QUANTITATIVE INHERITANCE OF RESISTANCE TO STRIPE RUST (*Puccinia striiformis* West.) IN WHEAT (*Triticum aestivum* L.)

A thesis presented
in partial fulfilment of the requirements
for the degree of Doctor of Philosophy (Ph.D.) in
Plant Breeding & Genetics
Department of Plant Science
Massey University

Mohammad Reza Ghannadha

1993

In the name of Almighty God,
the most Compassionate, the most merciful,

Praise be to God, Lord, Master, Cherisher and Sustainer
of the universe

ABSTRACT

Fifteen wheat (*Triticum aestivum L.*) cultivars, of widely varying resistance to stripe rust, were tested by measuring infection type, latent period, pustule size and pustule density in the glasshouses to study the inheritance of resistance to three pathotypes of stripe rust (*Puccinia striiformis* West.). Large differences were demonstrated between cultivars by using ANOVA and MANOVA. There were highly significant positive genotypic and phenotypic correlations amongst infection type, pustule size and pustule density whereas there were highly significant negative correlations amongst latent period with other components. Pathotype 232E137A- was more aggressive than the other pathotypes on these cultivars. Heritability was moderately high for infection type and latent period in comparison with pustule size and pustule density.

Genetic studies were made of seedling resistant to two pathotypes of stripe rust by means of a diallel cross among five seedling resistant cultivars and one susceptible. Parents and F_1 progenies were assessed for the four characters previously mentioned. Hayman's and Griffing's diallels demonstrated that the additive component of variation was the major component although dominance was important, in which case it was usually partial dominance. High heritability was observed for all characters showing the effectiveness of the breeding programme. By using W_r/V_r graphical analysis, it was observed that Tiritea which is susceptible to both pathotypes had reversal gene action. Reversal gene action was also observed amongst seedling resistant cultivars. In general, if a cultivar was susceptible to a pathotype it would show recessive gene action, and if it was resistant it would show dominance.

To study the interaction of seedling resistant gene(s) with adult plant resistant gene(s), four adult plant resistant with five seedling resistant cultivars were intercrossed in a factorial mating fashion. Parents and F_1 progenies were tested by two pathotypes of stripe rust in the glasshouse, measuring the four characters mentioned before. Estimates of genetic components of variance indicated that the major genetic effect controlling all characters was additive, and that the additive component resulting from seedling resistant cultivars was by far greater than that

which resulting from adult plant resistant cultivars. This suggests that even in crosses of seedling resistant cultivars transgressive segregation will be observed. In general, those genes in adult plant resistant cultivars were masked by those in seedling resistant cultivars.

Four adult plant resistant cultivars and one susceptible were intercrossed in a diallel fashion. Parents and F_1 progenies were tested with three pathotypes in the glasshouse, measuring the four previous characters. Griffing's and Hayman's analyses indicated that general combining ability explained a high proportion of the variability of most of the characters. This was substantiated by high narrowsense heritability estimates. Specific combining ability was small but significant, suggesting that dominance and nonallelic interaction played a minor role. In general, partial dominance was observed for most characters but in response to pathotype 232E137A-, full dominance and overdominance also were observed. The number of genes could be changed for any one character by changing the pathotype.The W_r/V_r graphic analysis of Hayman indicated that Tiritea which is susceptible to all pathotypes had reversal dominance. Reversal gene action were also observed in adult plant resistance cultivars but Briscard had constant recessiveness for most of characters against all pathotypes.

All possible crosses, except reciprocal, were made among four adult plant resistant cultivars and one susceptible to study in more detail the inheritance of infection type and latent period in the glasshouse by using generation mean analysis. This involves parents, F_1, F_2, and backcross populations. Testing was at the seedling stage, using the common pathotype (106E139A-) in New Zealand. All crosses showed transgressive segregation in both resistance and susceptibility. These results indicate that most of the genes conditioning resistance (low infection type or long latent period) in adult plant resistant cultivars are different from one another. This is collaborated by the number of genes conditioning both characters, which were also different. Over ten crosses, the broadsense and narrowsense heritabilities averaged 0.76 and 0.61, respectively for infection type. These were greater than the heritabilities for latent period. For both characters, simple additive-dominance model was not sufficient and epistatic gene action for resistance was significant for all crosses. This suggested the action of more than one gene. Additive and additive x

additive gene action were important for both characters.

Inheritance of stripe rust severity was studied in the field, by means of diallel and generation mean analysis of all possible crosses among four adult plant resistant cultivars and one susceptible. The diallel showed that the additive component of variation again was the major gene action. This was confirmed by a high narrowsense heritability. In the adult stage, resistant cultivars showed dominance which was partial for resistance. Results of generation means analysis confirmed the diallel results. Over all ten crosses, significant epistatic gene effects were present, suggesting polygenic inheritance. Transgressive segregation was observed, indicating different genes amongst the parents were controlling the resistance. The average broadsense and narrowsense heritabilities were 0.73 and 0.51, respectively. Genes conditioning rust severity in adult plant resistant cultivars showed more frequent (partial) dominance in the adult stage than in the seedling stage. Also the numbers of genes in the adult stage were greater than in the seedling stage.

Knowledge of the type of gene action involved in the expression of a character is helpful in deciding on the breeding procedures to be used for improvement of the character. The study showed that selection among F_2 plants of almost all crosses should be effective for higher levels of resistance than the parents and would be useful sources of resistance in breeding programme.

ACKNOWLEDGEMENTS

I would like to express my deepest gratitude to my chief supervisor, Dr. I.L. Gordon, my co-supervisors Dr. J.M. McEwan (Crop Research Division, ex DSIR, Palmerston North) and Dr. M. G. Cromey (Plant Protection, ex DSIR, Lincoln) for their untiring quantitative, pathological and grammatical guidance, close supervision, enthusiastic encouragement, great assistance, invaluable criticism, patient efforts, advice, valuable discussion, wise counsel, unforgettable friendship, helpful suggestions on content and presentation during all phases of this study.

Special thanks must go to Head and all members of the ex Agronomy: Prof. J. Hodgson, Dr. P. Kemp (former liaison person), Dr. I. Valentine, Dr. C. Matthew, Mrs C. Gwynne and Mrs J. Cave for their valuable assistance in many ways.

I am particularly indebted to Plant Science Dept., specially Prof. M. Hill (Head of Seed and Crop Group), Dr H. Behboudian (liaison person), Dr. P. Long, J. Milner, Dr. K. Harington and Mrs C. Gwynne for their helpful advice and kindness at various stages of the study.

I wish to thank the Crop & Food research Institute, Lincoln: (1) Plant Protection for providing me with excellent laboratory and support during my study at Lincoln and (2) Crop Research Division for supplying seeds for the experiment.

This study could not have been fully undertaken without the technical assistance of a number of people whose help I gratefully acknowledge. Skilled assistance was provided by : D. Sollit, C. McKenzie and F. Brown for their technical and organisational assistance: Mr T. Lynch, G. Evanse and M. Osbone for field technical assistance.

I would also wish to express my grateful thanks to Ray Johnstone, Charlie Forbes and Giles Russell and all members of Massey Plant Growth Unit for their co-operative manner, willingness to help, friendly attitude and prompt dealing with problems which have made a substantial contribution to the success of this study. In particular, I was impressed by Mr Ray Johnstone who usually said "everything is

possible" whenever I have faced any problem.

Special thanks and appreciation must be due to all postgraduate students of Plant Science Dept. and the families of my Iranian friends who made our (my and my family) being in N.Z. pleasant and memorable by their invaluable friendship, joyful company, humour and help.

I would like to extend my sincere appreciation to my sponsor, Ministry of Culture and Higher Education of Islamic Republic of Iran, for financial support during my stay in New Zealand.

My deepest gratitude and heartfelt thanks to my dear relatives : my mother, sister, brothers, and their families for their love, moral support, encouragement and patience while I was in New Zealand. I especially wish to appreciate the help of my caring brother, Farshad, for his considerable time, effort and patience.

Last, but certainly not least, I am forever grateful to my wife, Maryam, and childem, Shima & Mehran, for their love, devoted support, consistent concem encouragement, understanding, forbearance and help throughout the course of my study.

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CHAPTER 1

INTRODUCTION

In the year 1650 the world's population on the earth was estimated at 500 million; providing about 3 ha of crop land per capita. Now the population is greater than 4 billion and the available crop land is less than 0.4 ha per capita. In the year 2000 the world population may be estimated to be about 6.8 billion (Poehlman, 1987), so the demand for food is increasing day by day.

Wheat (*Triticum aestivum* L.) is grown in a wide range of environments around the world, in almost every country from within the Arctic Circle to the equator, and more than 20% of our food comes from wheat (Poehlman, 1987) This suggests that it is the world's most important crops (Fig. 1).

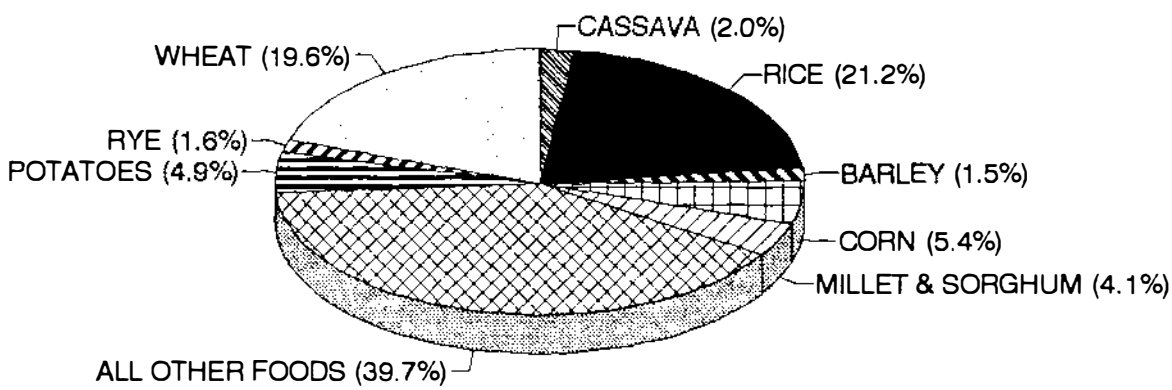
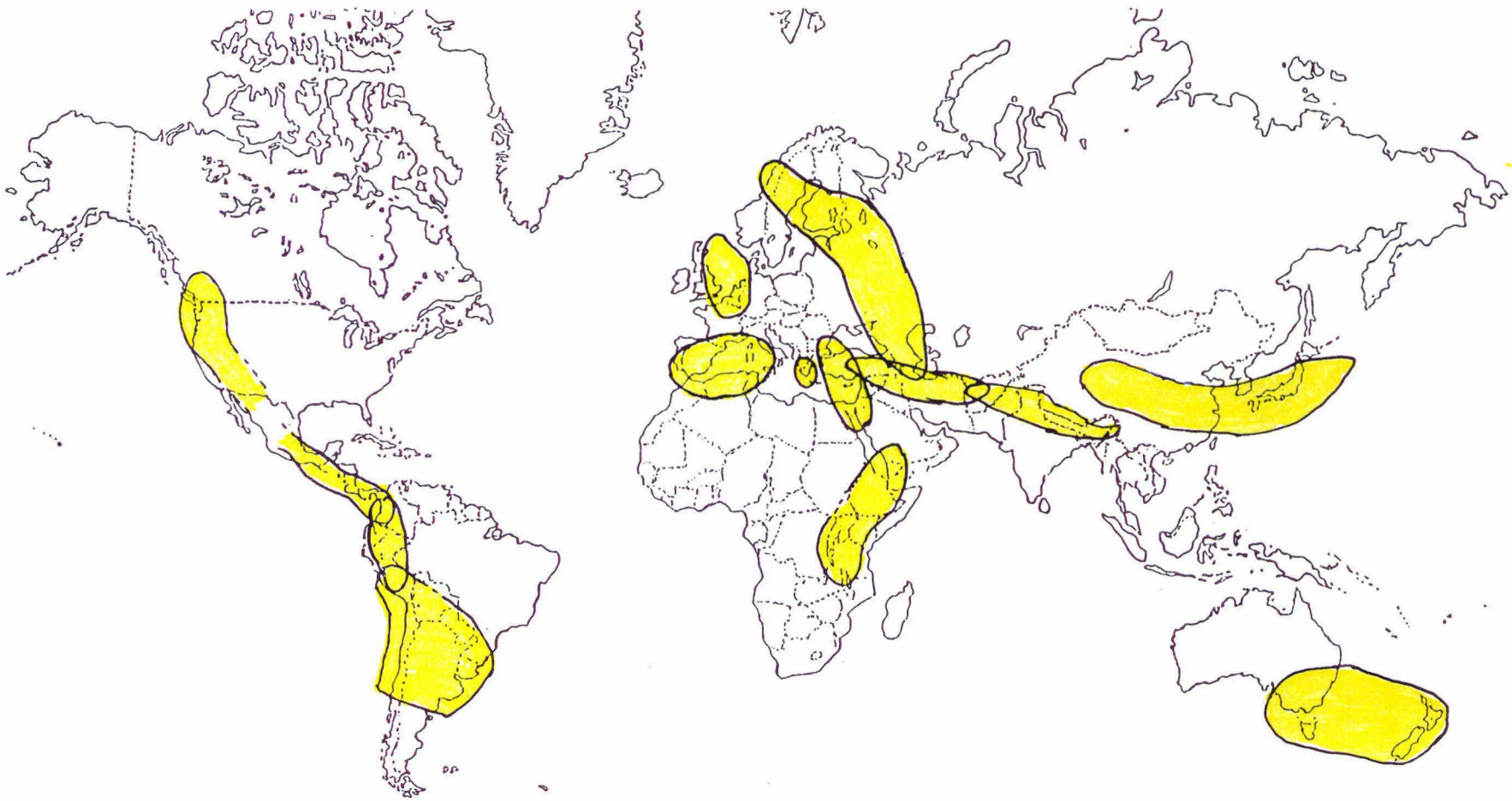


Fig. 1. The percentage of human food composition in the world (Poehlman, 1987)

Stripe rust (yellow rust) caused by *Puccinia striiformis* West. f.sp. *tritici*, is reported to be the most serious rust of wheat in many areas of the world where wheat is grown in cool, damp climate (Roelfs *et al.*, 1992). The world geographical distribution of stripe rust races is shown in Fig. 2 (Stubbs, 1988).

Apart from yield improvement, genetic resistance has been the main focus of wheat breeders throughout the world. In Australasia, wheat stripe rust was first reported in Australia in October 1979 (O'Brien *et al.*, 1980). The rapid establishment and

Fig. 2. The world geographical distribution of stripe rust races



widespread distribution of this disease in the first season of occurrence suggested that the pathogen may have been present, but undetected, for several years (Wellings *et al.*, 1987). These authors postulated that an international traveller might have imported the first race of stripe rust from Europe to Australia in 1979. The rust appeared in New Zealand, in 1980, apparently having been windborne from Australia (Beresford, 1982; McIntosh and Wellings, 1986). The relationship between pathotypes in New Zealand is presented schematically in Fig. 3 (Cromey and Munro, 1992).

