	6.55***	2.78*	5.54***	1.04NS	5.44***	2.23*	2.07(NS)	3.84**	2.66*
D	36.34	40.66	40.13	50.39	104.97	140.33	117.64	69.71	59.64
$H_1$	-61.07	3.53	-43.38	-10.50	-53.44	-35.86	36.39	34.48	-43.53
$H_2$	-36.31	-18.45	-14.76	-29.63	-49.35	-54.82	33.02	32.75	-105.84
F	-76.01	-45.68	-65.27	-5.20	-11.73	-34.97	-16.26	-118.24	-90.55
E (pool)	62.43	34.18	57.38	35.63	49.16	36.18	56.53	77.18	81.11
E (parents)	51.76		62.90		33.89		67.26	63.44	
E (hybrids)	66.59		49.59		55.80		51.10	88.83	
$\sqrt{H_1/D}$	a	0.29					0.56	0.70	
$uv (H_2/4H_1)$		0.15	-1.31	0.09	0.71	0.23	0.38	0.23	0.61
$0.5F/\sqrt{D(H_1 - H_2)}$	c		-0.76		-0.08		-0.34	-0.41	-0.74
dom./rec. genes	a		-0.31				0.78	-0.09	
$h^2$ (narrow)		45.10	64.68	41.70	56.96	60.46	81.21	51.44	52.63
$h^2$ (broad)		35.74	59.17	37.69	45.67	47.21	69.75	57.63	57.17
$r (\bar{P}_R, W_R + V_R)$	d	0.13NS	-0.42NS				0.29	0.38*	
$\sigma_A^2/\sigma_D^2$	b						7.13	4.26	
K(effective factors) <sup>b</sup>							1.32	0.96	

a - not calculated where  $H_1$  was negative; b - not calculated where  $H_2$  was negative; c - not calculated where  $(H_1 - H_2)$  was negative; d - generally not calculated where all previous analyses indicated that dominance was trivial.

NS =  $P > 0.10$ ; (NS) =  $0.10 \geq P > 0.05$ ; \* =  $0.05 \geq P > 0.01$ ; \*\* =  $0.01 \geq P > 0.001$ .



**Figure 3.1:** Regressions of  $W_r$  on  $V_r$  for top canopy score;  
 (a) all five parents, (b) parent 2 omitted.

additive-dominance model while no significant difference between estimates of  $(W_r + V_r)$  denoted that dominance was trivial. Regression slope ( $\hat{\beta}_1$ ) deviated significantly ( $P \leq 0.001$ ) from unity, indicating graphical distortion due to one or more of epistasis, correlated gene distributions (Mather and Jinks, 1971; Jana, 1973) and environment. Hence, there was lack of agreement between results of the two analyses with respect to complexity of the genetical models suggested.

Mather and Jinks (1971) stressed the need to have both analyses indicating adequacy of a simple additive-dominance model before estimating genetic components of variation. As this was not so for the regression slope test, diallel data sets with one array removed in turn, were reanalysed (Section 2.8.3). By this procedure, it was endeavoured to remove major disturbances causing non-conformance to an additive-dominance model. Regression equations and appropriate statistics for the reduced diallel data sets are presented in Table 3.8. All regressions were significant. Array 5 (Manchuria and its crosses) was not deleted since Manchuria was the only net blotch resistant genetic stock used in the study. Inclusion of array 5 was therefore necessary to obtain information on resistance to the disease.

From comparison between the four regression equations and their respective statistics, deletion of array 2 (Zephyr and its crosses) and reanalysis of remaining data gave a result which conformed most closely to an additive-dominance model. For this data, slope was closest to unity, standard error (S.E.) of regression was the smallest and R-square was relatively high. Although the t-test for departure of slope from unity (Table 3.7) was still significant ( $P \leq 0.05$  compared with  $P \leq 0.001$  for original data set), the significance level was lower. It was therefore decided to use this reduced diallel data set for estimation of genetic components. Analysis of variance of  $(W_r + V_r)/(W_r - V_r)$  is

Table 3.8

$W_R/V_R$  regression equations and associated statistics for analyses conducted on reduced diallel data sets for top canopy score  
(transformed to  $\sin^{-1} \sqrt{X/10}$ )

Array deleted	Regression equation	S.E.	F-test	R-square	S.E. ( $\hat{\beta}_1$ )
1	$W_R = 0.54V_R + 18.71$	32.61	73.45***	0.84	0.06
2	$W_R = 0.72V_R - 0.23$	22.03	48.68***	0.78	0.10
3	$W_R = 0.59V_R + 14.04$	40.75	40.18***	0.74	0.09
4	$W_R = 0.59V_R + 3.79$	38.79	31.23***	0.69	0.11

\*\*\* =  $P \leq 0.001$

given for data with array 2 deleted in Appendix 5.2 and significance of the major sources of variation are shown in Table 3.7. The  $W_r/V_r$  regression plot for the reanalysed data is presented in Figure 3.1(b).

Genetical components of variation plus other relevant statistics (Mather and Jinks, 1971) are presented in Table 3.7 for original and reanalysed data. Both data sets gave relatively large D components, indicating a preponderance of additive genetic variance. Negative estimates of  $H_1$  and  $H_2$  from analysis of original data suggested that dominance was trivial since these components have positive expectations as seen from the formulae,  $H_1 = 4uvh^2$  and  $H_2 = 16u^2v^2h^2$  (Mather and Jinks, 1971). Estimates of  $\sqrt{H_1/D}$ ,  $uv$ ,  $0.5F\sqrt{D(H_1-H_2)}$  and ratio of dominance to recessive genes, were therefore also regarded as trivial. For reanalysed data,  $H_1$  was slightly positive indicating a relatively small dominance component. Both data sets gave large negative estimates of F which suggested inequality of gene frequencies with an excess of recessive over dominant alleles.

It should be noted that negative estimates of  $H_1$  and  $H_2$  did not necessarily indicate that dominance was small or even absent. Rather, they showed that correction for environment (Table 2.3) was greater than the combination of other basic array statistics in the relevant equations. That is, equations gave net results of the balance between environment and other statistics.

With regard to analysis of original top canopy score data, error mean squares in Table 3.7 were relatively large compared with the D estimate. With parents (n) equal to five in equations estimating H components (Table 2.3), environment corrections were approximately quadrupled ( $H_1$ ) and tripled ( $H_2$ ). However, following reanalysis of data, error was approximately halved and D estimate unchanged. A positive  $H_1$  estimate resulted mainly from reduced error component and

slight reduction in environment correction factor due to decreased size of  $n$ . It was concluded that reduction in error enabled greater precision in estimation of genetic components.

The  $\sqrt{H_1/D}$  ratio was 0.29 using reanalysed data which indicated partial dominance. A similar estimate was unable to be calculated for original data since  $H_1$  was negative, as noted previously.

Narrowsense and broadsense heritability estimates were around 40 to 60 per cent. Narrowsense heritability was greater than broadsense heritability using original and reanalysed data, due to influence of negative estimates of  $H_1$  and/or  $H_2$ . The results further suggested that genetic variance was predominantly additive.

Correlations between common parent mean and  $(W_r + V_r)$  for each array (Table 3.7), for original and reanalysed data, were very low (0.13) and moderate (-0.42), respectively. Both correlations were nonsignificant. These findings indicated that distribution of dominant to recessive alleles was only slightly correlated with common parent phenotype. The result was not unexpected as previous analyses suggested that dominance was trivial.

A ranking of parental arrays with respect to proportion of dominant alleles may be obtained from the  $W_r/V_r$  plots (Figures 3.1(a) and (b)). Smallest  $(W_r, V_r)$  points, closest to the origin, correspond to parental arrays with the greatest proportion of dominant alleles. However, it should be noted that when R-square values for regression are low due to epistasis, correlated gene distributions and/or environment, array rankings in terms of dominance should be interpreted cautiously. In such cases, scattering of the  $(W_r, V_r)$  points may be attributable mainly to forms of non-additive genetic variation other than dominance.

For the present character, R-square (0.78) was quite acceptable. Regression of original data showed that Manchuria (5) had the highest

proportion of dominant alleles while the highly susceptible cultivar Zephyr (2), had least dominant alleles. All parental array points, except for Zephyr, coincided approximately with each other. This could indicate that additivity was of greater importance than dominance, as suggested by analysis of variance of  $(W_R + V_R)/(W_R - V_R)$  and D component. Indeed, in the absence of interaction and other disturbing influences,  $W_R/V_R$  points coincide, apart from sampling variation, when only additive variation is present (Mather and Jinks, 1971). Order of parental array means was very similar to that for respective phenotypic parental means (Table 3.2).

It is worthy of note that positions of array points on the regression line are relative and ranking of arrays gives no quantification of the proportion of dominance alleles. Ratios of distances between array points could be used to give quantitative measures of relative proportions of dominance genes. However, these would be tedious to obtain and valid only when R-square equals one and t-test  $(\hat{\beta}_1 - 1)$  is nonsignificant. The superior approach is correlation of parental phenotypic means against  $(W_R + V_R)$  (Mather and Jinks, 1971), as conducted previously.

When array 2 (Zephyr and its crosses) was deleted and remaining data reanalysed, order of points on the  $W_R/V_R$  graph (Figure 3.1(b)) changed slightly. Although relative positions of arrays 5, 3 and 1 were unchanged, array 4, previously at a higher regression corrected  $(W_R, V_R)$  value than these arrays, was transferred to the lowest position. That is, its relative proportion of dominance genes increased upon reanalysis.

In summary, net blotch resistance as measured by top canopy score, was controlled predominantly by genes acting additively. Dominance and epistasis and/or correlated gene distributions were of

relatively minor importance. Analysis of reanalysed data suggested partial dominance.

As regards heterosis and heterobeltiosis for top canopy score, significant ( $P \leq 0.05$ ) estimates were identified by use of appropriate L.S.D.'s (Table 3.2). It should be noted that all net blotch measurements in this study were expressed in terms of susceptibility. Therefore, from a resistance viewpoint, acceptable heterosis and heterobeltiosis estimates were highly negative. Of the ten crosses, there was one significant ( $P \leq 0.05$ ) estimate of heterosis. This was for Kaniere x Hassan cross and was positive. A significant and positive estimate of heterobeltiosis was also obtained for this cross. That is, heterosis and heterobeltiosis were for net blotch-susceptibility.

#### 3.1.4 BOTTOM CANOPY SCORE

Genotype means and relevant statistics are presented in Table 3.9.

Manchuria was significantly ( $P \leq 0.05$ ) more net blotch-resistant than the other parents. This contrasts slightly with the result for top canopy score where Manchuria was significantly more resistant than only two of the other four parents. All crosses involving Manchuria, with the exception of Manchuria x Mata cross, were not significantly different from Manchuria. Zephyr x Kaniere cross was significantly ( $P \leq 0.05$ ) less resistant to net blotch than Manchuria and all its crosses. The cross between Zephyr and Manchuria was significantly more resistant than its relatively susceptible parent.

Estimates of variances and covariances of arrays and their sums and differences, are presented in Tables 3.10 and 3.11 respectively. Basic array statistics and the  $W_r/V_r$  regression equation together with associated statistics are shown in Tables 3.5 and 3.6 respectively. The  $W_r/V_r$  regression plot is presented in Figure 3.2 (a).

Table 3.9

Genotype means (upper triangle) and estimates of heterosis and heterobeltiliosis (bottom triangle) for bottom canopy scores (transformed to  $\sin^{-1} \sqrt{X/10}$ )

Male/Female	1	2	3	4	5
1	21.6 abc #	34.0 a	31.2 ab	29.8 ab	19.9 bcd
2	8.5 33.3 (4.6) † (15.6)	29.4 ab	31.2 ab	31.0 ab	16.9 cd
3	9.2 39.5 (8.7) (38.7)	5.3 20.4 (1.8) (6.1)	22.5 abc	23.6 abc	19.5 bcd
4	6.8 29.6 (5.4) (22.1)	4.1 15.2 (1.6) (5.4)	0.2 0.9 (-0.8) (-3.3)	24.4 abc	21.1 bc
5	4.5 29.2 (-1.7) (-7.9)	-2.4 -12.4 (-12.5) (-43.0)	3.7 23.3 (-3.0) (-13.3)	4.3 25.6 (-3.3) (-13.5)	9.2 d

$\bar{F}_1 = 25.8$      $\bar{P} = 21.4$     Grand mean = 24.4    L.S.D.(5%) = 10.8    L.S.D.(1%) = 14.5

Heterosis:    S.E. = 4.5    L.S.D.(5%) = 9.1    Heterobeltiliosis:    S.E. = 5.4    L.S.D.(5%) = 10.8

† Estimates of heterosis and heterobeltiliosis (latter in brackets) expressed as deviations and percentages (left and right sets of figures for each cross, respectively).

# Figures underscored by different letters differ at the 5% level of significance.

Table 3.10

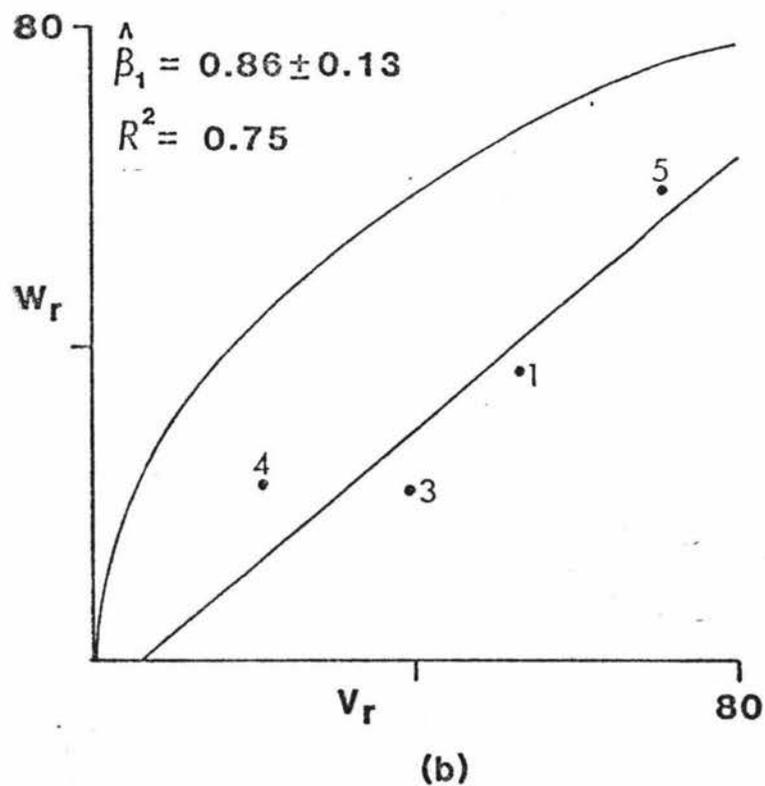
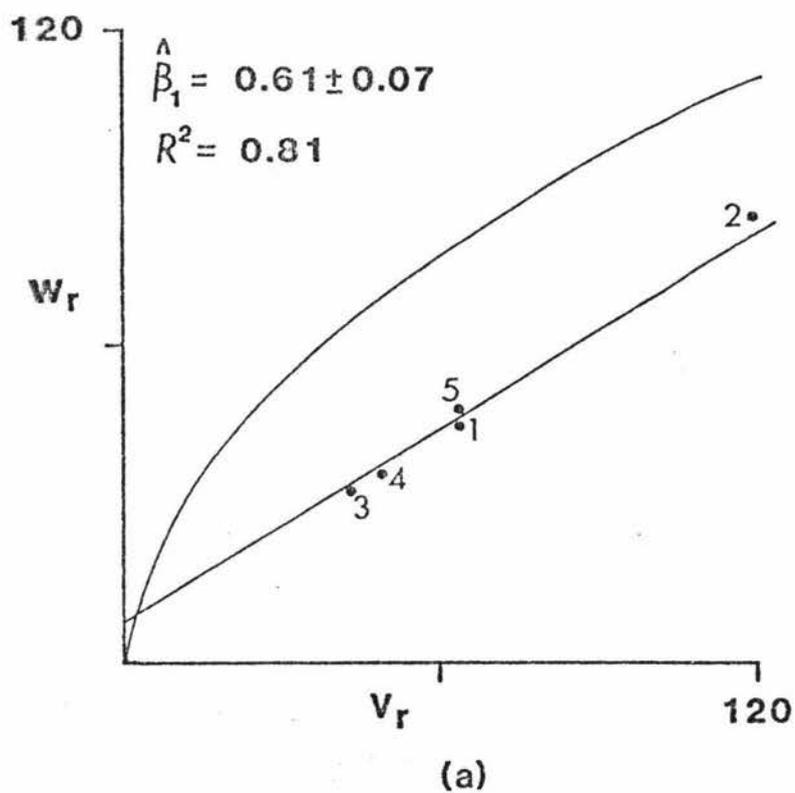
Estimates of variances ( $V_r$ ) and covariances ( $W_r$ ) of arrays for  
 bottom canopy score (transformed to  $\sin^{-1} \sqrt{X/10}$ )

Array	Block 1		Block 2		Block 3		Block 4	
	$V_r$	$W_r$	$V_r$	$W_r$	$V_r$	$W_r$	$V_r$	$W_r$
1	80.34	5.25	5.70	-2.19	88.48	76.48	130.80	98.75
2	29.77	14.84	25.53	20.78	70.34	110.33	352.29	193.18
3	13.25	8.40	5.70	5.67	78.28	66.10	78.28	50.76
4	27.82	11.37	16.58	2.20	45.84	77.98	103.05	50.37
5	19.88	-7.93	6.07	-1.62	139.45	125.67	87.66	72.74

Table 3.11

Estimates of  $(W_R + V_R)$  and  $(W_R - V_R)$  for bottom canopy score  
 (transformed to  $\sin^{-1} \sqrt{X/10}$ )

Array	Block 1		Block 2		Block 3		Block 4	
	$W_R+V_R$	$W_R-V_R$	$W_R+V_R$	$W_R-V_R$	$W_R+V_R$	$W_R-V_R$	$W_R+V_R$	$W_R-V_R$
1	35.59	-25.10	3.51	-7.90	164.96	-12.00	229.56	-32.05
2	44.61	-14.92	46.31	-4.75	180.67	39.99	545.47	-159.12
3	21.65	-4.85	11.37	-0.04	144.33	-12.18	129.03	-27.52
4	39.18	-16.45	18.78	-14.38	123.79	32.12	153.43	-52.68
5	11.95	-27.81	4.45	-7.69	265.33	-13.58	160.40	-14.92



**Figure 3.2:** Regressions of  $W_r$  on  $V_r$  for bottom canopy score; (a) all five parents, (b) parent 2 omitted..

Significances of t-test for slope and major sources of variation in analysis of variance of  $(W_R + V_R)/(W_R - V_R)$ , are given in Table 3.7. The latter analysis of variance is detailed in Appendix 5.2.

$(W_R + V_R)$  and  $(W_R - V_R)$  were not significantly different between arrays (Table 3.7), indicating that the data were adequately accounted for by an additive-dominance model. Dominance was trivial since variation due to  $(W_R + V_R)$  was not significant. There was a highly significant ( $P \leq 0.001$ ) deviation of  $\hat{\beta}_1$  from unity, indicating disturbances due to one or more of epistasis, correlated gene distributions and environment. Hence, the regression analysis suggested a more complex genetical model than the simple additive-dominance model implied by the analysis of variance of  $(W_R + V_R)$  and  $(W_R - V_R)$ . A similar conclusion was reached for top canopy score.

In view of this lack of agreement between analyses, data were reanalysed (Section 2.8.3). Regression equations and appropriate statistics for the reduced diallel data sets are shown in Table 3.12. All regressions were significant. Regression information for the four reduced diallel data sets was compared. Deletion of array 2 (Zephyr and its crosses) and reanalysis of remaining data gave the most favourable result in that  $\hat{\beta}_1$  was not significantly different from unity (Table 3.7). Analysis of variance of  $(W_R + V_R)$  and  $(W_R - V_R)$  is given for data with array 2 deleted in Appendix 5.2 and significance of appropriate sources of variation is presented in Table 3.7.  $(W_R + V_R)$  and  $(W_R - V_R)$  were not significantly different between arrays. Hence, both analyses indicated that an additive-dominance model was adequate to account for the diallel data set with array 2 deleted. The  $W_R/V_R$  regression plot for this reanalysed data is shown in Figure 3.2(b).

Genetic components and other appropriate statistics (Mather and Jinks, 1971) are given in Table 3.7 for original and reanalysed data.

Table 3.12

$W_R/V_R$  regression equations and associated statistics for analysis conducted on reduced diallel data sets for bottom canopy score.

(transformed to  $\sin^{-1}\sqrt{X/10}$ )

Array deleted	Regression equation	S.E.	F-test	R-square	S.E. ( $\hat{\beta}_1$ )
1	$W_R = 0.63V_R + 18.32$	41.72	34.44***	0.71	0.11
2	$W_R = 0.86V_R - 4.99$	24.84	41.69***	0.75	0.13
3	$W_R = 0.60V_R + 14.65$	36.03	45.89***	0.77	0.09
4	$W_R = 0.64V_R + 11.19$	25.20	89.88***	0.87	0.07

\*\*\* =  $p \leq 0.001$

Both data sets gave positive, relatively large, D components indicating that additivity was important. Negative estimates of  $H_1$  and  $H_2$  for original and reanalysed data suggested that dominance was trivial. Non-significance of  $(W_R + V_R)$  between arrays also supported this. Error mean square used in the analysis of reanalysed data was approximately half that for original data and resulted in a lower, more positive estimate of  $H_1$  but a doubled estimate of  $H_2$ . In analysis of original data,  $E(\text{pool})$  was approximately 1.5 times the size of D suggesting that environment was a major source of variation. This was also found for top canopy scores. Negative estimates of F indicated an excess of recessive over dominant alleles.

Estimates of  $uv$ ,  $0.5F/\sqrt{D(H_1 - H_2)}$  and the ratio of dominant to recessive genes  $((\sqrt{4DH_1} + F)/(\sqrt{4DH_1} - F))$  were trivial due to unimportance of dominance.  $\sqrt{H_1/D}$  could not be estimated for either original or reanalysed data as  $H_1$  was negative in both instances.

Heritability estimates, both narrow sense and broad sense, ranged similarly to those of top canopy score. Narrow sense was greater than broad sense heritability for both original and reanalysed data, indicating preponderance of additive genetic variance. It is noted that heritabilities were estimated using genetical components including  $H_1$  and  $H_2$ . Although these latter component estimates were negative, they were not deleted from the appropriate equations or assumed as zero, since either approach would cause likely bias in the results. Correlations between common parent mean and  $(W_R + V_R)$  for each array, were not estimated as dominance was trivial according to all previous analyses and statistics.

Rankings of arrays with respect to proportion of dominant alleles in the parents, may be obtained from the  $W_R/V_R$  plots (Figures 3.2(a) and (b)). R-square estimates for original (0.81) and reanalysed (0.75) data

were quite respectable. Caution should be exercised in interpreting the distribution of arrays in terms of dominance, however, as previous results indicated that dominance was trivial. With this in mind, a brief appraisal of the results is presented in the following.

In Figure 3.2(a), array 2 (Zephyr and its crosses) was isolated distinctly from the other arrays and it was indicated that Zephyr had the highest proportion of recessive alleles. Approximately the same ( $W_R$ ,  $V_R$ ) position for the other four arrays suggested a relatively large additive component as discussed previously for top canopy score (Section 3.1.3). Following removal of array 2 and reanalysis of remaining data, array order on the  $W_R/V_R$  graph (Figure 3.2(b)) changed, as did distance between array points. Manchuria (5) displayed the lowest proportion of dominant alleles. Array 4 was at the lowest ( $W_R$ ,  $V_R$ ) position which was also found for reanalysed top canopy scores.

The number of effective factors ( $K$ ) and the ratio of additive to dominance genetic variance (Section 2.8.5) were not estimated as dominance was indicated as trivial. Both these statistics involve  $H_2$  for their calculation.

To summarise, net blotch resistance as measured by bottom canopy score, was conditioned mainly by additivity. This was also found for top canopy score. Dominance was trivial. According to Mather and Jinks (1971), the highly significant ( $P < 0.01$ ) departure of the regression slope from unity (original diallel data set) suggested disturbances due to epistasis and/or correlated gene distributions. However, no indication of these disturbances was shown by an analysis of variance of ( $W_R - V_R$ ) and it is concluded that the roles of epistasis and/or correlated gene distributions were equivocal.

Estimates of heterosis and heterobeltiosis for all crosses are presented in Table 3.9. Heterosis for Kaniera x Hassan cross was

significant ( $P \leq 0.05$ ) and positive as found for top canopy score. Manchuria x Zephyr cross had a significant, negative estimate of heterobeltiosis.

### 3.1.5 FLAG LEAF AREA

Genotype means plus associated statistics are presented in Table 3.13.

The most notable feature amongst parents was that Manchuria was significantly ( $P \leq 0.05$ ) more resistant to net blotch than all others. Among the other four parents, Zephyr (2) was significantly less resistant than Kaniere (1) and Hassan (3), but not Mata (4). As regards crosses, Manchuria crosses were as net blotch resistant as Manchuria, since they had a common significance group (f). Manchuria x Hassan cross was an exception. All crosses between the relatively susceptible parents were significantly ( $P \leq 0.05$ ) less resistant than Manchuria. Manchuria x Zephyr cross was significantly more resistant to net blotch than all other Zephyr crosses. Net blotch infection on the flag leaf of Zephyr is illustrated in Plate 3.1.

$V_R/W_R$  and  $(W_R + V_R)/(W_R - V_R)$  estimates are presented in Tables 3.14 and 3.15 respectively. Basic array statistics and the  $W_R/V_R$  regression equation plus associated statistics, are shown in Tables 3.5 and 3.6 respectively. The regression of  $W_R/V_R$  is plotted in Figure 3.3 (a). Relevant results of t-test for slope and analysis of variance of  $(W_R + V_R)$  and  $(W_R - V_R)$ , are presented in Table 3.7. The latter analysis of variance is given in Appendix 5.2.

$(W_R + V_R)$  and  $(W_R - V_R)$  were not significantly different between arrays (Table 3.7), suggesting that an additive-dominance model was applicable. Dominance was negligible as indicated by the test for  $(W_R + V_R)$ , which was not significant at the five per cent level.

**Table 3.13**

Genotype means (upper triangle) and estimates of heterosis and heterobeltiosis (bottom triangle) for flag leaf area (transformed to  $\sin^{-1} \sqrt{X/100}$ )

Male/Female	1	2	3	4	5
1	15.8 bcde #	22.3 abc	20.1 abcd	16.6 bcde	8.8 ef
2	-0.6    -2.6 (-7.6) † (-25.4)	29.9 a	25.2 abc	26.6 ab	10.4 def
3	5.1    33.9 (4.3) (27.2)	3.1    14.0 (-4.7) (-15.7)	14.3 cde	21.9 abc	15.8 bcde
4	-2.2    -11.7 (-5.2) (-23.9)	0.8    3.1 (-3.3) (-11.0)	3.9    21.6 (0.1) (0.5)	21.8 abc	8.0 ef
5	0.4    4.8 (-7.0) (-44.3)	-5.1    -33.0 (-19.5) (-65.2)	8.2    107.2 (1.5) (10.5)	-3.4    -29.8 (-13.8) (-63.3)	1.0 f

$\bar{F}_1 = 17.6$      $\bar{P} = 16.6$     Grand mean = 17.2    L.S.D.(5%) = 10.0    L.S.D.(1%) = 13.4

Heterosis: S.E. = 4.3    L.S.D.(5%) = 8.6    Heterobeltiosis: S.E. = 5.0    L.S.D.(5%) = 10.0

† Estimates of heterosis and heterobeltiosis (latter in brackets) expressed as deviations and percentages (left and right sets of figures for each cross, respectively).

# Figures underscored by different letters differ at the 5% level of significance.



Plate 3.1      Net blotch infection on the  
flag leaf of Zephyr.

Table 3.14

Estimates of variances ( $V_r$ ) and covariances ( $W_r$ ) of arrays for  
flag leaf area (transformed to  $\sin^{-1} \sqrt{X/100}$ )

Array	Block 1		Block 2		Block 3		Block 4	
	$V_r$	$W_r$	$V_r$	$W_r$	$V_r$	$W_r$	$V_r$	$W_r$
1	36.06	41.22	41.56	70.77	80.00	45.20	74.68	79.08
2	19.01	27.49	64.45	74.31	84.93	124.95	228.10	143.68
3	20.51	23.21	13.79	12.51	12.93	42.76	119.29	72.16
4	19.81	25.08	70.18	83.32	61.91	107.35	259.42	127.08
5	19.75	25.50	28.98	-2.56	59.42	95.72	79.91	25.13

Table 3.15

Estimates of  $(W_r + V_r)$  and  $(W_r - V_r)$  for flag leaf area  
(transformed to  $\sin^{-1} \sqrt{X/100}$  )

Array	Block 1		Block 2		Block 3		Block 4	
	$W_r + V_r$	$W_r - V_r$						
1	77.28	5.16	112.33	29.21	75.20	15.19	153.76	4.40
2	46.50	8.48	138.76	9.85	209.88	40.02	371.78	-84.41
3	43.73	2.69	26.30	-1.28	55.69	29.82	191.45	-47.13
4	44.89	5.27	153.50	13.14	169.26	45.45	386.50	-132.34
5	45.25	5.74	26.42	-31.54	155.15	36.30	105.04	-54.79

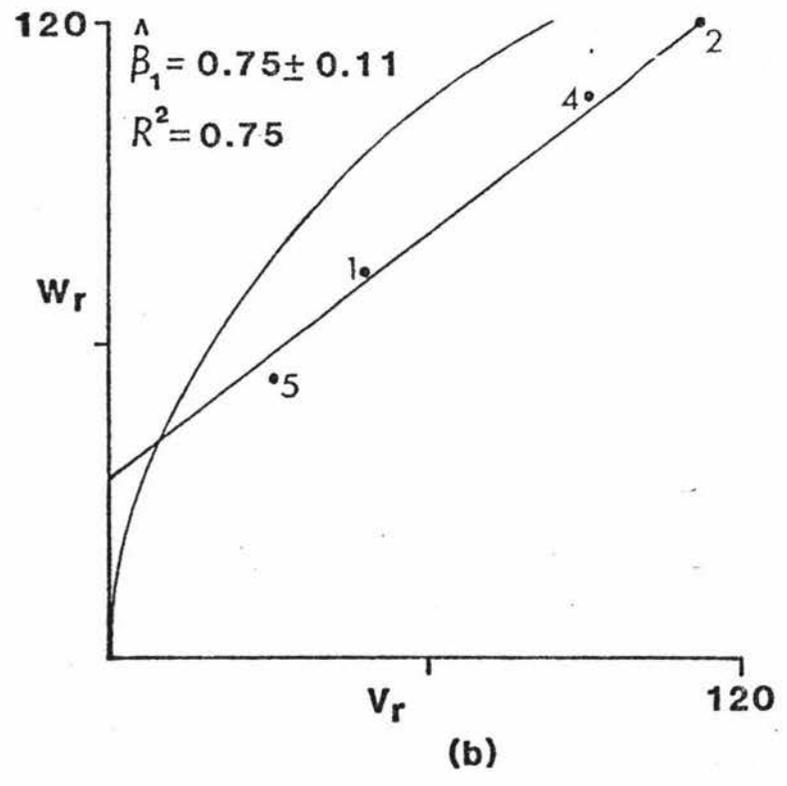
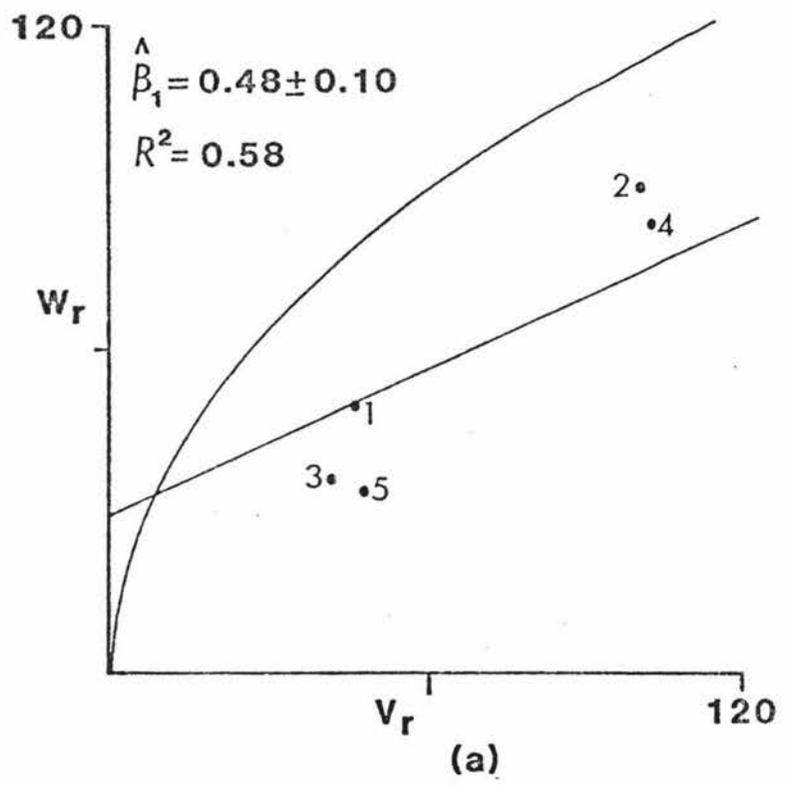


Figure 3.3: Regressions of  $W_r$  on  $V_r$  for flag leaf area;  
(a) all five parents, (b) parent 3 omitted.