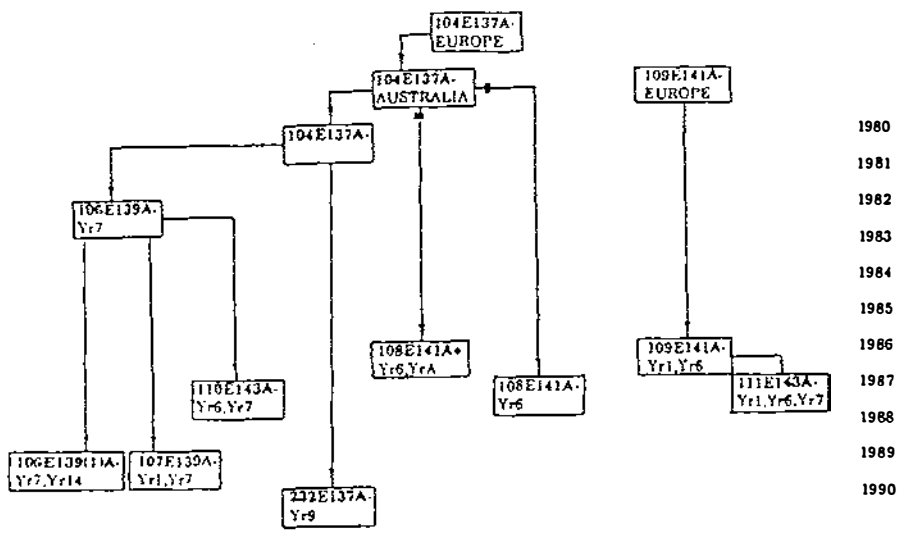


Fig. 3. Stripe rust recorded in New Zealand, their year of identification, and virulence

Stripe rust is a continual threat to wheat in many areas of the world. Crop losses in susceptible cultivars were as high as 50-60% in Australia (McIntosh and Wellings, 1986) and up to 60% in New Zealand (Beresford, 1982). As well as infecting leaves, stripe rust can attack wheat spikes. In New Zealand, spike infection can be widespread in crops of susceptible wheat cultivars, resulting in significant yield losses where the disease is not controlled adequately (Cromey, 1989a). Spike infection has been recorded in the foliar-resistant cultivar in the United States (Purdy and Allan, 1965).

There are three approaches to the control of rust disease: firstly agronomic practices such as removing the "green bridge" (volunteer plants), secondly, chemical control and thirdly, genetic control via the host. Chemical control, although often

effective, may pose environmental hazards, is relatively expensive. It may be especially uneconomic in developing countries. However, control through host genetic resistance to rusts is both economically feasible and environmentally safe.

The use of resistant cultivars is the most feasible means to prevent stripe rust damage. Two different types of resistance are present amongst the cultivars: overall or seedling resistance and adult plant resistance. The first effective at all stages of plant growth, which the second is effective only at post-seedling growth stages (van der Plank, 1982). Cultivars that contain only specific major genes for resistance are vulnerable. Race-specific resistance can be identified easily and is effective in the seedling test. However, it causes directional selection and pressure towards greater virulence in rust populations and consequently that major gene(s), whether seedling resistance or adult plant resistance, causes "boom and bust cycle" of cereal cultivars (Fig. 4).

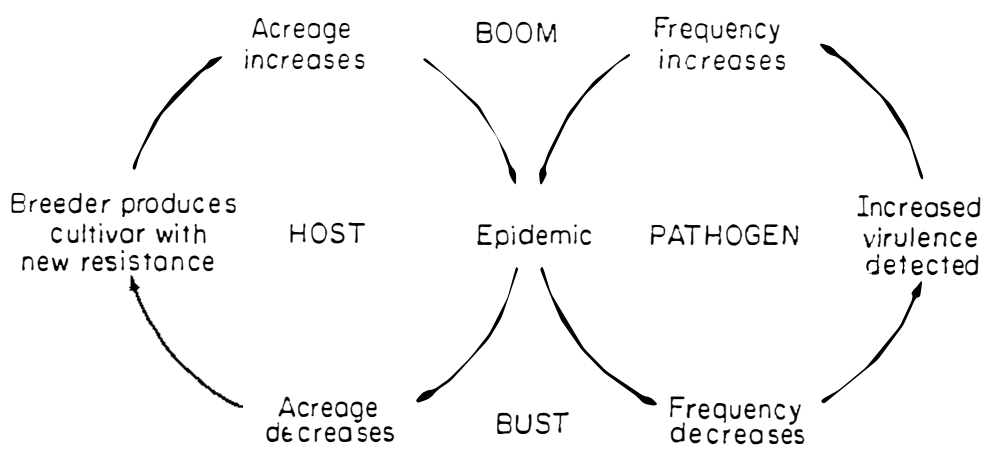


Fig. 4. The "boom and bust" cycle (Priestley, 1978).

Consequently, breeders have turned their attention to other forms of resistance, such as adult plant resistance (but polygenes and durable type), that are not so dramatically effective. Adult plant resistance is defined as resistance that is absent in young seedlings but develops as the plant matures. If the resistance is controlled by several to many genes and does not result in differential selection in the pathogen, the pathogen may have greater difficulty in developing effective virulence. Successful use of such resistance is enhanced by a knowledge of its genetic nature.

Since 1980, stripe rust resistance has been a major aim in New Zealand wheat breeding programmes and a number of cultivars with improved resistance have been released (Cromey, 1989b). However the resistance of some cultivars often has been short-lived due to the occurrence of new virulent races. Recently, breeding programmes have focused on the aim of developing of cultivars with more durable resistance. Johnson (1984) defines durable resistance as "resistance that remains effective during its prolonged and widespread use in an environment favourable to the disease". The test for durable resistance must include two elements, time (long) and area (large). By these criteria, it is difficult to know whether some new cultivars possess durable resistance. Allard (1960) pointed out that potential problems inherent in incorporation of resistance from unadapted cultivars into a breeding program are the possible introduction of unknown susceptibility to a nontarget pathogen, or linkage between the genes controlling disease resistance and agronomically undesirable traits such as late maturity, height, and weak straw. Also studies in the USA (Hendriksen and Pope, 1971; Krupinsky and sharp, 1979) and United Kingdom (Wallwork and Johnson, 1984) suggested that improved levels of resistance can be obtained from intercrosses among moderately resistant and moderately susceptible wheats. These, presumably, were of a polygenic nature.

The present study aims at estimating the quantitative genetics of adult and seedling resistant cultivars to stripe rust with a local, but diverse wheat germplasm. This knowledge should enhance the possibility of constructing multiple gene combinations, as well as demonstrating how major gene(s) and minor genes interact.

Estimating components of quantitative genetic variation may be useful in at least three ways: (1) understanding fundamental genetic phenomena, (2) predicting genetic advance under selection, and (3) identifying parents which might increase resistance for F_1 hybrids. The third reason may become increasingly important in cereal breeding if hybrid vigour is of useful level. Sprague (1963) listed three major factors that must be considered and which may limit progress in the analysis of quantitative variation: the number of genes involved, the type of gene action, and the genotype environment interaction.

Therefore, objectives of this study were:

1. To estimate seedling responses and genotypic parameters cultivars with respect to components of resistance separately for three pathotypes and over all pathotypes collectively;

2. To estimate quantitative genetics of seedling-resistance with two pathotypes (using two glasshouse diallel, one per pathotype);

3. To estimate quantitative genetics of combinations of seedling and adult plant resistance with two pathotypes (using two glasshouse factorial- matings , one per pathotype);

4. To estimate quantitative genetics of adult plant resistance over three pathotypes (using three glasshouse diallels, one per pathotype);

5. To estimate more detailed quantitative genetics of adult plant resistance, using parents, F_1, F_2, and backcross to both parents progenies with common pathotype as seedlings in ten generation means mating designs;

6. To estimate quantitative genetics of adult plant resistance in adult stages with common pathotype (using one field diallel);

7. To estimate more detailed quantitative genetics of adult plant resistance, using parents, F_1, F_2, F_3, backcross and backcross selfing to both parents progenies in adult stages with common pathotype in ten generation means mating designs.

CHAPTER 2

LITERATURE REVIEW

2.1. Pathogen

2.1.1. Introduction

Several thousands of rust species attack a wide range of higher plants. A number of them cause serious economic losses in crops, but none more so than the three rusts that attack wheat, the world's most important crop.

2.1.1.1. Wheat stem rust

Stem rust, which is also called black rust, is caused by *Puccinia graminis* Pers. The name black rust refers to the black teliospores which are formed towards the end of the growing season. Within *P. graminis*, specialization on particular host genera has occurred to produce formea specials. Three of the most important are : *P. graminis* f.sp.*avenae*, which is specific to oats and some related grasses, *P. graminis* f.sp.*secalis* on rye and some related grasses, and, most important, *P. graminis* f.sp.*tritici* on wheat, barley, and many of other relatives of wheat.

2.1.1.2. Wheat leaf rust

Wheat leaf rust, sometimes called brown rust, is caused by *Puccinia recondita* Rob. ex Desm. It also shows specialization for specific hosts and the wheat leaf rust fungus is commonly designated *P. recondita* f.sp.*tritici*.

2.1.1.3. Wheat stripe rust

Stripe rust often called yellow rust (it was accepted that the American name, stripe rust rather than European name, yellow rust was the adopted name throughout Australia and New Zealand, McIntosh and Wellings, 1986), is caused by *Puccinia Striiformis* Westend, and is one of the most important rust diseases of wheat. This rust requires relatively cool temperatures for good growth, and so is found wherever wheat is grown in a cool, damp climate. It is sensitive to environmental factors such as high

temperature, low moisture and air pollution (Stubbs, 1985). The world distribution of yellow rust virulence and the geographical distribution of yellow rust were reviewed by Stubbs in 1985 and 1988, respectively. It is most common in regions where the climate is cool (temperate about 15 °C and less) and is generally associated with high elevations, northern latitudes or cooler years, or in tropical areas where wheat is grown in the cool, moist winter season. It takes its name from the characteristic stripe of uredinia that produce yellow coloured urediniospores.

2.1.2. Taxonomy and specificity

The wheat rust pathogens belong to the genus *Puccinia* of the family *Pucciniaceae* of the order *Uredinales* of the class *Basidiomycetes* (Littlefield, 1981). In the *Basidiomycetes*, meiosis occurs in a basidium and results in the production of four haploid, single-celled basidiospores. The order *Uredinales* includes the rust fungi, which are highly specialized plant pathogens. Depending on the taxonomist, the *Uredinales* are divided into two or more families based on the characters of the teliospores. By far the largest number of rust species belong to the *Pucciniaceae* and *Puccinia* is the largest genus in the family. Because the rust fungi are highly specialized and have narrow host ranges, identification of the hosts is an important aid in identifying a rust fungus. Cereal rusts are highly specialized pathogens and resistance, whether race-specific or non-race-specific, operates to one pathogen species; so they are pathogen specific. All three rusts differ in morphology, life cycle, and optimal environmental conditions for growth.

2.1.3. Variability in the rust pathogen

Within a rust species, the first level of variability is "formae specials". They are defined by their ability to attack a particular host species or a group of related species but do not differ morphologically. When a progeny of the pathogen exhibits a characteristic that is different from those present in the parental individuals, it is called "variant". The population of genetically identical individuals produced by the variant is called a "biotype". The biotype with certain defined characteristics such as pathogenicity on a particular set of host differentials is called a "physiological race" or "strain" or "pathotype" (Robinson, 1969). The origin of pathogenic variability is mutation, somatic recombination, parasexuality and sexual state. So far mutation and somatic

recombination have been associated with variability in stripe rust (Stubbs, 1985).

2.1.4. Life cycle of stripe rust

Since no alternate host has been identified for stripe rust, only three spores stages are known; urediospores, teliospores and basidiospores. The life cycle of stripe rust involves repeated cycles of the asexual uredinal stage. A major difference between stripe rust and the other two rusts infecting wheat is that in stripe rust a single infection on a leaf can produce a long stripe containing many uredia whereas for the other rusts, a single uredinium is usually produced from one infection (Fig. 5).

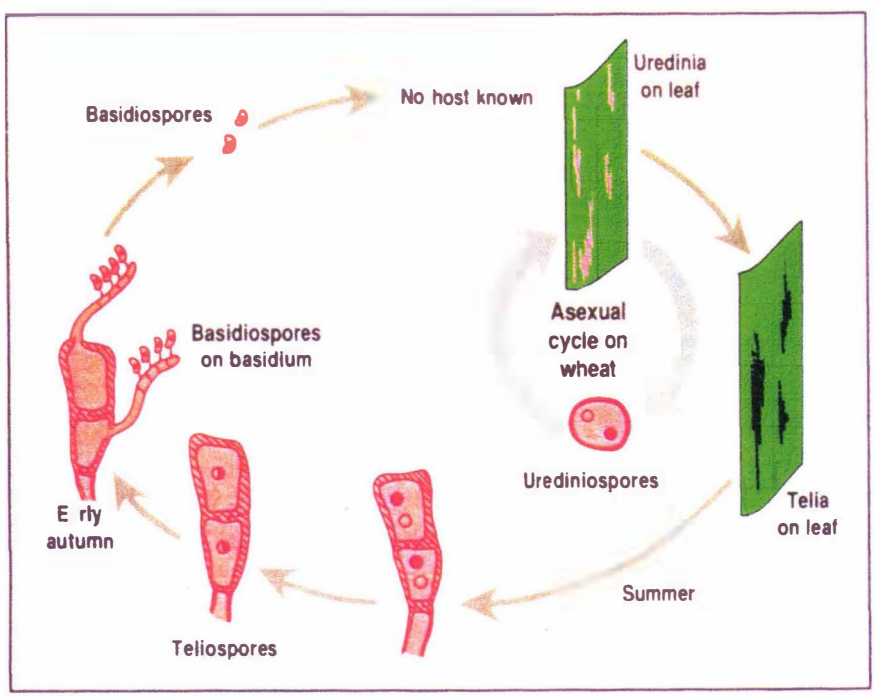


Fig. 5. Life and disease cycles for wheat stripe rust (Roelfs *et al.*, 1992)

2.2. Host-pathogen systems

These host-pathogen systems can be divided into two categories: Specific and Non-specific.

Race-specific or vertical resistance implies resistance to some pathotypes or races and not to others; the presence of differential genetic interactions between host and pathogen genotypes, there is the possibility to identify races, relatively simple

inheritance. Non-specific, or horizontal, resistance implies resistance to all isolates of the disease organism; the absence of genetic interactions between host and pathogen genotypes, and no possibility to discern pathogen races and often polygenic inheritance. So if a pathogen isolate has overcome non-specific resistance it must be reclassified as specific. Specific resistance has also been defined as resistance to infection or hypersensitive resistance, while non-specific resistance permits infection but reduces colonization or spread of the disease (van der Plank, 1968b; Nelson, 1978).

High levels of resistance based on single genes are often very vulnerable to the occurrence and spread of new races of pathogens, while polygenic resistance (which is controlled by several genes and is difficult or impossible to identify the effect of individual genes, Knott, 1988) is not usually affected by this problem of being overcome by new races. Resistance that have sometimes been characterized as adult plant resistance (resistance is not fully expressed at the seedling stage), slow rusting (a reduced rate of epidemic development), partial resistance (a reduced rate of epidemic development despite a high or susceptible infection type), minor genes, etc. are usually placed in non-specific group.

2.2.1. Race specificity and gene-for-gene theory

Flor (1942, 1946, 1947) was the first person to study both the inheritance of pathogenicity in a pathogen and the inheritance of resistance in its host. Working with flax rust (*Melampsora lini* Desm.) and its host, flax (*Linum usitatissimum* L.), he demonstrated that if a flax cultivar carried a single gene for resistance, then virulence in the rust was also conditioned by a single gene. Similarly, if resistance in a cultivar was conditioned by two genes, then virulence in the rust was conditioned by two genes. Flor (1956) used the term complementary genes for the interacting genes in the host and pathogen. However, since this term has a specific and different genetic meaning, the terms corresponding or matching genes are now used.

2.2.2. Expression of specific resistance

1. Hypersensitivity. This is an active resistance mechanism in which the rapid death of the host cells around the point of infection prevents colonization (Robinson,

1976).

2. Immunity. An immune response is indicated by the absence of visible lesion on the host plant (Stakman *et al*., 1962) and an immune plant is a non-host (Robinson, 1976). Since an immune or hypersensitive reaction depends on host genetic background and/or the rust culture used, the presence or absence of visible lesions does not imply two different types of specific genes.

3. Intermediate. Resistance does not prevent colonization but reduces the rate of spread of the pathogen,and is usually expressed both in seedling and in the adult plant growth stages.

4. Adult plant resistance. The resistance which is apparent in post seedling growth stages and also termed mature-plant or field resistance. The onset of adult-plant resistance has been reported from the third leaf stage (Anderson, 1966) to after the emergence of the flag leaf (Samborski and Ostapyk, 1959). Since genes for adult-plant resistance may often be effective against a wide spectrum of rust races, Robinson (1976) stated that all adult-plant resistance is of the horizontal type. However, race specificity has been found for several of the adult plant genes for resistance e.g. in stripe rust, see Priestley (1978). Wallwork and Johnson (1984) reported that Cappelle Desprez which has several specific genes for seedling resistance and several for adult-plant resistance, is a durable cultivar. Some of the adult-plant factors appear to be responsible for durable resistance.

2.2.3. Theoretical aspects of non-race-specificity

The recognition of races and resistance genes are possible in the case of genetic interactions between host and pathogen genotypes based on a gene-for-gene system. Typical race-specific and typical non-race-specific resistance form the extremes of a continuum; the size of the race-specific effects depend on the size of the gene effects. This is well supported by the wheat-stripe rust system where the effects of the many resistance genes vary from small to large (Röbbelen and Sharp, 1978). Zadoks (1972), studying various wheat cultivar-stripe rust race combinations, observed a continuum between instances of near non-race-specific resistance to instances of clear race-specific

resistance. Parlevliet (1985) stated that non-specific resistance can arise from two genetic systems : when the host and the pathogen genes have small effects and operate on a gene-for-gene basis and when the host and pathogen genes, whether of small or large effect, do not operate on a gene-for-gene basis. Genes with large effects operating in a non-specific way are believed not to exist in host-specialized pathogen systems. In cereal-cereal rust systems resistance genes with large effects most likely operate on a gene-for-gene system and are race specific. Resistance genes with small effects, too small to study them individually (polygenes), give a non-race-specific pattern whether they operate on a gene-for-gene system or not.

2.2.4. Expression of non-specific resistance

In recent years there has been increasing interest in resistance that reduces the rate of epidemic development by reducing the number and rate of growth of pustules, the final pustule size, the spore production and confers a longer latent period (reviewed by Parlevliet, 1979, 1983, 1985). The effect is often called slow rusting and resistance is thought to be nonspecific. However, slow rusting is simply one manifestation of resistance and is not necessarily nonspecific. Specific genes conditioning intermediate levels of resistance or conditioning adult-plant resistance also reduce the rate of epidemic development because of reduced spore production and possibly longer latent periods as well (Parlevliet, 1977a).

2.3. General expression of resistance

Since 1971, much more work has been done on more complex types of resistance such as slow rusting, and the number of papers reporting genes having small effects has increased greatly. Because individual genes have small effects in such resistance, it is much more difficult to identify them and to determine whether a gene-for-gene relationship exists. However, for barley leaf rust (*Hordeum vulgare; Puccinia hordei*), Parlevliet (1977a) obtained evidence for a differential interaction and suggested that minor genes operate on a gene-for-gene basis. Exceptions to the gene-for-gene hypothesis have been reported, but can usually be resolved if the hypothesis is described in terms of the interaction of gene products rather than the interaction of genes.

2.3.1. Temperature sensitivity

Temperature can affect the expression of many genes; some genes become in effective at high temperatures Green *et al.* (1960), while others become ineffective under low temperatures Lewellen and Sharp (1968). The wheat cultivar P.I.178383, which has minor additive genes for resistance to stripe rust, showed greatest resistance at high temperatures, while other minor genes confer more resistance at a lower temperature (Lewellen and Sharp 1968). Sharp *et al.* (1976) selected plants among infected plant population grown at different temperatures that were resistant over a wide range of temperatures . Evidently, these differences in sensitivity to temperature are due to diverse physiological functions of the minor genes concerned (Röbbelen and Sharp, 1978).

2.3.2. Gene interaction

A cultivar with two genes, each determining a different level of resistance, usually exhibits the rust reaction phenotype of the most effective gene, and the gene conferring the least resistance is masked. The most effective gene is epistatic to those that condition a less resistant reaction. Furthermore, a cultivar with two or more genes will be resistant to all of the rust races to which the genes are effective separately. Sharp *et al.* (1967) found that each of the two cultivars PI178383 and Chinese 166 had a different dominant major gene. Each gene gave a high level of resistance that was largely unaffected by the environment. However, F_2 plants lacking the major genes segregated for additional genes that gave some resistance themselves or acted as modifiers of the heterozygous major genes. Up to three minor genes were accumulated in lines with good levels of resistance, and these gene are generally sensitive to stripe rust.

2.3.3. Inhibitory effects

Genes conditioning host resistance can also be inhibited or suppressed by nonallelic genes (McIntosh and Dyck, 1975; Kerber and Green, 1980; Johnson and Dyck, 1984).

2.3.4. Background effects

The genetic background can affect the expression of specific genes for resistance (Dyck and Samborski, 1974). A gene for resistance may be dominant in one genetic background and recessive in another. Consequently, the susceptible parent in a cross can influence the degree of dominance of a gene (Anderson, 1966). The reaction conferred by a gene may be dominant relative to one race of a pathogen, and recessive to another (Knott and Anderson, 1955; Lupton and Macer, 1962). It has been suggested that this phenomenon may be due to two closely linked genes, the expression of one being dominant and the other recessive (Hooker and Saxena, 1971). Further Johnson (1981b) indicated that the genetic background is important to the expression of durability.

2.3.5. Allelism

When two or more genes are either tightly linked or are alleles (at the same locus on a chromosome). Such tight linkage, or multiple allelism may restrict the number of genes that can be combined into one cultivar (Dyck and Samborski, 1970; Johnson *et al.*, 1986).

2.3.6. Reduced infection rate

van der Plank (1963, 1968a) concluded that horizontal resistance was characterized by a reduced apparent infection rate (slow rusting) but race-specific resistance can also be expressed as slow rusting (Johnson and Tylor, 1972, 1980; Parlevliet, 1979); many genes controlling specific resistance have intermediate effects and produce results similar to slow rusting. Because of difficulties in recognizing non-specific resistance Parlevliet and Van Ommeran (1975) defined partial resistance as that resistance which is manifest by a high infection type and slow rusting. This partial resistance is assumed to be by and large non-specific and was reported in wheat to stem rust (Wilcoxson *et al.* 1974, 1975), leaf rust (Caldwell *et al.*, 1970; Shaner and Finney, 1980), and stripe rust (Hendriksen and Pope, 1971; Dehn 1977, quoted by Parlevliet 1985).

2.4. Durability

Race-specific resistance genes, with few exceptions, are not durable. When they exposed over large areas for long periods, races develop that neutralize the effect of the resistance gene. With genes for specific resistance, an pathotype attacks only those cultivars that carry the matching gene for resistance (and cultivars with no resistance gene) and no other unmatched gene. With polygenes where an isolate overcomes only part of the resistance of a cultivar regardless of whether the resistance is specific or not, so polygenic resistance is much more likely to be durable. A major problem with the concept of non-specific resistance is the difficulty in proving its occurrence. A resistance may appear to be non-specific or effective against all races until a race of the pathogen is discovered to which it is susceptible. In other words, resistance is non-specific until it is found to be specific. To prove non-specificity, every member of the pathogen population would need to be evaluated, which of course is impossible, so proving these resistances are non-specific is limited to the pathogen isolates at hand. To solve this problem, durable resistance has been defined as that resistance that has remained effective in a cultivar during its widespread cultivation for a long sequence of generations or period of time, in an environment favourable to a disease (Johnson and Law, 1973; Johnson 1979, 1981a, 1983). This can be judged only in retrospect, and can be either complexly or simply inherited. Further Johnson (1981b) also indicated that the genetic background is important to the expression of durability. Knott (1988) stated that two reasons why polygenic resistance can be durable for two reasons : firstly, the pathogen can not develop a highly virulent or aggressive race or, if one is produced, it is not competitive, and secondly, a highly virulent or aggressive race, for whatever reason, does not come into contact with the resistance host.

2.5. Assessment

2.5.1. Infection type

The infection type, which is the product of the interaction of the host and pathogen, has been used extensively for the assessment of resistance to many biotrophic disease, such as rusts. Line *et al.* (1974) expanded to a 0 to 9 scale from the basic scale with four classes (Gassner and Straib, 1932, quoted by Line *et al.* 1974) for stripe rust. A 0 indicates immunity, 1 to 3 indicate variation for low infection type, 4 to

6 indicate variation for intermediate infection type, and 7 to 9 indicate variation for high infection type. This expanded scale has the advantage that the data on infection type are better suited for statistical calculations.

2.5.2. Latent period

Variation for latent period (days from inoculation until the first pustule erupted) has been reported for many rust disease(Parlevliet, 1975, 1977b, 1980; Ohm and Shaner, 1976; Martin *et al.*, 1979), including stripe rust (Park and Rees, 1989; Dehne, 1977, quoted by Parlevliet, 1985; and Cromey, 1992a). The growth stage of the plant and the age of leaves can play an important role in that variation. At the heading growth stage, the latent period is usually longest and decreases with the leaves down the stem. Younger and older plants have shorter latent periods, with the seedling stage showing the shortest latent period and smallest differences (Parlevliet, 1975, 1977b).

2.5.3. Pustule size

Because of difficulties in measuring spore production, it is often estimated by pustule size, assuming a close association between spore production and size. Differences in pustule size amongst cultivars have been reported (Ohm and Shaner, 1976; Johnson and Wilcoxson, 1978; Kuhn *et al.*, 1978), including stripe rust in wheat (Cromey, 1992a).

2.5.4. Pustule density

Differences in pustule density among cultivars have been reported by Ohm and Shaner (1976); Kuhn *et al.* (1978), including stripe rust in wheat (Cromey, 1992a). Parlevliet and Kuiper (1977a) found that differences in pustule density varied with the development stage.

2.5.5. Percentage severity

The percent area of plant organs covered with rust pustules can be measured once at the peak of epidemic development or several times from the beginning to the

end of the epidemic. The former is assumed to represent the cumulative result of all infection or resistance during the progress of the epidemic (Parlevliet and van Ommeren, 1975); the latter enables the computation of the apparent infection rate "r" (van der Plank, 1963, 1968a) or the area under the disease progress curve (AUDPC) (Wilcoxson *et al.*, 1975). In general, AUDPC is considered to be a better criterion for measuring slow rusting than r (Rees *et al.*, 1979a, b; Shaner and Finney, 1980), because the r value depended not only on the slow rusting of cultivars, but also on the stage of the development of the epidemic (Parlevliet, 1985). Earliness, interplot interferences and presence of major race-specific genes can change the evaluation of slow rusting (Parlevliet 1985, 1988).

Infection type is applicable to both specific and non-specific resistance. Latent period, pustule size and number are suitable to measure components of resistance in the glasshouse, and longer latent period, smaller pustule size and lower pustule density are associated with slow rusting. AUDPC can be used to measure slow rusting in the field.

2.6. Inheritance of resistance to stripe rust

Rust resistance is a complex character. Person and Sidhu (1971) surveyed 301 papers dealing with rusts ,on all crops, in which, resistance was reported to be due to dominant genes in 292 papers and recessive genes in 36. It was reported to be due to major genes in 291 papers and minor genes in 19. Gene interaction was reported in 43 papers and allelism in 27. Much research has been published on the inheritance of resistance, studied using seedling infection with known isolates, under controlled favourable environmental conditions. The resistance of adult plants in the field is just as important from an epidemiological and breeding standpoint. Many cultivars which are resistance in the seedling growth stage is dependent on major gene, and remain resistant against the same rust pathotypes throughout their entire life cycle; This is termed seedling or overall resistance (Zadoks, 1961). Some cultivars are susceptible in greenhouse tests, but demonstrate extensive resistance at the adult growth stage; and this is termed field or adult resistance (Zadoks, 1961; Purdy and Allan, 1963).

2.6.1. Simple inheritance

2.6.1.1. Recessive genes

Biffen (1905), working with stripe rust, provided the first evidence that resistance to a pathogen could be controlled by resistance factors acting in accordance with Mendel's laws. By crossing susceptible and resistant wheat cultivars, he found that stripe rust resistance reacted as if a single recessive gene was involved (Biffen, 1907,1911,1912). Armstrong (1922) continued Biffen's work and described simple recessive Mendelian factors conditioning resistance in crosses between susceptible and resistant wheat cultivars. Pal (1951) found that resistance to natural infection to stripe rust in the field was controlled by a single recessive gene in the mature plants. Reports of recessive resistance were also found by Favret and Vallega (1953), Sikka *et al.* (1960) and Sawhney and Bakshi (1965). Lupton and Macer (1962) found that resistance of seedlings of cv. Cappelle Desprez to pathotype 8B was due to the recessive allele Yr4a, whereas the resistance of Heines Peko was conditioned by Yr6. Allan and Purdy (1967) determined that recessive alleles controlled resistance of the winter wheat Kansas 587023 as well as Heines Kolbon and Spalding Prolific, using a single spore culture. However, Spalding Prolific only exhibited a clear-cut 1 recessive:3 dominance segregation ratio in F_2, when Gaines was used as the susceptible wheat parent. In progeny tests of Spalding's Prolific x Omar the intermediate type had to be included with the susceptible class to obtain satisfactory agreement for segregation expected with a single recessive gene.

2.6.1.2. Dominant genes

Dominant monogenic resistance appears to occur much more frequently with stripe rust than does resistance due to single recessive genes. Isenbeck (1931) and Hubert (1932), quoted by Röbbelen and Sharp (1978), reported partial dominance of a single gene for resistance in the F_2 population for stripe resistance in hybrid combinations with Chinese 166 in the greenhouse. On the other hand, Favret and Vallega (1953) showed dominance for the same resistance factor from Chinese 166, using a different pathotype in a field test. Further cases of monogenic dominant resistance have been reported for Cometa Klein (Ghosh *et al.*, 1958), for cultivars Rio Negro, Centeria and La Prévision (Singh and Dhillon, 1963), and for cultivars Andes,

Bowie and Narino 59 (Omar *et al.*, 1970). Lewellen *et al.* (1967) showed that stripe rust resistance is controlled by two types of resistance gene. They crossed a susceptible cultivar with two resistant cultivars, both of which were shown to possess a different dominant major gene for resistance against the pathotypes used. In the homozygous state each gene conditioned a high level of resistance which was largely independent of environment or cultural conditions. Singh and Johnson (1988) ran an experiment to understand the reaction of two European and one Indian cultivar to British and non-British races of stripe rust. Genetic analysis indicated that all three cultivars possess the gene Yr2 and that the European cultivars that possess at least one additional gene for resistance to the non-British races. The data for Heines VII are especially important because of its use as a differential cultivar.

2.6.1.3. Reversal of dominance

Dominance and recessiveness, just as resistance, are not absolute attributes of a host plant, but are only the expression of its specific interaction with a certain pathogen. Accordingly, Lupton and Macer (1962) reported that the resistance gene Yr3c in Minister reacted as a dominant against the weakly aggressive pathotypes 5 and 8, but manifested itself as recessive against the aggressive pathotypes 2B and 8B. The same reversal in dominance could also be shown for the genes Yr3a from Cappelle desprez and Yr6 from Peko (Macer, 1966b). In explanation, the authors pointed out the possibility of a dosage effect. In heterozygous plants a single allele could be sufficient for the expression of resistance against a weak pathotype but not effective against a strong one. The alternative explanation that resistance, in such cases, is due to two closely linked genes, one acting as dominant against one pathotype and the other acting as recessive against the other pathotype, has not yet been demonstrated for stripe rust; examples of this have been described only for maize rust, *Puccinia sorghi* (Hooker and Saxena, 1967). Additional evidence for a dosage effect was indicated by the results of Lupton and Macer (1962). They reported the recessive Yr3c gene was expected to segregate with 25% resistance lines in the F_2 population for the progeny of Minster x Cappelle Desprez. However, in the F_2, 167 plants were found to be resistant and only 70 susceptible. One can also hypothesize that the Yr3c gene from Cappelle Desprez which alone confers no resistance against the 2B-pathotype, does complement the effect of the Yr3c gene from Minister, resulting in an effective resistance of the heterozygous individuals. The reversal

in dominance was also seen from seedling to adult plant in Favret and Vallega (1953). Changes in dominance were also reported with stem rust where a gene behaved as recessive in the seedling stage and as a dominant in a population of older plants (Hooker, 1967).