Regression slope ( $W_R/V_R$ ) deviated highly significantly ( $P \leq 0.001$ ) from unity indicating disturbances due to environment and/or non-additivity other than dominance. Hence, there was a lack of agreement between these analyses again, regarding the complexity of the genetical models that they indicated.

In view of this and the comments made in Section 3.1.3, data were reanalysed (Section 2.8.3). Regression equations and relevant statistics for the reduced diallel data sets are presented in Table 3.16. All regressions were significant. Regression information for the four reduced diallel data sets was compared. Deletion of array 3 (Hassan and its crosses) and reanalysis of remaining data gave a result which conformed most adequately to an additive-dominance model. For this data, slope was closest to one and was less significantly ( $P \leq 0.05$ ) different from unity than for original data (Table 3.7). Standard error of regression was relatively low and R-square was acceptable. This result differed from those for top and bottom canopy scores where deletion of array 2 (Zephyr and its crosses) was most efficacious. Analysis of variance of  $(W_R + V_R)$  and  $(W_R - V_R)$  is given for data with array 3 deleted in Appendix 5.2 and significance of the appropriate sources of variation is presented in Table 3.7.  $(W_R + V_R)$  and  $(W_R - V_R)$  were not significantly different between arrays. The  $W_R/V_R$  regression plot for this reanalysed data is shown in Figure 3.3(b).

Genetic components of variation plus other relevant statistics (Mather and Jinks, 1971) are given in Table 3.7 for original and reanalysed data. Estimates of D were similar for both data sets and were relatively large compared with error mean squares obtained from analysis of variance with all genotypes included. Estimates were at least twice those calculated for top and bottom canopy scores.

Table 3.16

$W_R/V_R$  regression equations and associated statistics for analyses  
 conducted on reduced diallel data sets for flag leaf area  
 (transformed to  $\sin^{-1} \sqrt{X/100}$ ).

Array deleted	Regression equation	S.E.	F-test	R-square	S.E. ( $\hat{\beta}_1$ )
1	$W_R = 0.48V_R + 39.38$	39.15	21.03***	0.60	0.10
2	$W_R = 0.47V_R + 11.89$	21.54	43.19***	0.76	0.07
3	$W_R = 0.75V_R + 34.96$	27.29	42.73***	0.75	0.11
4	$W_R = 0.62V_R + 33.10$	32.74	18.84***	0.57	0.14

\*\*\* =  $P \leq 0.001$

H components were all negative due to correction for environment being greater than the combination of basic array statistics in the appropriate equations (Table 2.3).

Hence, additivity was of major importance while dominance was trivial. This was also indicated by analysis of variance of  $(W_R + V_R)$  and  $(W_R - V_R)$  for original and reanalysed data. As dominance was trivial, estimates of  $\sqrt{H_1/D}$ ,  $uv$ ,  $0.5F\sqrt{D(H_1 - H_2)}$  and ratio of dominant to recessive genes (Table 3.7), were therefore also regarded as trivial.  $F$  for both data sets was negative indicating a greater proportion of recessive than dominant alleles in parents. Results were similar to those for top and bottom canopy scores.

As regards heritability estimates, narrow sense was greater than broad sense heritability for original and reanalysed data (Table 3.7). This indicated a preponderance of additive genetic variance, which agreed with the findings for top and bottom canopy scores. Correlations between common parent mean and  $(W_R + V_R)$  for each array, were not calculated since dominance was trivial according to all previous analyses and statistics.

In view of the triviality of dominance indicated above, graphical order of the parental arrays (Figures 3.3(a) and (b)) is not discussed. Furthermore, the number of effective factors ( $K$ ) and the ratio of additive to dominance genetic variance were not estimated as they depend on positive expectations of  $H_2$ .

Heterosis and heterobeltiosis estimates for all crosses are presented in Table 3.13. Heterosis was not significant for any cross. However, for heterobeltiosis, estimates for Manchuria  $\times$  Zephyr and Manchuria  $\times$  Mata crosses were significant and negative. Manchuria  $\times$  Zephyr cross also gave a similar result for bottom canopy score.

### 3.1.6 SECOND LEAF AREA

Genotype means plus relevant statistics are given in Table 3.17.

Manchuria was significantly ( $P \leq 0.05$ ) more resistant to net blotch than the other parents and Zephyr was significantly less resistant than Kaniere and Hassan. Manchuria and crosses between Manchuria and Kaniere/Mata were not significantly different. Manchuria  $\times$  Zephyr cross was significantly more resistant than Zephyr but significantly less resistant than Manchuria. This result was not found previously. All crosses between relatively susceptible parents were not significantly different in their reactions.

Estimates of  $V_R/W_R$  and  $(W_R + V_R)/(W_R - V_R)$  are shown in Tables 3.18 and 3.19, respectively. Basic array statistics plus the  $W_R/V_R$  regression equation and associated statistics are shown in Tables 3.5 and 3.6 respectively. The  $W_R/V_R$  regression plot is shown in Figure 3.4. Significances of t-test for slope and major sources of variation in analysis of variance of  $(W_R + V_R)/(W_R - V_R)$  are presented in Table 3.7. The latter analysis of variance is given in Appendix 5.2.

$(W_R - V_R)$  were not significantly different between arrays indicating that an additive-dominance model was adequate to account for the data. Dominance was suggested, as variation due to  $(W_R + V_R)$  was significant ( $P \leq 0.05$ ). The  $W_R/V_R$  regression slope was not significantly different from unity, suggesting absence of non-additivity other than dominance. Hence, the analyses indicated jointly that an additive-dominance model was adequate and that dominance was present. This is the first instance where dominance was indicated clearly in these analyses (Table 3.7). Furthermore, no reanalysis was necessary (Mather and Jinks, 1971)!

Genetic components plus associated statistics (Mather and Jinks, 1971) are given in Table 3.7. D component was approximately four times

Table 3.17

Genotype means (upper triangle) and estimates of heterosis and heterobeltilosis (bottom triangle) for second leaf area (transformed to  $\sin^{-1} \sqrt{X/100}$ )

Male/Female	1		2		3		4		5
1	17.8 bcd #		28.3 ab		33.0 a		27.7 ab		10.5 de
2	2.3 (-5.9) †	8.8 (-17.3)	34.2 a		26.3 ab		30.0 ab		14.0 cd
3	14.1 (13.0)	74.6 (65.0)	-0.8 (-7.9)	-3.0 (-23.1)	20.0 bcd		26.1 ab		22.2 abc
4	7.3 (4.6)	35.7 (19.9)	1.4 (-4.2)	4.9 (-12.3)	4.6 (3.0)	21.3 (13.0)	23.1 abc		9.0 de
5	0.6 (-7.3)	6.1 (-41.0)	-4.1 (-20.2)	-21.5 (-59.1)	11.2 (2.2)	101.8 (11.0)	-3.6 (-14.1)	-28.7 (-61.0)	2.0 e

$\bar{F}_1 = 22.7$     $\bar{P} = 19.4$    Grand mean = 21.6   L.S.D.(5%) = 10.7   L.S.D.(1%) = 14.4

Heterosis: S.E. = 4.6   L.S.D.(5%) = 9.3   Heterobeltilosis: S.E. = 5.3   L.S.D.(5%) = 10.7

† Estimates of heterosis and heterobeltilosis (latter in brackets) expressed as deviations and percentages (left and right sets of figures for each cross, respectively).

# Figures underscored by different letters differ at the 5% level of significance.

Table 3.18

Estimates of variances ( $V_r$ ) and covariances ( $W_r$ ) of arrays for second leaf area ( transformed to  $\sin^{-1} \sqrt{X/100}$  )

Array	Block 1		Block 2		Block 3		Block 4	
	$V_r$	$W_r$	$V_r$	$W_r$	$V_r$	$W_r$	$V_r$	$W_r$
1	109.40	62.57	51.22	90.06	139.84	163.27	157.13	97.75
2	13.49	6.29	120.74	133.59	147.47	132.23	246.45	123.60
3	24.04	-22.96	25.12	51.93	68.74	-6.63	73.96	47.96
4	58.50	11.08	110.73	103.92	126.06	174.65	183.30	103.43
5	61.87	43.81	58.52	57.33	73.83	119.48	87.60	33.37

Table 3.19

Estimates of  $(W_R + V_R)$  and  $(W_R - V_R)$  for second leaf area(transformed to  $\sin^{-1} \sqrt{X/100}$  )

Array	Block 1		Block 2		Block 3		Block 4	
	$W_R + V_R$	$W_R - V_R$						
1	171.98	-46.83	141.28	38.84	303.10	23.43	254.89	-59.38
2	19.78	-7.20	254.32	12.85	279.70	-15.24	370.05	-122.85
3	1.08	-46.99	77.05	26.81	62.11	-75.37	121.92	-26.00
4	69.58	-47.42	214.65	-6.81	300.70	48.59	286.73	-79.87
5	105.68	-18.06	115.85	-1.20	193.31	45.64	120.97	-54.23

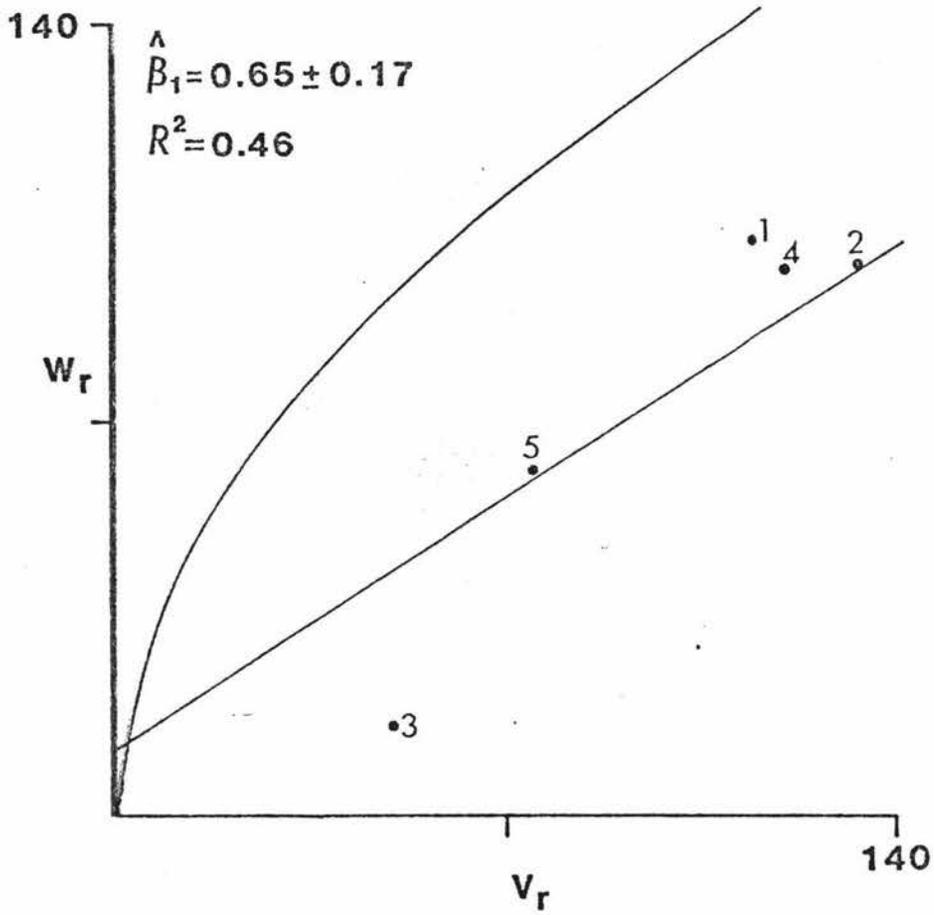


Figure 3.4: Regression of  $W_r$  on  $V_r$  for second leaf area.

the size of positive values of  $H_1$  and  $H_2$ , suggesting that additivity was of major importance. Positive values of both  $H_1$  and  $H_2$  were not estimated previously and indicated that dominance was relatively important. This supported the significance of  $(W_r + V_r)$  between arrays found earlier.  $F$  was negative and relatively small compared with estimates of  $H$  and  $D$  components and indicated that there was a greater proportion of recessive than dominant alleles in parents. Ratio of additive genetic variance to dominance genetic variance ( $\sigma_A^2/\sigma_D^2$ ) was estimated assuming no epistasis and equal gene frequencies (suggested presently). Additive genetic variance was approximately seven times dominance genetic variance and confirmed previous findings in this study that additivity was of major importance in controlling resistance to net blotch.

Ratio  $\sqrt{H_1/D}$  was 0.56 indicating partial dominance (Table 3.7). Gene frequencies of increasing and decreasing alleles were approximately equal ( $uv = 0.23$ ). Ratio of dominance to additive effects was not constant over all loci, nor was it fully independently distributed (absolute value of  $(0.5F/\sqrt{D(H_1 - H_2)})$  was between 0 and 1). Hence, partial dominance probably resulted from complete dominance at some loci and no dominance at other loci, rather than partial dominance at all loci (Mather and Jinks, 1971). Proportion of dominant to recessive alleles over all parents was 0.78 to 1 (Table 3.7) and was in agreement with interpretation of the negative  $F$  estimate. Number of effective factors was estimated as approximately one (1.32).

The correlation between common parent mean and  $(W_r + V_r)$  for each array (Table 3.7) was 0.29 which was not significantly different from zero. The small, positive, correlation indicated that there was a slight association between increasing dominance alleles and increasing resistance to net blotch.

Rankings of arrays with respect to the proportion of dominant alleles in the parents, may be obtained from the  $W_R/V_R$  plot presented in Figure 3.4. R-square value of 0.46 suggested caution in interpreting rankings. Order of arrays was Hassan, Manchuria, Kaniere, Mata and Zephyr, indicating that Hassan had the highest proportion of dominant alleles and Zephyr the lowest. Manchuria's ( $W_R, V_R$ ) estimate was relatively small compared to Zephyr's.

Narrowsense heritability (51.44 per cent) was slightly less than broadsense heritability (57.63 per cent), which never occurred previously. Close similarity between estimates suggested that additivity was of major importance, while dominance was less important. The preponderance of additivity was also indicated by previous analyses.

In summary, net blotch resistance as measured by second leaf area infected, was conditioned by a single, partially dominant effective factor. Additive genetic variance accounted for the majority of genetic variation. Non-additivity, other than dominance, was not indicated by any analyses.

Estimates of heterosis and heterobeltiosis accompany genotype means in Table 3.17. Heterosis was significant ( $P \leq 0.05$ ) for Manchuria x Hassan and Kaniere x Hassan crosses. Both estimates were positive. Heterobeltiosis for Manchuria x Mata and Manchuria x Zephyr crosses was significant ( $P \leq 0.05$ ) and negative. The result for the latter cross was found previously for bottom canopy score and flag leaf area.

### 3.1.7 LEAF SECTION AREA

Genotype means and relevant statistics are presented in Table 3.20. Measurements were made on a percentage scale as for flag and second leaf areas (Section 2.6.1) and transformed similarly using arcsin (Section 2.7). Grand mean (13.5) was lower than that for top and bottom canopy

Table 3.20 Genotype means (upper triangle) and estimates of heterosis and heterobeltiosis (bottom triangle) for leaf section area (transformed to  $\sin^{-1} \sqrt{X/100}$  )

Male/Female	1	2	3	4	5
1	19.0 abcd #	31.3 a	31.3 a	16.5 bcde	6.9 def
2	17.5 126.4 (12.3) † (64.7)	8.7 bcdef	21.4 abc	16.6 bcde	3.9 ef
3	10.4 49.8 (8.5) (37.3)	5.7 36.2 (-1.4) (-6.1)	22.8 ab	13.5 bcdef	2.6 ef
4	3.3 25.0 (-2.5) (-13.2)	8.6 106.8 (7.9) (90.8)	-1.6 -10.6 (-9.3) (-40.8)	7.4 cdef	0.0 f
5	-2.6 -27.4 (-12.1) (-63.7)	-0.5 -11.5 (-4.8) (-55.2)	- 8.8 -77.2 (-20.2) (-88.6)	-3.7 -100.0 (-7.4) (-100.0)	0.0 f

$\bar{F}_1 = 14.4$      $\bar{P} = 11.6$     Grand mean = 13.5    L.S.D. (5%) = 12.5    L.S.D. (1%) = 16.8

Heterosis: S.E. = 5.5    L.S.D. (5%) = 11.1    Heterobeltiosis: S.E. = 6.1    L.S.D. (5%) = 12.5

† Estimates of heterosis and heterobeltiosis (latter in brackets expressed as deviations and percentages (left and right sets of figures for each cross, respectively).

# Figures underscored by different letters differ at the 5% level of significance.

scores, and than flag and second leaf areas infected.

Manchuria leaf sections were fully resistant to net blotch, as no symptoms were detected. This result did not occur for intact plant tissue. With respect to parents, Zephyr and Mata were in the same significance group as Manchuria (group "f") while only Kaniere and Hassan were significantly ( $P \leq 0.05$ ) less resistant. No significant difference between Manchuria and Zephyr in their net blotch reactions, was not found previously. Array 4 (Mata and crosses), array 5 (Manchuria and crosses) and Zephyr were not significantly different in their reactions. Manchuria and its crosses were significantly ( $P \leq 0.05$ ) distinct from only Hassan and three crosses. With the exception of Manchuria x Kaniere cross, the former genotypes were also different from Kaniere.

Estimates of variances and covariances of arrays and their sums and differences are given in Tables 3.21 and 3.22 respectively. Basic array statistics are shown in Tables 3.5 and 3.6' respectively. The  $W_r/V_r$  regression plot is presented in Figure 3.5(a). Relevant results of t-test for slope and analysis of variance of  $(W_r + V_r)$  and  $(W_r - V_r)$ , are presented in Table 3.7. The latter analysis of variance is detailed in Appendix 5.2.

$(W_r - V_r)$  were significantly ( $P \leq 0.05$ ) different between arrays while source of variation attributable to  $(W_r + V_r)$  was not significant (Table 3.7). This indicated that non-additivity, other than dominance, was present and therefore data could not be adequately accounted for by an additive-dominance model alone (Mather and Jinks, 1971). Regression slope departed highly significantly ( $P \leq 0.01$ ) from unity and supported this finding.

Data were therefore reanalysed (Section 2.8.3) and regression equations and appropriate statistics for the reduced diallel data sets

Table 3.21

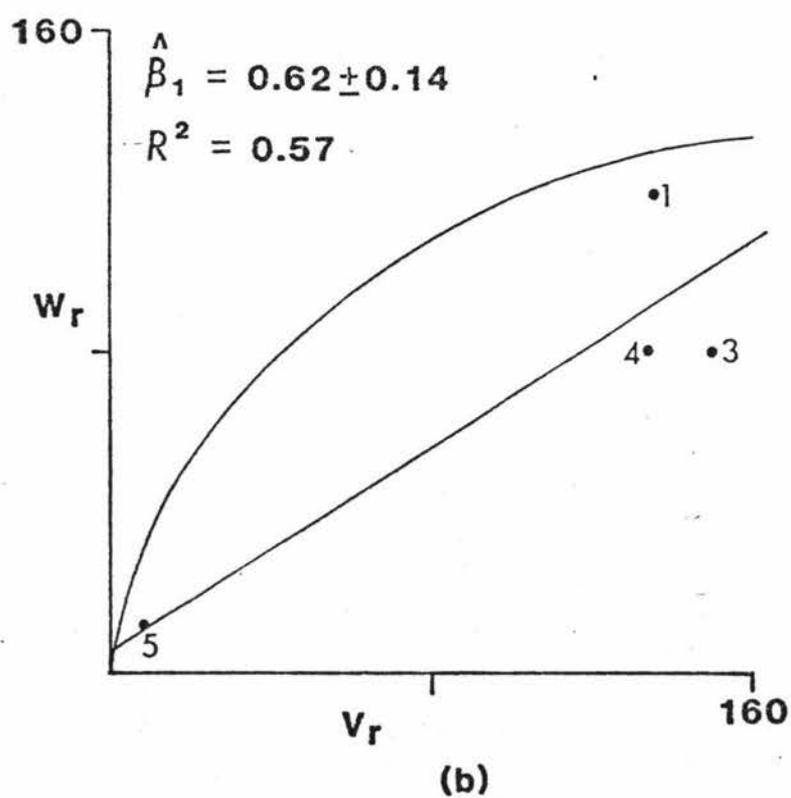
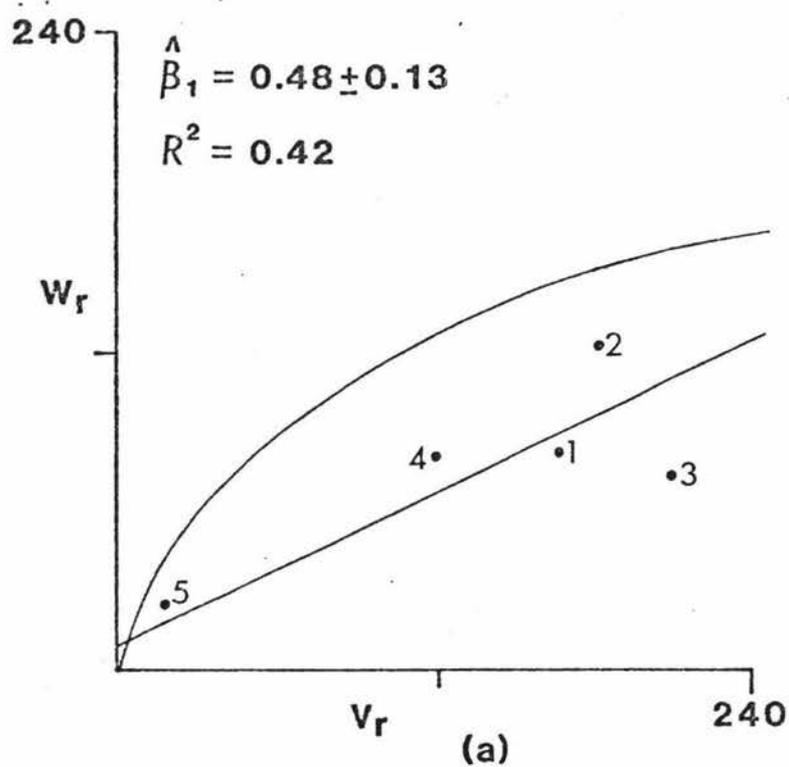
Estimates of variances ( $V_r$ ) and covariances ( $W_r$ ) of arrays for leaf section area  
(transformed to  $\sin^{-1} \sqrt{X/100}$  )

Array	Block 1		Block 2		Block 3		Block 4	
	$V_r$	$W_r$	$V_r$	$W_r$	$V_r$	$W_r$	$V_r$	$W_r$
1	266.93	104.64	44.80	-17.10	203.50	91.43	150.23	144.91
2	304.29	109.02	245.70	203.02	82.91	53.70	97.74	134.11
3	165.58	52.09	143.27	-27.19	256.62	52.80	276.30	223.01
4	129.58	93.44	203.12	170.27	82.40	-13.93	62.11	74.26
5	0.00	0.00	42.46	71.43	21.41	12.95	8.21	9.17

Table 3.22

Estimates of  $(W_r + V_r)$  and  $(W_r - V_r)$  for leaf section area  
 (transformed to  $\sin^{-1} \sqrt{X/100}$  )

Array	Block 1		Block 2		Block 3		Block 4	
	$W_r+V_r$	$W_r-V_r$	$W_r+V_r$	$W_r-V_r$	$W_r+V_r$	$W_r-V_r$	$W_r+V_r$	$W_r-V_r$
1	371.57	-162.29	27.69	-61.90	294.93	-112.07	295.14	-5.32
2	413.31	-195.28	448.72	-42.67	136.61	-29.21	231.85	36.36
3	217.67	-113.49	116.08	-170.46	309.42	-203.81	499.30	-53.29
4	223.01	-36.14	373.39	-32.85	68.47	-96.34	136.37	12.15
5	0.00	0.00	113.90	28.97	34.36	-8.46	17.38	0.97



**Figure 3.5:** Regressions of  $W_r$  on  $V_r$  for leaf section area; (a) all five parents, (b) parent 2 omitted.

are shown in Table 3.23.

All regressions were significant. Regression information for the four reduced diallel data sets was compared. Deletion of array 1 (Kaniere and its crosses) and reanalysis of remaining data gave a result which conformed most adequately to an additive-dominance model. For this data, slope was closest to one, standard error (S.E.) of regression was relatively low and R-square was moderate. Despite t-test for departure of regression slope from unity still being significant (Table 3.7), the significance level was lower ( $P \leq 0.05$  compared with  $P \leq 0.01$  for original data). Analysis of variance of  $(W_R + V_R)$  and  $(W_R - V_R)$  is given for data with array 1 deleted in Appendix 5.2 and significances of relevant sources of variation are presented in Table 3.7.  $(W_R + V_R)$  and  $(W_R - V_R)$  were not significantly different between arrays, indicating, now, that an additive-dominance model was adequate. As data conformed more closely to a simple genetic model following deletion of array 1, genetic components and other statistics were estimated for this reduced data set as well as for original data. The  $W_R/V_R$  regression plot for the reanalysed data is shown in Figure 3.5(b).

Genetic components plus associated statistics (Sections 2.8.4 and 2.8.5) are presented in Table 3.7 for original and reanalysed data. Both data sets gave relatively large D components suggesting that additivity was important in the genetic system. This was in agreement with the results for analysis of variance of  $(W_R + V_R)$  and  $(W_R - V_R)$  for reanalysed data.  $H_1$  and  $H_2$  (original data) were each approximately half that of D while dominance was trivial for reanalysed data (negative estimates of  $H_1$  and  $H_2$ ). The importance of additive genetic variance was shown for original data by the ratio of  $\sigma_A^2/\sigma_D^2$  being 4.26 (Table 3.7). Relatively high negative estimates of F indicated a greater proportion of recessive than dominant alleles in the parents. Error mean squares were

Table 3.23

$W_R/V_R$  regression equations and associated statistics for analysis conducted on reduced diallel data sets for leaf section area

(transformed to  $\sin^{-1} \sqrt{X/100}$ )

Array deleted	Regression equation	S.E.	F-test	R-square	S.E. ( $\hat{\beta}_1$ )
1	$W_R = 0.62V_R + 5.92$	48.58	18.76***	0.57	0.14
2	$W_R = 0.58V_R + 9.83$	56.20	22.02***	0.61	0.12
3	$W_R = 0.45V_R + 22.81$	36.80	20.68***	0.60	0.10
4	$W_R = 0.48V_R + 9.06$	58.23	15.39**	0.52	0.12

\*\* =  $0.01 \geq P > 0.001$ ;

\*\*\* =  $P \leq 0.001$ .

larger than D and H components.

As regards original data, partial dominance ( $\sqrt{H_1/D} = 0.70$ ) and approximately equal frequencies of increasing and decreasing alleles ( $uv = 0.24$ ) were indicated (Table 3.7). An estimate of the consistency of expression of the degree of dominance across all segregating loci ( $0.5F/\sqrt{D(H_1 - H_2)}$ ), was unable to be interpreted as it was well beyond absolute value limits of 0 and 1. Ratio of dominant to recessive alleles ( $(\sqrt{4DH_1} + F)/(\sqrt{4DH_1} - F)$ ) was negligible. The number of effective factors (Table 3.7) was approximately one (0.96). For reanalysed data, dominance was trivial (Table 3.7) and therefore the above statistics were also regarded as trivial.

Estimates of narrow-sense heritability (52.63 per cent) and broad-sense heritability (57.17 per cent) for original data indicated that additive genetic variance accounted for approximately 90 per cent of total genetic variance. This provided further support for the importance of additivity found previously. For reanalysed data narrow-sense heritability was greater than broad-sense heritability and this result was interpreted similarly. There was very good agreement between heritability estimates obtained from original data and second leaf area data.

Correlation between common parent mean and  $(W_r + V_r)$  for each array is presented for original data in Table 3.7. Although analysis of variance of  $(W_r + V_r)$  did not indicate dominance,  $H_1$  and  $H_2$  were positive and this suggested that dominance could have been present. However, an estimate for reanalysed data was not calculated as dominance was indicated as trivial (Table 3.7). The correlation for original data (0.38) was positive, low to moderate and significantly ( $P \leq 0.05$ ) different from zero. This suggested that there was a moderate association between increasing dominance and reduction in leaf section area infected, that is,

increasing net blotch resistance. A similar result was found for second leaf area. However, the reanalysed data did not indicate any dominance.

Rankings of arrays with respect to the proportion of dominant alleles in the parents may be obtained from the  $W_R/V_R$  plots (Figures 3.5(a) and (b)). For original data (Figure 3.5(a)), Manchuria (array 5) was closest to the origin indicating that it had the greatest proportion of dominant alleles. In decreasing order of dominance, the other parents were Mata, Kaniere, Zephyr and Hassan. The distinction between Manchuria and its crosses (5) and other array estimates presented in Figure 3.5(a), was not shown using other assessment techniques in this study. As regards reanalysed data (Figure 3.5(b)), all previous analyses indicated that dominance was trivial. Therefore, Figure 3.5(b) is not discussed, other than to comment that Manchuria had the lowest ( $W_R, V_R$ ) estimate. This feature was noted above for the original data plot.

Using the leaf section area technique, then, it was indicated that net blotch resistance was conditioned by a single, partially dominant, effective factor. Additivity was relatively important.

Heterosis and heterobeltiosis estimates are presented in Table 3.20. Kaniere x Zephyr cross exhibited significant ( $P \leq 0.05$ ) and positive heterosis. This was not found previously. For heterobeltiosis, only Manchuria x Hassan cross was significant. The estimate was negative.

### 3.1.8 OTHER ASPECTS OF NET BLOTCH

In addition to investigating resistance to net blotch, studies were also conducted to examine the effect of the disease on several agronomic features of barley. This included formulation of a relationship between net blotch intensity on the flag and second leaves, and grain yield loss. Flag and second leaves may make significant contributions

to grain yield, particularly the former (Thorne, 1973; Evans and Wardlaw, 1976) and it was therefore of interest to examine this feature. The studies were detailed in Materials and Methods and involved fungicide treated (control) and untreated (diseased) diallel parents. Only the high nitrogen environment was used for the studies as net blotch was absent in the other environment.

#### 3.1.8.1 CONTROL VS. DISEASE IN DIALLEL PARENTS

Treatment means and significance of F-test for treatments, are presented in Table 3.24 for each character. Pooled analysis of variance over control and disease treatments is given in Appendix 6.

A major feature of the results was the nonsignificant difference between disease and control (fungicide treated) treatments for all characters except heading date. The latter was significant at the 5 per cent level. The results indicated that net blotch prevalence and intensity were relatively uniform across both treatments and/or that residual error was large. Where relatively large errors occurred (Appendix 6), greater differences between treatment means were necessary to achieve significance (Steel and Torrie, 1980). That is, large errors decreased the ability to detect significant treatment differences.

Table 3.24 also shows that net blotch infection on intact plant tissue was low. This is supported by treatment and pooled means for top and bottom canopy scores, and flag and second leaf areas infected. Possible reasons for this are given in Chapter 4.

#### 3.1.8.2 YIELD LOSS RELATIONSHIPS

Estimates of losses of grain yield and 100 kernel weight between control and disease treatments, expressed as deviations and percentages, are presented for each diallel parent in Table 3.25. L.S.D.'s (5 per cent level of significance) are included.

Table 3.24

Treatment means, pooled means (plus associated standard errors in parentheses) and variance ratio significance for treatments (control vs. disease).

Character	Disease treatment		Control treatment		Pooled mean		F-test sig.
Top canopy score (arcsin score)	21.0	(1.6)	16.5	(1.3)	18.8	(1.0)	(NS)
Bottom canopy score (arcsin score)	21.4	(1.8)	19.1	(1.0)	20.3	(1.0)	NS
Flag leaf area (arcsin percentage)	16.6	(1.3)	11.5	(1.0)	14.0	(0.8)	NS
Second leaf area (arcsin percentage)	19.4	(1.8)	14.2	(1.3)	16.8	(1.1)	NS
Leaf section area (arcsin percentage)	11.6	(1.8)	15.4	(2.5)	13.5	(1.5)	NS
Heading date (days)	61.2	(0.2)	63.6	(0.4)	62.4	(0.2)	*
Plant height (cm)	104.5	(0.6)	104.5	(1.2)	104.5	(0.7)	NS
Tillers per plant	30.2	(0.5)	25.8	(1.7)	28.0	(0.9)	NS
Spikelet number per ear	26.7	(0.3)	26.8	(0.3)	26.8	(0.2)	NS
Grain number per ear ( $10 \log_{10}$ number)	12.9	(0.3)	13.1	(0.3)	13.0	(0.2)	NS
Grain yield (g)	13.08	(0.66)	10.62	(0.91)	11.85	(0.56)	NS
100 kernel weight (g)	4.26	(0.10)	3.86	(0.20)	4.06	(0.11)	NS
Physiological brown spot (arcsin percentage)	26.8	(1.1)	29.9	(1.1)	28.4	(0.8)	NS

NS =  $P > 0.10$ (NS) =  $0.10 \geq P > 0.05$ \*  $0.05 \geq P > 0.01$

Table 3.25.

Grain yield and 100 kernel weight losses for control and disease treatments.

Attribute		Kaniere	Zephyr	Hassan	Mata	Manchuria	Mean
GRAIN YIELD (g)	Control mean	12.83	7.13	7.90	10.27	15.01	10.62
	Disease mean	13.93	11.18	14.41	7.14	18.76	13.08
	Yield loss	- 1.10 NS	- 4.05 NS	- 6.51 *	3.13 NS	- 3.75 NS	- 2.46
	Yield loss (%)	-16.49	-56.73	-82.49	30.42	-25.02	-23.16
100 KERNEL WEIGHT (g)	Control mean	4.04	3.48	3.68	4.25	3.84	3.86
	Disease mean	4.54	4.45	4.21	4.29	3.80	4.26
	100 kernel weight loss	- 0.50 NS	- 0.97 NS	- 0.53 NS	- 0.04 NS	0.04 NS	- 0.40
	100 kernel weight loss (%)	-12.18	-28.04	- 0.94	- 0.94	1.04	-10.36

L.S.D. (yield loss) = 5.48 ( $P \leq 0.05$ )    L.S.D. (100 kernel weight loss) = 1.10 ( $P \leq 0.05$ )NS =  $P > 0.10$ \* =  $0.05 \geq P > 0.01$

From Table 3.25, most estimates of grain yield and 100 kernel weight losses were negative, but they were non-significant. That is, there were generally no changes in grain yield and 100 kernel weight between disease and control treatments. Only Hassan exhibited a significant ( $P \leq 0.05$ ) gain in grain yield. Results were probably an artifact arising from the poor onset of disease, noted previously.

Grain yield ( $Y$ ) in the disease treatment was regressed against net blotch infection on the flag ( $X_1$ ) and second ( $X_2$ ) leaves to determine the relative importance of each leaf in contributing to yield. This was conducted across all genotypes and the results for the multiple regression (stepwise) are presented in Tables 3.26 (analysis of variance) and 3.27. Included in the latter Table are R-square increments, partial regression coefficients ( $\hat{\beta}$ ) and their standard errors, and standardised partial regression coefficients (Beta) (Steel and Torrie, 1980). The latter estimates were unaffected by original scales, and thereby enabled the relative importance of  $X_1$  and  $X_2$  to be determined by comparison (Steel and Torrie, 1980). The R-square change resulting from inclusion of  $X_2$  in the regression equation also provided information on importance. This was possible since regression was conducted in a stepwise manner (Steel and Torrie, 1980).

The regression was highly significant ( $P \leq 0.01$ ) (Table 3.26) and the R-square value of 0.55 was moderate. Standardised partial regression coefficients (Table 3.27) showed that  $X_1$  was approximately three times as important as  $X_2$  in determining grain yield ( $Y$ ). Relative importance was also indicated by low R-square change resulting from inclusion of  $X_2$  in the regression equation. It is concluded that  $X_2$  was trivial in its contribution to grain yield and may therefore be omitted. There was a decrease in  $Y$  with increase in  $X_1$ , as indicated by a partial regression coefficient of  $-0.47$  (Table 3.27). That is, an inverse

Table 3.26 Multiple regression of grain yield against flag and second leaf areas infected with net blotch in disease treatment - analysis of variance.

Source of variation	D.F.	Mean square	F	Signif.
Regression	2	128.16	10.32	**
Residual	17	12.42		
			$R^2 = 0.55$	

Table 3.27 Multiple regression statistics for grain yield against flag and second leaf areas infected with net blotch in disease treatment.

Variable	$R^2$ increment	$\hat{\beta}$	Standard error ( $\hat{\beta}$ )	Beta
Flag leaf ( $X_1$ )	0.52	- 0.47	0.16	-1.04
Second leaf ( $X_2$ )	0.03	0.14	0.13	0.36
Constant		18.11		

relationship existed between the variables. But, the relationship is weak, as shown by the large variation unexplained by regression (48%).

### 3.2 OTHER CHARACTERS

#### 3.2.1 ANALYSIS OF VARIANCE

Remaining characters were analysed for low and high nitrogen levels firstly, and then pooled phenotypic analyses over nitrogen levels were conducted, using PHANIE. Analyses (single nitrogen levels and pooled) for each character are presented in Appendix 7. Analyses for parents ( $n$ ), hybrids, and parents plus hybrids ( $n(n + 1)/2$ ) are included for single nitrogen levels. However, only pooled analyses conducted with all genotypes (parents and hybrids) included, are presented since most error mean squares for parents and hybrids for respective characters in single nitrogen levels, were not significantly different (Appendix 2). Genotype means for single and pooled nitrogen levels are given in Appendix 8. Pooled analyses over nitrogen levels are discussed presently, following examination of the assumption of homogeneous error variances.

##### 3.2.1.1 HOMOGENEITY OF ERROR VARIANCES AND COEFFICIENTS OF VARIATION

Results of chi-square tests for homogeneity of error variances (Bartlett, 1947; Steel and Torrie, 1980) are given in Table 3.28. Heading date, plant height, tillers per plant, spikelet number per ear, grain yield, 100 kernel weight and physiological brown spot had heterogeneous error variances over the two pooled environments (nitrogen levels). This indicated that estimates of significance levels, especially for genotype-nitrogen means, and variance components for these characters, were possibly biased (Snedecor and Cochran, 1967; Steel and Torrie, 1980).

Table 3.28. Homogeneity of error variances,  
and coefficients of variation.

Character	Chi <sup>2</sup>	Probability*	C.V. %
Heading date	9.46	0.00	2.76
Plant height	22.24	0.00	5.29
Tillers per plant	57.28	0.00	19.10
Spikelet number per ear	17.72	0.00	5.66
Grain number per ear	40.11	0.74	5.67
Grain yield	44.15	0.00	20.60
100 kernel weight	14.24	0.00	6.50
Physiological brown spot	4.41	0.03	21.84

\* Probabilities <0.05 mean that error variances are heterogeneous.

Probabilities >0.05 mean that error variances are homogeneous.

Nevertheless, these estimates were used for comparative purposes with other characters as the pooled statistics are still the best under the circumstances.

Coefficients of variation (Table 3.28) ranged from 2.76 per cent for heading date to 21.84 per cent for physiological brown spot. These estimates indicate that the experiment had acceptable precision (Balaam, 1972).

### 3.2.1.2 SIGNIFICANCE OF SOURCES OF VARIATION

Environment (nitrogen) means and pooled grand means are presented in Table 3.29 for all characters. Included are appropriate standard errors and F-test for nitrogen effects. Nitrogen effects were significant for all characters except heading date.

Estimates of genotypes, genotype-nitrogen, blocks (nitrogen) and error variance components are given in Table 3.30. Accompanying these are significance levels (F-test) and standard errors of variance components. Ratios of variance components to error variance are also included for each character and indicate relative importance of respective variance components.

Genotypes were significantly different for all characters except plant height, grain yield and physiological brown spot (Table 3.30). Apart from heading date, tillers per plant and grain number per ear, all genotype - nitrogen interactions were significant (Table 3.30). Genotypic variance was relatively more important than genotype - nitrogen variance for five characters. The latter variance was of greatest importance for physiological brown spot and grain yield.

Blocks (within nitrogen levels) were significantly different for four of the eight characters (Table 3.30), indicating lack of uniform environmental conditions within each room. The direction of blocking

Table 3.29. Environmental means, pooled grand means (plus associated standard errors in parentheses) and variance ratio significance for environments (nitrogen)

Character	Low nitrogen	High nitrogen	Pooled grand mean	F-test sig.
Heading date	54.8 (0.2)	55.7 (0.1)	55.2 (0.1)	NS
Plant height	68.9 (0.4)	103.9 (0.8)	86.4 (0.4)	***
Tillers per plant	13.9 (0.2)	29.2 (0.7)	21.6 (0.4)	***
Spikelet number per ear	22.0 (0.1)	26.5 (0.2)	24.2 (0.1)	***
Grain number per ear	11.4 (0.1)	12.7 (0.1)	12.1 (0.1)	***
Grain yield	8.94 (0.12)	12.37 (0.38)	10.66 (0.20)	*
100 kernel weight	5.94 (0.03)	4.59 (0.05)	5.26 (0.03)	***
Physiological brown spot	17.3 (0.5)	24.1 (0.7)	20.7 (0.4)	*

NS =  $P > 0.10$ ; \* =  $0.05 \geq P > 0.01$ ; \*\*\* =  $P \leq 0.001$ .

Table 3.30

Estimates of components of variance, together with their standard errors (below) and significances

Character	$\hat{\sigma}_G^2$	$\hat{\sigma}_{G-N}^2$	$\hat{\sigma}_{B(N)}^2$	$\hat{\sigma}$	$\hat{\sigma}_G^2/\hat{\sigma}^2$	$\hat{\sigma}_{G-N}^2/\hat{\sigma}^2$
Heading date	73.73 *** 26.24	0.39 (NS) 0.36	0.19 * 0.17	2.32 0.35	31.78	0.17
Plant height	9.16 NS 8.76	19.42 *** 8.75	-0.51 NS 0.49	20.93 3.19	0.44	0.93
Tillers per plant	12.97 ** 6.03	3.15 (NS) 2.69	0.61 NS 0.89	16.96 2.59	0.76	0.19
Spikelet number per ear	7.98 *** 3.08	0.94 ** 0.50	0.09 NS 0.11	1.88 0.29	4.24	0.50
Grain number per ear	1.75 *** 0.64	0.02 NS 0.05	0.00 NS 0.02	0.47 0.07	3.72	0.04
Grain yield	0.24 NS 1.28	3.66 *** 1.73	1.91 *** 1.12	4.82 0.73	0.05	0.76
100 kernel weight	0.16 ** 0.07	0.04 ** 0.03	0.05 *** 0.03	0.12 0.02	1.33	0.33
Physiological brown spot	1.18 NS 12.67	44.36 *** 17.51	1.92 * 1.66	20.49 3.13	0.06	2.16

NS =  $P \geq 0.10$

\* =  $0.05 \geq P > 0.01$

(NS) =  $0.10 \geq P > 0.05$

\*\* =  $0.01 \geq P > 0.001$

\*\*\* =  $P \leq 0.001$

was from the door to back of the rooms. Results indicated that barley trials conducted in climate rooms should be adequately blocked to take account of such environmental non-uniformity.

As regards single environments (Appendix 7), genotypes were significantly ( $P \leq 0.001$ ) different for all characters except plant height in high nitrogen environment. Significant genotypic variation indicated that at least one type of genetic variation was present. Only characters exhibiting significant genotypic differences in the single nitrogen level analyses of variance, were subjected to diallel analysis. As net blotch occurred only in the high nitrogen level, there was no justification for conducting diallel analyses pooled over nitrogen levels, since all other characters were of minor importance.

### 3.2.2 DIALLEL ANALYSIS

#### 3.2.2.1 DATA CONFORMITY TO ADDITIVE-DOMINANCE MODEL

Basic array statistics and analysis of variance of ( $W_r + V_r$ ) and ( $W_r - V_r$ ) are presented in Appendix 9 for characters which had significant genotypic differences. Results for the  $W_r/V_r$  regression analyses of these characters are given in Tables 3.31 (low nitrogen) and 3.32 (high nitrogen). Included are the regression equation, R-square value, F-test and its significance and standard errors of regression and slope. Where F-test was significant ( $P \leq 0.05$ ), a t-test to judge the significance of departure of the regression slope from unity (Mather and Jinks, 1971), was conducted. This result together with the significance levels (F-test) for differences between ( $W_r + V_r$ ) and ( $W_r - V_r$ ) arrays, are given in Table 3.33. These two analyses are essential for determining applicability of an additive-dominance model to account for diallel cross data (Mather and Jinks, 1971).

Table 3.31

$W_r/V_r$  regression equations and associated statistics  
for characters in low nitrogen environment

Character	Regression equation	S.E.	F-test	R-square	S.E. ( $\hat{\beta}_1$ )
Heading date	$W_r = 0.24V_r - 24.58$	17.36	5.23 *	0.23	0.10
Plant height	$W_r = 0.79V_r + 7.85$	8.83	114.91 ***	0.86	0.07
Tillers per plant	$W_r = 0.66V_r + 3.74$	2.32	40.56 ***	0.69	0.10
Spikelet number per ear	$W_r = 0.98V_r + 2.08$	1.19	246.25 ***	0.93	0.06
Grain number per ear	$W_r = 1.38V_r + 0.49$	0.33	63.14 ***	0.79	0.17
Grain yield	$W_r = 0.79V_r - 0.66$	1.37	10.88 **	0.38	0.24
100 kernel weight	$W_r = 0.59V_r - 0.01$	0.07	16.69 ***	0.48	0.14
Physiological brown spot	$W_r = 0.48V_r - 8.41$	17.93	7.98 *	0.31	0.17

\* =  $0.05 \geq P > 0.01$

\*\* =  $0.01 \geq P > 0.001$

\*\*\* =  $P \leq 0.001$

Table 3.32

$W_r/V_r$  regression equations and associated statistics for characters in high nitrogen environment

Character	Regression equation	S.E.	F-test	R-square	S.E. ( $\hat{\beta}_1$ )
Heading date	$W_r = 0.03V_r - 2.80$	6.14	0.32 NS	0.02	0.05
Tillers per plant	$W_r = 0.28V_r + 1.43$	13.31	8.82 **	0.33	0.09
Spikelet number per ear	$W_r = 1.10V_r + 2.95$	2.14	48.49 ***	0.73	0.16
Grain number per ear	$W_r = 0.89V_r + 0.47$	0.50	17.65 ***	0.50	0.21
Grain yield	$W_r = 0.16V_r + 0.91$	11.23	6.52 NS	0.03	0.23
100 kernel weight	$W_r = 0.16V_r - 0.04$	0.18	0.77 NS	0.04	0.18
Physiological brown spot	$W_r = 0.75V_r + 15.06$	22.35	50.12 ***	0.74	0.11

NS =  $P > 0.10$

\*\* =  $0.01 \geq P > 0.001$

\*\*\* =  $P \leq 0.001$

Table 3.33A

Genetic statistics for heading date, plant height,  
tillers per plant, spikelet number per ear and  
grain number per ear

Statistic	Heading date		Plant height		Tillers per plant			Spikelet number per ear		Grain number per ear (transformed to 10 log <sub>10</sub> X)	
	LN	HN	LN (original)	LN (reanalysed)	LN (original)	LN (reanalysed)	HN	LN	HN	LN	HN
$W_r + V_r$ (sig.)	**	**	**	**	NS	NS	NS	***	NS	NS	*
$W_r - V_r$ (sig.)	***	***	(NS)	NS	NS	NS	NS	NS	NS	NS	NS
t test ( $\hat{\beta}_1 - 1$ ) <sup>a</sup>	7.36**		2.26 **	1.55 NS	3.32**	1.74 NS	7.72**	0.30 NS	0.64 NS	2.21*	0.51 NS
D	3.56	0.64	61.58	78.53	19.60	24.80	23.71	16.90	26.44	5.16	3.67
H <sub>1</sub>	326.50	293.61	40.55	51.74	10.29	5.99	24.18	6.33	1.00	0.20	0.87
H <sub>2</sub>	220.27	197.27	31.07	41.15	8.02	4.19	20.28	4.86	1.50	0.19	0.60
F	37.93	4.22	-42.87	-37.73	0.21	1.47	-11.08	9.00	6.51	1.33	1.87
E (pool)	3.36	1.28	7.51	8.20	2.33	2.45	31.59	0.78	2.99	0.44	0.49
E (parents)	5.81	1.14	5.36		1.07		5.73	0.55	2.01	0.75	0.70
E (hybrids)	2.36	1.35	8.59		2.29		44.69	0.81	3.21	0.27	0.45
$\sqrt{H_1/D}$	9.58	21.39	0.81	0.81	0.72	0.49	1.01	0.61	0.19	0.20	0.49
$uv(H_2/4H_1)$	0.17	0.17	0.19	0.20	0.19	0.17	0.21	0.19	0.37	0.24	0.17
$0.5F/\sqrt{D(H_1-H_2)}$	0.98	0.27	-0.89	-0.65	0.02	0.11	-0.58	0.90	0.90	2.77	0.95
dom./rec. genes	3.51	1.36	0.40	0.54	1.02	1.13	0.62	2.54	4.43	4.92	3.21
h <sup>2</sup> (narrow)	38.08	47.83	78.85	77.43	71.42	78.20	34.55	73.82	74.31	66.21	61.55
h <sup>2</sup> (broad)	96.44	98.68	89.60	89.98	84.64	84.73	43.59	92.99	77.17	67.81	70.58
$r(\bar{P}_r, W_r + V_r)$	0.81***	0.35 NS	-0.62***	-0.69**	0.32 NS	0.27 NS	-0.04 NS	-0.88***	-0.19 NS	0.07 NS	0.42*
$\sigma_A^2/\sigma_D^2$	0.03	0.01	3.96	3.82	9.80	11.84	2.34	6.95	17.63	51.60	12.23
K(effective factors)	1.41	1.40	1.24	1.97	0.18	0.09	0.39	1.39	0.24	0.84	0.27

a - not calculated where  $W_r/V_r$  regression non-significant.

NS =  $P > 0.10$

(NS) =  $0.10 > P > 0.05$

\* =  $0.05 > P > 0.01$

\*\* =  $0.01 > P > 0.001$

\*\*\* =  $P < 0.001$

Table 3.33B

Genetic statistics for grain yield, 100 kernel weight and physiological brown spot

Statistic	Grain yield		100 kernel weight		Physiological brown spot (transformed to $\sin^{-1}\sqrt{X}/100$ )			
	LN	HN	LN	HN	LN (original)	LN (reanalysed)	HN (original)	HN (reanalysed)
$W_r + V_r$ (sig.)	*	*	**	NS	**	NS	(NS)	(NS)
$W_r - V_r$ (sig.)	NS	NS	*	NS	NS	NS	(NS)	**
t test ( $\hat{\beta}_1 - 1$ ) <sup>a</sup>	0.70 NS		2.87*		3.01**	1.23 NS	2.37*	0.81 NS
D	2.60	16.25	0.12	0.04	17.50	26.76	158.69	130.67
H <sub>1</sub>	4.14	37.97	0.37	0.88	72.74	-4.47	68.10	26.85
H <sub>2</sub>	2.87	29.42	0.33	0.64	48.41	-15.28	67.47	-3.59
F	3.82	24.94	-0.19	0.16	27.07	22.44	65.32	12.31
E (pool)	0.93	8.70	0.05	0.18	14.02	11.17	26.96	31.19
E (parents)	1.24	8.64	0.06	0.18	19.49		22.68	
E (hybrids)	0.76	9.55	0.04	0.18	12.29		31.48	
$\sqrt{H_1/D}$ <sup>b</sup>	1.26	1.53	1.76	4.86	2.04		0.66	0.45
$uv(H_2/4H_1)$	0.17	0.19	0.22	0.18	0.17	0.86	0.25	-0.03
$0.5F/\sqrt{D(H_1-H_2)}$	1.05	1.06	-1.29	0.87	0.66	0.66	3.26	0.10
dom./rec. genes <sup>b</sup>	3.79	3.02	0.38	2.66	2.22		1.92	1.23
$h^2$ (narrow)	3.00	-0.42	16.97	13.93	22.02	50.71	51.75	71.06
$h^2$ (broad)	93.17	45.58	24.90	54.60	58.14	25.10	70.32	70.20
$r(\bar{P}_r, W_r + V_r)$ <sup>d</sup>	0.59**	0.31 NS	-0.54**	-0.50*	0.13 NS		0.36 NS	0.59**
$\sigma_A^2/\sigma_D^2$ <sup>c</sup>	1.81	1.10	0.73	0.13	0.72		4.70	
K (effective factors) <sup>c</sup>	0.02	0.15	2.79	1.56	1.53		0.95	

a - not calculated where  $W_r/V_r$  regression non-significant; b - not calculated where  $H_1$  was negative; c - not calculated where  $H_2$  was negative; d  $r$  not calculated where all previous analyses indicated that dominance was trivial.

NS =  $P > 0.10$ ; (NS) =  $0.10 \geq P > 0.05$ ; \* =  $0.05 \geq P > 0.01$ ; \*\* =  $0.01 \geq P > 0.001$ .

$(W_r - V_r)$  were not significantly ( $P \leq 0.05$ ) different between arrays for plant height, tillers per plant, spikelet number and grain number per ear and grain yield (Table 3.33). This was true for both single nitrogen level analyses and indicated that non-allelic interactions (epistasis) and/or correlated gene distributions were unimportant (Hayman, 1954a; Mather and Jinks, 1971). Hence, an additive-dominance model accounted adequately for these data. Highly significant ( $P \leq 0.001$ ) epistatic effects were demonstrated for heading date in each nitrogen level. Significant  $(W_r + V_r)$  array differences occurred in at least one nitrogen level for all characters, except tillers per plant, indicating dominance (Mather and Jinks, 1971).

Plots of  $(W_r, V_r)$  estimates, which had significant regression analyses (Tables 3.31 and 3.32), are presented for heading date (Figure 3.6), plant height (Figure 3.7), tillers per plant (Figure 3.8), spikelet number per ear (Figure 3.9), grain number per ear (Figure 3.10), grain yield (Figure 3.11), 100 kernel weight (Figure 3.12), and physiological brown spot (Figure 3.13). Accompanying each plot are the limiting parabola (Mather and Jinks, 1971) and regression slope and its standard error.

Regression slopes deviated significantly from unity (Table 3.33) for heading date, plant height, grain number per ear and 100 kernel weight (all low nitrogen), and tillers per plant and physiological brown spot (low and high nitrogen). This suggested inadequacy of an additive-dominance model for these data sets due to epistasis and/or correlated gene distributions (Mather and Jinks, 1971).

There was a lack of agreement between results of analysis of variance of  $(W_r - V_r)$  and t-test for slope, with respect to complexity of genetical models indicated. This occurred for plant height and grain number per ear (low nitrogen) and tillers per plant and physiological

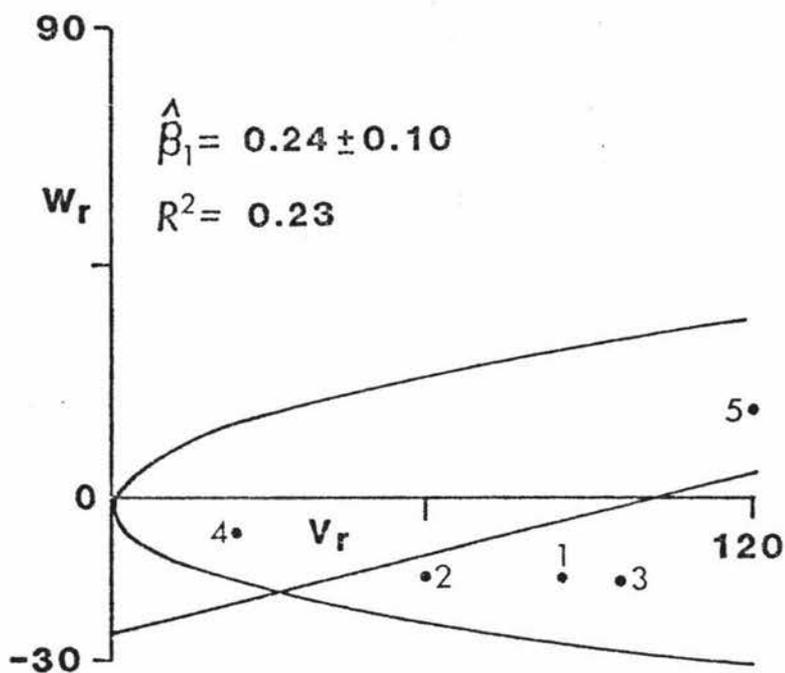


Figure 3.6: Regression of  $W_r$  on  $V_r$  for heading date in low nitrogen environment.

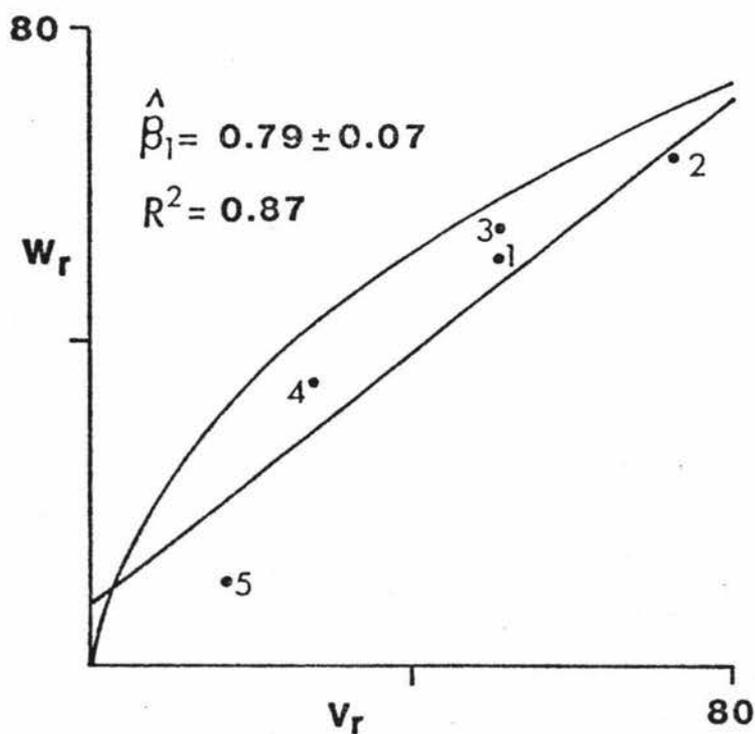


Figure 3.7: Regression of  $W_r$  on  $V_r$  for plant height in low nitrogen environment (all five parents).

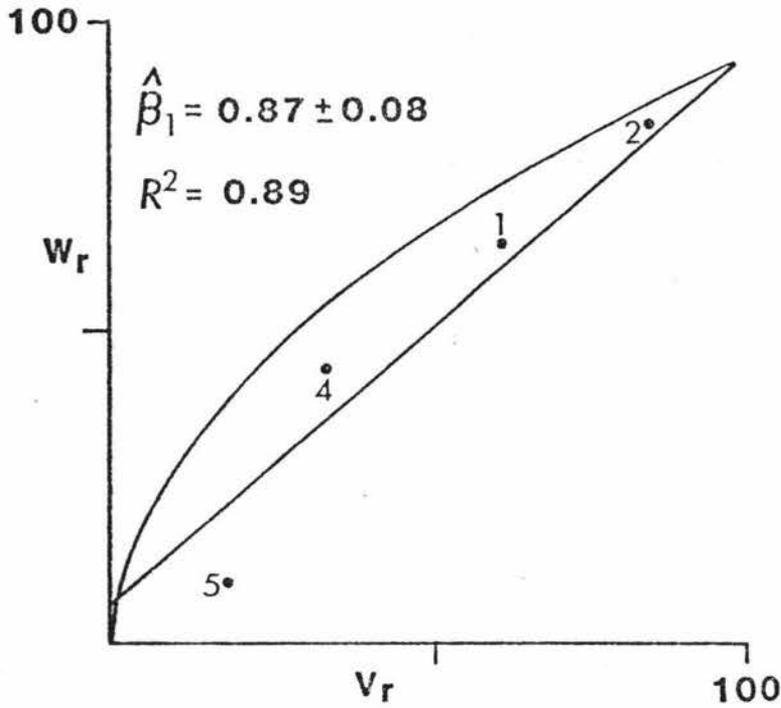


Figure 3.7: Regression of  $W_r$  on  $V_r$  for plant height in low nitrogen environment (parent 3 omitted).