2.6.1.4. Mapping of resistance genes

The allocation of resistance genes to individual chromosomes is difficult for stripe rust even when the required aneuploids are available, because of the significant environmental influences on the infection phenotype with this pathogen. Singh and Swaminathan (1959) crossed the 21 monosomic of Chinese Spring with the resistant cultivar Cometa Klein and tested the F_2 as seedlings in the greenhouse with the pathotypes H and 13. They postulated a recessive gene on each of chromosomes 4A (IV) and 6A (VI) operating against pathotype H and another gene on chromosome 5A (IX) conditioning resistance to pathotype 13. Using the same technique Macer (1966a, b) showed that the dominant Yr1 locus of Chinese 166 was on chromosome 2A (II). In substitution lines (after replacement of a single chromosome pair of Chinese Spring with a the homologous chromosome pair from Thatcher). Stubbs (1966) located one dominant resistant gene of Thatcher on chromosome 2B (XIII). This was later confirmed by Johnson *et al.* (1969) to be Yr7. Welsh *et al.* (1964), however, found that none of the single substitution lines of Thatcher gave full resistance. Their results showed that chromosome 2A (II), 5B (V) and 6B (X) all contained factors contributing to resistance in different degrees. At a relatively low temperature the chromosomes 3B, 5B and 6B were effective, while at a relatively high temperature resistance was even more accentuated by 2A, 3B and 5B. Almost all of the resistance genes determined for individual wheat chromosomes so far have occurred exclusively on the genomes A and B (Singh and Swaminathan, 1959). Stripe rust resistance associated with the D genome has been found only for Compair (Riley *et al.*, 1968a, b). Law *et al.* (1978) presented a review of genetic control of resistance to stripe rust among a series of varieties having a history of durable resistance to this pathogen. Among these varieties the major contribution to adult plant resistance has been shown to be determinate by a translocated chromosome $5B^S$-$7B^S$ on which at least three genes affecting different components of resistance have been identified. They also described the manipulation of this chromosome in breeding programmes.

2.6.1.5. Specificity in resistance

In discussing this topic, the results in general present valuable information on the genetic basis for stripe rust resistance in crosses between resistant and susceptible cultivars. They, however, do not give any information on the relationships of resistance genes present in different cultivars. Zadoks (1961) proceeded from the theoretical position that Person (1959) had proposed. He ascertained the reaction of 17 mature field-grown wheat cultivars to 15 different pathotypes and classified them for stripe rust resistance using B, U, X, L, M and T as gene symbols. Lupton and Macer (1962) and Macer (1964, 1966a) introduced Yr (Yellow rust) in order to designate genes for resistance to *P. striiformis.* They evaluated an extensive number of progenies from intervarietal crosses using at first four and then an additional three pathotypes of yellow rust. Their results showed a preponderance of independent and mainly dominant genes for resistance.

Since the 1930's, it has been known that pathotypes differ in pathogenicity towards individual wheat cultivars, as well as to related species and genera (Johnson, 1988). This specificity operates according to the gene-for-gene hypothesis, with "Yr" genes proving to be race-specific (Johnson, 1988). Not only there is specificity in seedlings, but specificity also exists in adult plants (Johnson and Tylor, 1977; Priestley, 1978; Zadoks, 1961). Lupton and Macer (1962) designated four independent loci (Yr1 to Yr4) controlling seedling resistance, including three proposed alleles at the Yr3 locus and two at the Yr4 locus. Macer (1966a) added a further three loci, Yr5 to Yr7. The gene Yr8 was introduced to wheat from *Aegilops comosa* (*Triticum comosum*) by Riley *et al.* (1968a). Macer (1975) designated a gene present in lines carrying the wheat-rye translocation chromosome IBL/1RS as Yr9 and a gene in the cultivar Moro as Yr10. Genes Yr11 to Yr14 were designated by McIntosh (1986) and controlled a set of race-specific interactions observed in adult plants of several cultivars of wheat to isolates of yellow rust collected in the United Kingdom. The gene Yr15 was derived from *Triticum dicoccoides* and is effective in seedling stage (Gerechter-Amitai *et al.*, 1989). The gene Yr16 controls adult plant resistance in Cappelle Desprez and was identified by the use of chromosome substitution and the development of homozygous recombination lines (Worland and Law, 1986). The genes Yr1 to Yr14 are all known to be race-specific but as yet there is no evidence for race specificity of Yr15 and Yr16. Many other specificities

are likely to be controlled by genes other than those so far designated (Knott, 1989). Stubbs (1985)numbered race-specific host differential cultivars from 0 to 15. For the numbers 1 to 15 these corresponded approximately with the named genes, but for the numbers 11 to 15 the specificities were different from those designated by the genes Yr11 to Yr15. The genes Yr5 and Yr7 are usually dominant and both are located on the long arm of chromosome 2B. Johnson and Dyck (1984) tested F_1 plants from crosses of *Triticum spelta* (Yr5) with Thatcher and Lee, both of which posses Yr7, with race 43E138 which possesses pathogenicity for Yr7 but not for Yr5. The F_1 with Lee was resistant but that with Thatcher was susceptible despite the presence of heterozygous Yr5. In the case of the *Triticum spelta* by Thatcher cross, segregation of susceptible plants occurred in the F_2 and F_3 generations when tested with a race lacking pathogenicity for both genes. Johnson and Dyck (1984) suggested that Thatcher may possess a dominant inhibitor of the gene Yr5. There were no susceptible plants in an F_2 of 200 plants or an F_3 of 200 families from the cross of *T. spelta* (Yr5) with Lee (Yr7), suggesting that Lee does not possess the inhibitor and supporting the evidence of linkage or allelism of these genes (Johnson *et al.*, 1986). Wellings *et al.* (1988) showed that phenotypic effect of YrA (an undescribed source of resistance) varied when exposed to contrasting light intensities in the post-inoculation phase.

2.6.1.6. Multiple alleles:

Just as with the dominant and recessive manifestations, the number of genes in a host population can only be determined with help of, and in dependence of, the available pathogen cultures. The first information on multiple alleles concerned the gene Yr5 according to later nomenclature (Macer, 1966b). Similar types of allelic series were then determined by Lupton and Macer (1962) at the Yr3 and Yr4 loci of wheat. A corresponding situation was established for barley with the Ps2 and Ps3 loci (Bakshi and Luthra, 1971). In all cases, such finding could also be due to a series of very closely linked genes; but since the lack of recombinant was deduced from the usual analysis of several hundred meiotic products only, the actual status is largely of theoretical interest. Much more significant was the finding that host cultivars which are not directly related quite frequently contain the same gene for resistance (Lupton and Macer, 1962). It is, however, always possible in such cases that appearing pathotypes may produce a differential reaction with the same host genotype. Thus the grouping of various sources

of resistance that carry the same allele can only be considered as a tentative or preliminary description.

2.6.1.7. Pyramiding

Combining race specific resistance genes, whether seedling genes or adult plant genes, has not been successful in controlling stripe rust in climates such as that in Britain (Johnson, 1988) where the environment is favourable to the pathogen in most years. It might however be successful in those climates which are less favourable to the pathogen. The International Maize and Wheat Improvement Centre (1981) reported that the pyramiding of primarily specific genes in breeding for resistance to stem rust of wheat has been reasonably successful in several countries. There is an increasing consensus that all types of resistance must be utilized in the development of a breeding strategy to produce cultivars with stable rust resistance (Dyck and Kerber 1985).

2.6.1.8. Artificial limit and intermediate monogenic expression

Many authors seemed to have started from what had to be proved, an explanation according to Mendelian rules is admitted a priori and the results are interpreted accordingly. This is true for all disease where quantitative differences play a role and many authors arbitrarily have fixed an artificial limit between susceptible and resistance F_3 families or have drawn conclusions as to the number of gene solely on the basis of F_2 data. Armstrong (1922) first spoke about recessive inheritance in a cross of susceptible and resistant lines, but with more consideration in later generations it was clearly indicated to be an intermediate expression of a monogenic resistance. Macer (1966b) adduced a similar example with the Yr1 gene in Chinese 166, which by superficial rating was designated as dominant in action, but with further investigation the revised designation of the observed reaction type thus fitted a 1:2:1 segregation ratio. Allan *et al.* (1966) found an intermediate expression in a cross of moderately resistant cultivars in a field test. Similar results in the greenhouse were reported by Allan and Purdy (1967). In the publication of Allan and Purdy (1970) F_2 progenies with an intermediate infection type were grouped with a resistant or susceptible type in order to arrive at a 15:1 ratio. Biffen (1905, 1907) and Armstrong (1922) considered all infection type O to belong to susceptible class, while other authors (Singh and Swaminathan,

1959; Omar *et al.*, 1970) additionally included type I-II into resistant category. Thus, it is easy to see why the first authors mainly considered resistance to be recessive, while the latter most frequently established the dominance of resistance. Therefore, the rule is adopted in most publications that the correctness of the infection type grouping must further be checked by F_3 segregation ratios (Lupton and Macer, 1962; Allan *et al.*, 1966).

2.6.1.9. Spectrum of pathotype effect on Mendelian classification

The identification of resistance genes in the host can be greatly influenced by the prevalent spectrum of pathotypes. McRostie (1921) demonstrated that two pathogen cultures each reacted in the F_2 to give 3:1 ratios, while in a mixture of the two, the same population exhibited 9:7 ratio. Thus, in field investigations, or by use of heterogenous uncontrolled spore mixtures, single Mendelian distributions can become masked or even completely indiscernible.

2.6.1.10. Stage specific differences

Various stages of plant development can affect the components of resistance. Stubbs (1968) observed that the first leaf was susceptible but that the second leaf showed moderate resistance when inoculated at a later date. However, if the tip of the secondary leaf was inoculated concomitantly with the primary leaf, the rust reaction was the same for both leaves. Stubbs (1968) explained this as being due to the influence of the endosperm, considering that the period of the observed shift in resistance correlated especially well with the duration of physiological dependence of wheat seedling on endosperm functions, known from other experiments (Friend, 1966). Similar organ-specific differences in resistance have been shown for later stages of plant development. Zadoks (1961) reported differences in primary leaves, stem leaves, stems and heads. There is little doubt that such organ-specific resistance reactions are under genetic control (Röbbelen and Sharp, 1978).

2.6.2. Complex inheritance

In total, the phenotypically recognisable resistance of a host plant is undoubtedly nothing less than the final expression of a complex of physiological processes, in which

the resistance gene controls only one point in a chain of events (Hooker and Saxena, 1971).

2.6.2.1. Digenic expression

Griffey and Allan (1988) studied the 14 sources of stripe rust resistance represented by near-isogenic lines developed in stripe rust susceptible Lemhi 53. Plant reactions of the lines were evaluated in F_1, F_2 and BC1 populations from resistant by susceptible crosses and in F_1 and F_2 populations from resistant by resistant crosses. In glasshouse and field studies they found both seedling and adult plant resistance genes. Segregation patterns from resistant by susceptible crosses suggested monogenic or digenic control of plant reaction to the pathogen with digenic expression more common in the field.

2.6.2.2. Complementary effects

Inheritance of resistance from oligogenes involves additive genes as well as other types of gene interactions. Allan *et al.* (1966) indicated two recessive resistance factors with complementary effects demonstrated in the an F_2 distribution. In contrast, their F_3 greenhouse results in another cross could be interpreted either as digenic complementary control or as hypostasis for resistance. Bahl and Kohli (1960) also found two dominant complementary factors for the high grade seedling resistance. Especially obvious was the complementary effect of factors for resistance in the cultivars Hope and Timstein. Both are very susceptible; but the F_1 from the cross between them was resistant in the field to all pathotypes in the Netherlands. Correspondingly, Stubbs (1966) reported that none of the 21 substitution lines of these parents into Chinese Spring was resistant to the Falco pathotype.

2.6.2.3. Epistasis

Sikka *et al.* (1960) described another mode of genetic interaction. They crossed a resistant cultivar to a susceptible one. In the F_1 all combinations expressed resistance as dominant and the F_2 showed a segregation of 13 resistant : 3 susceptible. They explained these results as being due to a recessive gene "s" directly conditioning

resistance and a dominant inhibitor "I" which suppressed susceptibility. With this hypothesis, the genotype of the resistant parent was considered as ssII and of the susceptible parent as SSii. In a similar manner, though with reversed symbols, Bakshi and Sawhney (1965) interpreted their greenhouse results. Similar epistasis relations can be seen in the publication of Omar *et al.* (1970). A diallel cross of 4 resistant and 3 susceptible was evaluated, and ratios of 3R:13S, 12R:4S were found.

2.6.2.4. Additive effects

Two or more independent or modifying factors were found with increasing frequency in later analyses of crosses (Bhullar *et al.*, 1967), because the first encouraging results prompted plant breeders to incorporate as many of them as possible in their breeding material. For example three dominant factors in the cultivar Bonza by Omar *et al.* (1970). Consequently, it became evident that not only the resistant parent contributed to resistance in the progeny but also combinations of recessive factors (Rudorf and Job, 1934, quoted by Röbbelen and Sharp, 1978). More frequently, similar cases occurred ,of course, in crosses between various moderately susceptible parents (Allan *et al.*, 1966).

2.6.2.5. Mendelian classification for more than two factors

In genetic analyses of stripe rust resistance, hypotheses based on more than two factors usually are more or less questionable because of the phenotypic instability of the reaction. Pal *et al.* (1956) proposed the presence of one independent dominant gene and two complementary genes; to each of the last two factors was ascribed only a small effect if alone, but both acting together were thought to be able to express full resistance. It, therefore, appears doubtful that these results are due solely to selected grouping of the reaction classes to fit the desired segregation ratios.

2.6.2.6. Transgressive segregation

In progeny tests, in which several genes segregate simultaneously, gene analysis is difficult not only because of the expected large number of different phenotypes. Much more significant is the occurrence, in many cases, of a large number of intermediate

gradations of resistance, instead of a sharp split between resistant and susceptible reactions. Often these intermediate plants also exhibit a hypersensitive type of reaction. Additionally, such intermediate reactions are easily influenced by the environment, especially temperature, to an extent that they may be considered at one time as resistant and at another time as susceptible (Isenbeck, 1931, quoted by Röbbelen and Sharp, 1978).

It was reported that resistance higher degree than in either parent can be selected in the progeny from crosses between susceptible or moderately resistant parents (Krupinsky and Sharp, 1979; Wallwork and Johnson, 1984). These two papers show that transgressive segregation for resistance to stripe rust can be selected in the progeny of many different wheat crosses. It could therefore be a useful way of increasing resistance by crossing among locally adapted wheats, rather than trying to transfer durable resistance. But transgressive segregation for resistance could arise from interactions or additive effects or race-specific genes, or from the transfer of race-specific genes from a suppressive to an expressive backgrounds (Wallwork and Johnson, 1984). It could be also arise from the accumulation of resistance genes of the type associated with durable resistance (Johnson, 1988). Certain wheat varieties may posses major genes for resistance against specific pathotypes and at the same time carry minor genes that determine the reaction to other rust isolates (Volin and Sharp, 1969). Crossing between two parents of moderate resistance can result in transgression for greater resistance (Lewellen et al, 1967; Lewellen and Sharp, 1968; Sharp, 1972; Lupton and Johnson, 1970). Pope (1968) could probably distinguish 20 polygenes in the 28 cultivars of wheat he investigated. They were all additive in action, but showed clear differences in their effective contribution to resistance. He attempted to obtain higher degrees of resistance by combining all the available small effects. Often a significant number of resistant plants occurred only after several generations of selfing, inoculation and repeated selection of the most resistance plants (Sharp *et al.*, 1976), indicating the presence of many genes conditioning resistance. Since major genes were excluded from the parental materials, the added gene increments could be readily followed. Zwer and Qualset (1991) intercrossed four spring wheat cultivars which differing in origin and reaction in the seedling stage to one race of stripe rust. They examined F_1, F_2 and F_3 generations in the greenhouse. They found digenic and transgressive segregation in all crosses. Grama *et al.* (1984) intercrossed seven selections of wild emmer with a minor

effect gene cultivar. The transgressive segregation towards resistance in the F_2 observed in all the combinations indicates that additive gene action for resistance indeed occurs in wild emmer.