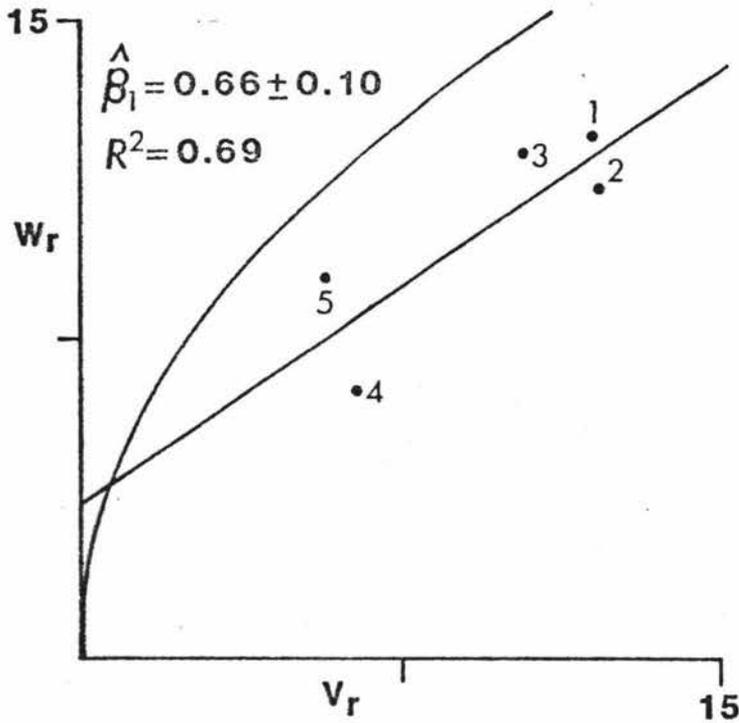
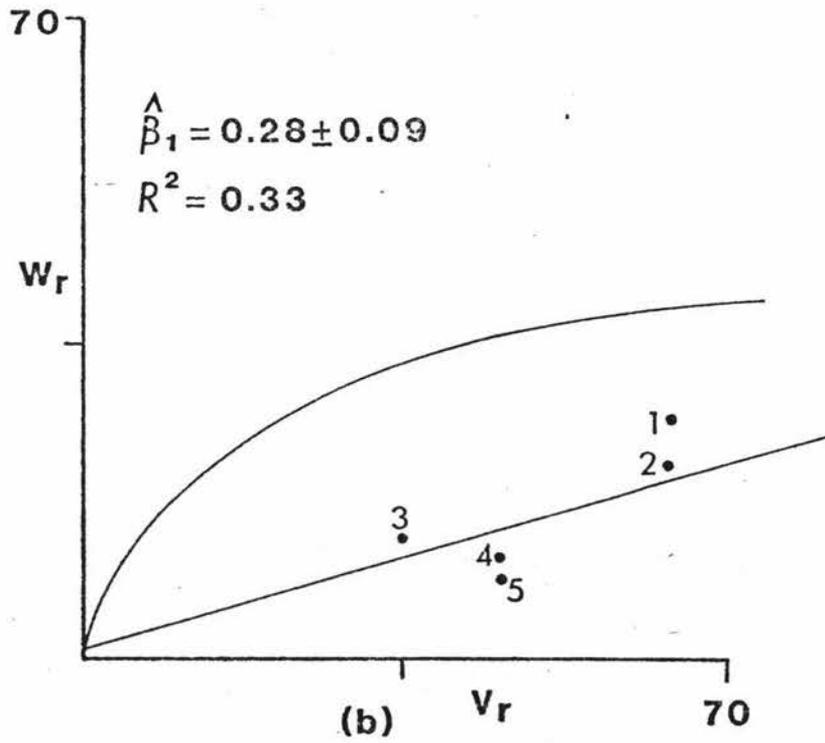
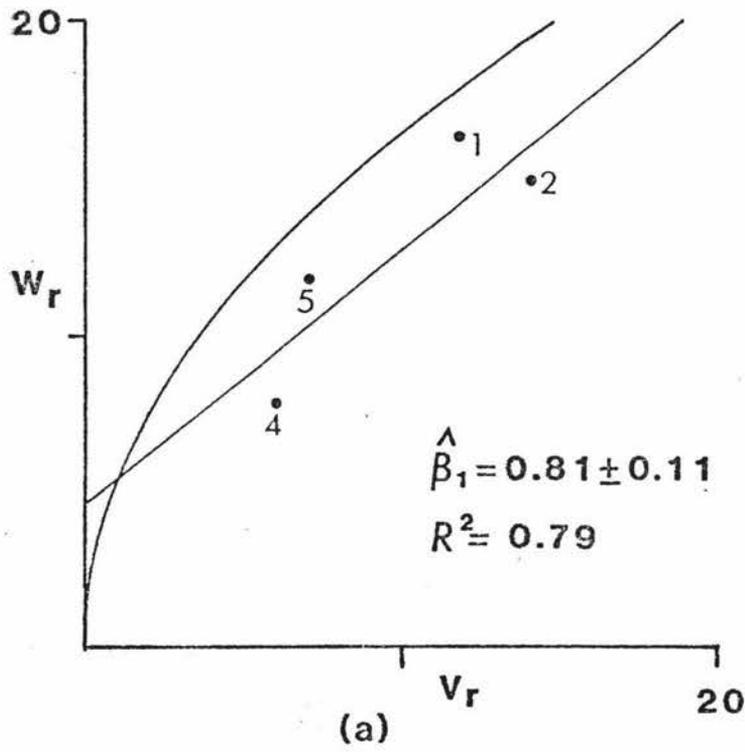
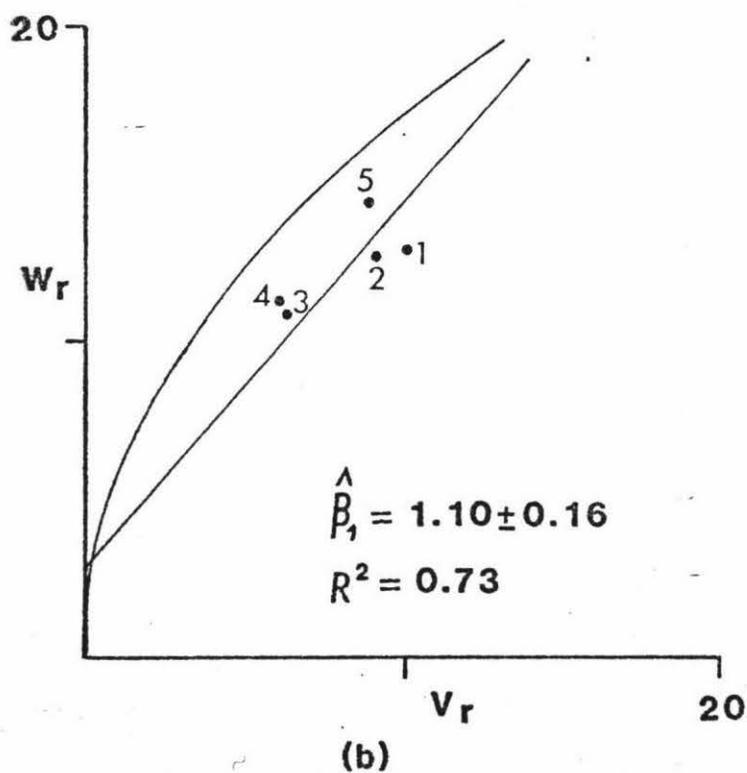
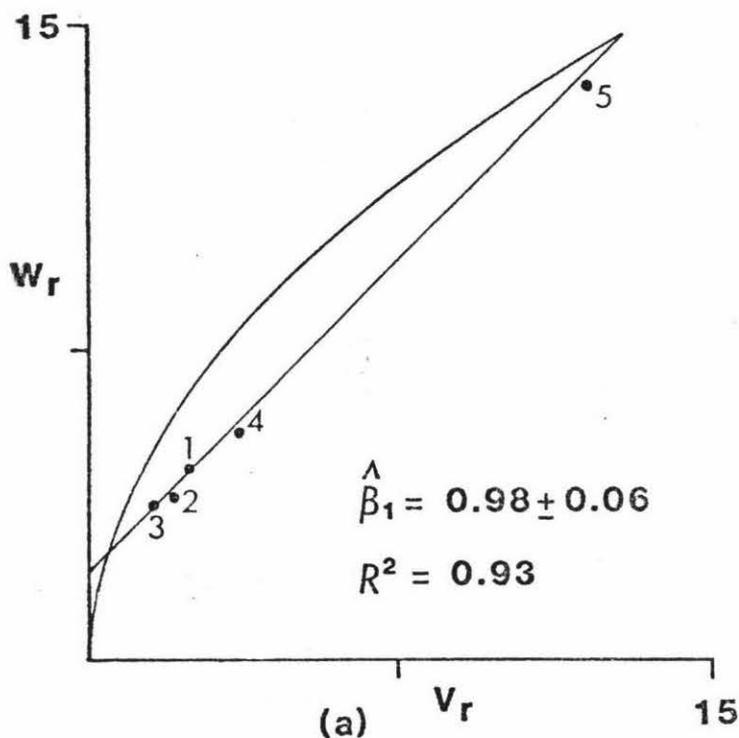


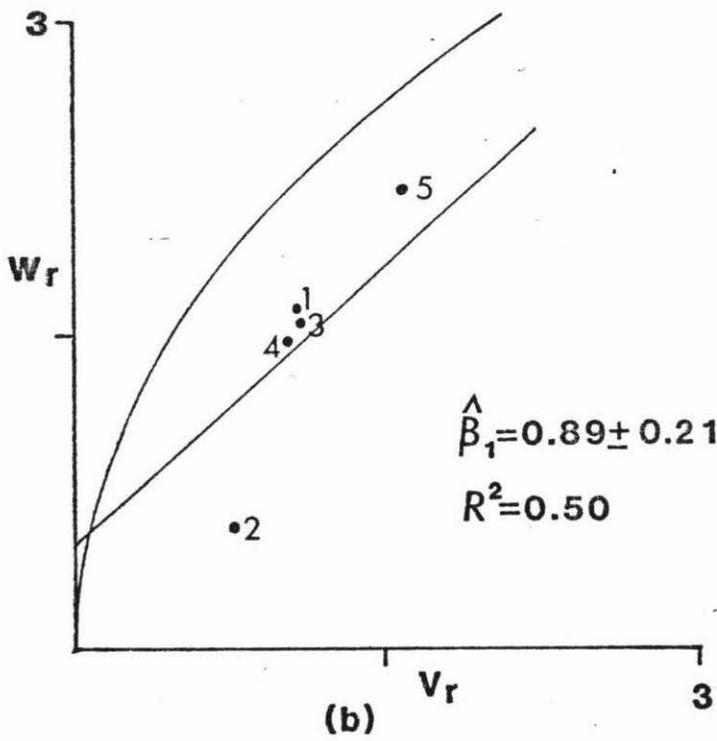
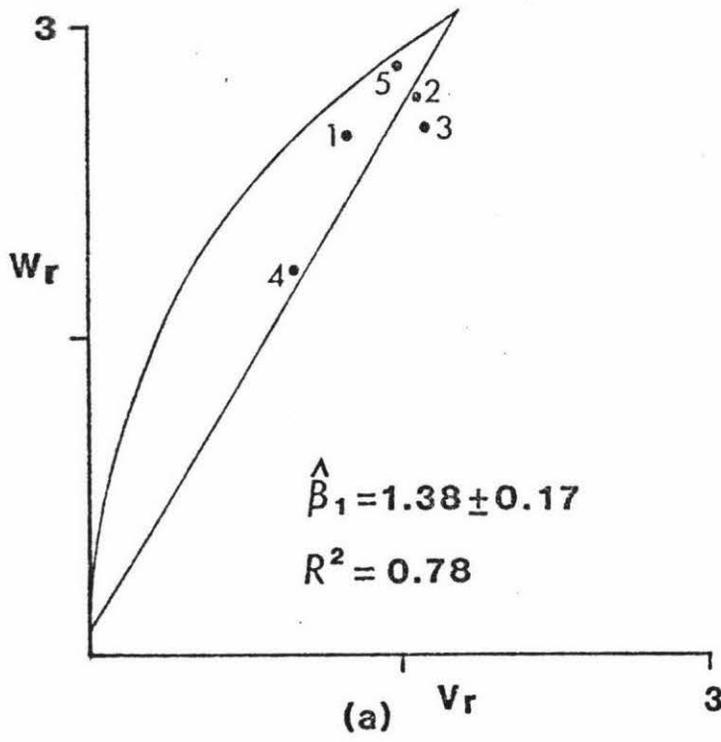
Figure 3.8: Regression of  $W_r$  on  $V_r$  for tillers per plant in low nitrogen environment (all five parents).



**Figure 3.8:** Regressions of  $W_r$  on  $V_r$  for tillers per plant;  
 (a) low nitrogen environment (parent 3 omitted),  
 (b) high nitrogen environment.



**Figure 3.9:** Regressions of  $W_r$  on  $V_r$  for spikelet number per ear;  
 (a) low nitrogen environment,  
 (b) high nitrogen environment.



**Figure 3.10:** Regressions of  $W_r$  on  $V_r$  for grain number per ear;  
 (a) low nitrogen environment,  
 (b) high nitrogen environment.

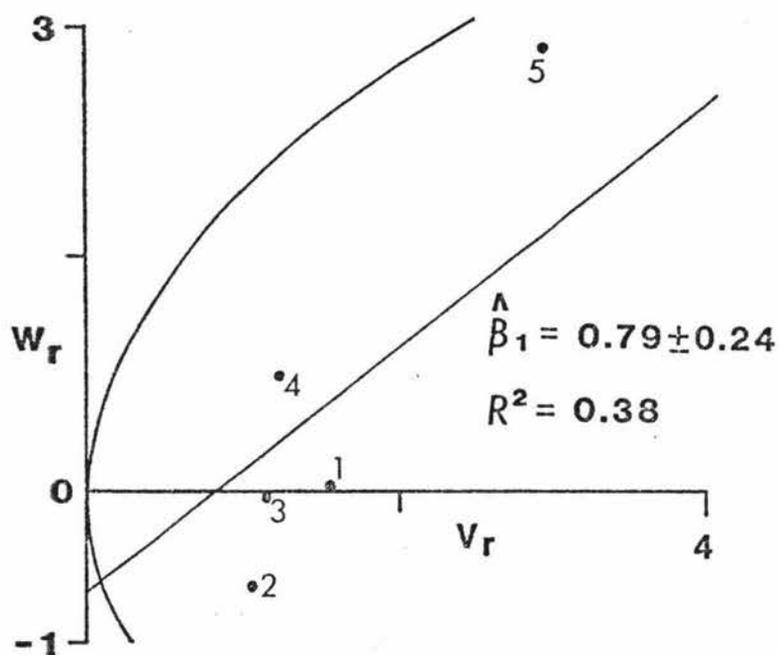


Figure 3.11: Regression of  $W_r$  on  $V_r$  for grain yield in low nitrogen environment.

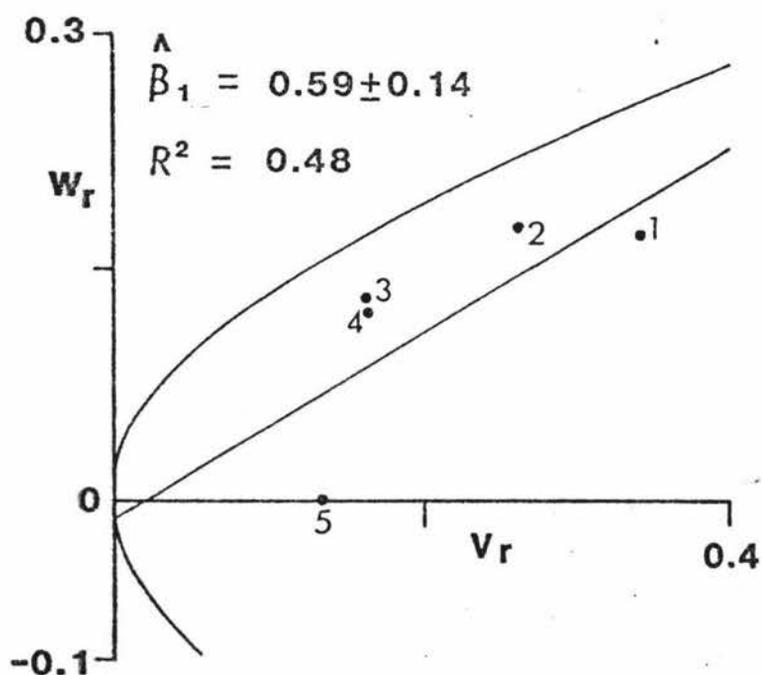
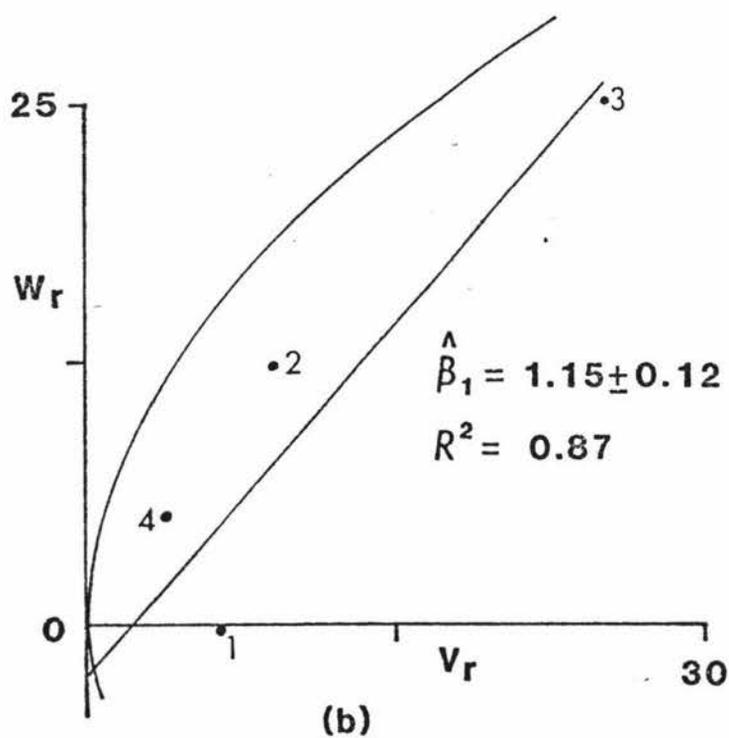
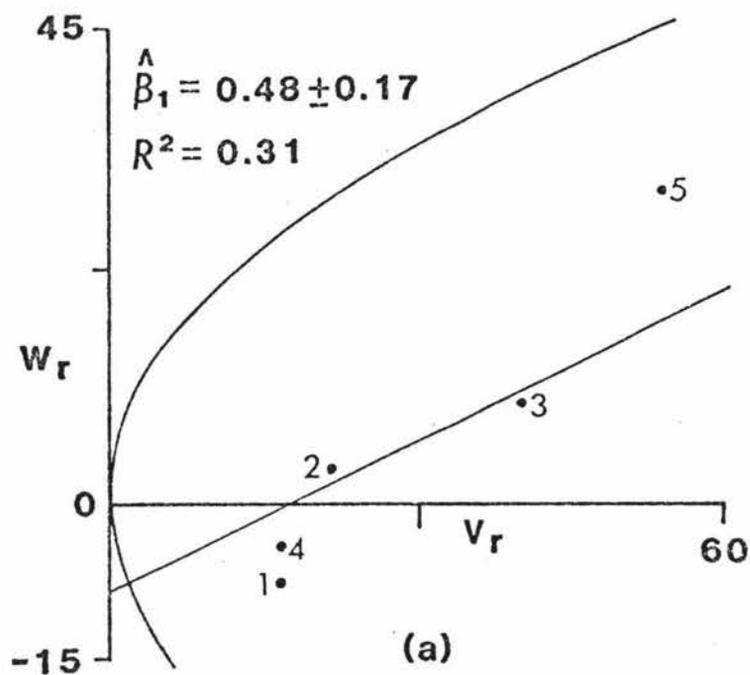
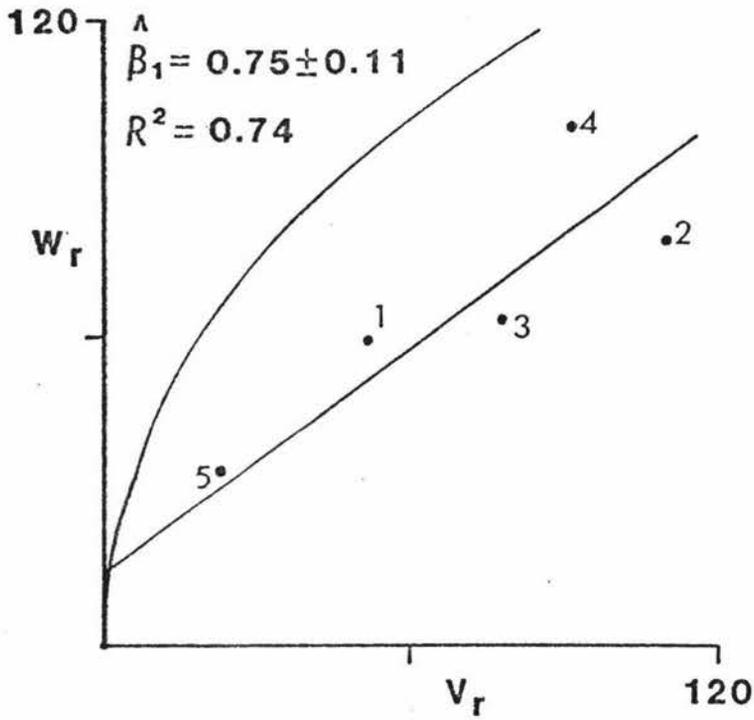


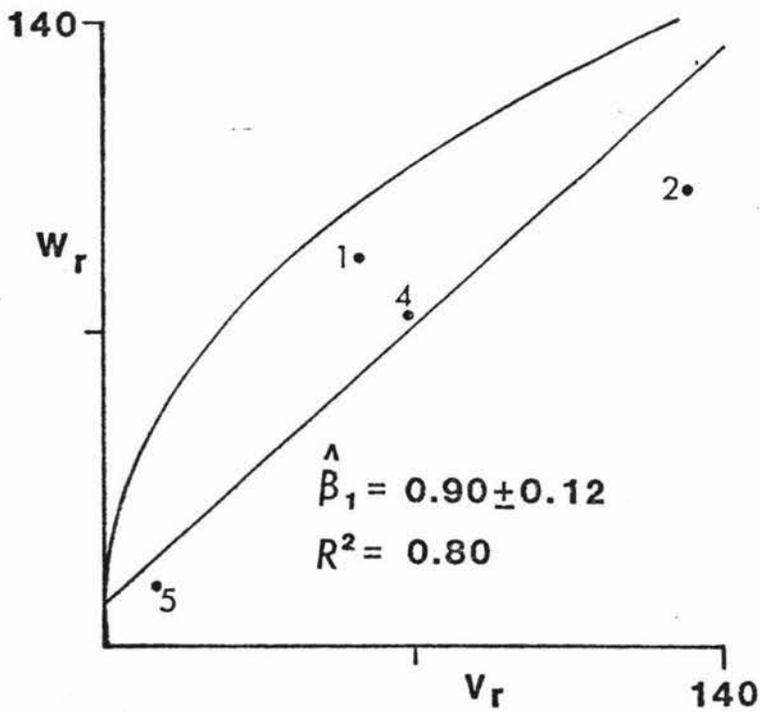
Figure 3.12: Regression of  $W_r$  on  $V_r$  for 100 kernel weight in low nitrogen environment.



**Figure 3.13:** Regressions of  $W_r$  on  $V_r$  for physiological brown spot;  
 (a) low nitrogen environment (all five parents),  
 (b) low nitrogen environment (parent 5 omitted).



(a)



(b)

**Figure 3.13:** Regressions of  $W_r$  on  $V_r$  for physiological brown spot;  
 (a) high nitrogen environment (all five parents),  
 (b) high nitrogen environment (parent 3 omitted).

brown spot (low and high nitrogen). An arbitrary sample of some of these data sets was reanalysed (Section 2.8.3). Regression information for each array deleted in turn, is presented in Appendix 10. A consensus opinion was made on the side of "acceptability" of an additive-dominance model. For each character, the regression equation and associated statistics for the reduced diallel data set which was most adequately accounted for by an additive-dominance model, are given in Table 3.34.

From Table 3.34, all regression equations for reanalysed data sets were highly significant ( $P \leq 0.001$ ), that is, slope was significantly different from zero. The t-test for departure of regression slope from unity was not significant in all cases (Table 3.33) and indicated that an additive-dominance model could not be rejected. Results for analysis of variance of  $(W_r + V_r)/(W_r - V_r)$  and t-test of slope for reanalysed data (Table 3.33), were in agreement, except for physiological brown spot (high nitrogen), in indicating that an additive-dominance model was adequate to account for the data.  $W_r/V_r$  regression plots for these data are presented together with their respective original data plots in Figure 3.7 (plant height), 3.8 (tillers per plant) and 3.13 (physiological brown spot). Included are the R-square value and regression slope plus its standard error.

#### 3.2.2.2 GENETIC COMPONENTS

Estimates of D,  $H_1$ ,  $H_2$ , F and E for original and reanalysed data, are presented in Table 3.33 for all characters. D estimates were positive and large, except for relatively low values for heading date and 100 kernel weight. Negative estimates of  $H_1$  and/or  $H_2$  were obtained for reanalysed data of physiological brown spot in low and high nitrogen levels (Table 3.33B). This was also encountered for most net blotch characters and resulted from environment (E) being greater than the

Table 3.34

$W_r/V_r$  regression equations and associated statistics for several reanalysed data sets of characters in low and high nitrogen environments

Character	Array deleted	Regression equation	S.E.	F-test	R-square	S.E. ( $\hat{\beta}_1$ )
Plant height (LN)	3	$W_r = 0.87V_r + 6.97$	10.80	111.89***	0.89	0.08
Tillers per plant (LN)	3	$W_r = 0.81V_r + 4.73$	2.82	54.02***	0.79	0.11
Physiological brown spot (LN)	5	$W_r = 1.15V_r - 2.21$	8.32	91.14***	0.87	0.12
(HN)	3	$W_r = 0.90V_r + 9.13$	31.61	56.08***	0.80	0.12

LN = Low nitrogen.

HN = High nitrogen.

\*\*\* =  $P \leq 0.001$ .

combination of all basic array statistics in the appropriate equations (Section 3.1.1). The results indicated that dominance was trivial and statistics involving negative H estimates were also regarded as trivial. Estimates of  $H_1$  and  $H_2$  were generally greater than D for heading date, grain yield and 100 kernel weight. In most instances, the reverse trend occurred for plant height, tillers per plant, spikelet number and grain number per ear, and physiological brown spot.

F estimates (Table 3.33) were positive for heading date, spikelet number and grain number per ear, grain yield and physiological brown spot, and indicated that there were more dominant than recessive alleles in the parents. Negative estimates (plant height, tillers per plant and 100 kernel weight) indicated the reverse pattern.

### 3.2.2.3 ADDITIONAL GENETIC STATISTICS

The statistics for degree of dominance ( $\sqrt{H_1/D}$ ), product of allele frequencies ( $H_2/4H_1$ ), dominance consistency ( $0.5F/\sqrt{D(H_1-H_2)}$ ), proportion of dominant to recessive alleles ( $(\sqrt{4DH_1} + F)/(\sqrt{4DH_1} - F)$ ), direction of dominance ( $r(\bar{P}_r, W_r + V_r)$ ), relative sizes of  $\sigma_A^2$  and  $\sigma_D^2$  ( $\frac{1}{2}D/\frac{1}{2}H_2$ ), and number of effective factors (K), are presented in Table 3.33.

Partial dominance ( $0 < \sqrt{H_1/D} < 1$ ) was indicated for plant height, tillers per plant, spikelet number and grain number per ear and physiological brown spot, while heading date, grain yield and 100 kernel weight showed overdominance ( $\sqrt{H_1/D} > 1$ ). The findings suggested that in spite of additivity being of major importance for many characters, as indicated previously, there was also a relatively strong influence by dominance.

The product of frequencies of increasing and decreasing alleles (uv) ranged from 0.10 to 0.20 for most characters, indicating gene asymmetry (Mather and Jinks, 1971). Estimates of greater than 0.25

(theoretical maximum) must be considered spurious. Consistency of degree of dominance across all segregating loci ( $0.5F/\sqrt{D(H_1-H_2)}$ ) varied considerably between some characters (Table 3.33). Estimates relatively close to an absolute value of one were obtained for heading date, spikelet number and grain number per ear, grain yield and 100 kernel weight. This indicated that the degree of dominance for these characters in one or both nitrogen levels was consistent over all segregating loci.

There were more dominant than recessive alleles over all the parents ( $(\sqrt{4DH_1} + F)/(\sqrt{4DH_1} - F)$ ) for every character except plant height, tillers per plant and 100 kernel weight. Estimates for some characters such as heading date, grain number per ear and 100 kernel weight, varied dramatically with nitrogen level. Relatively high estimates were obtained for spikelet number and grain number per ear and grain yield. The relative importance of dominance for grain yield was shown previously by analysis of variance of  $(W_r + V_r)$ ,  $H_1$  and  $H_2$  greater than  $D$  in each nitrogen level,  $F$  ratio and overdominance ( $\sqrt{H_1/D} > 1$ ).

Characters with positive correlations between common parent mean and  $(W_r + V_r)$  (Table 3.33) were heading date, tillers per plant, grain number per ear, grain yield and physiological brown spot. Negative correlations were estimated for plant height, spikelet number per ear and 100 kernel weight. The latter correlations indicated that dominance was associated with increasing phenotypic expression while positive correlations indicated the reverse trend (Mather and Jinks, 1971). The largest correlation was  $-0.88$  for spikelet number per ear in low nitrogen level (Table 3.33A). This was a very high correlation and highly significantly ( $P \leq 0.001$ ) different from zero. Previous analyses indicated that dominance was relatively important (analysis of variance

of  $(W_r + V_r)$  was significant ( $P \leq 0.01$ ),  $H_1$ ,  $H_2$  and  $F$  were positive and there was partial dominance ( $\sqrt{H_1 D} < 1$ ) and therefore this result may be interpreted with confidence.

Estimates of the ratio of additive genetic variance ( $\sigma_A^2$ ) to dominance genetic variance ( $\sigma_D^2$ ), calculated using  $\frac{1}{2}D/\frac{1}{4}H_2$ , are presented in Table 3.33. The ratio was greater than or equal to unity for plant height, tillers per plant, spikelet number and grain number per ear, grain yield and physiological brown spot. For number of effective factors ( $K$ ), estimates differed considerably (Table 3.33) and were disappointingly low for tillers per plant, grain number per ear and grain yield. The formula used presently (Jinks, 1954) estimated only factors with large dominance and this may have accounted for some of the very low estimates.

#### 3.2.2.4 HERITABILITY

Narrow-sense ( $h_N^2$ ) and broad-sense ( $h_B^2$ ) heritability estimates for all characters are presented in Table 3.33.

Broad-sense heritabilities were large in low and/or high nitrogen levels for heading date, plant height, tillers per plant, spikelet number per ear and grain yield. Physiological brown spot and 100 kernel weight had low to middling estimates. Narrow-sense estimates were similar to those of  $h_B^2$  for plant height, tillers per plant and grain number per ear. This indicated that total genetic variance was predominantly additive genetic variance with only minor dominance and/or epistasis.

However, there were discrepancies between  $h_N^2$  and  $h_B^2$  for other characters and this suggested presence of relatively large non-additive genetic variance, in addition to additive genetic variance. These findings generally supported those found from analysis of variance of

$(W_r + V_r)/(W_r - V_r)$ , t-test of departure of regression slope from unity and relative sizes of D and H and related statistics.

For each character, there were often large differences between heritability estimates obtained for individual nitrogen levels. This emphasises their limited usefulness when estimates obtained in one environment are translated to another environment, even when using identical material and recording the same character. The problem is due partly to genotype x nitrogen interaction since  $\sigma_G^2$  from one environment is actually  $(\sigma_G^2 + \sigma_{GE}^2)$ .

### 3.2.3 HETEROSIS AND HETEROBELTIOSIS

Estimates of heterosis ( $\bar{F}_1$ -mid parent mean (MP)) and heterobeltiosis ( $\bar{F}_1$ -better parent mean ( $\bar{P}_1$ )) are presented for each character in Appendix 8. Statistics for single nitrogen levels and pooled analyses over nitrogen levels are included. Standard errors accompany all estimates.

Heterosis and/or heterobeltiosis were significant ( $P \leq 0.05$ ) for at least one cross for the following characters in the low and high nitrogen levels and the pooled analysis: heading date, tillers per plant, spikelet number and grain number per ear, and 100 kernel weight. One or more significant ( $P \leq 0.05$ ) estimates occurred for grain yield and physiological brown spot in the low and high nitrogen levels only. For plant height, several heterosis and heterobeltiosis estimates were significant solely in the low nitrogen level. The results indicated that significant heterosis and heterobeltiosis occurred widely.

CHAPTER 4

CHAPTER 4.GENERAL DISCUSSION4.1 NET BLOTCH EXPRESSION

Net blotch symptoms occurred solely in the high nitrogen environment (Section 3.1), indicating that there was a dramatic reduction in resistance to the disease with a relatively large increase in nitrogen concentration. Similar findings were reported by Singh (1963) and Piening (1967). Absence of net blotch in the low nitrogen environment suggested that a minimum level of internal plant nitrogen may be necessary before net blotch symptoms develop. The result highlighted the importance of providing optimum nutrition when undertaking disease resistance studies.

Lesion infection types (Table 3.1) predominating on Manchuria foliage were typical of those found on many net blotch resistant genetic stocks (Buchannon and McDonald, 1965; Khan and Boyd, 1969a, b, c; Shipton *et al.*, 1973). Restriction of full lesion development was the key feature for this stock. In contrast, highly susceptible reactions occurred on the foliage of Kaniere, Mata and Zephyr. Zephyr's reaction was in agreement with numerous studies (Evans, 1968; Douglas, 1979; Hampton, *per comm.*, McEwan, *pers. comm.*).

4.2 QUANTITATIVE GENETICS OF NET BLOTCH RESISTANCE

Biometrical analyses of the diallel data sets for top and bottom canopy scores, flag and second leaf areas infected and leaf section area infected (petri dish), were in agreement in showing that additivity was of major importance in conditioning net blotch resistance (Table 3.7). D components were relatively large, narrow sense heritability ( $h_N^2$ ) estimates

were greater than their respective broadsense ( $h_B^2$ ) estimates in most cases, and for second leaf and leaf section areas infected the ratio of  $\sigma_A^2$  to  $\sigma_D^2$  was relatively high (7.13 and 4.26 respectively). These findings were undocumented previously.

The relationship between narrow-sense and broad-sense heritability estimates noted above was due to negative estimates of H, particularly  $H_2$  (Table 3.7). The formulae used to estimate narrow-sense and broad-sense heritabilities were:

$$h_N^2 = \frac{\frac{1}{2}D + \frac{1}{2}H_1 - \frac{1}{2}H_2 - \frac{1}{2}F}{\frac{1}{2}D + \frac{1}{2}H_1 - \frac{1}{4}H_2 - \frac{1}{2}F + E} \quad \text{and}$$

$$h_B^2 = \frac{\frac{1}{2}D + \frac{1}{2}H_1 - \frac{1}{4}H_2 - \frac{1}{2}F}{\frac{1}{2}D + \frac{1}{2}H_1 - \frac{1}{4}H_2 - \frac{1}{2}F + E}$$

Both formulae are identical except for a difference in the numerator coefficient of  $H_2$ . Hence, when  $H_2$  is positive or zero, narrow-sense heritability will be less than or equal to broad-sense heritability, respectively. However, a negative estimate of  $H_2$  causes a greater increase in the numerator of the formula estimating  $h_N^2$  than that for  $h_B^2$ , resulting in the former being relatively larger.

Negative estimates of H were calculated for most analyses (Table 3.7), indicating that dominance was trivial. This was also suggested by non-significance of  $(W_r + V_r)$  between arrays (Mather and Jinks, 1971) for all analyses except that for second leaf area infected. Where positive values occurred, estimates of  $\sqrt{H_1/D}$  were calculated (Table 3.7). These were 0.29 (top canopy score), 0.56 (second leaf area) and 0.70 (leaf section area) and suggested partial dominance for resistance. Conditioning of net blotch resistance by partial dominance was also found in several qualitative studies (Schaller, 1955; Mode and Schaller, 1958; Khan and

Boyd, 1969a; Bockelman et al., 1977).

The number of effective factors (Mather and Jinks, 1971) involved in resistance to the disease was estimated for only second leaf (1.32) and leaf section (0.96) areas infected (Table 3.7), as the formula used for estimation (Jinks, 1954) required positive expectations of  $H_2$ . Unfortunately the number of effective factors rarely equals gene number (Mather and Jinks, 1971) and therefore it is of doubtful value to compare results of this study with those of previous qualitative investigations in which one or more alleles and/or modifying factors conditioned resistance to net blotch (Schaller, 1955; Mode and Schaller, 1958; Omar et al., 1970). Nevertheless, taking due cognizance of these comments, it is tentatively suggested that resistance in the present study was controlled by a single, partially dominant gene. Verification of involvement of several alleles and/or modifying factors was not possible as only a single, physiological race of *Drechslera teres* (Khan and Boyd, 1969a; Shipton et al., 1973; Tekauz and Buchannon, 1977; Khan and Portmann, 1979) was used.

It should be noted that the preponderance of additivity found in this study (Table 3.7) did not necessarily indicate that dominance was lacking. Additivity is the variance of average allele effects and therefore does not mean, literally, additive action of genes. That is, additive genetic variance in no way implies that dominance and/or epistasis are absent (Falconer, 1981). It is commented that dominant genes, with the exception of those that exhibit overdominance, have at least half of their effect estimated as additive (Mather and Jinks, 1971). A partially dominant gene, which was suggested presently as conditioning resistance to net blotch, has most of its effect calculated as additive action (Mather and Jinks, 1971).

#### 4.3 INTERPRETATION DIFFICULTIES

Analyses differed in their sensitivity for detecting epistasis and/or correlated gene distributions (Table 3.7). Results of analysis of variance of  $(W_R - V_R)$  indicated, almost without exception, that there was no non-additivity. However, there was lack of agreement between these results and those of significance tests of departure of regression slope from unity. It was concluded that epistasis and/or correlated gene distributions were present, although they were relatively unimportant.

Mather and Jinks (1971) noted the need for agreement between results of analysis of variance of  $(W_R + V_R)/(W_R - V_R)$  and t-test for deviation of regression slope from unity, in indicating that an additive-dominance model adequately accounted for the data. Lack of agreement between these results indicates that suitability of the model is equivocal. Furthermore, the evidence for disturbance is generally weak. Therefore, it is appropriate to proceed with the analysis and estimate genetic components and other statistics, bearing in mind possible bias. This approach was adopted presently and the results were given in Table 3.7. The recommended procedure for situations where disagreement occurs between results of analysis of variance of  $(W_R + V_R)/(W_R - V_R)$  and t-test  $(\hat{\beta}_1 - 1)$ , is array deletion (Hayman, 1954a; Allard, 1956; Mather and Jinks, 1971) and this was also conducted.

Examination of biometrical analyses for net blotch (Table 3.7) showed that only second leaf area infected (original data) and bottom canopy score (reanalysed data) had agreement between results of analysis of variance of  $(W_R + V_R)/(W_R - V_R)$  and t test  $(\hat{\beta}_1 - 1)$ . However, genetic interpretations on net blotch resistance obtained from these two data sets were compared with those from biometrical analyses conducted on the other data sets and it was concluded that all interpretations were similar. Reanalysis of reduced diallel data sets was unrewarding as it

offered no notable improvement in genetic information. It is postulated that an overall examination of analysis of variance of  $(W_r + V_r)/(W_r - V_r)$ , t-test ( $\hat{\beta}_1 - 1$ ), genetic components and associated statistics, should be conducted before making genetic interpretations.

It is noteworthy that residual error variances associated with several net blotch assessment techniques (Table 3.7) were relatively large compared with respective estimates of D and  $H_1/H_2$ . This indicated that caution should be exercised in interpreting the relevant biometrical results.

#### 4.4 BREEDING IMPLICATIONS

In view of the probable unimportance of non-allelic interactions in net blotch resistance, as noted above, there should be no fundamental genetic difficulties in manipulation of net blotch resistance genes in a breeding programme. The present study indicated that incorporation of a resistance gene from Manchuria into agronomically acceptable cultivars was successful. This was particularly so in the cross of Manchuria with Zephyr, a highly susceptible New Zealand cultivar. The genetical simplicity suggested that a backcross breeding programme (Allard, 1960; Thomas, 1973) could be used advantageously for development of net blotch resistant cultivars.

#### 4.5 PETRI DISH TECHNIQUE

Results of the petri dish study (after Spiers, 1978) were of particular interest as this was the first time the technique had been used to investigate net blotch resistance. The biometrical analysis results were compared with those obtained for the other net blotch data sets (Table 3.7) and it was concluded that genetic interpretations were similar. Biometrical analyses conducted on leaf section area infected

indicated that resistance was conditioned by a partially dominant, effective factor. Results were very similar for second leaf area infected (Table 3.7).

In view of the success of the technique in this study, the leaf section area method of assessing net blotch resistance, should be very useful in future investigations. The technique has several advantages over methods of intact plant tissue assessment (top and bottom canopy scores and flag and second leaf areas infected). Foremost among these is that disease reaction is assessed for a known leaf area against a constant volume of inoculum and therefore variability in genotype reaction reflects variation in the degree of resistance. Further advantages include: rapid technique, relatively unlimited replication in time and space thereby permitting collection of voluminous data within a normal host growing season, ready ability to test effects of environmental variation on expression of resistance, choice of physiological race, and cheapness in comparison to the use of controlled environment facilities.

#### 4.6 AGRONOMIC SIDE EFFECTS

The effect of net blotch on several agronomic characteristics of barley was examined using control (fungicide treated) and disease treatments (Table 3.24). The disease had no significant effect on the host other than shortening the time to heading. This was undocumented previously. Results for grain number per ear and stem length (height) were in agreement with those of previous studies (Hampton, 1977; Hampton and Arnst, 1978). An unexpected result was the non-significant effect of net blotch on grain yield and 100 kernel weight, as reductions in grain yield (Shipton, 1966; Shipton *et al.*, 1973; Hampton and Arnst, 1978) and kernel weight (Buchannon and Wallace, 1962; Hampton, 1977; Hampton and Arnst, 1978), which may be attributed to the disease, have

been documented widely.

Multiple regression of grain yield in the disease treatment against net blotch infection on the flag ( $X_1$ ) and second ( $X_2$ ) leaves (Section 3.1.8.2), showed that  $X_2$  was unimportant relative to  $X_1$  in contributing to yield. This supported the well documented role of the flag leaf as a major contributor to grain yield (Thorne, 1966; Evans and Wardlaw, 1976; Teng and Gaunt, 1980). Net blotch infection on the flag leaf also had a marked effect on grain yield in other New Zealand studies (Hampton and Arnst, 1978; Hampton, in preparation).

Several reasons for uniformity between control and disease treatments, particularly with respect to grain yield and 100 kernel weight estimates, are suggested. Inability to achieve total net blotch control (Table 3.24) was the most important reason for uniformity between the two treatments. This has been a difficulty in numerous studies (Shipton, 1966; James, 1974; Hampton and Arnst, 1978). Interplot interference (Large, 1966; James, 1974) likely occurred between adjacent plots. Control plants were randomised within blocks, together with disease plants and were therefore often in close proximity to disease sources. Hence, *D. teres* had greater opportunity to infect unprotected foliage produced between spray applications.

Low net blotch intensity may also be a reason for the uniform conditions. Pooled treatment means (Table 3.24) for net blotch intensity assessments conducted on intact plant tissue were all under 21.0 (approximately 13.0 per cent for untransformed data). It is suggested that beneficial effects of the fungicides used in the control treatment were not demonstrated due to these low intensities.

With respect to grain yield and 100 kernel weight losses, it may be noted that net blotch intensity was insufficient for assessment until after Growth Stage 11.1 (Large, 1954) and just before colour change of

grain. Grain-filling was at least midway to fully completed, depending on genotype and therefore the disease had little opportunity to adversely affect either grain yield or 100 kernel weight.

#### 4.7 NET BLOTCH ESTABLISHMENT DIFFICULTIES

As noted above, poor net blotch establishment occurred, particularly in the early to middle stages of the study. This may have resulted from several factors, foremost among these being displacement of conidia from leaf surfaces by overhead misting (Section 2.5.2). Frequency and duration of misting were important in maintaining consistent wetting of leaf surfaces and determined jointly the amount of droplet formation and runoff from foliage. In this study, it is suggested that an optimum frequency/duration combination of misting, offering consistent wetness and minimum runoff, was not identified. This optimum misting regime should be accurately defined in future studies.

Air circulation within the high nitrogen environment may have been inadequate for distribution of conidia throughout the room, thereby discouraging rapid establishment of the disease. Measurements of conidial load per unit volume of air, were not taken. Environmental factors of light intensity, temperature and initial relative humidity, were similar to those used successfully in other studies (Buchannon and McDonald, 1965; Khan and Boyd, 1969b; Tekauz and Mills, 1974; Keeling and Bantari, 1975; Douglas, 1979) and therefore it is suggested that factor(s) other than these resulted in poor net blotch establishment. It may be concluded that there is a very intimate relationship between the host and pathogen. Furthermore, net blotch outbreaks in the field are difficult to simulate under controlled environmental conditions.

#### 4.8 OTHER CHARACTERS AND THEIR BIOMETRICAL ANALYSES

An increase in nitrogen concentration had a highly significant (at least  $P \leq 0.05$ ) effect on all characters, except heading date (Section 3.2.1.2). Such dramatic responses to nitrogen have been widely documented (Littler *et al.*, 1969; Briggs, 1978; McGuire *et al.*, 1979). Interactions of genotype with nitrogen were significant for plant height, spikelet number per ear, grain yield, 100 kernel weight and physiological brown spot (Table 3.30). The results were in agreement with previous studies (Finlay and Wilkinson, 1963; Riggs and Hayter, 1973; Hayes and Paroda, 1974; Johnson and Whittington, 1977; Surma, 1978) which showed that G x E interactions occur widely for many barley characters.

Genetic interpretations of biometrical analyses for the other characters were based on all the appropriate analyses, genetic components and associated statistics (Table 3.33 A,B). Additivity was most important for plant height, tillers per plant, spikelet number and grain number per ear, and physiological brown spot. Role of additivity in controlling these characters has been reported (Riggs and Hayter, 1973, 1975; Jana, 1976; Surma, 1978; Nikitenko *et al.*, 1979), although relative importance of additivity compared to dominance and/or non-allelic interactions was not always clear.

Previous studies showed that physiological brown spot (Clark *et al.*, 1979) on Awnless Atlas barley was conditioned by a dominant gene (Schaller and Qualset, 1975; Faris, 1977), although the degree of dominance was unstated. Dominance occurred presently as evidenced by mostly positive expectations of H (Table 3.33B).

Past investigations indicated overdominance for yield (Johnson and Aksel, 1959; Virk and Verma, 1973; Nikitenko *et al.*, 1979), mainly overdominance for kernel weight (Riggs and Hayter, 1975; Surma, 1978; Nikitenko *et al.*, 1979) and partial dominance and overdominance for grain number per ear (Riggs and Hayter, 1973, 1975;

Nikitenko *et al.*, 1979) and spikelet number per ear (Surma, 1978).

Results of the present study were in general agreement with these findings.

#### 4.9 OTHER APPROACHES TO QUANTITATIVE INHERITANCE

The  $F_1$   $W_R/V_R$  regression analysis used in this study has differential sensitivity in detection of the various types of non-allelic interaction (Jinks, 1954, 1956). This is also true presumably for relationships between gene distributions. The  $F_2$  diallel overcomes this difficulty and is therefore desirable for obtaining more reliable information on underlying genetic mechanisms. It has been adopted in several studies (Jinks, 1956; Johnson and Aksel, 1959; Riggs and Hayter, 1973; Greenberg, 1977).

However, if proceeding to  $F_2$  generation, there is justification for undertaking extra work to obtain first generation backcrosses. With three generations ( $F_1$ ,  $F_2$ ,  $B_1$ ), generation means analysis (Hayman, 1958; Mather and Jinks, 1971) is possible which provides detailed information on additive, dominance and the three kinds of epistatic (additive by additive, dominance by dominance and additive by dominance) variation. Estimates of the latter are unobtainable in diallel analysis.

Generation means analysis was not conducted presently as there were no  $F_2$  or backcross generations of the appropriate material. There was only one season available for crossing and this only permitted production of  $F_1$  seed.

An alternative and probably more useful analysis of diallel cross data is that involving general and specific combining abilities (Griffing, 1956a, b). Combining abilities may be interpreted genetically as additive, dominance and various types of epistatic genetic variance and therefore the predominant type of genetic variance may be ascertained. This is of fundamental interest to a breeder as it determines largely the

breeding policy adopted.

In the analysis, total genotypic variance ( $\sigma_G^2$ ) may be represented symbolically in terms of the variances of general combining abilities ( $\sigma_{gca}^2$ ) and specific combining abilities ( $\sigma_{sca}^2$ ), as (Griffing, 1956a):

$$\sigma_G^2 = 2 \sigma_{gca}^2 + \sigma_{sca}^2$$

Furthermore, assuming no epistasis and a random mating population, the combining ability variances may be interpreted genetically with respect to additive and non-additive (dominance) genetic variances. The following relationships exist:

$$2 \sigma_{gca}^2 = \sigma_A^2$$

and

$$\sigma_{sca}^2 = \sigma_D^2$$

Hence, the relative importance of additive and non-additive genetic variances may be determined as well as the average degree of dominance, as given by the ratio of  $\sigma_D^2/\sigma_A^2$ . With epistasis,  $2 \sigma_{gca}^2$  and  $\sigma_{sca}^2$  contain, in addition to the above mentioned variances, contributions due to epistatic variance (Griffing, 1956a). However, the predominant role of additive or non-additive (dominance and epistasis) gene action is still readily assessed.

Combining abilities may be related to the genetic components estimated in the present study, as follows (Hayman, 1960, 1963; Mather and Jinks, 1971):

$$\sigma_G^2 = 2 \sigma_{gca}^2 + \sigma_{sca}^2 = \frac{1}{2}D - \frac{1}{2}F + \frac{1}{2}H_1 - \frac{1}{4}H_2$$

$$\sigma_{gca}^2 = \frac{1}{4}D - \frac{1}{4}F + \frac{1}{4}H_1 - \frac{1}{4}H_2 = \frac{1}{4}D_R$$

and

$$\sigma_{sca}^2 = \frac{1}{4}H_2 = \frac{1}{4}H_R$$

$H_R$  and  $D_R$  are the random mating ( $u = v = \frac{1}{2}$ ) forms of D and H and epistasis

is assumed to be absent (Mather and Jinks, 1971). Other terms were defined previously (Section 2.8.4).

It is pertinent to note several advantages which the combining abilities analysis (Griffing, 1956a,b) has over that used in this study (Mather and Jinks, 1971). From above, combining ability variances provide a simple and concise account of the genetic situation and the genetic model on which the analysis is based provides for the existence of epistasis. This is in contrast with the analysis of Mather and Jinks (1971) in which absence of epistasis is an important assumption. However, epistasis occurs widely (Hayman, 1958) and its absence is probably rarely, if ever, realised.

Griffing's analysis may be generalised to any number of alleles per locus and any number of loci. A further assumption of the analysis used presently is no multiple allelism (Mather and Jinks, 1971). Instead, the genetic model is developed for one diallelic gene which may be extended to many loci only when the data conform to several strict assumptions (Jinks, 1954; Hayman, 1954, 1960; Mather and Jinks, 1971).

Arunachalam (1976) compared and contrasted the combining abilities analysis and that of Mather and Jinks (1971) and concluded that the former provided "all the information that a breeder will need from a diallel cross".

## CONCLUSIONS

CONCLUSIONS

1. Net blotch resistance was conditioned by a partially dominant, single, effective factor which was tentatively suggested as one gene. Incorporation of this gene, from Manchuria, into agronomically acceptable New Zealand cultivars could be accomplished via backcrossing.
2. Diallel analysis provided more detailed genetical information on net blotch resistance than previous qualitative analyses. This included estimates of genetic components (D,  $H_1$ ,  $H_2$  and F), heritabilities and findings of epistasis and/or correlated gene distributions.
3. Assessment of leaf section area infected in petri dishes was conducted successfully and biometrical analysis of the data gave results which were similar to those obtained for intact plant tissue. The technique may be useful in future disease resistance studies.
4. In order for yield loss investigations to be conducted successfully, net blotch epidemics must be well established by the stage of maximum grain filling. Delay in disease establishment gives disappointing results.
5. Additive genetic variance was more important than dominance and epistatic genetic variances for most characters.
6. Nitrogen had a profound effect on the majority of barley characters. Net blotch resistance generally decreased dramatically in the high nitrogen environment.

7. The diallel analysis of Mather and Jinks (1971) is based on several assumptions, some of which are difficult to satisfy. Griffing's (1956a, b) combining abilities analysis is less restrictive and provides all essential information for the plant breeder.

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APPENDICES

Stock solution concentrations ( $\text{g } 10^{-3} \text{ m}^{-3}$ )			
Stock Solution	Molecular Weight (g)	High Nitrogen*	Low Nitrogen†
$\text{Ca}(\text{NO}_3)_2 \cdot 4\text{H}_2\text{O}$	236.15	295.19	31.59
$\text{CaSO}_4 \cdot 2\text{H}_2\text{O}$	168.14	-	62.87
Sequestrene 330 (10% DTPA NaFe)	468.20	10.40	10.40
$\text{KH}_2\text{PO}_4$	136.08	34.02	34.02
KCl	74.56	1.58	41.60
$\text{KNO}_3$	101.11	126.39	9.02
$\text{MgSO}_4$	246.50	123.24	123.24
MICRO $\text{H}_3\text{BO}_3$	61.82	0.715	0.715
$\text{MnCl}_2 \cdot 4\text{H}_2\text{O}$	197.92	0.453	0.453
$\text{ZnSO}_4 \cdot 7\text{H}_2\text{O}$	287.55	0.055	0.055
$\text{CuSO}_4 \cdot 5\text{H}_2\text{O}$	249.68	0.020	0.020
$\text{NaMoO}_4 \cdot 2\text{H}_2\text{O}$	241.93	0.007	0.007

\*  $2 \times 10^{-6} \text{ m}^{-3}$  stock solution per  $10^{-3} \text{ m}^{-3}$  water to give final solution.

†  $4 \times 10^{-6} \text{ m}^{-3}$  stock solution per  $10^{-3} \text{ m}^{-3}$  water to give final solution.

APPENDIX I (contd.)Nutrient supply by final solutions ( $\text{mg } 10^{-3} \text{ m}^{-3}$ ).

Nutrient	High Nitrogen	Low Nitrogen
N	105.06	20.00
P	15.49	30.97
K	118.95	140.31
S	32.08	112.10
Ca	100.20	81.41
Fe	2.08	4.16
Mg	24.32	48.64
B	0.250	0.500
Mn	0.251	0.502
Cu	0.010	0.020
Zn	0.025	0.050
Mo	0.005	0.010
Cl	1.822	79.770
Na	1.023	2.046

Variance ratios (VR) involving parental ( $E_p$ ) and  $F_1$  ( $E_F$ ) error mean squares for all characters in low and high nitrogen levels.

	Low nitrogen				High nitrogen			
	$E_F$	$E_p$	Ratio of respective d.f.	VR and signif.	$E_F$	$E_p$	Ratio of respective d.f.	VR and signif.
Net blotch top canopy score					66.59	51.76	27/12	1.29 NS
Net blotch bottom canopy score					49.59	62.90	12/27	1.27 NS
Net blotch flag leaf area			Not calculated as net blotch was absent		55.80	33.89	27/12	1.65 NS
Net blotch second leaf area					51.10	67.26	12/27	1.32 NS
Net blotch leaf section area					88.83	63.44	27/12	1.40 NS
Heading date	2.36	5.81	12/27	2.46*	1.35	1.14	27/12	1.18 NS
Plant height	8.59	5.36	27/12	1.60 NS	43.49	8.31	27/12	5.23 **
Tillers per plant	2.29	1.07	27/12	2.14 (NS)	44.69	5.73	27/12	7.80 **
Spikelet number per ear	0.81	0.55	27/12	1.48 NS	3.21	2.01	27/12	1.60 NS
Grain yield	0.76	1.24	12/27	1.64 NS	9.55	8.64	27/12	1.11 NS
100 kernel weight	0.04	0.06	12/27	1.42 NS	0.19	0.18	27/12	1.02 NS
Grain number per ear	0.27	0.75	12/27	2.74 *	0.45	0.70	12/27	1.55 NS
Physiological brown spot	12.29	19.49	12/27	1.59 NS	31.48	22.68	27/12	1.39 NS

NS =  $P > 0.10$ ; (NS) =  $0.10 \geq P > 0.05$ ; \* =  $0.05 \geq P > 0.01$ ; \*\* =  $0.01 \geq P > 0.001$ .

APPENDIX 3.Modifications of equations which estimate genetic components for a full diallel to those suitable for a half diallel.

Equations given by Mather and Jinks (1971) on page 269 estimated genetic components ( $H_1$ ,  $H_2$  and  $F$ ) for a full diallel and were therefore unsuitable in this study. Equations estimating basic array statistics (mostly on page 253) were modified by removal of a half in all coefficients of the  $F_1$  error mean square ( $E_F$ ) since there was only one set of  $F_1$ 's. Hence, equations for a half diallel which relate basic array statistics to genetic and environmental components are:

$$V_p = D + E_p$$

$$V_{\bar{r}} = \frac{1}{2}D + \frac{1}{2}H_1 - \frac{1}{2}H_2 - \frac{1}{4}F + \frac{(n-1)}{n^2}E_F + \frac{1}{n^2}E_p$$

$$\bar{V}_r = \frac{1}{2}D + \frac{1}{2}H_1 - \frac{1}{4}F + \frac{(n-1)}{n}E_F + \frac{1}{n}E_p$$

$$\bar{W}_r = \frac{1}{2}D - \frac{1}{4}F + \frac{1}{n}E_p$$

Appropriate combinations of these equations are used below to estimate  $H_1$ ,  $H_2$  and  $F$  for a half diallel.

Estimation of  $H_1$ :

Using  $D = V_p - E_p$

$$\bar{W}_r = \frac{1}{2}D - \frac{1}{4}F + \frac{1}{n}E_p$$

$$\bar{V}_r = \frac{1}{2}D + \frac{1}{2}H_1 - \frac{1}{4}F + \frac{(n-1)}{n}E_F + \frac{1}{n}E_p$$

Substituting  $(V_p - E_p)$  for  $D$  in equations estimating  $\bar{W}_r$  and  $\bar{V}_r$

$$\bar{W}_r = \frac{1}{2}V_p - \frac{1}{2}E_p - \frac{1}{4}F + \frac{1}{n}E_p$$

$$\bar{V}_r = \frac{1}{2}V_p - \frac{1}{2}E_p + \frac{1}{2}H_1 - \frac{1}{4}F + \frac{(n-1)}{n}E_F + \frac{1}{n}E_p$$

$$\bar{W}_r - \bar{V}_r = \frac{1}{2}V_p - \frac{1}{2}E_p - \frac{1}{2}H_1 - \frac{(n-1)}{n}E_F$$

$$(x 4) \quad 4\bar{W}_r - 4\bar{V}_r = V_p - E_p - H_1 - \frac{4(n-1)}{n}E_F$$

$$H_1 = 4\bar{V}_r + V_p - 4\bar{W}_r - \left(\frac{4(n-1)}{n}E_F + E_p\right)$$

If  $E_F$  is not significantly different from  $E_p$ ,  $E_{POOL}$  may be used.

Hence,

$$H_1 = 4\bar{V}_r + V_p - 4\bar{W}_r - \left(\frac{3n-2}{n}\right)E_{POOL}$$

Estimation of  $H_2$ :

$$\text{Using } V_{\bar{r}} = \frac{1}{2}D + \frac{1}{2}H_1 - \frac{1}{2}H_2 - \frac{1}{2}F + \frac{(n-1)}{n^2}E_F + \frac{1}{n^2}E_p$$

$$\bar{V}_r = \frac{1}{2}D + \frac{1}{2}H_1 - \frac{1}{2}F + \frac{(n-1)}{n}E_F + \frac{1}{n}E_p$$

$$\bar{V}_r - V_{\bar{r}} = \frac{1}{2}H_2 + \left(\frac{(n-1)}{n} - \frac{(n-1)}{n^2}\right)E_F + \left(\frac{1}{n} - \frac{1}{n^2}\right)E_p$$

Simplifying environmental components -

$$\left(\frac{(n-1)}{n} - \frac{(n-1)}{n^2}\right)E_F = \left(\frac{(n-1)^2}{n^2}\right)E_F$$

$$\left(\frac{1}{n} - \frac{1}{n^2}\right)E_p = \left(\frac{(n-1)}{n^2}\right)E_p$$

Hence,

$$\bar{V}_r - V_{\bar{r}} = \frac{1}{2}H_2 + \frac{(n-1)^2}{n^2}E_F + \frac{(n-1)}{n^2}E_p$$

$$(x 4) \quad 4\bar{V}_r - 4V_{\bar{r}} = H_2 + \frac{(n-1)^2}{n^2}E_F + \frac{(n-1)}{n^2}E_p$$

$$H_2 = 4\bar{V}_r - 4V_{\bar{r}} - \left(\frac{4(n-1)^2}{n^2}E_F + \frac{4(n-1)}{n^2}E_p\right)$$

If  $E_F$  is not significantly different from  $E_p$ ,  $E_{POOL}$  may be used.

Hence,

$$H_2 = 4\bar{V}_r - 4V_{\bar{r}} - 4E_{POOL} \left(1 - \frac{1}{n}\right)$$

Estimation of F.

Using  $D = V_p - E_p$

$$\bar{W}_r = \frac{1}{2}D - \frac{1}{4}F + \frac{1}{n}E_p$$

Substituting  $(V_p - E_p)$  for  $D$  in equations estimating  $\bar{W}_r$  and  $\bar{V}_r$

$$\bar{W}_r = \frac{1}{2}V_p - \frac{1}{2}E_p - \frac{1}{4}F + \frac{1}{n}E_p$$

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

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

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

If  $E_p$  is not significantly different from  $E_F$ ,  $E_{POOL}$  may be used.

Hence,

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

APPENDIX 4.

Derivation of formulae which estimate standard errors of  
heterosis and heterobeltiosis for single environments  
and pooled analysis over environments.

Heterosis ( $\bar{F}_1 - \bar{MP}$ ):

Standard error of heterosis for parents and crosses in a replicated trial (b blocks) in a single environment is

$$\sqrt{\frac{\sigma_{F_1}^2}{b} + \frac{\sigma_p^2}{2b}}$$

after the procedures of Steel and Torrie (1980).  $\sigma_{F_1}^2$  and  $\sigma_p^2$  are error mean squares of  $F_1$ 's and parents, respectively. The factor of one half in the ratio  $\sigma_p^2/2b$  is due to the mid-parent (MP) being an average of the two parents involved in a particular cross. In the pooled analysis over environments (e) both error mean squares are also divided by e to account for the additional replication.