2.6.2.7. The possibility of the detection of adult plant resistance genes in the seedling growth stage

Allan *et al.* (1966) analyzed 8 different progenies of wheat and determined 2 complementary or epistatic factors for resistance in greenhouse tests using seedling plants at the 1 or 2 leaf stage. However, classification of the same progeny in the field 8 days before heading indicated, in most cases, 3 loci. The supplementary factor for resistance in the mature plant stage apparently was derived from the susceptible parent. By contrast, loci conditioning resistance in the field were generally not detectable in greenhouse tests. But there is evidence that some adult plant resistance genes are detectable in seedling.

2.6.2.8. Is adult plant resistance certainly non-specific?

The work of Manners (1950) and Zadoks (1961) demonstrated that resistance developed by plants after the seedling stage could be race-specific. But as many other publications have suggested that field or adult resistance is race non-specific; a claim valid only so long as no corresponding virulent pathotype is found. The wheat cultivar Joss Cambier, in spite of its high grade adult (field) resistance, showed a considerable increase in rust attack in England for the first time in 1971 (Priestley and Doling, 1974), and also specificity for other cultivars (Priestley and Doodson, 1976). Such establishment that certain type of field resistance can be overcome by certain pathotypes, does not necessarily signify that pathotype non-specific components of resistance do not exist. Even by genetic means, pathotype non-specific resistance cannot simply be identified by the presence of minor genes of polygenes *per se.* Lupton *et al.* (1971), therefore, began basic inheritance studies on such situations of uniform resistance by analysing the results of diallel crossing with the same cultivars. Their results indicated that pathotype independent-resistance, present in their material, was not necessarily determined by polygenes, since lines showing the resistance reaction of the better parent appeared more frequently than expected.

2.6.2.9. The inheritance of adult plant resistance

With the increased interest in horizontal or general resistance, minor gene, adult plant resistance, slow rusting, tolerance, etc., stimulated by van der Plank (1963), many more cases of complex resistance have been reported. Since these types of resistance have more complex inheritance, they are more difficult to analyze genetically, and special methods are often necessary to measure them: measurement of latent period, pustule size, spore production, or area under the disease progress curve.

Wilcoxson (1981) reviewed slow rusting in cereals, finding that most studies investigated the progenies of crosses between slow and fast rusting line were investigated. The segregation patterns were quantitative in nature, and transgression was observed regularly. In wheat infected with *Puccinia striiformis*, minor genes have been found that additively reduced the infection type. These minor genes appear to be temperature-sensitive and to act independently of the race-specific major genes (Pope, 1968; Lupton and Johnson, 1970; Sharp and Volin, 1970; Hendriksen and Pope, 1971; Sharp *et al.*, 1976; Röbbelen and Sharp, 1978; Krupinsky and Sharp, 1979). Minor or polygenes apparently are as much a part of the resistance complex of cereals to rust as the major genes are. It should be mentioned that slow rusting can be due to: a) race-specific, adult plant or incomplete resistance; b) a low frequency of pathogenicity for a race specific gene in a mixed population of races, so that the cultivar possessing the gene receive a low frequency of matching infection; c) slow rusting of a durable, apparently race-non-specific type.

2.6.2.10. Polygenes

Hypersensitivity and distinct infection types, as a rule, are conditioned by one or a few major genes. Quantitative gradations in resistance, which can be measured in terms of rates or degree of host infection or spore yield of the pathogen (Johnson and Tylor, 1976a), generally arise from the additive effect of the phenotypically less striking reactions of a larger number of resistance genes (polygenes). The number of reported cases of polygenic systems conditioning stripe rust resistance is small in comparison to the number of publications on oligogenic inheritance. Complex resistance to stripe rust of wheat has frequently been reported (reviewed by Röbbelen and Sharp, 1978).

Typically, the number of resistant progeny in the early generations of crosses is low, there are many intermediate types of reaction, and there is no distinct separation between resistant and susceptible plants. Resistance is often affected by environment, particularly temperature. Lewellen *et al.* (1967) and Lewellen and Sharp (1968) studied F_1, F_2, F_3 and testcross populations from several crosses at two temperature profiles, 2°C night / 18°C day and 15°C night / 24°C day, in a growth chamber. The results indicated that resistance was controlled by two types of genes. In addition to dominant major genes (in one case complementary dominant genes), there were several recessive minor genes. In cultivar Rego the minor genes were effective at lower temperatures, but in P.I.178383 they were effective at higher temperatures. The minor genes from the two cultivars could be combined to give good resistance at both temperature profiles. The F_2 plants, however, lacking major genes or heterozygous for them, disclosed additional segregation of factors for resistance. These factors could be effective alone or act as a modifiers, intensifying the resistant reaction of the heterozygous major gene. The results also indicated the presence of a large number of such hypostatic minor genes; at least three such modifiers segregated in the F_2 and in selected F_3 lines from the cross Lemhi x PI178383. With the F_3 progeny derived from fully susceptible F_2 plants from that cross, it was further demonstrated that in the absence of a major gene, minor genes can be combined to give usable, effective resistance. One, two and three minor genes conditioned infection types of 2, O or even OO at a relatively high temperature or of types 3, 2 and O, respectively, at a lower temperature (Sharp and Hehn, 1967; Sharp, 1968). These authors showed one or more temperature-sensitive, recessive genes provided for greater resistance levels, but at lower temperature. Manners (1950) also described temperature sensitivity in stripe rust. Evidently, these differences in sensitivity to temperature are due to diverse physiological functions of the minor genes concerned.

From the cross, Itana (susceptible) x P.I.178383, Sharp and Volin (1970) selected lines carrying 1, 2 or 3 minor genes. At 2°C / 18°C they gave infection types 3, 2, and 0, respectively, on a 0-4 scale and at 15°C / 24°C they gave infection types 2, 0 and 0⁻. The lines gave similar reactions to 11 isolates of stripe rust, and the authors suggested that the resistance was nonspecific.

Krupinsky and Sharp (1978) studied the parents and F_1 plants from a 6x6 set of diallel crosses involving six minor gene lines, and from a 7x7 set of diallel crosses

involving three minor gene lines and four susceptible winter wheats. They concluded that general combining ability (additive gene effects) was much larger than specific combining ability, and heritabilities were above 90%. Maternal and reciprocal effects were also significant.

Pope (1968) did field studies on numerous crosses and indicated that resistance to stripe rust was frequently due to a minimum of five genes having small, additive effects. He suggested that a minimum of 20 genes was involved and that they functioned in gene complexes. Pope (1968) considered minor genes not to be resistance genes in the true sense, but to be modifiers in a gene complex which determined the resistance reaction of the host plants.

These findings regarding a lower vulnerability of minor genes to the occurrence of specific pathotypes provided only a tentative indication that the resistance which they conditioned might be basically different from that conferred by major genes. Indeed, a resistance may be only be non-specific until a new gene for virulence or a new race appears in the pathogen population. Nevertheless, there is much in favour of the hypothesis that minor genes are the constituting genetic elements for a horizontal type of resistance as advocated by van der Plank (1963, 1968a,b), and also (Stubbs, 1977).

In England a number of older wheat cultivars are susceptible to stripe rust as seedlings but have maintained adequate resistance in the field. Lupton and Johnson (1970) crossed the resistant Little Joss with the susceptible Nord Desprez. The F_1 plants were severely rusted and only 5% of 2500 F_2 plants showed some field resistance. Their F_3 progenies showed varying degrees of resistance. The authors concluded that resistance was recessive and complex in inheritance.

Wallwork and Johnson (1984) intercrossed Maris Bilbo, Joss Cambier and Nord Desprez which are an susceptible to race 104E137 in the field. Transgressive segregation for resistance occurred in all three crosses. Three of the most resistant lines from Joss Cambier x Nord Desprez showed more resistance than their parents to 12 races. The authors suggested that resistance was recessive and that the number of genes was not large since resistance was accumulated quickly.

Dijk *et al.* (1988) tested 29 older cultivars with durable resistance and eight recent non-durably resistant cultivars in the seedling growth stages and as adult-plants against 12 West-European stripe rust races and against some non-European races in the seedling growth stages only. They concluded winter wheat cultivars released in the Netherlands before 1930 carried durable resistance to stripe rust and cultivars released in the period between 1930 and 1950 often were durably resistant while recent cultivars infrequently showed durable resistance. This durable resistance was not difficult to transfer to new cultivars. They also concluded that as in the early years the selection was done in the field in dependence of the occurrence of the pathogen, which tended towards a mild selection against susceptibility. Gradually the selection became more efficient by introducing the pathogen and using highly susceptible spreader cultivars. In this latter situation selection is more clearly for resistance and the selection pressure is more severe. Parlevliet (1983) stated that the former situation is more conducive to the selection of complex of polygenic resistance, the latter more for the selection of simply inherited resistances due to genes with large effects, which often appear to be of the non-durable type. Thus, the changed procedures for selecting stripe rust resistant winter wheats apparently resulted in erosion of partial resistance. This is in accordance with van der Plank's (1968b) concept of the Vertifolia effect.

Milus and Line (1986a, b) initiated an approach to quantitative data in plant disease. They intercrossed three durable and one susceptible cultivars. They evaluated parental, F_1 (and reciprocal), F_2 and both backcrosses populations in the field for intensity of rustiness. Hayman's generation means were used to analyze data for the mode of gene action. Number of genes and heritability were calculated as well. Estimations based on three quantitative formulae indicated that rust intensity was controlled by two or three genes in Nugaines and Luke. Their results showed resistance in the three cultivars was partially recessive with no maternal inheritance. Epistasis was significant in Nugaines, but most gene action among loci was additive. They did a comprehensive study on quantitative aspects of stripe rust inheritance.

2.6.2.11. Procedures for studying complex resistance

Resistance that is complex in inheritance is difficult to study genetically. Individual genes have small effects and distributions in segregating generations of crosses is

usually continuous. Thus, normal genetic analyses using F_2 populations, F_3 families, and backcrosses are more difficult than for simply inherited resistance. If resistance is measured on an appropriate scale, such as the percent rust intensity in field tests, then quantitative genetic methods can be used. Depending on the experimental design, additive, dominance and epistatic effects, heritability and the number of effective factors can be estimated. Particularly for the number of effective factors, the assumptions made in the analyses are often unrealistic and the estimates are subject to considerable error.

2.7. The constant search for resistance

Race-specific resistance is conditioned by the interaction of specific genes in the host with those in the pathogen. Flor (1955) showed that a cultivar is resistant or susceptible to a physiologic race of the pathogen depending on the genotype for resistance or susceptibility of the host and genotype for virulence or avirulence of the pathogen. This gene-for-gene system seems a logical consequence of the coevolution of a host and its obligate parasite in nature. Johnson (1961) in man-guided evolution showed the pathogen adapt repeatedly to overcome the resistance of new host cultivars. This ability of the pathogen to generate new virulent forms necessitates an ongoing search for new sources and types of resistance that can be utilized in breeding for disease resistance. In a breeding programme, it is desirable to understand the genetic variation in both host and pathogen; knowing how the resistance is expressed should make it easier to design appropriate tests and follow them through a breeding programme. It can be dangerous to breed for resistance without any knowledge of the genes carried by the parents. Because of genetic variation in rust, breeding for resistance is a on-going task.

2.8. Sources of genetic disease resistance

Due to continual evolution of rust pathogens to form new and virulent biotypes, there must be a constant search for germplasm possessing resistance to stripe rust within cultivated and primitive cultivars (e.g. Sharp *et al.*, 1976). Because of great genetic diversity of primitive cultivars and land races, they are more likely sources of new resistance genes than material derived from breeding programmes. The known genes for rust resistance in most of the cereal crops are now being utilized on an international

scale, consequently, few new genes for resistance can be expected from advanced breeding stocks. Additional sources of new resistance genes may be found in alien and related cultivated species, or induced mutation.

2.9. Quantitative genetics

Genetic variability is essential for the success of a plant breeding programme. Quantitative genetic research measures this variability and examines the action, interaction and linkage relationships of genes. Phenotypic statistics (mean, variances, covariances and measures of skewness and kurtosis) may be employed for this purpose (Breese, 1971; Mather and Jinks, 1977, 1982).

Information of total phenotypic variation that is conditioned by the joint action of genetic and environmental forces is very important for the breeder in making decisions for the allocation of resources and expected response to selection. For estimation of components of variance, we will consider mating designs that develop progenies for evaluation. All mating designs include progenies that involve relationships among relatives having known genetic components of variance.

2.9.1. Diallel analysis

Diallel crossing is one type of important mating system enjoying universal application in genetics specially plant breeding. The diallel mating design has been utilized extensively to obtain genetic variance estimates (Moll and Stuber, 1974). A full diallel cross consists of all possible crosses between a number of varieties (lines). If there are p varieties, there will be p^2 combinations, consisting of p parent and $p(p-1)$ crosses. This includes reciprocal crosses which may be differentiated by matemal or paternal effects. As p increases, p^2 may become impossibly large. For this reason, many methods have been developed for examination of partial diallel crosses. This mating design can fit into at least two genetic models : one essentially developed for a single diallelic gene, but extended to many genes under restrictions (Jinks, 1954; Hayman, 1954a, b, 1957, 1958b, 1960c; Mather and Jinks, 1977). The other is a more general model (Griffing, 1956). Gilbert (1958) has listed the assumptions needed for diallel analyses, following Hayman's (1954a, b) method. These are : diploid segregation; no

reciprocal differences; independent action of non-allelic genes; no multiple allelism; alleles independently distributed at random between the parents. Reciprocal differences and polyploidy may readily be allowed for, but the other assumptions are more problematic. The analysis of Griffing (1956), being made in terms of combining ability, has less demanding genetical assumptions, and is probably to be preferred. This information is sufficient for plant breeding.

2.9.1.1. Griffing's method

Griffing (1956) described four possible experimental methods : (1) included parents and both sets of F_1's; (2) parents and only one set of F_1's (no reciprocal); (3) both sets of F_1's are included, but not the parents combinations; and (4) only one set of F_1's but neither parents nor reciprocals are included. Regard to the sampling assumptions, Griffing (1956) considered the different mean-squares necessary for the fixed and random models. In an analysis based on a fixed model, one is concerned with comparisons of the combining abilities of the actual parents used in the experiment and the identification of superior combinations. In an analysis based on a random model, inferences are to be made about the population from which the parents were sampled and these inferences are made from estimates of components of variance. In Griffing's method, genetic variance between crosses is partitioned into two components. The first is variance of general combining abilities (σ^2_{gca}) which contains only additive genetic variance and epistatic interactions of additive x additive, and so on. The second component is variance of specific combining abilities (σ^2_{sca}) 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 line involved." Estimates of (σ^2_{gca}) and (σ^2_{sca}) are derived from covariances of full- and half-sibs (Kempthome, 1957). Formulae for estimation of covariances of full- and half-sibs are :

$$Cov_{F.S.} = [(1+F)/2]\ \sigma^2_A + [(1+F)/2]^2\ \sigma^2_D + [(1+F)/2]^2\ \sigma^2_{AA} + [(1+F)/2]^3\ \sigma^2_{AD} + [(1+F)/2]^4\ \sigma^2_{DD} + \ldots$$

$$Cov_{H.S.} = [(1+F)/4]\,\sigma^2_A + [(1+F)/4]^2\,\sigma^2_{AA} + [(1+F)/4]^3\,\sigma^2_{AAA} + \ldots$$

where

σ^2_A = additive genetic variance of a random mating, non-inbred (i.e. panmictic)

σ^2_D = dominance genetic variance of a panmictic

σ^2_{AA} = epistatic interaction of additive x additive type

σ^2_{DD} = epistatic interaction of dominance x dominance type

σ^2_{AD} = epistatic interaction of additive x dominance type

σ^2_{AAA} = epistatic interaction of additive x additive x additive type

F = inbreeding coefficient amongst the parents

Variance of g.c.a. (σ^2_{gca}) and s.c.a. (σ^2_{sca}) may then be calculated as (Kempthorne, 1957):

σ^2_{gca} = $Cov_{H.S.}$(half-sib), because half-sib sets are being compared

σ^2_{sca} = $Cov_{F.S.}$(full-sib) - 2 $Cov_{H.S}$

There are two models : I. fixed and II. random. In model I the parents are the population, whereas in model II the parents are a sample from a population. The distinction between the two models is important not only for analysis of variance but for the information derived from the analysis of variance. Because the parents are the population for model I, estimation of components of variance is not appropriate but estimation of the effects of each pair of parents for specific crosses (SCA) and for all crosses that include a common parents (GCA) is appropriate and valid. GCA and SCA effects are more informative than components of variance for the model I analysis (Hallauer and Miranda, 1981). Also, estimated effects are applicable only for the parents included and would be different if the parents were tested with a different group of parents. For the model II analysis, estimation of the components of variance is of prime interest. to relate the variance components to types of gene action in the reference population, it is helpful to write the expected mean square in terms of genetic relationships of relatives and translate from covariances of relatives to the genetic components of variance.

2.9.1.2. Hayman-Jinks' method

Hayman and Jinks, in a series of papers from 1953, 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 the average degree of dominance and hence dominance type (partial, complete, or overdominance); the relative proportion of dominant and recessive genes in the parents, and genetical diversity among the parents. In Hayman's method, some of the assumptions may be violated with impunity; others remain essential, especially that of random distribution of genes among parents (Baker,1978). Some may be tested using features of diallel itself such as analysis of variance of W_r-V_r. Hayman (1954b), following Mather (1949), showed with a graphical analysis how to approach the problem of dominance. If the assumptions are met, it can be shown that for a *p* x *p* diallel that the variance of all the offspring of the i_{th} parent, i.e. of the i_{th} complete array , V_r is simply related to the covariance between these offspring and their non-recurrent parents, W_r. In fact,

$$W_r = V_r + 1/2\ V_A - \Sigma 2pqd^2$$
$$= V_r + 1/2\ V_A - 1/(2pq)\ V_D$$

if gene frequencies at all loci are the same. A plot of W_r against V_r will therefore display the relationship between V_A and a function of V_D. If dominance is uniform, linearity should be seen. Furthermore, the parabola $W_r^2 = V_P * V_r$, delimits the area in which the results may occur (where V_P is variance of parents. The position of the pairs of points W_r, V_r relative to the regression line of unit slope through the mean $\bar{V}r$, $\bar{W}r$ indicates the nature of dominance. The line through the origin indicates complete dominance : the greater the intercept with the abscissa, the more the tendency to overdominance. Gilbert (1958) noted that W_r and V_r do not fulfil the basic assumptions of independence and normality required for regression analysis, so that departure from simple linearity, used by Hayman to justify ranking the dominance of parents by the sum W_r+V_r, may not provide this justification. He suggested that the test of homogeneity of variance might be applied to V_r.

In the presence of non-allelic interactions and/or correlated gene distributions, this linear relationship is lost. Certain types of disturbance cause graphical distortion in characteristic ways which may permit their detection. For example, complementary interaction bends the W_r/V_r line concavely upwards, while duplicate interaction has the reverse effect (Mather and Jinks, 1977, 1982).

By this method we can estimate four genetic components as follow :

$$D = \sum 4uvd^2 \qquad H_1 = \sum 4uvh^2$$

$$F = \sum 8uv(u-v)dh \qquad H_2 = \sum 16u^2v^2h^2$$

where D is twice the panmictic 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. Σ signifies a sum over all segregating loci. Equations which estimate D, H_1, H_2 and F come from one or more combinations of V_P (variance of parents entering the diallel), $\bar{Vr}$ (mean variance of parental arrays), (mean of covariances between the family means within each array and the phenotypes of their respective non-recurrent parents) and $V_{\bar{r}}$ (variance of parental array means). Additive genetic variance (σ^2_A) and dominance genetic variance (σ^2_D) 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 assumptions, 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, we obtain :

$$\sigma^2_A = 1/2\ D \qquad \sigma^2_D = H_1\ (H_R)$$

$$\sigma^2_D = 1/4\ H_2\ (H_R)$$

these estimates show the proportion of genetic variation attributable to additive and dominance genetic variances.

Analysis of variance of array statistics can be done 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. Significance and non-significance of W_r+V_r indicate presence and absence of dominance, respectively, and significance of W_r-V_r shows that the data did not conform adequately to the model due to epistasis and/or correlated gene distributions (Mather and Jinks, 1977, 1982).Some statistics can be estimated from genetical components of variation which will be explained in the materials and methods.

2.9.2. Factorial mating designs

The Design II mating design or Factorial design was described by Comstock and Robinson (1948, 1952). The expectations of males and females for design II are equivalent to g.c.a., and the male x female source is equivalent to s.c.a. of diallel analysis (Hallauer and Miranda, 1981). In this design, because we have two different sets of parents, we have two independent estimates of g.c.a.. Appropriate F-tests can be made to test for the differences among males and among females and for their interactions. Its genetic information is similar to diallel, despite their being different sets of parents for each sex. With increasing number of parents, the number of crosses increases but not as much as for diallel. This is a advantage of this design, especially if one wishes to estimate the genetic parameters of a reference population. The genetic interpretation of the variance components is (Becker, 1984) :

$$\sigma^2_m = COV_{HS(M)} = 1/4\ V_A + 1/16\ V_{AA} + 1/32\ V_{AAA}$$

$$\sigma^2_f = COV_{HS(F)} = 1/4\ V_A + 1/16\ V_{AA} + 1/32\ V_{AAA} + 1\ V_M$$

$$\sigma^2_{mf} = COV_{FS} - COV_{HS(M)} - COV_{HS(F)} = 1\ V_D + 14/16\ V_{AA} + 1\ V_{AD} + 1\ V_{DD} + 30/32\ V_{AAA}$$

where

- σ^2_m, σ^2_f and σ^2_{mf} are variance of male, female and their interaction, respectively
- $COV_{HS(M)}$, $COV_{HS(F)}$ and COV_{FS} are covariance of half sib for male, female and covariance of full sib

- V_A, V_{AA} and V_{AAA} are additive variance and its interactions, respectively
- V_D, V_{DD} are dominance variance and its interaction, respectively
- V_{AD} is additive x dominance interaction
- V_M is maternal variance
- V_E is environmental variation

This design is well-adapted for use in most cross-pollinated species and a few self-pollinated species, such as tobacco, in which an adequate amount of seed is easily produced from each mating. Usually, the costly, time-consuming effort required to produce sufficient seed for replicated evaluation trials has essentially precluded the use of this and similar designs in self-pollinating species, such as small grains and soybean. However, Stuber (1970) has outlined a procedure for estimation of additive, dominance, and additive x additive genetic variance using inbred progeny evaluation. The inbred relatives are generated by bulk selfing of the F_1 progenies in mating designs such as design I and II. These selfed progenies are grown and evaluated in trial similar to those used for non-inbred progenies. Although the progenies evaluated are inbred to varying levels, genetic variances defined for the non-inbred reference population can be estimated with no restrictions on gene frequencies or number of alleles per locus (Fehr and Hadley, 1980). Design II, a factorial design, is essentially a modification of design I (Comstock and Robinson, 1948, 1952). It is used to estimate genetic variance and to evaluate inbred lines for combining ability. Each member of a group of parents used as males to each member of another group of parents used as females. Usually, reciprocal crosses are assumed to be identical genetically, therefore, seed of reciprocals is bulked for planting progeny evaluation tests. In the analyses of variance, male and female designations are purely arbitrary to facilitate the analyses (Fehr and Hadley, 1980).

2.9.3. Generation means analysis

In the study of quantitative traits, important consideration is given to the gene action that control these traits. The generation mean analysis is used to study gene effects by using different generations derived from a cross between homozygous parents. The use of different generation means to estimate magnitude of gene action and the expression of a character to an additive-dominance model was proposed by Cavalli (1952) and has been illustrated by Hayman (1958a, 1960b) and Mather and Jinks (1977,

1982). Generation mean analysis is an application of weighted least squares contrast that estimate relative genetic effects from the means of different generations. The weights are based on the internal variance of each generation. A weighted ANOVA would usually precede it in order to provide significance test and error variance. The most common sets of generations are as follow:

P_1 : the higher scoring parent
P_2 : the lower scoring parent
F_1 : $P_1 \times P_2$
F_2 : F_1 selfed
BC_1 : P_1 x F_1
BC_2 : P_2 x F_1

P_1 : the higher scoring parent
P_2 : the lower scoring parent
F_2 : F_1 selfed
F_3 : F_2 selfed
BCS_1 : (P_1 x F_1) selfed
BCS_2 : (P_2 x F_1) selfed

More complex experiments can be produced by inter-crossing and selfing these generations further (Mather and Jinks, 1982). A bulk of progenies of each generation is evaluated in a replicated experiment. Sufficient sampling of segregating generations is necessary to have a representative sample of genotypes. In parental and F_1 generations no sampling is involved, but F_2, F_3 and backcross generations will be segregating and sample size has to be considered. The resultant internal variances form the bases of the weighing.

Several different possibilities exist for the type and number of generations that can be included in a generation mean experiment. If the two parents, F_1, F_2 and both backcrosses are evaluated, we have six means for comparison. Expectations of each generation can be determined and a weighted least squares analysis made to estimate m, [d], [h], [i], [j] and [l] with a fair degree of precision. We can also make a goodness of fit test (observed means compared with predicted means) to determine the adequacy of the model for m, [d] and [h] to explain the differences among the generation means.

Hayman (1958a; 1960b) defined the base population as the F_2 population (rather than mid-parent in Mether and Jinks' method) resulting from a cross of two inbred lines, which differ by any number of unlinked loci, has expectation of m in term of genetic effect and the following expectations for other generations:

	m	d	h	i	j	l
P_1	1	1	-1/2	1	-1	1/4
P_2	1	-1	-1/2	1	1	1/4
F_1	1	1/2	0	0	0	1/4
F_2	1	0	0	0	0	0
F_3	1	0	-1/4	0	0	1/16
BC_1	1	1/2	0	1/4	0	0
BC_2	1	-1/2	0	1/4	0	0
BCS_1	1	1/2	-1/4	1/4	-1/4	1/16
BCS_2	1	-1/2	-1/4	1/4	1/4	1/16

The general model used is :

$$Y = m + \alpha d + \beta h + \alpha^2 i + 2\alpha\beta j + \beta^2 l$$

where the mean of a generation Y is a function of the mean (m) of all generations in a cross and five coefficients (α, β, α^2, $2\alpha\beta$, β^2) as products of genetic parameters : pooled additive effect [d], pooled dominance [h], pooled interaction between additive effects [i], pooled interaction between additive and dominance [j], and pooled interaction between dominance effects [l]. The base in the model of Mather and Jinks (1982) is m and the expectations of generations will be presented in materials and methods.

A cross producing an F_1 heterozygous for more than one locus can be made in two ways. If the increasing alleles occur together in one of the parents and the decreasing alleles in the other, the genes may being said to be associated. Or if each parent might carry the increasing allele of one or some genes and the decreasing allele of the another or others, the genes being said to be dispersed (Mather and Jinks, 1982). Under the dispersion state the genes tend to balance one another out, so the additive effect is the sum of the additive effects at all loci, taking the sign into account. Similarly the dominant effect is the sum of dominant effects of the individual genes taking the sign into account. But for dominant effect the sign of h does not depend on gene association nor dispersion but on the direction of the dominance itself (Mather and Jinks, 1977). Gamble (1962a, b) used other symbols for gene effects and showed that his symbols can be related with other symbols.

Generation mean analysis has been widely used to estimate the genetic parameters for various characters in many crops. In wheat stripe rust, it was used to study the inheritance of resistance in three durable, high temperature and adult plant resistant cultivars by Milus and Line (1986a).

Rowe and Alexander (1980) clarified computational aspects of the methodology and indicated the generalization to more complex genetic models. The computational presentation by Mather and Jinks does not specifically indicate the general statistical nature of the methodology nor the potential for generalization to more complex genetic models.

Generation mean analysis has some advantages. Because we are working with means (first order statistics) rather than variances (second order statistics), the errors are inherently smaller. We can easily extend generation mean analysis to more complex models that include epistasis, but the main effects (d and h) are not unique when epistatic effects are present. Generation mean analysis is equally applicable to cross- and self-pollinating species. Smaller experiments are required for generation mean analysis to obtain the same degree of precision. It also has some disadvantages which are. An estimate of heritability can not directly be obtained and one can not predict genetic advance because estimates of genetic variances are not available. Cancellation of effects may be a significant disadvantage because, for example, dominance effects may be present but opposing at various loci in the two parents and cancel each other.

2.9.4. Generation variance analysis

Mather and Jinks (1977) also developed a generation variance analysis which allow us to calculate heritability and degree of dominance. Their expectations are as follows:

$$V_{P1} = E_w$$
$$V_{P2} = E_w$$
$$V_{F1} = E_w$$
$$V_{F2} = 1/2D + 1/4H + E_w$$
$$V_{BC1} = 1/4D + 1/4H - 1/2F + E_w$$

$$V_{BC2} = 1/4D + 1/4H + 1/2F + E_w$$

where

E_w : non-heritable component of variation

D : additive component of variation

H : dominance component of variation

F : dependent contribution of d and h over all loci

Estimates are obtained from these expectation as follows:

$$E_w = 1/4\ (V_{P1} + V_{P2} + 2V_{F1})$$
$$D = 4V_{F2} - 2\ (V_{BC1} + V_{BC2})$$
$$H = 4\ (V_{BC1} + V_{BC2} - V_{F2} - E_w)$$
$$F = V_{BC1} - V_{BC2}$$

The dominance ratio can be estimated as $\sqrt{}(H/D)$ and the estimation $F/\sqrt{}(D*H)$ can provide evidence of dominance deviations at different loci. Having only four equations for the estimation of four parameters we obtain a prefect fit solution to them, and we can neither calculate the standard deviation of the estimates of D, H, E, and F, nor indeed can we test the goodness of fit of the additive-dominance model as a whole.

Hayman (1960a) solved the problem mentioned above by using the procedure which is essentially the same method of weighted least squares already used for the analysis of means. The expectations for other generations such as F_3, BCS_1 and BCS_2 which may be needed in Hayman's method are as follows :

$$V_{1F3} = 1/2\ D + 1/16\ H + E_b + 1/n\ V_{2F3}$$
$$V_{2F3} = 1/4\ D + 1/8\ H + E_w$$
$$V_{1BCS1} + V_{1BCS2} = 1/2\ D + 1/8\ H + 2E_b + 1/n\ V_2$$
$$V_{2BCS1} = V_{2BCS2} = 1/4\ D + 1/8\ H + E_w$$

where

V_{1F3} : the variance amongst F_3 lines

V_{2F3} : the mean variance of within lines

E_b : non-heritable variation between families

E_w : non-heritable variation within families

n : number of plant per line

V_{1BCS1} : the variance amongst the selfed backcross with P_1

V_{1BCS2} : the variance amongst the selfed backcross with P_2

$V_{1BCS2} = V_{1BCS2}$: the mean variance of within second selfing backcross with P_1 and P_2, respectively.