Heterobeltiosis ( $\bar{F}_1 - \bar{P}$ ):

As heterobeltiosis is the superiority of an  $F_1$  over its better parent, rather than mid-parent, there is no division of  $\sigma_p^2$  by two as for heterosis. Furthermore, if  $\sigma_{F_1}^2$  is not significantly different from  $\sigma_p^2$ , a pooled error mean square ( $\sigma_{POOL}^2$ ) may be used. Hence, standard error formulae for heterobeltiosis, based on the methods of Steel and Torrie (1980), are

$$\sqrt{\frac{2 \sigma_{POOL}^2}{b}} \quad (\text{single environment})$$

and

$$\sqrt{\frac{2 \sigma_{POOL}^2}{be}} \quad (\text{pooled analysis over environments}).$$

APPENDIX 5.1

Analysis of variance (for parents + crosses, parents and crosses)

for all methods of assessment of net blotch infection

Top Canopy Score (transformed to  $\sin^{-1}\sqrt{x/10}$ )

1) Original data

Source	DF	Mean Square	F-test	Signif.
Blocks	3	114.13	1.83	NS
Genotypes	14	197.86	3.17	**
ERROR	42	62.43		
Standard deviation = 7.90		Coefficient of variation = 0.33		
S.E. genotype means = 3.95				
Blocks	3	3.20	0.06	NS
Parents	4	197.13	3.81	*
ERROR	12	51.76		
Standard deviation = 7.19		Coefficient of variation = 0.34		
S.E. parent means = 3.60				
Blocks	3	178.69	2.68	(NS)
Crosses	9	183.33	2.75	*
ERROR	27	66.59		
Standard deviation = 8.16		Coefficient of variation = 0.31		
S.E. cross means = 4.08				

2) Reanalysed data (array 2 deleted):

Source	DF	Mean Square	F-test	Signif.
Blocks	3	13.25	0.39	NS
Genotypes	9	191.77	5.61	***
ERROR	27	34.18		
Standard deviation = 5.85		Coefficient of variation = 0.26		
S.E. genotype means = 2.92				

## APPENDIX 5.1 (contd.)

Bottom Canopy Score (transformed to  $\sin^{-1}\sqrt{X/10}$ )

1) Original data:

Source	DF	Mean Square	F-test	Signif.
Blocks	3	198.97	3.47	*
Genotypes	14	181.41	3.16	**
ERROR	42	57.38		
Standard deviation = 7.58		Coefficient of variation = 0.31		
S.E. genotype means = 3.79				
Blocks	3	7.14	0.11	NS
Parents	4	223.43	3.55	*
ERROR	12	62.90		
Standard deviation = 7.93		Coefficient of variation = 0.37		
S.E. parent means = 3.97				
Blocks	3	297.48	6.00	**
Crosses	9	154.24	3.11	*
ERROR	27	49.59		
Standard deviation = 7.04		Coefficient of variation = 0.27		
S.E. cross means = 3.52				

2) Reanalysed data (array 2 deleted):

Source	DF	Mean Square	F-test	Signif.
Blocks	3	62.02	1.74	NS
Genotypes	9	145.76	4.09	**
ERROR	27	35.63		
Standard deviation = 5.97		Coefficient of variation = 0.27		
S.E. genotype means = 2.98				

## APPENDIX 5.1 (contd.)

Flag Leaf Area (transformed to  $\sin^{-1}\sqrt{X/100}$ )

1) Original data:

Source	DF	Mean Square	F-test	Signif.
Blocks	3	267.14	5.43	**
Genotypes	14	248.28	5.05	***
ERROR	42	49.16		
Standard deviation = 7.01		Coefficient of variation = 0.41		
S.E. genotype means = 3.51				
Blocks	3	29.88	0.88	NS
Parents	4	453.82	13.39	***
ERROR	12	33.89		
Standard deviation = 5.82		Coefficient of variation = 0.35		
S.E. parent means = 2.91				
Blocks	3	287.75	5.16	**
Crosses	9	183.19	3.28	**
ERROR	27	55.80		
Standard deviation = 7.47		Coefficient of variation = 0.43		
S.E. cross means = 3.74				

2) Reanalysed data (array 3 deleted):

Source	DF	Mean Square	F-test	Signif.
Blocks	3	72.08	1.99	NS
Genotypes	9	334.50	9.24	***
ERROR	27	36.18		
Standard deviation = 6.02		Coefficient of variation = 0.37		
S.E. genotype means = 3.01				

## APPENDIX 5.1 (contd.)

Second Leaf Area (transformed to  $\sin^{-1}\sqrt{X/100}$ )

Source	DF	Mean Square	F-test	Signif.
Blocks	3	115.00	2.03	NS
Genotypes	14	351.49	6.22	***
ERROR	42	56.53		
Standard deviation = 7.52		Coefficient of variation = 0.35		
S.E. genotype means = 3.76				
Blocks	3	57.62	0.86	NS
Parents	4	537.81	8.00	**
ERROR	12	67.26		
Standard deviation = 8.20		Coefficient of variation = 0.42		
S.E. parent means = 4.10				
Blocks	3	120.03	2.35	(NS)
Crosses	9	291.61	5.71	***
ERROR	27	51.10		
Standard deviation = 7.15		Coefficient of variation = 0.31		
S.E. cross means = 3.57				

Leaf Section Area (transformed to  $\sin^{-1}\sqrt{X/100}$ )1) Original data:

Source	DF	Mean Square	F-test	Signif.
Blocks	3	13.47	0.17	NS
Genotypes	14	431.38	5.59	***
ERROR	42	77.18		
Standard deviation = 8.79		Coefficient of variation = 0.65		
S.E. genotype means = 4.39				
Blocks	3	29.22	0.46	NS
Parents	4	342.28	5.40	*
ERROR	12	63.44		
Standard deviation = 7.97		Coefficient of variation = 0.69		
S.E. parent means = 3.98				
Blocks	3	11.56	0.13	NS
Crosses	9	507.24	5.71	***
ERROR	27	88.83		
Standard deviation = 9.43		Coefficient of variation = 0.65		
S.E. cross means = 4.71				

APPENDIX 5.1 (contd.)2) Reanalysed data (array 1 deleted):

Source	DF	Mean Square	F-test	Signif.
Blocks	3	15.47	0.19	NS
Genotypes	9	288.41	3.56	**
ERROR	27	81.11		

Standard deviation = 9.01                      Coefficient of variation = 0.93  
S.E. genotype means = 4.50

APPENDIX 5.2

Analysis of variance of  $(W_r + V_r)/(W_r - V_r)$  for all  
methods of assessment of net blotch infection

Top Canopy Score (transformed to  $\sin^{-1}\sqrt{X/10}$ )

1) Original data:

Source	DF	Mean Square	F-test	Signif.
Blocks	3	83569.00	10.42	**
Arrays( $W_r+V_r$ )	4	8641.50	1.08	NS
ERROR	12	8017.58		
Standard deviation = 89.54		Coefficient of variation = 0.72		
Blocks	3	8400.87	8.55	**
Arrays( $W_r-V_r$ )	4	1691.28	1.72	NS
ERROR	12	982.23		
Standard deviation = 31.34		Coefficient of variation = 1.08		

2) Reanalysed data (array 2 deleted):

Source	DF	Mean Square	F-test	Signif.
Blocks	3	34831.67	10.33	**
Arrays( $W_r+V_r$ )	3	2607.33	0.77	NS
ERROR	9	3371.33		
Standard deviation = 58.06		Coefficient of variation = 0.60		
Blocks	3	675.07	1.06	NS
Arrays( $W_r-V_r$ )	3	929.73	1.46	NS
ERROR	9	635.94		
Standard deviation = 25.22		Coefficient of variation = 1.54		

APPENDIX 5.2 (contd.)Bottom Canopy Score (transformed to  $\sin^{-1}\sqrt{X/10}$ )1) Original data:

Source	DF	Mean Square	F-test	Signif.
Blocks	3	61618.67	8.09	**
Arrays ( $W_R+V_R$ )	4	10464.25	1.37	NS
ERROR	12	7620.50		
Standard deviation = 87.30		Coefficient of variation = 0.75		
Blocks	3	3799.00	2.92	(NS)
Arrays ( $W_R-V_R$ )	4	355.00	0.27	NS
ERROR	12	1300.00		
Standard deviation = 36.06		Coefficient of variation = 1.92		

2) Reanalysed data (array 2 deleted):

Source	DF	Mean Square	F-test	Signif.
Blocks	3	22138.33	4.40	*
Arrays ( $W_R+V_R$ )	3	5702.00	1.13	NS
ERROR	9	5035.22		
Standard deviation = 70.96		Coefficient of variation = 0.86		
Blocks	3	1984.97	6.75	*
Arrays ( $W_R-V_R$ )	3	235.66	0.80	NS
ERROR	9	294.22		
Standard deviation = 17.15		Coefficient of variation = 1.49		

## APPENDIX 5.2 (contd.)

Flag Leaf Area (transformed to  $\sin^{-1}\sqrt{X/100}$ )

1) Original data:

Source	DF	Mean Square	F-test	Signif.
Blocks	3	33547.67	8.21	**
Arrays( $W_R+V_R$ )	4	12662.25	3.10	(NS)
ERROR	12	4086.50		
Standard deviation = 63.93		Coefficient of variation = 0.50		
Blocks	3	8344.67	9.43	**
Arrays( $W_R-V_R$ )	4	528.95	0.60	NS
ERROR	12	884.60		
Standard deviation = 29.74		Coefficient of variation = 5.95		

2) Reanalysed data (array 3 deleted):

Source	DF	Mean Square	F-test	Signif.
Blocks	3	21158.67	2.92	(NS)
Arrays( $W_R+V_R$ )	3	18686.33	2.58	NS
ERROR	9	7241.89		
Standard deviation = 85.10		Coefficient of variation = 0.54		
Blocks	3	2629.10	4.28	*
Arrays( $W_R-V_R$ )	3	231.17	0.38	NS
ERROR	9	614.68		
Standard deviation = 24.79		Coefficient of variation = 1.47		

## APPENDIX 5.2 (contd.)

Second Leaf Area (transformed to  $\sin^{-1}\sqrt{X/100}$ )

Source	DF	Mean Square	F-test	Signif.
Blocks	3	27307.67	6.91	**
Arrays( $W_R+V_R$ )	4	20456.50	5.17	*
ERROR	12	3954.17		
Standard deviation = 62.88		Coefficient of variation = 0.36		
Blocks	3	7221.70	5.18	*
Arrays( $W_R-V_R$ )	4	531.50	0.38	NS
ERROR	12	1395.00		
Standard deviation = 37.35		Coefficient of variation = 1.81		

Leaf Section Area (transformed to  $\sin^{-1}\sqrt{X/100}$ )1) Original data:

Source	DF	Mean Square	F-test	Signif.
Blocks	3	5798.32	0.27	NS
Arrays( $W_R+V_R$ )	4	44950.58	2.11	NS
ERROR	12	21335.44		
Standard deviation = 146.07		Coefficient of variation = 0.68		
Blocks	3	9996.00	3.72	*
Arrays( $W_R-V_R$ )	4	11033.25	4.10	*
ERROR	12	2690.75		
Standard deviation = 51.87		Coefficient of variation = 0.84		

2) Reanalysed data (array 1 deleted):

Source	DF	Mean Square	F-test	Signif.
Blocks	3	13397.66	0.77	NS
Arrays( $W_R+V_R$ )	3	46421.00	2.68	NS
ERROR	9	17319.11		
Standard deviation = 131.60		Coefficient of variation = 0.73		
Blocks	3	5225.33	2.25	NS
Arrays( $W_R-V_R$ )	3	4365.33	1.88	NS
ERROR	9	2322.11		
Standard deviation = 48.19		Coefficient of variation = 1.38		

APPENDIX 6Pooled analysis of variance over control(fungicide treated) and diseased treatmentsHeading Date

Source	Mean Square	F-test	Signif.	Test DF
Treatments	58.81	5.82	*	(1,10)
Blocks (Trt.)	5.11	2.66	*	
Genotypes	11.74	2.21	NS	(4,4)
Genotype-treatment	5.32	2.77	*	
ERROR	1.92			
Standard deviation = 1.39		Coefficient of variation = 0.02		
S.E. genotype means = 0.82				

Plant Height

Source	Mean Square	F-test	Signif.	Test DF
Treatments	0.03	0.16	NS	(24,10)
Blocks (Trt.)	81.04	4.33	**	
Genotypes	220.10	6.14	(NS)	(4,4)
Genotype-treatment	35.85	1.92	NS	
ERROR	18.70			
Standard deviation = 4.32		Coefficient of variation = 0.04		
S.E. genotype means = 2.12				

Tillers per Plant

Source	Mean Square	F-test	Signif.	Test DF
Treatments	191.41	2.49	NS	(1,7)
Blocks	25.49	0.80	NS	
Genotypes	165.15	2.56	NS	(4,4)
Genotype-treatment	64.39	2.01	NS	
ERROR	31.97			
Standard deviation = 5.65		Coefficient of variation = 0.20		
S.E. genotype means = 2.84				

## APPENDIX 6 (contd.)

Top Canopy Score (transformed to  $\sin^{-1}\sqrt{X/10}$ )

Source	Mean Square	F-test	Signif.	Test DF
Treatments	198.78	4.85	(NS)	(1,10)
Blocks (Trt.)	28.97	0.66	NS	
Genotypes	530.31	12.16	*	(4,4)
Genotype-treatment	20.98	0.48	NS	
ERROR	43.60			

Standard deviation = 6.60                      Coefficient of variation = 0.35  
S.E. genotype means = 1.62

Bottom Canopy Score (transformed to  $\sin^{-1}\sqrt{X/10}$ )

Source	Mean Square	F-test	Signif.	Test DF
Treatments	52.72	1.43	NS	(3,8)
Blocks (Trt.)	21.55	0.52	NS	
Genotypes	640.26	14.38	*	(4,4)
Genotype-treatment	44.53	1.07	NS	
ERROR	41.74			

Standard deviation = 6.46                      Coefficient of variation = 0.32  
S.E. genotype means = 2.36

Flag Leaf Area (transformed to  $\sin^{-1}\sqrt{X/100}$ )

Source	Mean Square	F-test	Signif.	Test DF
Treatments	256.04	2.94	NS	(1,10)
Blocks (Trt.)	50.86	1.87	NS	
Genotypes	621.71	13.71	*	(4,4)
Genotype-treatment	45.35	1.66	NS	
ERROR	27.26			

Standard deviation = 5.22                      Coefficient of variation = 0.37  
S.E. genotype means = 2.38

Second Leaf Area (transformed to  $\sin^{-1}\sqrt{X/100}$ )

Source	Mean Square	F-test	Signif.	Test DF
Treatments	267.91	2.34	NS	(1,8)
Blocks (Trt.)	45.49	0.91	NS	
Genotypes	789.89	8.73	*	(4,4)
Genotype-treatment	90.48	1.81	NS	
ERROR	49.92			

Standard deviation = 7.07                      Coefficient of variation = 0.42  
S.E. genotype means = 3.36

## APPENDIX 6 (contd.)

Leaf Section Area (transformed to  $\sin^{-1}\sqrt{X/100}$ )

Source	Mean Square	F-test	Signif.	Test DF
Treatments	142.62	0.92	NS	(3,8)
Blocks (Trt.)	96.91	1.02	NS	
Genotypes	646.51	4.02	NS	(4,4)
Genotype-treatment	160.79	1.69	NS	
ERROR	95.03			

Standard deviation = 9.75                      Coefficient of variation = 0.72  
S.E. genotype means = 4.48

Spikelet Number per Ear

Source	Mean Square	F-test	Signif.	Test DF
Treatments	0.03	0.20	NS	(25,9)
Blocks (Trt.)	5.19	2.44	(NS)	
Genotypes	210.27	36.17	**	(4,4)
Genotype-treatment	5.81	2.73	(NS)	
ERROR	2.13			

Standard deviation = 1.46                      Coefficient of variation = 0.05  
S.E. genotype means = 0.85

Grain Number per Ear (transformed to  $10 \log_{10} X$ )

Source	Mean Square	F-test	Signif.	Test DF
Treatments	0.62	1.11	NS	(6,9)
Blocks (Trt.)	1.03	1.05	NS	
Genotypes	24.98	25.47	**	(4,4)
Genotype-treatment	0.42	0.42	NS	
ERROR	0.98			

Standard deviation = 0.99                      Coefficient of variation = 0.08  
S.E. genotype means = 0.23

Grain Yield

Source	Mean Square	F-test	Signif.	Test DF
Treatments	60.4422	1.48	NS	(1,9)
Blocks (Trt.)	22.6990	1.79	NS	
Genotypes	90.6534	3.38	NS	(4,4)
Genotype-treatment	26.8096	2.12	NS	
ERROR	12.6554			

Standard deviation = 3.5574                      Coefficient of variation = 0.30  
S.E. genotype means = 1.8306

APPENDIX 6 (contd.)100 Kernel Weight

Source	Mean Square	F-test	Signif.	Test DF
Treatments	1.5880	2.24	NS	(2,10)
Blocks (Trt.)	0.5963	1.17	NS	
Genotypes	0.3521	0.69	NS	(4,4)
Genotype-treatment	0.3404	0.67	NS	
ERROR	0.5104			

Standard deviation = 0.7144                      Coefficient of variation = 0.18  
S.E. genotype means = 0.2063

Physiological Brown Spot (transformed to  $\sin^{-1}\sqrt{x/100}$ )

Source	Mean Square	F-test	Signif.	Test DF
Treatments	97.34	1.24	NS	(2,10)
Blocks (Trt.)	61.05	2.63	*	
Genotypes	1022.11	28.23	**	(4,4)
Genotype-treatment	36.20	1.56	NS	
ERROR	23.20			

Standard deviation = 4.82                      Coefficient of variation = 0.17  
S.E. genotype means = 2.13

APPENDIX 7Analysis of variance (for parents + crosses, parents and crosses)  
for all minor characters in single environments (nitrogen levels)Low NitrogenHeading Date.

Source	DF	Mean Square	F-test	Signif.
Blocks	3	8.83	2.63	(NS)
Genotypes	14	295.53	87.95	***
ERROR	42	3.36		
Standard deviation = 1.83		Coefficient of variation = 0.03		
S.E. genotype means = 0.92				
Blocks	3	8.25	1.42	NS
Parents	4	20.05	3.45	*
ERROR	12	5.81		
Standard deviation = 2.41		Coefficient of variation = 0.04		
S.E. parent means = 1.21				
Blocks	3	3.17	1.35	NS
Crosses	9	335.76	142.38	***
ERROR	27	2.36		
Standard deviation = 1.54		Coefficient of variation = 0.03		
S.E. cross means = 0.77				

Plant Height1) Original data:

Source	DF	Mean Square	F-test	Signif.
Blocks	3	4.72	0.63	NS
Genotypes	14	231.93	30.88	***
ERROR	42	7.51		
Standard deviation = 2.74		Coefficient of variation = 0.04		
S.E. genotype means = 1.37				
Blocks	3	2.71	0.51	NS
Parents	4	251.68	46.97	***
ERROR	12	5.36		
Standard deviation = 2.31		Coefficient of variation = 0.03		
S.E. parent means = 1.16				
Blocks	3	8.38	0.98	NS
Crosses	9	235.36	27.39	***
ERROR	27	8.59		
Standard deviation = 2.59		Coefficient of variation = 0.04		
S.E. cross means = 1.47				

## APPENDIX 7 (contd.)

2) Reanalysed data (array 3 deleted):

Source	DF	Mean Square	F-test	Signif.
Blocks	3	2.41	0.29	NS
Genotypes	9	261.45	31.87	***
ERROR	27	8.20		
Standard deviation = 2.86		Coefficient of variation = 0.04		
S.E. genotype means = 1.43				

Tillers per Plant1) Original data:

Source	DF	Mean Square	F-test	Signif.
Blocks	3	1.76	0.76	NS
Genotypes	14	46.82	20.11	***
ERROR	42	2.33		
Standard deviation = 1.53		Coefficient of variation = 0.11		
S.E. genotype means = 0.76				
Blocks	3	7.27	6.80	**
Parents	4	79.47	74.36	***
ERROR	12	1.07		
Standard deviation = 1.03		Coefficient of variation = 0.08		
S.E. parent means = 0.52				
Blocks	3	2.22	0.97	NS
Crosses	9	37.00	16.17	***
ERROR	27	2.29		
Standard deviation = 1.52		Coefficient of variation = 0.11		
S.E. cross means = 0.76				

2) Reanalysed data (array 3 deleted):

Source	DF	Mean Square	F-test	Signif.
Blocks	3	0.81	0.33	NS
Genotypes	9	53.86	21.95	***
ERROR	27	2.45		
Standard deviation = 1.57		Coefficient of variation = 0.12		
S.E. genotype means = 0.78				

## APPENDIX 7 (contd.)

Spikelet Number per Ear

Source	DF	Mean Square	F-test	Signif.
Blocks	3	4.38	5.63	**
Genotypes	14	28.30	36.37	***
ERROR	42	0.78		
Standard deviation = 0.88		Coefficient of variation = 0.04		
S.E. genotype means = 0.44				
Blocks	3	1.10	2.02	NS
Parents	4	67.85	124.12	***
ERROR	12	0.55		
Standard deviation = 0.74		Coefficient of variation = 0.04		
S.E. parent means = 0.37				
Blocks	3	4.70	5.80	**
Crosses	9	11.15	13.78	***
ERROR	27	0.81		
Standard deviation = 0.90		Coefficient of variation = 0.04		
S.E. cross means = 0.45				

Grain Number per Ear (transformed to  $10 \log_{10}X$ )

Source	DF	Mean Square	F-test	Signif.
Blocks	3	0.47	1.06	NS
Genotypes	14	9.00	20.22	***
ERROR	42	0.44		
Standard deviation = 0.67		Coefficient of variation = 0.06		
S.E. genotype means = 0.33				
Blocks	3	0.31	0.41	NS
Parents	4	21.37	28.58	***
ERROR	12	0.75		
Standard deviation = 0.86		Coefficient of variation = 0.08		
S.E. parent means = 0.43				
Blocks	3	0.95	3.49	*
Crosses	9	4.41	16.13	***
ERROR	27	0.27		
Standard deviation = 0.52		Coefficient of variation = 0.05		
S.E. cross means = 0.26				

## APPENDIX 7 (contd.)

Grain Yield

Source	DF	Mean Square	F-test	Signif.
Blocks	3	9.1913	9.86	***
Genotypes	14	4.5560	4.89	***
ERROR	42	0.9319		
Standard deviation = 0.9654		Coefficient of variation = 0.11		
S.E. genotype means = 0.4827				
Blocks	3	1.4201	1.14	NS
Parents	4	11.6322	9.37	**
ERROR	12	1.2409		
Standard deviation = 1.1139		Coefficient of variation = 0.12		
S.E. parent means = 0.5570				
Blocks	3	9.0392	11.94	***
Crosses	9	1.8985	2.51	*
ERROR	27	0.7573		
Standard deviation = 0.8702		Coefficient of variation = 0.10		
S.E. cross means = 0.4351				

100 Kernel Weight

Source	DF	Mean Square	F-test	Signif.
Blocks	3	0.2171	4.00	*
Genotypes	14	0.9356	17.24	***
ERROR	42	0.0543		
Standard deviation = 0.2330		Coefficient of variation = 0.04		
S.E. genotype means = 0.1165				
Blocks	3	0.0298	0.49	NS
Parents	4	0.5400	8.91	**
ERROR	12	0.0606		
Standard deviation = 0.2461		Coefficient of variation = 0.04		
S.E. parent means = 0.1231				
Blocks	3	0.3215	7.55	**
Crosses	9	0.8715	20.46	***
ERROR	27	0.0426		
Standard deviation = 0.2064		Coefficient of variation = 0.03		
S.E. cross means = 0.1032				

## APPENDIX 7 (contd.)

Physiological Brown Spot (transformed to  $\sin^{-1}\sqrt{X/100}$ )1) Original data:

Source	DF	Mean Square	F-test	Signif.
Blocks	3	73.77	5.26	**
Genotypes	14	91.86	6.55	***
ERROR	42	14.02		
Standard deviation = 3.75		Coefficient of variation = 0.22		
S.E. genotype means = 1.87				
Blocks	3	16.09	0.83	NS
Parents	4	89.47	4.59	*
ERROR	12	19.49		
Standard deviation = 4.42		Coefficient of variation = 0.31		
S.E. parent means = 2.21				
Blocks	3	65.49	5.33	**
Crosses	9	75.72	6.16	***
ERROR	27	12.29		
Standard deviation = 3.51		Coefficient of variation = 0.19		
S.E. cross means = 1.75				

2) Reanalysed data (array 5 deleted):

Source	DF	Mean Square	F-test	Signif.
Blocks	3	74.85	6.70	**
Genotypes	9	34.01	3.04	*
ERROR	27	11.17		
Standard deviation = 3.34		Coefficient of variation = 0.21		
S.E. genotype means = 1.67				

## APPENDIX 7 (contd.)

High NitrogenHeading Date

Source	DF	Mean Square	F-test	Signif.
Blocks	3	1.49	1.17	NS
Genotypes	14	302.07	236.52	***
ERROR	42	1.28		
Standard deviation = 1.13		Coefficient of variation = 0.02		
S.E. genotype means = 0.57				
Blocks	3	1.38	1.21	NS
Parents	4	3.71	3.25	*
ERROR	12	1.14		
Standard deviation = 1.07		Coefficient of variation = 0.02		
S.E. parent means = 0.53				
Blocks	3	1.29	0.96	NS
Crosses	9	366.49	271.52	***
ERROR	27	1.35		
Standard deviation = 1.16		Coefficient of variation = 0.02		
S.E. cross means = 0.58				

Plant Height

Source	DF	Mean Square	F-test	Signif.
Blocks	3	21.78	0.63	NS
Genotypes	14	38.58	1.12	NS
ERROR	42	34.35		
Standard deviation = 5.86		Coefficient of variation = 0.06		
S.E. genotype means = 2.93				
Blocks	3	53.10	6.39	**
Parents	4	71.00	8.55	**
ERROR	12	8.31		
Standard deviation = 2.88		Coefficient of variation = 0.03		
S.E. parent means = 1.44				
Blocks	3	25.01	0.58	NS
Crosses	9	27.16	0.62	NS
ERROR	27	43.49		
Standard deviation = 6.59		Coefficient of variation = 0.06		
S.E. cross means = 3.30				

## APPENDIX 7 (contd.)

Tillers per Plant

Source	DF	Mean Square	F-test	Signif.
Blocks	3	50.39	1.60	NS
Genotypes	14	116.05	3.67	***
ERROR	42	31.59		
Standard deviation = 5.62			Coefficient of variation = 0.19	
S.E. genotype means = 2.81				
Blocks	3	21.35	3.72	*
Parents	4	100.58	17.55	***
ERROR	12	5.73		
Standard deviation = 2.39			Coefficient of variation = 0.08	
S.E. parent means = 1.20				
Blocks	3	46.16	1.03	NS
Crosses	9	132.87	2.97	*
ERROR	27	44.69		
Standard deviation = 6.69			Coefficient of variation = 0.23	
S.E. cross means = 3.34				

Spikelet Number per Ear

Source	DF	Mean Square	F-test	Signif.
Blocks	3	2.18	0.73	NS
Genotypes	14	46.81	15.67	***
ERROR	42	2.99		
Standard deviation = 1.73			Coefficient of variation = 0.07	
S.E. genotype means = 0.86				
Blocks	3	1.33	0.66	NS
Parents	4	107.79	53.57	***
ERROR	12	2.01		
Standard deviation = 1.42			Coefficient of variation = 0.05	
S.E. parent means = 0.71				
Blocks	3	5.69	1.77	NS
Crosses	9	24.71	7.69	***
ERROR	27	3.21		
Standard deviation = 1.79			Coefficient of variation = 0.07	
S.E. cross means = 0.90				

## APPENDIX 7 (contd.)

Grain Number per Ear (transformed to  $10 \log_{10} X$ )

Source	DF	Mean Square	F-test	Signif.
Blocks	3	0.45	0.91	NS
Genotypes	14	0.13	12.43	***
ERROR	42	0.49		
Standard deviation = 0.70		Coefficient of variation = 0.06		
S.E. genotype means = 0.35				
Blocks	3	0.25	0.36	NS
Parents	4	15.38	22.13	***
ERROR	12	0.70		
Standard deviation = 0.83		Coefficient of variation = 0.06		
S.E. parent means = 0.42				
Blocks	3	0.26	0.57	NS
Crosses	9	2.63	5.86	***
ERROR	27	0.45		
Standard deviation = 0.67		Coefficient of variation = 0.05		
S.E. cross means = 0.34				

Grain Yield

Source	DF	Mean Square	F-test	Signif.
Blocks	3	57.7775	6.64	***
Genotypes	14	36.2998	4.17	***
ERROR	42	8.7009		
Standard deviation = 2.9497		Coefficient of variation = 0.24		
S.E. genotype means = 1.4749				
Blocks	3	23.0885	2.67	(NS)
Parents	4	73.6300	8.52	**
ERROR	12	8.6418		
Standard deviation = 2.9397		Coefficient of variation = 0.22		
S.E. parent means = 1.4698				
Blocks	3	35.9962	3.77	*
Crosses	9	22.0687	2.31	*
ERROR	27	9.5486		
Standard deviation = 3.0901		Coefficient of variation = 0.26		
S.E. cross means = 1.5450				

## APPENDIX 7 (contd.)

100 Kernel Weight

Source	DF	Mean Square	F-test	Signif.
Blocks	3	1.5814	8.80	***
Genotypes	14	0.9404	5.23	***
ERROR	42	0.1798		
Standard deviation = 0.4240		Coefficient of variation = 0.09		
S.E. genotype means = 0.2120				
Blocks	3	0.5971	3.30	(NS)
Parents	4	0.3300	1.83	NS
ERROR	12	0.1807		
Standard deviation = 0.4251		Coefficient of variation = 0.10		
S.E. parent means = 0.2125				
Blocks	3	1.1150	6.03	**
Crosses	9	0.9458	5.12	***
ERROR	27	0.1848		
Standard deviation = 0.4299		Coefficient of variation = 0.09		
S.E. cross means = 0.2149				

Physiological Brown Spot (transformed to  $\sin^{-1}\sqrt{X/100}$ )1) Original data:

Source	DF	Mean Square	F-test	Signif.
Blocks	3	24.83	0.92	NS
Genotypes	14	313.47	11.63	***
ERROR	42	26.96		
Standard deviation = 5.19		Coefficient of variation = 0.22		
S.E. genotype means = 2.60				
Blocks	3	10.90	0.48	NS
Parents	4	657.45	28.98	***
ERROR	12	22.68		
Standard deviation = 4.76		Coefficient of variation = 0.18		
S.E. parent means = 2.38				
Blocks	3	17.88	0.57	NS
Crosses	9	171.14	5.44	***
ERROR	27	31.48		
Standard deviation = 5.61		Coefficient of variation = 0.25		
S.E. cross means = 2.81				

APPENDIX 7 (contd.)2) Reanalysed data (array 3 deleted):

Source	DF	Mean Square	F-test	Signif.
Blocks	3	65.50	2.10	NS
Genotypes	9	297.65	9.54	***
ERROR	27	31.19		

Standard deviation = 5.59                      Coefficient of variation = 0.26  
S.E. genotype means = 2.79

Analysis of variance over pooled nitrogen levels(all genotypes included)Heading Date

Source	Mean Square	F-test	Signif.	Test DF
Envir (Nitrogen)	21.68	2.65	NS	(1,15)
Blks (Env)	5.16	2.23	*	
Genotypes	593.71	152.30	***	(14,14)
Genot-Nitrogen	3.90	1.68	(NS)	
ERROR	2.32			

Standard deviation = 1.52                      Coefficient of variation = 0.03  
S.E. Genotype means = 0.70

Plant Height

Source	Mean Square	F-test	Signif.	Test DF
Envir (Nitrogen)	36610.13	327.46	***	(1,17)
Blks (Env)	13.25	0.63	NS	
Genotypes	171.89	1.74	NS	(14,14)
Genot-Nitrogen	98.62	4.71	***	
ERROR	20.93			

Standard deviation = 4.58                      Coefficient of variation = 0.05  
S.E. Genotype means = 3.51

APPENDIX 7 (contd.)Tillers per Plant

Source	Mean Square	F-test	Signif.	Test DF
Envir (Nitrogen)	7061.00	127.20	***	(1,18)
Blks (Env)	26.07	1.54	NS	
Genotypes	133.30	4.51	**	(14,14)
Genot-Nitrogen	29.57	1.74	(NS)	
ERROR	16.96			
Standard deviation = 4.12		Coefficient of variation = 0.19		
S.E. Genotype means = 1.92				

Spikelet Number per Ear

Source	Mean Square	F-test	Signif.	Test DF
Envir (Nitrogen)	612.64	68.92	***	(1,20)
Blks (Env)	3.28	1.74	NS	
Genotypes	69.48	12.33	***	(14,14)
Genot-Nitrogen	5.63	2.99	**	
ERROR	1.88			
Standard deviation = 1.37		Coefficient of variation = 0.06		
S.E. Genotype means = 0.84				

Grain Number per Ear (transformed to  $10 \log_{10} X$ )

Source	Mean Square	F-test	Signif.	Test DF
Envir (Nitrogen)	50.31	49.39	***	(1,18)
Blks (Env)	0.46	0.98	NS	
Genotypes	14.56	25.61	***	(14,14)
Genot-Nitrogen	0.57	1.21	NS	
ERROR	0.47			
Standard deviation = 0.68		Coefficient of variation = 0.06		
S.E. Genotype means = 0.27				

## APPENDIX 7 (contd.)

Grain Yield

Source	Mean Square	F-test	Signif.	Test DF
Envir (Nitrogen)	354.3891	6.78	*	(1,13)
Blks (Env)	33.4844	6.95	***	
Genotypes	21.3844	1.10	NS	(14,14)
Genot-Nitrogen	19.4714	4.04	***	
ERROR	4.8164			
Standard deviation = 2.1946		Coefficient of variation = 0.21		
S.E. Genotype means = 1.5601				

100 Kernel Weight

Source	Mean Square	F-test	Signif.	Test DF
Envir (Nitrogen)	54.6615	46.44	***	(1,10)
Blks (Env)	0.8992	7.68	***	
Genotypes	1.5956	5.69	**	(14,14)
Genot-Nitrogen	0.2804	2.40	**	
ERROR	0.1170			
Standard deviation = 0.3421		Coefficient of variation = 0.07		
S.E. Genotype means = 0.1872				

Physiological Brown Spot (transformed to  $\sin^{-1}\sqrt{X/100}$ )

Source	Mean Square	F-test	Signif.	Test DF
Envir (Nitrogen)	1374.86	5.64	*	(1,19)
Blks (Env)	49.30	2.41	*	
Genotypes	207.39	1.05	NS	(14,14)
Genot-Nitrogen	197.93	9.66	***	
ERROR	20.49			
Standard deviation = 4.53		Coefficient of variation = 0.22		
S.E. Genotype means = 4.97				

APPENDIX 8.Genotype means and estimates of heterosis and heterobeltiosis  
for other characters - single and pooled nitrogen levelsLow Nitrogen.APPENDIX 8.1.Genotype means (upper triangle) and estimates of heterosis and  
heterobeltiosis (bottom triangle) for heading date (days)

Male/Female	1	2	3	4	5
1	60.3 bc#	58.4 c	58.9 bc	57.9 c	38.6 f
2	-0.9 (-1.9) <sup>†</sup>	58.3 c	58.6 c	58.9 bc	41.6 e
3	-2.1 (-2.7)	-1.4 (-3.0)	61.6 ab	59.1 bc	38.1 f
4	-1.9 (-2.4)	0.1 (-0.4)	-1.4 (-2.5)	59.3 bc	48.5 d
5	-23.6 (-25.4)	-19.6 (-22.4)	-24.7 (-25.9)	-13.2 (-15.5)	64.0 a

$\bar{F}_1 = 51.9$        $\bar{P} = 60.7$       Grand mean = 54.8      L.S.D. (5%) = 2.6  
L.S.D. (1%) = 3.5

Heterosis: S.E. = 1.2      Heterobeltiosis: S.E. = 1.3

<sup>†</sup> Estimates of heterosis and heterobeltiosis (latter in brackets) expressed as deviations.