2.9.5. Heritability

Heritability is a coefficient of determination indicating what proportion of phenotypic variance is genetic. The mean gen-flow via sexual reproduction (breeding value) depends largely on heritability, and is a major component of predicting genetic advance in selection. The accuracy of prediction would be low, for example, for a trait whose measurements show little genetic variance but a relatively high degree of environmental variance. For parents to transmit characteristics of their offspring in some predictable degree, it is obvious that environmental variance should be low, and genetic variance high, the trait should be only slightly affected by environmental differences but greatly affected by genetic differences. The variation exhibited in any character constitutes the phenotypic variance (V_P). This may be divided into genetic variance (V_G), non-genetic or environmental variance (V_E) and their interaction (V_{GE}). The genetic variance (V_G) is composed of three major components: additive genetic variance (V_A), dominance variance (V_D) and nonallelic interaction variance (V_I). The additive portion of phenotypic variance is of greater importance in the average resemblance between relatives than the dominance portion. Because of this, the relationship between the additive variance the total phenotypic variance is commonly used in predicting the results of selection of variates. There are two meanings of heritability :

$$h^2_{BS} = V_G / V_P \qquad h^2_{NS} = V_A / V_P$$

since V_A is, at most equal, smaller than V_G, it may be called the narrow sense heritability in contrast to the broad sense type.

In a Hayman-Jinks diallel cross heritability is estimable as follows :

$$h^2_{BS} = (1/2\ D + 1/2\ H_1 - 1/4\ H_2 - 1/2\ F) / (1/2\ D + 1/2\ H_1 - 1/4\ H_2 - 1/2 \quad F + E)$$

$$h^2_{NS} = (1/2\ D + 1/2\ H_1 - 1/2\ H_2 - 1/2\ F) / (1/2\ D + 1/2\ H_1 - 1/4\ H_2 - 1/2 \quad F + E)$$

where E is the pooled error mean square from analysis of variance of parents and F_1's.

For a Griffing's diallel, it may be estimated for :

$$h^2_{BS} = (2\ \sigma^2_{gca} + \sigma^2_{sca}) / (2\ \sigma^2_{gca} + \sigma^2_{sca} + \sigma^2_e)$$

$$h^2_{NS} = 2\ \sigma^2_{gca} / (2\ \sigma^2_{gca} + \sigma^2_{sca} + \sigma^2_e)$$

Heritability can also be estimated in terms of the parent-offspring regression techniques, which indicates narrow sense heritability (Robinson *et al.*, 1949; Falconer, 1981) and on the basis of variance components from replicated plots (Hanson, 1963).

2.9.6. Number of genes

The most common problem in applied genetics is the size of the initial sample to be screened; the more populations sampled, the more chance of obtaining rare alleles having the properties desired. But on the other hand there is less ability to assess the material adequately. If a trait is controlled by n genes, there are 3^n kinds of digenic genotype possible in the F_2, and the smallest adequate population in the F_2 would be 4^n (Pope and Dewey, 1978). The number of genes involved in the inheritance of quantitative characters influences the limits of progress from selection. Many statistical methods exist for the estimation of the number of genes determining a trait, a number of biometrical procedures have been developed for estimating the number of genes governing quantitative traits in autogamous diploids, from the early Castel-Wright segregation index (Wright, 1952) to genotype assay (Jinks and Towey, 1976). Actually, we estimate the number of effective factors influencing a trait, rather than the number of gene. The effective factors may be defined in terms of the cross between two homozygous lines, P_1 and P2, at the loci which differ between the lines. Some alleles tending to increase the trait of interest will be homozygous in one line, some in the other. If for the k loci at which P_1 and P_2 differ, P_1 contains only alleles increasing the trait,

while P_2 contains only alleles decreasing it, then the differences between P_1 and P_2 will be 2ka. The estimate of additive genetic variance of the trait from the F_2 will be

$$V_A = \sum_i^k 2pqa^2 \{=V_{F2}-V_E\}$$

and $p=q=0.5$

so that $V_A=ka^2/2$ or $a^2=2V_A/k$

$(P_1-P_2)=2ka$ or $k^2=(P_1-P_2)^2/4a^2$

Hence $k = (P_1-P_2)^2 / 8V_A$

The estimator k must be the minimum number of genes affecting a trait, as it has been assumed equality of effects as well as independence of all gene loci. Mather and Jinks (1982) have shown that if one strain has 'x' loci fixed for the '+' allele and 'k-x' fixed for the '-' allele, and conversely for the other strain, then setting r = (k-2x)/k, the estimate is reduced by a factor r^2. Furthermore, if the gene effects are not all equal, and their variance is $V_{\Delta a}$, then the estimate will be reduced independently by a factor $(1+ V_{\Delta a})^{-1}$.

Lande (1981) outlined a procedure to estimate the minimum number of freely segregating genetic factors contributing to the differences in a quantitative character between two populations that have diverged by artificial or natural selection. He presented all standard errors for those formula.

The method of moments (Castel, 1921; Burton, 1951; Wright, 1968) was among the first procedures for analysing the differences in a quantitative trait between two homozygous parents. Subsequent procedures included Mather's k, (Mather 1949), the partitioning method (Powers *et al.*, 1950; Powers, 1963), discriminant analysis (Weber, 1959), the inbred backcross procedure (Wehrhahn and Allard, 1965), the convolution approach (Tan and Chang, 1972), genotype assay (Jinks and Towey, 1976; Towey and Jinks, 1977), and the doubled haploid method (Choo and Reinbergs, 1982). Methods

vary in their assumptions, in time and resources required, and in the precision and reliability of estimates of the number of genes.

Panse (1940) introduced another method using F_2 and F_3 second order statistics (genetic variances) to estimate k.

A recent method, called genotype assay by Jinks and Towey (1976) and Towey and Jinks (1977), is designed to overcome various problems such as large standard error because of linkage, sensitivity to unequal gene effects and so on, which are seen in previous methods. They emphasized that because the method detects only genes with effects sufficient to yield a significant difference, given the variability in the material, the range shown will be an underestimate. This method requires an estimate of proportion (P_h) of randomly selected F_n plants, derived from crossing two homozygous parents, that are homozygous for at least one locus. The proportion P_h is estimated by testing for unequal means of F_{n+2} lines derived from two or more randomly selected F_{n+1} progeny of each assay plant.

Wehrhahn and Allard (1965) used a technique, called "inbred back-cross line", which has the advantage that the magnitude of effects of the genes detected can be measured.This method involves the production of inbred lines following several backcrosses to the recurrent parent and their subsequent classification in replicated field trials as different from or not different from the recurrent parent. Unless many genes govern the trait, most inbred backcross lines are expected either to be genotypically identical to the recurrent parent or single gene deviates.

Park (1977a, b) has developed further related methods which are designed to assess the number of genes affecting a trait over the early generations of selection in order to estimate likely total response limits to selection depending on the number of loci involved.

2.9.7. Genetic correlation

It was established in classical genetics that many genes have manifold effects; i.e. some genes seem to affect traits that are unrelated. Genes that have manifold

effects are pleiotropic. In pleiotropic the same gene affects different traits in a complementary way; whereas in epistasis different genes affect the same trait. The existence of pleiotropic effects of genes in classical genetic analysis would logically imply the existence of pleiotropic effects for traits. Then it is possible that selection may be exerted on secondary traits that have greater heritabilities than the primary trait. Success of selection, however, also depends on association between the traits; if association is not large the effect of indirect selection for second trait by use of first trait will not be successful. Linkage also is another cause of a correlation between traits.

Genetic correlations are of interest to determine degree of association between traits and how they may enhance selection. Genetic correlations are useful if indirect selection gives greater response to selection for a trait than direct selection for the same trait. This depends on estimates of heritabilities for each trait and genetic correlation between them (Hallauer and Miranda, (1981). Success from combination selection depends on level of genetic correlation among traits included for selection.

2.10. Multivariate analysis

Having introduced correlated characters and response including the selection index, it is clear that multivariate methods generally may be of use to the geneticist and breeder.

Multivariate analysis is the branch of statistics concerned with analysing multiple measurements on one or more populations. It is a technique of data summary and reduction, grouping and analysing which can also discriminant populations on the basis of many associated attributes.

Analysis of variance (ANOVA) has been a widely used and useful approach to study differences among several populations or treatments. But ANOVA handles only one variable, which is assumed to be normally distributed with the same variance in each population. If one wants to deal with many characters together, ANOVA cannot do it and the multiple analysis of variance (MANOVA) is needed to solve this kind of problem. there is also the problem of redundancy in univariate F-test because of ..... with other attributes. In MANOVA one is concerned with the multivariate generalisation

(vector variable) of analysis of variance, which is the study of group differences with a number of variable combined (Bose, 1977). Since MANOVA deals with the vector variables rather than scalars, it looks at the whole dispersion of the variables, i.e. variance and covariance. It is a more suitable method of handling combined variables. It is also a very important tool for genetic studies because most quantitative genetic information derives from partitioning variance and covariance of phenotypic values, and Manova can generate a dispersion matrix for this purpose. The distinctive nature of MANOVA design is that dependent variable is a vector variable. This dependent vector variable is assumed to be multivariate, normal in distribution and with the same dispersion, or variance-covariance matrix, for each population. Equality of dispersions is the Manova extension of the assumption of homogeneity of variance in Anova design. In Manova the research issues concern the realness of the differences among the population centroids, or the mean vector (Bose, 1977).

There are two null hypotheses established for Manova. Hypothesis one is used to test that the populations have a common dispersion; whereas hypothesis two is to identify whether the populations have a common centroid.

2.10.1. Discriminant analysis

When two or more populations have been measured for several characters, special interest to certain linear function called discriminant functions, by which the populations are best discriminated. Since Manova cannot make a comparison of various means, we need to use discriminant function to get test scores for a means comparison. So it is of great importance in multivariate analysis, and it has been widely used in social science (Manly, 1986)

2.10.2. Canonical Correlation

The canonical correlation is the maximum correlation between the linear functions of the variables in two data sets. Canonical analysis is the most general of the multivariate techniques, multiple regression and Manova are all special cases in it. It is a descriptive or screening procedure rather than a hypothesis-testing one. Since canonical correlation analyses the relationship between two sets of variable, it is suitable

for dealing with relationships of characters which being measured.

2.10.3. Cluster analysis

The cluster analyst's objective is to find out which objects are similar and dissimilar to each other. Cluster analysis is widely applicable in research to determine clusters of similar objects. Cluster analysis may be worthwhile owing to grouping of similar objects or for data reduction. Many algorithms have been proposed for cluster analysis. Here attention will be restricted Ward's minimum variance clustering method. Ward's (1963) describes a class of hierarchical clustering methods including the minimum variance method. Anderberg (1973) showed that Ward's method would appear to be the more suitable than the other methods of clustering, and also conceptually attractive basis of this method meant that it was chosen as the method to be used for this data set. Ward's method tends to join clusters with a small number of observations and is strongly biased toward producing clusters with roughly the same number of observations. It is also very sensitive to outliers (Milligan, 1980). Ward's method followes a series of clustering steps that begin with t clusters, each containing one object, and it ends with one cluster containing all objects. At each step it makes whichever merger of two clusters that will result in the smallest increase in the value of an sum-of-squares index, or variance. This meant that at each clustering step we must try all possible mergers of two clusters, compute the value of sum-of-square index for each, and select that one whose value of sum-of-square index is the smallest. Then we go on to the next clustering step and repeat the process (Romesburg, 1984). The result of hierarchical clustering can be represented in a dendrogram (tree diagram). A practical problem in performing a cluster is deciding on the number of clusters to obtain. In hierarchical clustering system the number of clusters which may be obtained from n, the number of individuals (objects) to one depending on the level at which the hierarchy is cut-off (Anderberg, 1973). To find a truncation point, there was some way to do that, e.g. based on previous knowledge of the structure of the data set (Anderberg, 1973), or making arbitrary cut-off points (Romesburg, 1984). Gordon (pers. comm.) implemented a cut-off measure based on the F ratio of amongst cluster sums of squares / within cluster sums of squares. The cluster membership at each stage of successive clustering can be used as treatment in a successive MANOVA analyses. Wilk's Lambda F-ratio can be used to get a joint significant test of all attributes simultaneously. That clustering stage which has

greatest significance (lowest probability) can be used as the cut-off point (Gordon, pers. comm.).

Pattern analysis as commonly employed (Williams, 1976) consists of the joint numerical classification and ordination of a set of entities (cultivars) on the basis of their attributes (number of rust assessments). This approach should find considerable application in the preliminary evaluation of rates of disease development on the very large numbers of genotypes tested in some plant breeding programmes.

CHAPTER 3

MATERIALS AND METHODS

To estimate the quantitative genetics of adult plant and seedling resistant cultivars to stripe rust, seven experiments were carried out at Lincoln (glasshouse) and Palmerston North (glasshouse and field), New Zealand, during 1991 - 1993. The reaction of fifteen cultivars in response to three pathotypes of stripe rust were studied in different stages.

3.1. Cultivars, hybrids and experiments

Fifteen cultivars were chosen for their differing reactions to stripe rust and were grown from authenticated seed. There were as follows:

Cultivar	Resistance	Yr genes	Reference	Origin
Oroua	specific	7	Cromey 1990	NZ
Pegasus	specific	1,6	Cromey 1990	UK
Sapphire	specific	6,7	Cromey 1990	UK
Batten	specific	9	Cromey 1990	UK
Karamu	durable	A	Johnson 1988	NZ
Briscard	nonspecific	-	Cromey 1990	UK
Domino	nonspecific	-	Cromey 1990	UK
Otane	nonspecific	-	Cromey 1990	NZ
Ruapuna	nonspecific	-	Cromey 1990	NZ
Tiritea	susceptible	-	Cromey 1990	NZ
Kotare	nonspecific	-	Cromey 1990	NZ
Takahe	susceptible	-	Cromey 1990	NZ
Elite Lep.	durable	2	Johnson 1988	FR
Yeoman	durable	13	Johnson 1988	UK
Flanders	durable	1	Johnson 1988	NDL

[1] New Zealand, United Kingdom, France and Netherlands, respectively

Seven experiments were done as follows:

3.1.1. Seedling tests

1. *Cultivars experiment.* Fifteen cultivars ,as mentioned above, were used to investigate components of resistance with cultures of three pathotypes of *Puccinia striiformis* (106E139A-, 111E143A-, 232E137A-).

2. *Specific diallel.* Five cultivars with specific resistance and one susceptible cultivar (Oroua, Pegasus, Sapphire, Batten, Karamu and Tiritea, respectively) were intercrossed in all combinations of a diallel set of crosses, without reciprocals. Six parents and fifteen F1 progenies were tested with cultures of two pathotypes (111E143A-, 232E137A-) in two separate diallels in glasshouse.

3. *Specific vs nonspecific factorial mating.* Five cultivars with specific resistance,as used in experiment two, and four cultivars with nonspecific resistance (Briscard, Domino, Otane, Ruapuna) were crossed in a factorial mating design. Nine parents and the twenty F1 progenies were tested with cultures of two pathotypes (111E143A-, 232E137A-) in two separate experiments in the glasshouse.

4. *Nonspecific glasshouse diallel.* Four cultivars with nonspecific and one susceptible cultivar (Briscard, Domino, Otane, Ruapuna and Tiritea, respectively) were intercrossed in all combinations of a diallel set of crosses, without reciprocals. Five parents and ten F1 progenies were tested with cultures of three pathotypes (106E139A-, 111E143A-, 232E137A-) in three separate diallels in the glasshouse.

5. *Nonspecific glasshouse generation mean.* Those cultivars used in experiment four were continued for further generations. All ten crosses were extended by other generations (P_1, P_2, F_1, F_2, BC_1 - backcrossed to P_1- and BC_2 -backcrossed to P_2-) to stablish ten families. These were tested with a common pathotype (106E139A-) and ten separate generation mean analyses were conducted in the glasshouse.

3.1.2. Adult plant tests

6. *Nonspecific field diallel.* The same materials as in experiment four were tested with one pathotypes (106E139A-) in a diallel mating design in the field.

7. *Nonspecific field generation mean.* The same materials as in experiment five plus F_3, BCS_1 (backcross selfing with P_1) and BCS_2 (backcross selfing with P_2) lines were tested in the field with the common pathotype (106E139A-), giving a further in a ten generation mean analyses.