# Figures underscored by different letters differ at the 5% level of significance.

## APPENDIX 8.2.

Genotype means (upper triangle) and estimates of heterosis and heterobeltiosis (bottom triangle) for plant height (cm).

Male/Female	1	2	3	4	5
1	65.3 cd#	65.3 cd	65.1 cd	67.0 c	80.3 a
2	1.9 (0.0) <sup>†</sup>	61.5 d	61.4 d	64.1 cd	81.4 a
3	-0.2 (-0.2)	-2.0 (-3.9)	65.3 cd	63.1 cd	78.5 a
4	3.4 (1.7)	2.4 (2.2)	-0.5 (-2.2)	61.9 d	73.4 b
5	7.3 (-0.5)	10.3 (0.6)	5.5 (-2.3)	2.1 (-7.4)	80.8 a

$\bar{F}_1 = 70.0$      $\bar{P} = 66.9$     Grand mean = 68.9    L.S.D. (5%) = 3.9  
L.S.D. (1%) = 5.2

Heterosis: S.E. = 1.7    Heterobeltiosis: S.E. = 1.9

<sup>†</sup> Estimates of heterosis and heterobeltiosis (latter in brackets) expressed as deviations.

# Figures underscored by different letters differ at the 5% level of significance.

## APPENDIX 8.3.

Genotype means (upper triangle) and estimates of heterosis and heterobeltiosis (bottom triangle) for tillers per plant.

Male/Female	1	2	3	4	5
1	13.8 bc#	17.0 a	17.3 a	15.6 ab	9.6 e
2	1.4 (-0.5) <sup>†</sup>	17.5 a	16.3 a	14.0 bc	10.6 de
3	3.6 (3.5)	0.8 (-1.2)	13.6 bc	17.5 a	10.9 de
4	0.5 (-0.9)	-3.0 (-3.5)	2.5 (1.0)	16.5 a	12.1 cd
5	-0.4 (-4.2)	-1.2 (-6.9)	1.1 (-2.7)	0.8 (-4.4)	6.1 f

$\bar{F}_1 = 14.1$      $\bar{P} = 13.5$     Grand mean = 13.9    L.S.D. (5%) = 2.2  
L.S.D. (1%) = 2.9

Heterosis: S.E. = 0.8    Heterobeltiosis: S.E. = 1.1

<sup>†</sup> Estimates of heterosis and heterobeltiosis (latter in brackets) expressed as deviations.

# Figures underscored by different letters differ at the 5% level of significance.

## APPENDIX 8.4.

Genotype means (upper triangle) and estimates of heterosis and heterobeltiosis (bottom triangle) for spikelet number per ear

Male/Female	1	2	3	4	5
1	22.7 bcde#	24.5 a	23.5 abc	23.3 abcd	20.8 f
2	1.8 (1.8)†	22.7 bcde	23.7 ab	24.1 a	21.6 ef
3	0.2 (-0.5)	0.4 (-0.3)	24.0 ab	22.0 def	21.6 ef
4	0.9 (0.6)	1.7 (1.4)	-1.1 (-2.0)	22.2 cde	19.2 g
5	2.6 (-1.9)	3.4 (-1.1)	2.7 (-2.4)	1.2 (-3.0)	13.8 h

$\bar{F}_1 = 22.4$      $\bar{P} = 21.1$     Grand mean = 22.0    L.S.D. (5%) = 1.3  
L.S.D. (1%) = 1.7

Heterosis: S.E. = 0.5    Heterobeltiosis: S.E. = 0.6

† Estimates of heterosis and heterobeltiosis (latter in brackets) expressed as deviations.

# Figures underscored by different letters differ at the 5% level of significance.

## APPENDIX 8.5.

Genotype means (upper triangle) and estimates of heterosis and heterobeltiosis (bottom triangle) for grain number per ear (transformed to  $10 \log_{10}X$ )

Male/Female	1	2	3	4	5
1	10.3 cd#	10.6 cd	10.3 cd	10.9 c	12.8 b
2	0.6 (0.3) <sup>†</sup>	9.7 d	11.0 c	10.8 c	12.7 b
3	-0.2 (-0.4)	0.8 (0.3)	10.7 cd	10.8 c	12.9 b
4	0.6 (0.6)	0.8 (0.5)	0.3 (0.1)	10.3 cd	12.6 b
5	0.0 (-2.5)	0.2 (-2.6)	-0.1 (-2.4)	-0.2 (-2.7)	15.3 a

$\bar{F}_1 = 11.5$      $\bar{P} = 11.3$     Grand mean = 11.4    L.S.D. (5%) = 1.0  
L.S.D. (1%) = 1.3

Heterosis: S.E. = 0.4    Heterobeltiosis: S.E. = 0.5

<sup>†</sup> Estimates of heterosis and heterobeltiosis (latter in brackets) expressed as deviations.

# Figures underscored by different letters differ at the 5% level of significance.

## APPENDIX 8.6.

Genotype means (upper triangle) and estimates of heterosis and heterobeltiosis (bottom triangle) for grain yield (g).

Male/Female	1	2	3	4	5
1	7.59 e#	9.79 b	9.20 bc	7.63 de	8.25 bcde
2	1.78 (1.35) <sup>†</sup>	8.44 bcde	9.35 bc	9.21 bc	8.44 bcde
3	1.41 (1.21)	1.14 (0.91)	7.99 cde	9.31 bc	8.34 bcde
4	0.76 (-1.55)	0.40 (0.03)	0.73 (0.13)	9.18 bcd	9.49 bc
5	-1.49 (-3.63)	-1.72 (-3.44)	-1.60 (-3.54)	-1.04 (-2.39)	11.88 a

$\bar{F}_1 = 8.90$      $\bar{P} = 9.01$     Grand mean = 8.94    L.S.D. (5%) = 1.38

L.S.D. (1%) = 1.84

Heterosis: S.E. = 0.59

Heterobeltiosis: S.E. = 0.68

<sup>†</sup> Estimates of heterosis and heterobeltiosis (latter in brackets) expressed as deviations.

# Figures underscored by different letters differ at the 5% level of significance.

APPENDIX 8.7.

Genotype means (upper triangle) and estimates of heterosis and heterobeltiosis (bottom triangle) for 100 kernel weight (g)

Male/Female	1	2	3	4	5
1	5.62 def#	5.64 def	5.92 cd	5.98 cd	6.94 a
2	0.17 (0.02) <sup>†</sup>	5.32 fg	5.51 efg	5.93 cd	6.52 b
3	0.49 (0.30)	0.23 (0.19)	5.25 g	5.74 de	6.17 c
4	0.30 (0.23)	0.40 (0.18)	0.24 (-0.01)	5.75 de	6.66 ab
5	1.05 (0.78)	0.78 (0.36)	0.47 (0.01)	0.71 (0.50)	6.16 c

$\bar{F}_1 = 6.10$      $\bar{P} = 5.62$     Grand mean = 5.94    L.S.D. (5%) = 0.33

L.S.D. (1%) = 0.44

Heterosis: S.E. = 0.13

Heterobeltiosis: S.E. = 0.16

<sup>†</sup> Estimates of heterosis and heterobeltiosis (latter in brackets) expressed as deviations.

# Figures underscored by different letters differ at the 5% level of significance.

## APPENDIX 8.8.

Genotype means (upper triangle) and estimates of heterosis and heterobeltiosis (bottom triangle) for physiological brown spot.  
(transformed to  $\sin^{-1}\sqrt{X/100}$ )

Male/Female	1	2	3	4	5
1	13.1 de#	16.4 bcd	16.5 bcd	14.9 cde	20.8 bc
2	3.3 (3.3) <sup>†</sup>	13.1 de	17.5 bcd	13.1 de	22.3 b
3	-1.3 (-6.0)	-0.3 (-5.0)	22.5 b	16.9 bcd	28.0 a
4	1.5 (1.1)	0.4 (-0.7)	-1.3 (-5.6)	13.8 de	21.4 b
5	9.3 (7.7)	10.8 (9.2)	6.9 (5.5)	9.6 (7.6)	9.9 e

$\bar{F}_1 = 18.8$      $\bar{P} = 14.5$     Grand mean = 17.3    L.S.D. (5%) = 5.3  
L.S.D. (1%) = 7.1

Heterosis: S.E. = 2.4    Heterobeltiosis: S.E. = 2.6

<sup>†</sup> Estimates of heterosis and heterobeltiosis (latter in brackets) expressed as deviations.

# Figures underscored by different letters differ at the 5% level of significance.

High NitrogenAPPENDIX 3.9.

Genotype means (upper triangle) and estimates of heterosis and heterobeltiosis (bottom triangle) for heading date (days)

Male/Female	1	2	3	4	5
1	62.3 a#	60.5 abcd	58.9 d	59.9 bcd	40.0 f
2	-0.5 (-1.8)†	59.6 cd	59.8 bcd	60.0 bcd	40.3 f
3	-3.0 (-3.4)	-0.7 (-1.6)	61.4 abc	61.3 abc	39.9 f
4	-1.8 (-2.4)	-0.4 (-1.1)	0.1 (-0.1)	61.1 abc	48.5 e
5	-21.9 (-22.3)	-20.3 (-21.2)	-21.6 (-21.6)	-12.8 (-13.0)	61.5 ab

$\bar{F}_1 = 52.9$      $\bar{P} = 61.2$     Grand mean = 55.7    L.S.D. (5%) = 1.6  
L.S.D. (1%) = 2.2

Heterosis: S.E. = 0.7    Heterobeltiosis: S.E. = 0.8

† Estimates of heterosis and heterobeltiosis (latter in brackets) expressed as deviations.

# Figures underscored by different letters differ at the 5% level of significance.

APPENDIX 8.10.Genotype means for plant height (cm).

Male/Female	1	2	3	4	5
1	105.0	100.8	105.1	103.1	103.5
2		100.5	100.4	104.3	102.6
3	Genotypes not		105.0	105.1	101.5
4	significantly different			101.0	109.3
	at the 5% level of				
	significance.				
5					111.0

$\bar{F}_1 = 103.6$      $\bar{P} = 104.5$     Grand mean = 103.9

## APPENDIX 8.11.

Genotype means (upper triangle) and estimates of heterosis and heterobeltiosis (bottom triangle) for tillers per plant.

Male/Female	1	2	3	4	5
1	30.0 abcde#	36.1 ab	32.0 abcd	36.9 a	24.0 def
2	5.3 (4.5) <sup>†</sup>	31.6 abcd	31.1 abcd	31.8 abcd	20.0 f
3	-0.7 (-3.3)	-2.4 (-4.2)	35.3 abc	26.5 cdef	27.4 bcdef
4	5.9 (4.8)	-0.1 (-0.3)	-7.2 (-8.8)	32.1 abcd	21.9 ef
5	-2.0 (-6.0)	-6.8 (-11.6)	-1.2 (-7.9)	-5.1 (-10.2)	21.9 ef

$\bar{F}_1 = 28.8$      $\bar{P} = 30.2$     Grand mean = 29.2    L.S.D. (5%) = 8.0  
L.S.D. (1%) = 10.7

Heterosis: S.E. = 3.5    Heterobeltiosis: S.E. = 4.0

<sup>†</sup> Estimates of heterosis and heterobeltiosis (latter in brackets) expressed as deviations.

# Figures underscored by different letters differ at the 5% level of significance.

## APPENDIX 8.12.

Genotype means (upper triangle) and estimates of heterosis and heterobeltiosis (bottom triangle) for spikelet number per ear.

Male/Female	1	2	3	4	5
1	29.8 a#	28.9 abc	28.5 abc	28.9 abc	23.8 e
2	-0.3 (-0.9) <sup>†</sup>	28.5 abc	27.0 bc	29.2 abc	23.0 e
3	-0.6 (-1.3)	-1.5 (-1.5)	28.4 abc	26.5 cd	23.5 e
4	-0.7 (-0.9)	0.3 (-0.2)	-2.4 (-2.9)	29.4 ab	24.4 de
5	0.2 (-6.0)	0.0 (-5.5)	0.6 (-4.9)	1.0 (-5.0)	17.5 f

$\bar{F}_1 = 26.4$      $\bar{P} = 26.7$     Grand mean = 26.5    L.S.D. (5%) = 2.5  
L.S.D. (1%) = 3.3

Heterosis: S.E. = 1.0    Heterobeltiosis: S.E. = 1.2

<sup>†</sup> Estimates of heterosis and heterobeltiosis (latter in brackets) expressed as deviations.

# Figures underscored by different letters differ at the 5% level of significance.

## APPENDIX 8.13.

Genotype means (upper triangle) and estimates of heterosis and heterobeltiosis (bottom triangle) for grain number per ear.

(transformed to  $10 \log_{10} X$ )

Male/Female	1	2	3	4	5
1	12.5 cdef#	11.8 efg	11.5 fg	12.4 def	13.6 bc
2	-0.5 (-0.7) <sup>†</sup>	12.0 efg	11.9 efg	12.7 bcde	13.3 bcd
3	-0.9 (-1.0)	-0.3 (-0.4)	12.3 defg	12.2 defg	13.5 bc
4	0.6 (-0.1)	1.1 (0.7)	0.5 (-0.1)	11.2 g	13.6 b
5	-0.8 (-2.7)	-0.9 (-3.0)	-0.8 (-2.8)	-0.2 (-2.7)	16.3 a

$\bar{F}_1 = 12.7$      $\bar{P} = 12.9$     Grand mean = 12.7    L.S.D. (5%) = 1.0  
L.S.D. (1%) = 1.3

Heterosis: S.E. = 0.5

Heterobeltiosis: S.E. = 0.5

<sup>†</sup> Estimates of heterosis and heterobeltiosis (latter in brackets) expressed as deviations.

# Figures underscored by different letters differ at the 5% level of significance.

## APPENDIX 8.14.

Genotype means (upper triangle) and estimates of heterosis and heterobeltiosis (bottom triangle) for grain yield (g).

Male/Female	1	2	3	4	5
1	13.93 bcd#	10.48 cde	9.35 de	16.43 ab	13.51 bcd
2	-2.08 (-3.45)†	11.18 cde	10.28 cde	14.06 bcd	10.93 cde
3	-4.82 (-5.06)	-2.52 (-4.13)	14.41 bc	9.66 cde	14.07 bcd
4	5.90 (2.50)	4.90 (2.88)	-1.12 (-4.75)	7.14 e	11.45 cde
5	-2.84 (-5.25)	-4.04 (-7.83)	-2.52 (-4.69)	-1.50 (7.31)	18.76 a

$\bar{P}_1 = 12.02$      $\bar{P} = 13.08$     Grand mean = 12.37    L.S.D. (5%) = 4.21  
L.S.D. (1%) = 5.63

Heterosis: S.E. = 1.86

Heterobeltiosis: S.E. = 2.09

† Estimates of heterosis and heterobeltiosis (latter in brackets) expressed as deviations.

# Figures underscored by different letters differ at the 5% level of significance.

APPENDIX 8.15.

Genotype means (upper triangle) and estimates of heterosis and heterobeltiosis (bottom triangle) for 100 kernel weight (g).

Male/Female	1	2	3	4	5
1	4.54 bcd#	4.49 bcd	4.82 bcd	4.96 bc	5.74 a
2	-0.01 (-0.05) <sup>†</sup>	4.45 bcde	4.27 cde	4.29 cde	5.13 b
3	0.45 (0.28)	-0.06 (-0.18)	4.21 de	4.13 de	4.78 bcd
4	0.55 (0.42)	-0.08 (-0.16)	-0.12 (-0.16)	4.29 cde	4.96 bc
5	1.57 (1.20)	1.01 (0.68)	0.78 (0.57)	0.92 (0.67)	3.80 e

$\bar{F}_1 = 4.76$      $\bar{P} = 4.26$     Grand mean = 4.59    L.S.D. (5%) = 0.61  
L.S.D. (1%) = 0.81

Heterosis: S.E. = 0.26    Heterobeltiosis: S.E. = 0.30

<sup>†</sup> Estimates of heterosis and heterobeltiosis (latter in brackets) expressed as deviations.

# Figures underscored by different letters differ at the 5% level of significance.

## APPENDIX 8.16.

Genotype means (upper triangle) and estimates of heterosis and heterobeltiosis (bottom triangle) for physiological brown spot (transformed to  $\sin^{-1}\sqrt{X/100}$ )

Male/Female	1	2	3	4	5
1	17.8 cd#	30.3 b	18.3 cd	16.4 cd	18.7 cd
2	0.9 (-10.8)†	41.1 a	28.7 b	28.0 b	18.0 cd
3	-10.9 (-22.2)	-12.1 (-12.4)	40.5 a	31.4 b	24.3 bc
4	-0.3 (-1.4)	-0.4 (-13.1)	3.4 (-9.1)	15.6 d	13.3 d
5	0.2 (-0.5)	-12.2 (-23.1)	-5.6 (-16.2)	-4.1 (-5.9)	19.2 cd

$\bar{F}_1 = 22.8$      $\bar{P} = 26.8$     Grand mean = 24.1    L.S.D. (5%) = 7.4  
L.S.D. (1%) = 9.9

Heterosis: S.E. = 3.3

Heterobeltiosis: S.E. = 3.7

† Estimates of heterosis and heterobeltiosis (latter in brackets) expressed as deviations.

# Figures underscored by different letters differ at the 5% level of significance.

Pooled Nitrogen LevelsAPPENDIX 8.17.

Genotype means (upper triangle) and estimates of heterosis and heterobeltiosis (bottom triangle) for heading date (days)

Male/Female	1	2	3	4	5
1	62.3 abc#	60.5 bcd	58.9 d	59.9 d	40.0 f
2	-0.5 (-1.8)†	59.6 d	59.8 cd	60.0 bcd	40.3 f
3	-3.0 (-3.4)	-0.7 (-1.6)	61.4 ab	61.3 bcd	39.9 f
4	-1.8 (-2.4)	-0.4 (-1.1)	0.1 (-0.1)	61.1 bcd	48.5 e
5	-21.9 (-22.3)	-20.3 (-21.2)	-21.6 (-21.6)	-12.8 (-13.0)	61.5 a

$\bar{F}_1 = 52.4$      $\bar{P} = 60.9$     Grand mean = 54.8    L.S.D. (5%) = 2.0  
L.S.D. (1%) = 2.6

Heterosis: S.E. = 0.7

Heterobeltiosis: S.E. = 0.8

† Estimates of heterosis and heterobeltiosis (latter in brackets) expressed as deviations.

# Figures underscored by different letters differ at the 5% level of significance.

APPENDIX 8.18.Genotype means for plant height (cm).

Male/Female	1	2	3	4	5
1	85.1	83.0	85.1	85.1	91.9
2		81.0	80.9	84.2	92.0
3	Genotypes not		85.1	84.1	90.0
4	significantly different			81.4	91.3
5	at the 5% level of				95.9
	significance.				

$$\bar{F}_1 = 86.8$$

$$\bar{P} = 85.7$$

$$\text{Grand mean} = 86.4$$

## APPENDIX 8.19.

Genotype means (upper triangle) and estimates of heterosis and heterobeltilosis (bottom triangle) for tillers per plant.

Male/Female	1	2	3	4	5
1	21.9 abcd#	26.6 a	24.6 ab	26.3 a	16.8 de
2	3.4 (2.0) <sup>†</sup>	24.6 ab	23.7 ab	22.9 abc	15.3 e
3	1.5 (0.2)	-0.8 (-0.9)	24.4 ab	22.0 abcd	19.1 bcde
4	3.2 (2.0)	-1.6 (-1.7)	-2.4 (-2.4)	24.3 ab	17.0 cde
5	-1.2 (-5.1)	-4.0 (-9.3)	-0.1 (-5.3)	-2.2 (-7.3)	14.0 e

$\bar{F}_1 = 21.4$      $\bar{P} = 21.8$     Grand mean = 21.6    L.S.D. (5%) = 5.4  
L.S.D. (1%) = 7.2

Heterosis: S.E. = 1.8    Heterobeltilosis: S.E. = 2.0

<sup>†</sup> Estimates of heterosis and heterobeltilosis (latter in brackets) expressed as deviations.

# Figures underscored by different letters differ at the 5% level of significance.

## APPENDIX 8.20.

Genotype means (upper triangle) and estimates of heterosis and heterobeltilosis (bottom triangle) for spikelet number per ear

Male/Female	1	2	3	4	5
1	26.2 a #	26.7 a	26.0 a	26.1 a	22.3 b
2	0.8 (0.5) <sup>†</sup>	25.6 a	25.3 a	26.6 a	22.3 b
3	-0.2 (-0.2)	-0.6 (-0.9)	26.2 a	24.2 ab	22.5 b
4	0.1 (-0.1)	0.9 (0.8)	-1.8 (-2.0)	25.8 a	21.8 b
5	1.4 (-3.9)	1.7 (-3.3)	1.6 (-3.7)	1.1 (-4.0)	15.6 c

$\bar{F}_1 = 24.4$      $\bar{P} = 23.9$     Grand mean = 24.2    L.S.D. (5%) = 2.4  
L.S.D. (1%) = 3.1

Heterosis: S.E. = 0.6

Heterobeltilosis: S.E. = 0.7

<sup>†</sup> Estimates of heterosis and heterobeltilosis (latter in brackets) expressed as deviations.

# Figures underscored by different letters differ at the 5% level of significance.

## APPENDIX 8.21.

Genotype means (upper triangle) and estimates of heterosis and heterobeltiosis (bottom triangle) for grain number per ear (transformed to  $10 \log_{10} X$ )

Male/Female	1	2	3	4	5
1	11.4 cd #	11.2 cd	10.9 d	11.6 cd	13.2 b
2	0.1 (-0.2) <sup>†</sup>	10.8 d	11.5 cd	11.8 c	13.0 b
3	-0.6 (-0.6)	0.4 (0.0)	11.5 cd	11.5 cd	13.2 b
4	0.5 (0.2)	1.0 (1.0)	0.4 (0.0)	10.8 d	13.1 b
5	-0.4 (-2.6)	-0.3 (-2.8)	-0.5 (-2.6)	-0.2 (-2.7)	15.8 a

$\bar{F}_1 = 12.1$      $\bar{P} = 12.1$     Grand mean = 12.1    L.S.D. (5%) = 0.7  
L.S.D. (1%) = 1.0

Heterosis: S.E. = 0.3

Heterobeltiosis: S.E. = 0.3

<sup>†</sup> Estimates of heterosis and heterobeltiosis (latter in brackets) expressed as deviations.

# Figures underscored by different letters differ at the 5% level of significance.

APPENDIX 8.22.Genotype means for grain yield (g).

Male/Female	1	2	3	4	5
1	10.76	10.13	9.28	12.03	10.88
2		9.81	9.81	11.63	9.68
3	Genotypes not		11.20	9.49	11.20
4	significantly different			8.16	10.47
5	at the 5% level of				15.32
	significance.				
$\bar{F}_1 = 10.46$	$\bar{P} = 11.05$	Grand mean = 10.66			

## APPENDIX 8.23.

Genotype means (upper triangle) and estimates of heterosis and heterobeltiosis (bottom triangle) for 100 kernel weight (g)

Male/Female	1	2	3	4	5
1	5.08 cd #	5.06 cd	5.37 bc	5.47 bc	6.34 a
2	0.08 (-0.02)†	4.89 cd	4.89 cd	5.11 cd	5.83 ab
3	0.47 (0.29)	0.08 (0.00)	4.73 d	4.93 cd	5.48 bc
4	0.42 (0.39)	0.52 (0.09)	0.06 (-0.09)	5.02 cd	5.81 b
5	1.31 (1.26)	0.90 (0.85)	0.63 (0.50)	0.81 (0.79)	4.98 cd

$\bar{F}_1 = 5.43$      $\bar{P} = 4.94$     Grand mean = 5.26    L.S.D. (5%) = 0.53  
L.S.D. (1%) = 0.70

Heterosis: S.E. = 0.15

Heterobeltiosis: S.E. = 0.17

† Estimates of heterosis and heterobeltiosis (latter in brackets) expressed as deviations.

# Figures underscored by different letters differ at the 5% level of significance.

APPENDIX 8.24.Genotype means for physiological brown spot(transformed to  $\sin^{-1}\sqrt{X/100}$ ).

Male/Female	1	2	3	4	5
1	15.4	23.4	17.4	15.7	19.8
2		27.1	23.1	20.5	20.1
3	Genotypes not		31.5	24.1	26.2
4	significantly different			14.7	17.4
5	at the 5% level of				14.5
	significance.				

 $\bar{F}_1 = 20.8$  $\bar{P} = 20.7$ 

Grand mean = 20.7

## APPENDIX 9.

A. Variances ( $V_r$ ) and covariances ( $W_r$ ) and their sums and differences for characters in low and high nitrogen levels.Low NitrogenHeading Date

ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	$V_r$	$W_r$	$V_r$	$W_r$	$V_r$	$W_r$	$V_r$	$W_r$
1	76.18	-23.95	60.93	-31.40	88.18	7.70	112.18	-13.43
2	68.83	-22.66	35.83	-24.33	58.05	6.08	71.13	-10.56
3	97.18	-24.93	71.30	-32.09	95.58	7.98	116.38	-15.06
4	15.08	-8.71	16.05	-15.54	33.95	4.35	28.45	-5.90
5	138.88	24.50	180.30	38.65	71.08	-9.23	136.18	17.74

ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	$W_r+V_r$	$W_r-V_r$	$W_r+V_r$	$W_r-V_r$	$W_r+V_r$	$W_r-V_r$	$W_r+V_r$	$W_r-V_r$
1	52.23	-100.13	29.53	-92.33	95.88	-80.48	98.75	-125.60
2	46.16	-91.49	11.50	-60.15	64.13	-51.98	60.56	-81.64
3	72.25	-122.10	39.21	-103.39	103.55	-87.60	101.31	-131.44
4	6.36	-23.79	0.51	-31.59	38.30	-29.60	22.55	-34.35
5	163.38	-114.38	168.95	-91.65	61.85	-80.30	153.91	-118.44

Plant Height

## 1) Original data:

ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	$V_r$	$W_r$	$V_r$	$W_r$	$V_r$	$W_r$	$V_r$	$W_r$
1	28.93	30.94	29.45	41.19	64.00	56.13	82.13	72.75
2	81.70	66.13	68.58	65.25	46.68	50.16	99.05	78.24
3	23.13	35.75	33.55	45.19	70.08	63.01	78.00	72.00
4	46.08	50.19	27.88	41.50	21.83	20.24	17.05	26.20
5	12.43	6.63	5.80	7.88	23.13	17.50	24.43	4.70

ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	$W_r+V_r$	$W_r-V_r$	$W_r+V_r$	$W_r-V_r$	$W_r+V_r$	$W_r-V_r$	$W_r+V_r$	$W_r-V_r$
1	59.86	2.01	70.64	11.74	120.13	-7.88	154.88	-9.38
2	147.83	-15.58	133.83	-3.33	96.84	3.49	177.29	-20.81
3	58.88	12.66	78.74	11.64	133.09	-7.06	150.00	-6.00
4	96.26	4.11	69.38	13.63	42.06	-1.59	43.25	9.15
5	19.05	-5.80	13.68	2.08	40.63	-5.63	29.13	-19.73

## APPENDIX 9 (contd.)

2) Reanalysed data (array 3 deleted):

ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	V <sub>r</sub>	W <sub>r</sub>						
1	33.17	40.50	35.00	48.92	84.92	74.92	94.50	92.25
2	89.67	86.75	85.42	79.88	54.17	67.25	115.00	99.25
4	60.08	66.54	33.42	49.71	21.75	27.33	20.90	33.27
5	10.23	8.02	6.23	6.94	27.08	23.08	31.75	7.38

ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>
1	73.67	7.33	83.92	13.92	159.83	-10.00	186.75	-2.25
2	176.42	-2.92	165.29	-5.54	121.42	13.08	214.25	-15.75
4	126.63	6.46	83.13	16.29	49.08	5.58	54.17	12.38
5	18.25	-2.21	13.17	0.71	50.17	-4.00	39.13	-24.38

Tillers per Plant1) Original data:

ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	V <sub>r</sub>	W <sub>r</sub>						
1	14.08	9.55	10.68	11.18	7.05	9.06	16.83	18.54
2	21.88	17.50	7.45	9.71	4.93	6.06	14.58	10.71
3	16.55	17.48	13.30	10.76	6.30	8.63	5.93	10.51
4	8.70	10.30	6.08	3.03	4.83	4.75	7.08	6.16
5	2.38	6.25	10.43	10.89	6.18	9.81	4.08	8.34

ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>
1	23.63	-4.53	21.85	0.50	16.11	2.01	35.36	1.71
2	39.38	-4.38	17.16	2.26	10.99	1.14	25.29	-3.86
3	34.03	0.93	24.06	-2.54	14.93	2.33	16.44	4.59
4	19.00	1.60	9.10	-3.05	9.58	-0.08	13.24	-0.91
5	8.63	3.88	21.31	0.46	15.99	3.64	12.41	4.26

## APPENDIX 9 (contd.)

2) Reanalysed data (array 3 deleted):

ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	V <sub>r</sub>	W <sub>r</sub>						
1	7.50	15.33	10.23	13.48	7.00	11.58	22.40	24.73
2	22.50	25.33	9.67	12.58	5.06	7.69	19.40	14.27
4	11.00	14.33	0.40	2.06	5.23	5.98	7.42	8.13
5	2.75	8.83	13.56	14.10	7.73	12.85	4.73	11.06

ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>
1	22.83	7.83	23.71	3.25	18.58	4.58	47.13	2.33
2	47.83	2.83	22.25	2.92	12.75	2.63	33.67	-5.13
4	25.33	3.33	2.46	1.67	11.21	0.75	15.54	0.71
5	11.58	6.08	27.67	0.54	20.58	5.13	15.79	6.33

Spikelet Number per Ear

ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	V <sub>r</sub>	W <sub>r</sub>						
1	1.58	3.42	1.45	4.31	3.28	7.46	3.28	3.43
2	0.79	3.72	1.47	4.27	1.47	4.06	4.07	3.91
3	3.17	7.01	1.10	3.71	1.47	3.69	0.44	0.95
4	4.81	7.07	0.64	0.97	8.64	10.91	0.81	2.61
5	13.35	15.77	12.01	13.95	10.32	11.70	12.13	13.30

ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>
1	5.00	1.83	5.76	2.87	10.74	4.18	6.71	0.15
2	4.51	2.92	5.74	2.81	5.53	2.59	7.98	-0.16
3	10.18	3.84	4.81	2.61	5.15	2.22	1.39	0.51
4	11.88	2.26	1.61	0.32	19.55	2.26	3.42	1.79
5	29.12	2.42	25.96	1.94	22.02	1.39	25.43	1.16

## APPENDIX 9 (contd.)

Grain Number per Ear (transformed to  $10 \log_{10} X$ )

ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	V <sub>r</sub>	W <sub>r</sub>						
1	1.49	2.22	1.12	2.58	1.13	2.28	1.23	2.59
2	0.74	1.09	2.59	4.12	1.24	2.18	1.56	2.92
3	1.26	2.21	1.87	2.54	1.25	2.23	1.98	2.90
4	0.92	1.45	0.86	1.25	1.37	2.58	1.02	1.80
5	1.64	2.92	1.37	2.61	1.63	2.82	1.19	2.61

ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>
1	3.71	0.74	3.69	1.46	3.41	1.16	3.82	1.36
2	1.83	0.34	6.71	1.53	3.42	0.93	4.48	1.36
3	3.48	0.95	4.41	0.66	3.48	0.99	4.87	0.92
4	2.37	0.53	2.10	0.39	3.95	1.22	2.81	0.78
5	4.56	1.28	3.98	1.24	4.45	1.19	3.80	1.42

Grain Yield

ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	V <sub>r</sub>	W <sub>r</sub>						
1	2.1087	0.4375	1.5770	0.8880	0.9055	-0.1349	1.5933	-1.0846
2	0.1217	0.4107	0.4120	-0.5220	1.3943	0.4824	2.3030	-2.9130
3	2.0743	0.4082	0.4180	-0.2760	0.8570	-0.8422	1.3742	0.6220
4	0.7663	1.0587	2.0643	1.1752	0.7532	1.0654	1.3043	-0.3361
5	2.7580	4.0605	0.6713	0.3531	1.9633	2.1766	6.2392	4.8236

ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>
1	2.5462	-1.6713	2.4650	-0.6890	0.7706	-1.0404	0.5086	-2.6779
2	0.5325	0.2890	-0.1100	-0.9340	1.8766	-0.9119	-0.6100	-5.2160
3	2.4825	-1.6660	0.1420	-0.6940	0.0148	-1.6993	1.9963	-0.7522
4	1.8250	0.2925	3.2395	-0.8890	1.8186	0.3121	0.9681	-1.6404
5	6.8185	1.3025	1.0244	-0.3181	4.1399	0.2134	11.0629	-1.4156

## APPENDIX 9 (contd.)

## 100 Kernel Weight

ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	V <sub>r</sub>	W <sub>r</sub>						
1	0.1991	0.0629	0.4849	0.2407	0.3248	0.1079	0.3625	0.2600
2	0.3174	0.1672	0.1874	0.2186	0.1918	0.0881	0.3582	0.2311
3	0.1005	0.0555	0.2861	0.2164	0.1927	0.0953	0.0960	0.1363
4	0.1649	0.0565	0.2511	0.2481	0.1398	0.0663	0.1158	0.1308
5	0.2243	-0.0058	0.1101	0.0115	0.1048	-0.0364	0.1124	0.0381

ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>
1	0.2621	-0.1362	0.7256	-0.2442	0.4328	-0.2169	0.6225	-0.1024
2	0.4846	-0.1503	0.4061	0.0312	0.2799	-0.1037	0.5893	-0.1270
3	0.1560	-0.0450	0.5024	-0.0697	0.2880	-0.0973	0.2323	0.0402
4	0.2213	-0.1084	0.4992	-0.0030	0.2060	-0.0735	0.2465	0.0150
5	0.2185	-0.2301	0.1216	-0.0987	0.0684	-0.1413	0.1405	-0.0844

Physiological Brown Spot (transformed to  $\sin^{-1}\sqrt{X/100}$ )

## 1) Original data:

ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	V <sub>r</sub>	W <sub>r</sub>						
1	29.06	-33.27	9.49	8.00	0.38	0.67	25.09	-7.02
2	19.01	25.21	6.84	-1.14	9.44	-0.08	51.30	-10.38
3	69.74	42.19	6.05	-6.42	47.64	8.83	34.53	-7.15
4	40.74	-15.54	1.58	-1.32	3.03	2.20	20.64	-5.15
5	86.28	59.41	51.98	22.85	46.79	26.96	50.83	8.72

ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>
1	-4.22	-62.33	17.49	-1.49	1.05	0.29	18.07	-32.12
2	44.22	6.21	5.70	-7.99	9.36	-9.52	40.93	-61.68
3	111.93	-27.55	-0.36	-12.47	56.47	-38.81	27.38	-41.67
4	25.20	-56.28	0.26	-2.90	5.23	-0.83	15.49	-25.79
5	145.69	-26.87	54.83	-9.13	73.75	-19.83	59.55	-42.10

## APPENDIX 9 (contd.)

2) Reanalysed data (array 5 deleted):

ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	V <sub>r</sub>	W <sub>r</sub>						
1	6.94	-14.40	12.55	11.85	0.48	1.11	4.92	-0.64
2	23.22	41.36	6.17	4.69	2.18	3.92	2.78	-0.60
3	74.19	79.28	2.22	0.19	16.17	20.37	7.22	0.65
4	7.57	15.60	1.78	0.28	1.05	5.09	8.74	0.22

ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>
1	-7.45	-21.34	24.40	-0.70	1.59	0.64	4.28	-5.55
2	64.58	18.13	10.86	-1.48	6.10	1.75	2.18	-3.38
3	153.48	5.09	2.41	-2.03	36.54	4.20	7.87	-6.57
4	23.18	8.03	2.06	-1.50	6.14	4.04	8.95	-8.52

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ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	V <sub>r</sub>	W <sub>r</sub>						
1	86.30	-9.58	60.93	-0.84	100.20	7.20	96.25	-0.50
2	12.33	-9.79	87.70	-1.18	64.93	5.65	86.93	0.64
3	100.08	-11.78	76.75	-1.63	82.43	5.73	80.88	0.19
4	24.58	-5.16	28.20	-1.01	27.33	4.45	40.08	-0.23
5	116.43	11.55	79.68	0.10	75.83	-7.43	88.55	3.49

ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>	W <sub>r</sub> +V <sub>r</sub>	W <sub>r</sub> -V <sub>r</sub>
1	76.73	-95.88	60.09	-61.76	107.40	-93.00	95.75	-96.75
2	62.54	-82.11	86.53	-88.88	70.58	-59.28	87.56	-86.29
3	88.30	-111.85	75.13	-78.38	88.15	-76.70	81.06	-80.69
4	19.41	-29.74	27.19	-29.21	31.78	-22.88	39.85	-40.30
5	127.98	-104.88	79.78	-79.58	68.40	-83.25	92.04	-85.06

## APPENDIX 9 (contd.)

Tillers per Plant

ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	V <sub>R</sub>	W <sub>R</sub>						
1	155.45	41.89	22.43	0.93	37.83	23.93	45.18	33.98
2	58.00	-3.50	55.38	31.25	45.20	26.51	98.70	30.80
3	25.33	19.36	31.80	10.58	46.58	14.39	39.20	7.39
4	81.45	-0.14	75.50	15.63	61.33	20.01	16.13	8.13
5	56.45	34.61	46.75	-18.38	8.58	-5.86	75.68	23.98

ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	W <sub>R</sub> +V <sub>R</sub>	W <sub>R</sub> -V <sub>R</sub>	W <sub>R</sub> +V <sub>R</sub>	W <sub>R</sub> -V <sub>R</sub>	W <sub>R</sub> +V <sub>R</sub>	W <sub>R</sub> -V <sub>R</sub>	W <sub>R</sub> +V <sub>R</sub>	W <sub>R</sub> -V <sub>R</sub>
1	197.34	-113.56	23.35	-21.50	61.75	-13.90	79.15	-11.20
2	54.50	-61.50	86.63	-24.13	71.71	-18.69	129.50	-67.90
3	44.69	-5.96	42.38	-21.23	60.96	-32.19	46.59	-31.81
4	31.31	-31.59	91.13	-59.88	81.34	-41.31	24.25	-8.00
5	91.06	-21.84	28.38	-65.13	2.71	-14.44	99.65	-51.70

Spikelet Number per Ear

ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	V <sub>R</sub>	W <sub>R</sub>						
1	7.33	8.52	15.05	17.33	8.52	14.68	9.61	10.65
2	7.38	11.35	5.08	5.24	13.74	18.31	11.24	15.29
3	8.19	10.27	4.36	9.35	5.71	9.12	7.02	11.59
4	7.96	15.26	7.76	12.68	5.65	6.81	3.23	5.68
5	9.85	17.96	7.05	11.10	12.92	17.05	6.52	11.70

ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	W <sub>R</sub> +V <sub>R</sub>	W <sub>R</sub> -V <sub>R</sub>	W <sub>R</sub> +V <sub>R</sub>	W <sub>R</sub> -V <sub>R</sub>	W <sub>R</sub> +V <sub>R</sub>	W <sub>R</sub> -V <sub>R</sub>	W <sub>R</sub> +V <sub>R</sub>	W <sub>R</sub> -V <sub>R</sub>
1	15.85	1.19	32.39	2.28	23.20	6.16	20.27	1.04
2	18.74	3.97	10.32	0.16	32.05	4.57	26.53	4.05
3	18.45	2.08	13.71	5.00	14.82	3.41	18.61	4.57
4	23.22	7.31	20.49	4.91	12.46	1.16	8.91	2.44
5	27.82	8.11	18.15	4.05	29.97	4.12	18.22	5.18

## APPENDIX 9 (contd.)

Grain Number per Ear (transformed to  $10 \log_{10} X$ )

ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	$V_R$	$W_R$	$V_R$	$W_R$	$V_R$	$W_R$	$V_R$	$W_R$
1	1.16	1.90	0.56	1.33	1.49	1.89	1.45	1.38
2	1.28	0.55	1.35	1.09	0.26	-0.06	0.21	0.86
3	0.98	1.65	0.85	1.66	1.86	1.29	1.10	1.55
4	2.04	2.28	1.05	1.22	0.45	1.03	0.85	1.32
5	1.19	1.86	1.97	2.91	1.33	1.69	1.90	2.73

ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	$W_R+V_R$	$W_R-V_R$	$W_R+V_R$	$W_R-V_R$	$W_R+V_R$	$W_R-V_R$	$W_R+V_R$	$W_R-V_R$
1	3.06	0.73	1.89	0.77	3.38	0.40	2.83	-0.07
2	1.83	-0.73	2.44	-0.26	0.21	-0.32	1.07	0.64
3	2.58	0.72	2.51	0.81	3.15	-0.57	2.65	0.45
4	4.32	0.23	2.27	0.17	1.48	0.58	2.17	0.48
5	3.05	0.66	4.89	0.94	3.02	0.36	4.63	0.83

## Grain Yield

ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	$V_R$	$W_R$	$V_R$	$W_R$	$V_R$	$W_R$	$V_R$	$W_R$
1	8.8829	-5.8257	30.1404	-13.8765	28.9057	-4.8284	8.8927	2.9018
2	21.4887	1.8200	13.9203	-9.9938	10.9123	-4.8231	4.6915	-4.1948
3	22.3170	5.1103	15.0361	21.6328	6.2104	1.9858	2.1963	4.0685
4	10.2780	12.5470	41.6999	-2.8774	36.4334	19.4560	5.5921	-0.2610
5	7.0805	3.8270	30.4221	30.4104	15.9024	14.0513	12.7515	1.2265

ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	$W_R+V_R$	$W_R-V_R$	$W_R+V_R$	$W_R-V_R$	$W_R+V_R$	$W_R-V_R$	$W_R+V_R$	$W_R-V_R$
1	3.0571	-14.7086	16.2639	-44.0169	24.0773	-33.7340	11.7945	-5.9910
2	23.3087	-19.6686	3.9265	-23.9141	6.0892	-15.7354	0.4967	-8.8863
3	27.4273	-17.2067	36.6689	6.5966	8.1962	-4.2247	6.2648	1.8722
4	22.8250	2.2691	38.8226	-44.5773	55.8895	-16.9774	5.3311	-5.8531
5	10.9076	-3.2535	60.8325	-0.0118	29.9536	-1.8511	13.9780	-11.5249

## APPENDIX 9 (contd.)

## 100 Kernel Weight

ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	V <sub>R</sub>	W <sub>R</sub>						
1	0.3056	-0.1838	0.3444	-0.1216	0.3292	-0.2015	0.3287	0.0064
2	0.5720	-0.2260	0.5155	-0.2166	0.0763	-0.0122	0.0990	0.0018
3	0.0413	-0.0054	0.2276	0.0208	0.1953	0.1129	0.2826	0.0835
4	0.6535	-0.2741	0.1264	0.1467	0.7253	0.2732	0.2780	0.0396
5	0.6323	0.3491	0.8471	0.2951	0.5672	0.1472	0.3056	0.0391

ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	W <sub>R</sub> +V <sub>R</sub>	W <sub>R</sub> -V <sub>R</sub>	W <sub>R</sub> +V <sub>R</sub>	W <sub>R</sub> -V <sub>R</sub>	W <sub>R</sub> +V <sub>R</sub>	W <sub>R</sub> -V <sub>R</sub>	W <sub>R</sub> +V <sub>R</sub>	W <sub>R</sub> -V <sub>R</sub>
1	0.1218	-0.4894	0.2228	-0.4660	0.1277	-0.5307	0.3351	-0.3223
2	0.3460	-0.7981	0.2988	-0.7321	0.0641	-0.0885	0.1008	-0.0972
3	0.0359	-0.0468	0.2483	-0.2068	0.3082	-0.0824	0.3661	-0.1990
4	0.3794	-0.9276	0.2731	0.0203	0.9985	-0.4521	0.3176	-0.2384
5	0.9815	-0.2832	1.1421	-0.5520	0.7144	-0.4200	0.3447	-0.2665

Physiological Brown Spot (transformed to  $\sin^{-1}\sqrt{X/100}$ )

## 1) Original data:

ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	V <sub>R</sub>	W <sub>R</sub>						
1	56.15	80.02	102.47	96.64	19.68	33.57	19.45	28.13
2	131.04	47.36	201.32	187.33	51.36	45.43	56.04	34.07
3	62.96	36.96	83.50	77.91	66.53	60.82	95.25	81.35
4	66.05	104.78	71.10	84.53	82.21	82.61	143.11	134.48
5	6.18	21.52	53.18	68.61	31.97	29.43	2.28	15.47

ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	W <sub>R</sub> +V <sub>R</sub>	W <sub>R</sub> -V <sub>R</sub>	W <sub>R</sub> +V <sub>R</sub>	W <sub>R</sub> -V <sub>R</sub>	W <sub>R</sub> +V <sub>R</sub>	W <sub>R</sub> -V <sub>R</sub>	W <sub>R</sub> +V <sub>R</sub>	W <sub>R</sub> -V <sub>R</sub>
1	136.17	23.87	199.11	-5.84	53.25	13.89	47.58	8.68
2	178.41	-83.68	388.65	-13.99	96.79	-5.92	90.11	-21.97
3	99.93	-26.00	161.41	-5.59	127.36	-5.72	176.60	-13.90
4	170.84	38.73	155.58	13.47	164.81	0.40	277.59	-8.63
5	27.70	15.33	121.79	15.43	61.40	-2.54	17.76	13.19

## APPENDIX 9 (contd.)

2) Reanalysed data (array 3 deleted):

ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	$V_r$	$W_r$	$V_r$	$W_r$	$V_r$	$W_r$	$V_r$	$W_r$
1	72.42	118.03	104.75	172.95	25.26	35.52	24.81	25.96
2	129.29	112.08	267.73	243.25	60.05	33.42	71.69	26.48
4	83.00	123.37	35.67	52.69	24.00	23.61	131.51	95.39
5	3.06	12.16	20.22	35.88	24.18	-0.93	2.26	10.98

ARRAY	BLOCK 1		BLOCK 2		BLOCK 3		BLOCK 4	
	$W_r+V_r$	$W_r-V_r$	$W_r+V_r$	$W_r-V_r$	$W_r+V_r$	$W_r-V_r$	$W_r+V_r$	$W_r-V_r$
1	190.46	45.61	277.69	68.20	60.79	10.26	50.77	1.15
2	241.37	-17.22	510.98	-24.48	93.47	-26.62	98.17	-45.21
4	206.37	40.37	88.36	17.01	47.61	-0.38	226.90	-36.12
5	15.22	9.10	56.10	15.66	23.25	-25.10	13.24	8.72

Summary of basic array statistics1) Low Nitrogen (means over blocks):

Character	$V_p$	$V_r$	$W_r$	$V_{\bar{r}}$
Heading Date	9.37	76.08	-6.54	18.58
Plant Height	66.94	44.19	42.58	30.07
Tillers/Plant	20.67	9.46	9.96	5.82
Spikelets/Ear	17.45	4.31	6.31	2.49
Grain No./Ear	5.90	1.37	2.39	1.03
Grain Yield	3.8387	1.5829	0.5927	0.1827
100 Kernel Weight	0.1804	0.2162	0.1190	0.0972
Physiological Brown Spot	36.99	29.52	5.88	16.43

APPENDIX 9 (contd.)2) High Nitrogen (means over blocks):

Character	$V_p$	$V_r$	$W_r$	$V_{\bar{r}}$
Heading Date	1.78	73.82	-0.51	23.45
Tillers/Plant	29.44	51.65	15.77	17.10
Spikelets/Ear	28.46	8.21	12.00	5.46
Grain No./Ear	4.37	1.16	1.51	0.62
Grain Yield	24.8889	16.6877	3.6178	1.8398
100 Kernel Weight	0.2180	0.3726	0.0137	0.0644
Physiological Brown Spot	181.37	70.09	67.55	29.44

3) Low and High Nitrogen levels (reanalysed data):

Character	$V_p$	$V_r$	$W_{r\bar{r}}$	$V_{\bar{r}}$
Physiological Brown Spot (LN)(5)#	37.93	11.14	10.56	3.78
Plant Height (LN)(3)	86.73	50.20	50.75	31.71
Tillers/Plant (LN)(3)	27.26	9.79	12.65	6.28
Physiological Brown Spot (HN)(3)	161.86	67.49	70.05	37.20

LN = low nitrogen; HN = high nitrogen; # = array deleted.

## APPENDIX 9 (contd.)

B. Analysis of variance of  $(W_R+V_R)/(W_R-V_R)$  for characters  
in low and high nitrogen levels

Low NitrogenHeading Date

Source	DF	Mean Square	F-test	Signif.
Blocks	3	1193.67	1.12	NS
Arrays ( $W_R+V_R$ )	4	7986.50	7.46	**
ERROR	12	1070.42		
Standard deviation = 32.71		Coefficient of variation = 0.47		
Blocks	3	1048.13	10.76	**
Arrays ( $W_R-V_R$ )	4	4361.37	44.77	***
ERROR	12	97.41		
Standard deviation = 9.87		Coefficient of variation = 0.12		

Plant Height

Source	DF	Mean Square	F-test	Signif.
Blocks	3	1456.00	1.36	NS
Arrays ( $W_R+V_R$ )	4	7591.25	7.07	**
ERROR	12	1074.33		
Standard deviation = 32.77		Coefficient of variation = 0.38		
Blocks	3	237.28	4.23	*
Arrays ( $W_R-V_R$ )	4	170.42	3.04	(NS)
ERROR	12	56.10		
Standard deviation = 7.49		Coefficient of variation = 4.68		

## APPENDIX 9 (contd.)

2) Reanalysed data (array 3 deleted):

Source	DF	Mean Square	F-test	Signif.
Blocks	3	1017.33	0.60	NS
Arrays ( $W_R+V_R$ )	3	14442.00	8.45	**
ERROR	9	1708.78		
Standard deviation = 41.34		Coefficient of variation = 0.41		
Blocks	3	135.15	1.48	NS
Arrays ( $W_R-V_R$ )	3	227.96	2.49	NS
ERROR	9	91.54		
Standard deviation = 9.57		Coefficient of variation = 17.58		

Tillers per Plant1) Original data:

Source	DF	Mean Square	F-test	Signif.
Blocks	3	111.65	1.94	NS
Arrays ( $W_R+V_R$ )	4	114.34	1.98	NS
ERROR	12	57.66		
Standard deviation = 7.59		Coefficient of variation = 0.39		
Blocks	3	6.81	0.92	NS
Arrays ( $W_R-V_R$ )	4	11.71	1.58	NS
ERROR	12	7.42		
Standard deviation = 2.72		Coefficient of variation = 5.47		

2) Reanalysed data (array 3 deleted):

Source	DF	Mean Square	F-test	Signif.
Blocks	3	142.87	1.10	NS
Arrays ( $W_R+V_R$ )	3	221.73	1.70	NS
ERROR	9	130.13		
Standard deviation = 11.41		Coefficient of variation = 0.51		
Blocks	3	11.54	1.85	NS
Arrays ( $W_R-V_R$ )	3	14.92	2.39	NS
ERROR	9	6.25		
Standard deviation = 2.50		Coefficient of variation = 0.87		

## APPENDIX 9 (contd.)

Spikelet Number per Ear

Source	DF	Mean Square	F-test	Signif.
Blocks	3	20.47	1.05	NS
Arrays ( $W_R+V_R$ )	4	289.66	14.82	***
ERROR	12	19.55		
Standard deviation = 4.42		Coefficient of variation = 0.42		
Blocks	3	4.06	3.99	*
Arrays ( $W_R-V_R$ )	4	0.35	0.34	NS
ERROR	12	1.02		
Standard deviation = 1.01		Coefficient of variation = 0.50		

Grain Number per Ear (transformed to  $10 \log_{10} X$ )

Source	DF	Mean Square	F-test	Signif.
Blocks	3	0.90	0.78	NS
Arrays ( $W_R+V_R$ )	4	1.32	1.15	NS
ERROR	12	1.15		
Standard deviation = 1.07		Coefficient of variation = 0.28		
Blocks	3	0.15	1.57	NS
Arrays ( $W_R-V_R$ )	4	0.20	2.02	NS
ERROR	12	0.10		
Standard deviation = 0.31		Coefficient of variation = 0.31		

Grain Yield

Source	DF	Mean Square	F-test	Signif.
Blocks	3	2.8270	0.56	NS
Arrays ( $W_R+V_R$ )	4	17.3748	3.46	*
ERROR	12	5.0218		
Standard deviation = 2.2410		Coefficient of variation = 1.03		
Blocks	3	4.2120	3.45	(NS)
Arrays ( $W_R-V_R$ )	4	1.9545	1.60	NS
ERROR	12	1.2226		
Standard deviation = 1.1060		Coefficient of variation = 1.12		

## APPENDIX 9 (contd.)

100 Kernel Weight

Source	DF	Mean Square	F-test	Signif.
Blocks	3	0.0421	2.70	(NS)
Arrays ( $W_R+V_R$ )	4	0.0844	5.42	**
ERROR	12	0.0156		
Standard deviation = 0.1247		Coefficient of variation = 0.37		
Blocks	3	0.0078	2.17	NS
Arrays ( $W_R-V_R$ )	4	0.0138	3.82	*
ERROR	12	0.0036		
Standard deviation = 0.0601		Coefficient of variation = 0.62		

Physiological Brown Spot (transformed to  $\sin^{-1}\sqrt{X/100}$ )1) Original data:

Source	DF	Mean Square	F-test	Signif.
Blocks	3	2152.93	3.31	(NS)
Arrays ( $W_R+V_R$ )	4	3911.93	6.01	**
ERROR	12	651.48		
Standard deviation = 25.52		Coefficient of variation = 0.72		
Blocks	3	1277.40	3.36	(NS)
Arrays ( $W_R-V_R$ )	4	76.75	0.20	NS
ERROR	12	380.63		
Standard deviation = 19.51		Coefficient of variation = 0.83		

2) Reanalysed data (array 5 deleted):

Source	DF	Mean Square	F-test	Signif.
Blocks	3	2431.67	1.99	NS
Arrays ( $W_R+V_R$ )	3	1595.33	1.31	NS
ERROR	9	1222.89		
Standard deviation = 34.97		Coefficient of variation = 1.61		
Blocks	3	66.62	0.94	NS
Arrays ( $W_R-V_R$ )	3	77.99	1.10	NS
ERROR	9	71.13		
Standard deviation = 8.43		Coefficient of variation = 14.70		

APPENDIX 9 (contd.)High NitrogenHeading Date

Source	DF	Mean Square	F-test	Signif.
Blocks	3	159.07	0.53	NS
Arrays ( $W_R+V_R$ )	4	2511.10	8.32	**
ERROR	12	301.84		
Standard deviation = 17.37		Coefficient of variation = 0.24		
Blocks	3	371.57	2.68	(NS)
Arrays ( $W_R-V_R$ )	4	2448.33	17.67	***
ERROR	12	138.53		
Standard deviation = 11.77		Coefficient of variation = 0.16		

Tillers per Plant

Source	DF	Mean Square	F-test	Signif.
Blocks	3	1077.00	0.48	NS
Arrays ( $W_R+V_R$ )	4	1461.75	0.65	NS
ERROR	12	2264.42		
Standard deviation = 47.58		Coefficient of variation = 0.71		
Blocks	3	448.60	0.47	NS
Arrays ( $W_R-V_R$ )	4	246.15	0.26	NS
ERROR	12	951.85		
Standard deviation = 30.85		Coefficient of variation = 0.86		

## APPENDIX 9 (contd.)

Spikelet Number per Ear

Source	DF	Mean Square	F-test	Signif.
Blocks	3	16.63	0.31	NS
Arrays ( $W_R+V_R$ )	4	51.49	0.97	NS
ERROR	12	52.95		
Standard deviation = 7.28		Coefficient of variation = 0.36		
Blocks	3	1.55	0.30	NS
Arrays ( $W_R-V_R$ )	4	4.13	0.78	NS
ERROR	12	5.27		
Standard deviation = 2.30		Coefficient of variation = 0.61		

Grain Number per Ear (transformed to  $10 \log_{10} X$ )

Source	DF	Mean Square	F-test	Signif.
Blocks	3	0.47	0.55	NS
Arrays ( $W_R+V_R$ )	4	3.17	3.68	*
ERROR	12	0.86		
Standard deviation = 0.93		Coefficient of variation = 0.35		
Blocks	3	0.17	0.80	NS
Arrays ( $W_R-V_R$ )	4	0.40	1.95	NS
ERROR	12	0.21		
Standard deviation = 0.45		Coefficient of variation = 1.34		

Grain Yield

Source	DF	Mean Square	F-test	Signif.
Blocks	3	0.0244	0.37	NS
Arrays ( $W_R+V_R$ )	4	0.2681	4.07	*
ERROR	12	0.0659		
Standard deviation = 0.2567		Coefficient of variation = 0.67		
Blocks	3	0.0722	1.06	NS
Arrays ( $W_R-V_R$ )	4	0.0664	0.97	NS
ERROR	12	0.0683		
Standard deviation = 0.2613		Coefficient of variation = 0.73		

## APPENDIX 9 (contd.)

100 Kernel Weight

Source	DF	Mean Square	F-test	Signif.
Blocks	3	519.1333	2.38	NS
Arrays ( $W_R+V_R$ )	4	365.8000	1.68	NS
ERROR	12	218.1333		
Standard deviation = 14.7682		Coefficient of variation = 0.73		
Blocks	3	205.5667	1.20	NS
Arrays ( $W_R-V_R$ )	4	335.4250	1.95	NS
ERROR	12	171.7250		
Standard deviation = 13.1034		Coefficient of variation = 1.00		

Physiological Brown Spot (transformed to  $\sin^{-1}\sqrt{X/100}$ )1) Original data:

Source	DF	Mean Square	F-test	Signif.
Blocks	3	10691.67	2.07	NS
Arrays ( $W_R+V_R$ )	4	12872.00	2.49	(NS)
ERROR	12	7088.50		
Standard deviation = 71.84		Coefficient of variation = 0.52		
Blocks	3	59.17	0.12	NS
Arrays ( $W_R-V_R$ )	4	1448.20	2.98	(NS)
ERROR	12	485.99		
Standard deviation = 22.05		Coefficient of variation = 8.69		

2) Reanalysed data (array 3 deleted):

Source	DF	Mean Square	F-test	Signif.
Blocks	3	24077.33	2.11	NS
Arrays ( $W_R+V_R$ )	3	29335.00	2.57	(NS)
ERROR	9	11410.33		
Standard deviation = 106.82		Coefficient of variation = 0.78		
Blocks	3	1528.03	4.76	*
Arrays ( $W_R-V_R$ )	3	2388.07	7.43	**
ERROR	9	321.28		
Standard deviation = 17.92		Coefficient of variation = 7.00		

APPENDIX 10Regression equations and associated statistics for  
reanalysed characters with one array deletedLow NitrogenPlant Height

Array deleted	Regression equation	S.E.	F-test	R-square	S.E. ( $\hat{\beta}_1$ )
1	$W_R = 0.78V_R + 12.96$	12.43	81.49***	0.85	0.09
2	$W_R = 0.75V_R + 13.22$	8.41	100.78***	0.88	0.08
3	$W_R = 0.87V_R + 6.97$	10.80	111.89***	0.89	0.08
4	$W_R = 0.81V_R + 6.97$	11.61	126.09***	0.90	0.07
5	$W_R = 0.20V_R + 0.18$	5.41	1.03 NS	0.07	0.20

Tillers per Plant

Array deleted	Regression equation	S.E.	F-test	R-square	S.E. ( $\hat{\beta}_1$ )
1	$W_R = 0.69V_R + 5.47$	3.26	27.26***	0.66	0.13
2	$W_R = 0.38V_R + 7.55$	2.47	7.50*	0.35	0.14
3	$W_R = 0.81V_R + 4.73$	2.82	54.02***	0.79	0.11
4	$W_R = 0.74V_R + 4.93$	1.77	152.90***	0.92	0.06
5	$W_R = 0.24V_R - 1.06$	2.90	1.01 NS	0.07	0.24

## APPENDIX 10 (contd.)

Physiological Brown Spot (transformed to  $\sin^{-1}\sqrt{X/100}$ )

Array deleted	Regression equation	S.E.	F-test	R-square	S.E. ( $\hat{\beta}_1$ )
1	$W_R = 0.51V_R - 8.26$	23.79	5.86*	0.30	0.21
2	$W_R = 0.61V_R - 15.44$	22.35	11.89**	0.46	0.18
3	$W_R = 0.10V_R - 4.11$	10.42	0.89 NS	0.06	0.10
4	$W_R = 0.61V_R - 13.08$	25.50	7.59*	0.35	0.22
5	$W_R = 1.15V_R - 2.21$	8.32	91.14***	0.87	0.12

High Nitrogen

Physiological Brown Spot (transformed to  $\sin^{-1}\sqrt{X/100}$ )

Array deleted	Regression equation	S.E.	F-test	R-square	S.E. ( $\hat{\beta}_1$ )
1	$W_R = 0.94V_R + 6.73$	36.13	35.79***	0.72	0.16
2	$W_R = 0.90V_R + 8.25$	22.02	52.35***	0.79	0.12
3	$W_R = 0.90V_R + 9.13$	31.61	56.08***	0.80	0.12
4	$W_R = 0.72V_R + 15.71$	23.30	50.66***	0.78	0.10
5	$W_R = 0.74V_R + 18.37$	31.63	19.07***	0.58	0.17