3.1.3. Crossing

For each cross, seed of respective cultivars were planted in 20 cm round plastic pots filled with a potting mixture (sand, peat and osmocote 5:2 parts and 250g, respectively) and grown in the greenhouse with 16 hr light. Planting was staggered in time to ensure that all cultivars would be at about the same growth stage for crossing. Standard plant breeding techniques for hand emasculation and for pollination were used for crossing (Plate 1). Parental and F_1 ears were harvested in August 1991 were threshed individually and were immediately planted in the glasshouse to produce backcross, F_2 and additional F_1 seed for nonspecific cultivars. The F_1 plants, as male parents, were crossed with their respective female parents to produce the backcross generations. Seed of the cultivars and progeny from the crosses between them were, in all cases, obtained from ears that had been enclosed in glassine bags at flowering time to prevent outcrossing.

Because of differences in heading date from other cultivars and lack of seed, five of the fifteen cultivars (Kotare, Takahe, Elite Lepeuple, Yeoman and Flanders) were not included in any crosses.

3.2. Cultures, storage and inoculation

Three cultures of the *Puccinia striiformis* from the rust culture collection of Crop & Food Research Institute at Lincoln, New Zealand, were used into this investigation.

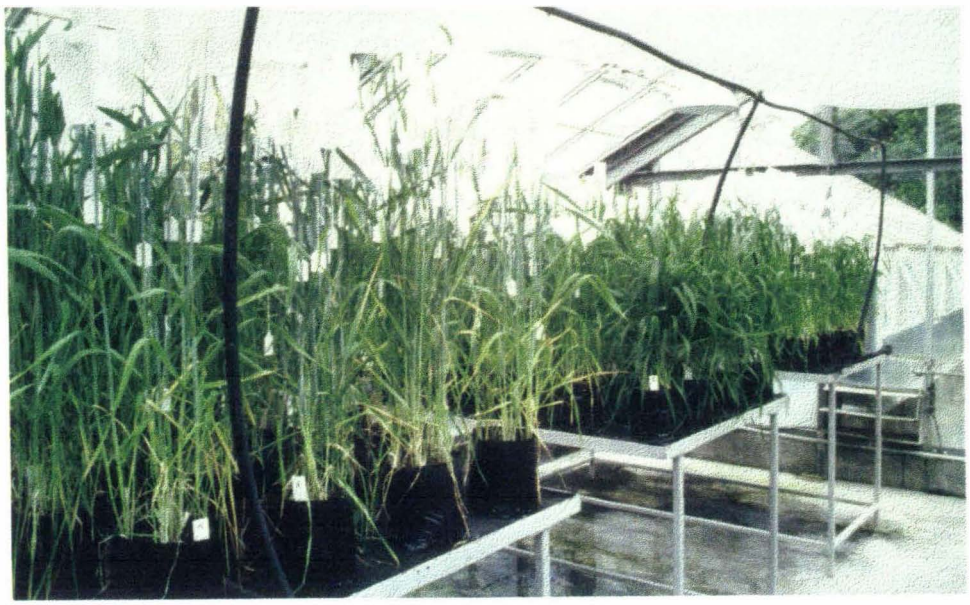
Culture WYR1 (pathotype 106E139A-) was pathotyped from volunteer wheat in Canterbury in 1986.

Culture WYR4 (pathotype 111E143A-) was pathotyped from cv. Pegasus in South Island in 1988.

Culture WYR9 (pathotype 232E137A-) was pathotyped from cv. Batten in Canterbury in 1990.

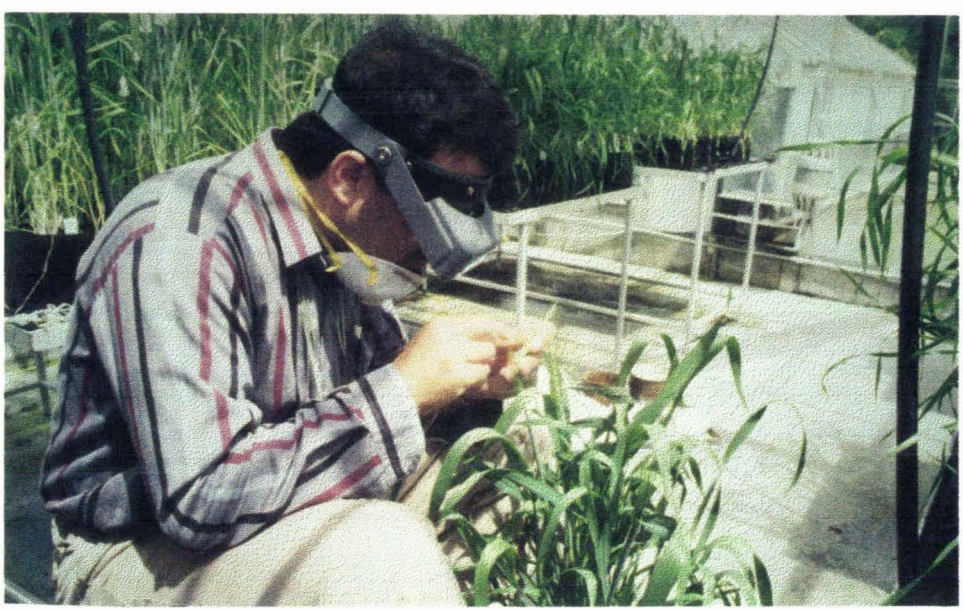
Plate 1. Overview of crossing block: (a) glasshouse location, (b) hand emasculation, and (c) pollination

(a)



(b)

(c)



Pathotype nomenclature follows the system described by Johnson *et al.* (1972), using the suffix by Wellings & McIntosh (1990).

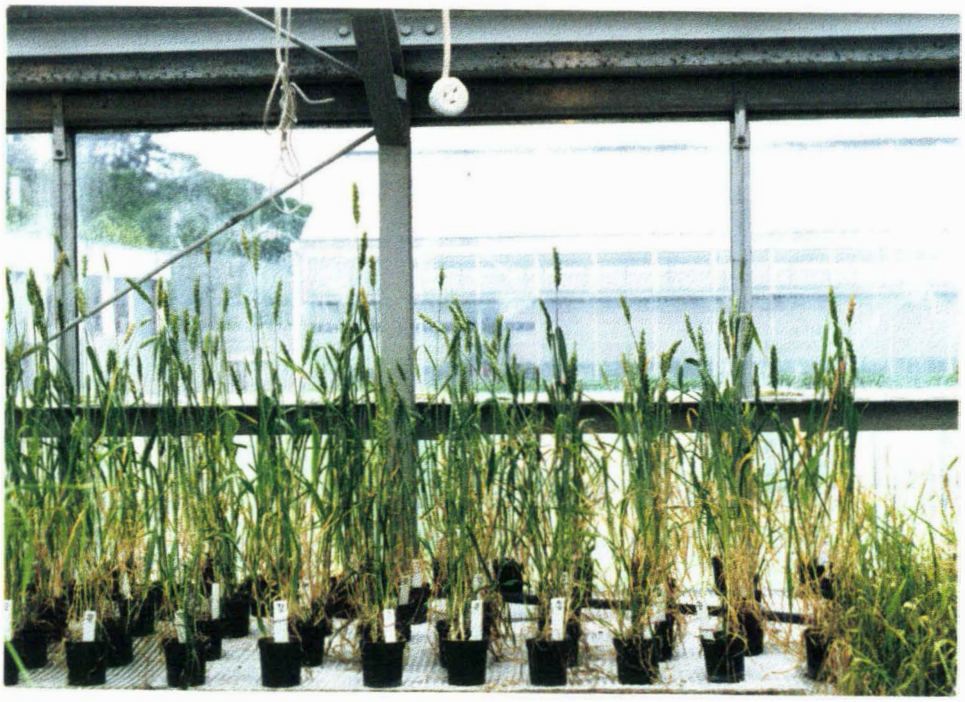
Uredospores of the three cultures were increased at three different times on the susceptible wheat cv. Tiritea grown in isolation in the glasshouse and controlled temperature room (Plate 2). Each day, inoculum was collected by holding the plants over a sheet of aluminium foil and gently tapping them, so that spores fell to the surface. They were partially dried (about 60 % r.h.) in desiccator containing silica gel, for 24 hours, and then sealed in plastic-lined aluminium foil bags and stored in an ultra-low freezer at -70 °c. Inoculum of each pathotype was heat-shocked by immersing in warm water (42 °c for 4 minute) before use. Plants in the glasshouse were sprayed with the fungicide Ethirimol, which has no any effect on stripe rust, to prevent powdery mildew infection. Any unwanted powdery mildew spores collected along with stripe rust spores, were inviable following storage at -70 °c.

Material for the seedling tests (experiments 1-5) was planted in 10 cm pots containing potting mixture. The pots were placed in the glasshouse with a 15 hr daily photoperiod at 15±2 °c. Inoculation was carried out when the first leaf was fully expanded and the second leaf was about half the length of the first leaf. For inoculation, all pots were sprayed as uniformly as possible using an atomizer with a spore suspension in distilled water with one drop of Tween 20 per litre and were left in a darkened moist chamber for 24 hr at 10±1 °c.

In the field (experiments 6-7), spreader rows of a highly susceptible variety, cv. Tiritea, were sown every five rows and also around the entire experimental field. When the second leaf become obvious all spreader rows were inoculated with a suspension of uredeospores in distilled water with one drop of Tween 20 per litre, using a knapsack sprayer at midnight. To ensure a good epidemic, all the material was inoculated two weeks after the first inoculation, with a mixture of spores and fine flour, particle size 100 µm, (Roelfs *et al.* 1992), using a knapsack motorized blower in the late evening. Spores were applied at the rate of 5-6g per hectare, which equates to approximately 1000 spores per plant (Stubbs et al, 1986). There was a good epidemic in the field (Plate 3).

Plate 2. Rust increase on susceptible wheat cv. Tiritea grown in the (a) glasshouse and (b) controlled temperature room

(a)



(b)

Plate 3. General view of the plots (a) no epidemic, (b) moderate epidemic, and (c) severe epidemic

(a)

(b)

(c)

3.3. Assessment and data collection

In the glasshouse, four attributes were measured, which were components of resistance in seedlings (experiments 1-5) : infection type, latent period, pustule size and pustule density. Daily assessments from the 7th day after inoculation were made for latent period (days from inoculation to first pustule eruption) by checking all leaves for visible pustules. Leaves with pustules were tagged with coloured wire, the date recorded, and these leaves omitted from further checking. Daily assessment continued until pustules were present on all leaves. Then, assessments for infection type were made based on a 0-9 ordinal scale (is shown in plate 4), according to the method described by Line *et al.* (1974), as follows :

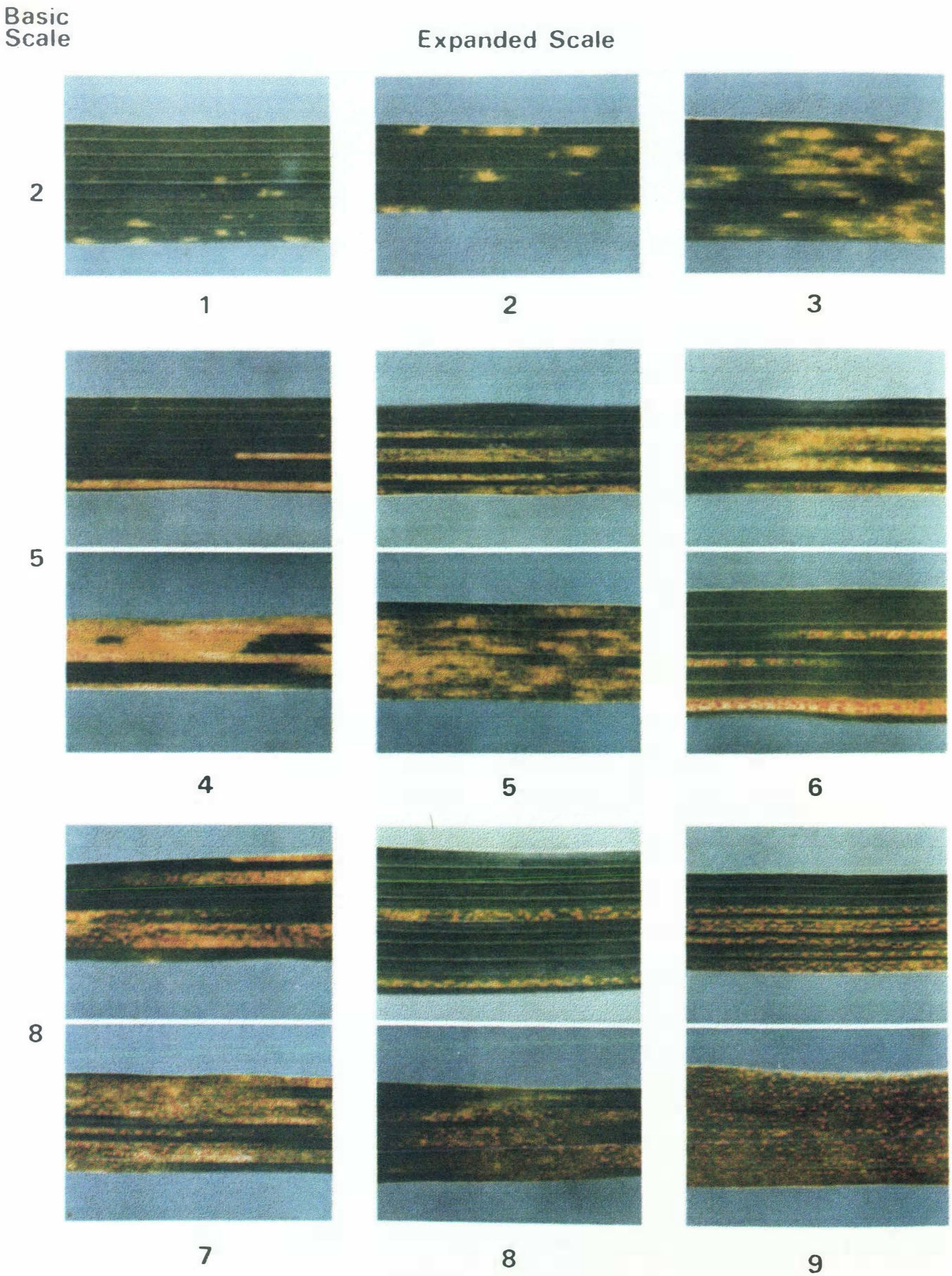
0	O	no visible signs or symptoms
1	VR	necrotic or chlorotic flecks, no sporulation
2	R	necrotic and/or chlorotic blotches[1] or stripes, no sporulation
3	MR	necrotic and/or chlorotic blotches or stripes, trace sporulation
4	LM	necrotic and/or chlorotic blotches or stripes, light sporulation
5	M	necrotic and/or chlorotic blotches or stripes, intermediate sporulation
6	HM	necrotic and/or chlorotic blotches or stripes, moderate sporulation
7	MS	necrotic and/or chlorotic areas, abundant sporulation
8	S	chlorosis behind sporulating area, abundant sporulation
9	VS	no chlorosis or necrosis, abundant sporulation

[1]Blotches occur on seedlings and stripes occur on plants in later stages of growth.

Finally, leaves were cut and preserved in jars which contained lactophenol:ethanol (1 : 2, v/v) solution for later assessment of pustules size (mm^2) and pustule density (number of pustules per mm^2 of infected leaf area).
Lactophenol was made as follows:

phenol (pure crystals)	20 g
lactic acid (SG 1.21)	20 g
glycerol	40 g

Plate 4. General classification of infection types for stripe rust



distilled water	20 g
ethanol (95%)	50 g

The phenol with water were warmed until dissolved and then the lactic acid and glycerol were added to it. A little dye such as cotton blue or picric acid may be added (0.05 g of cotton blue per 100 ml or substitute a saturated solution of picric acid for water when preparing the lactophenol).

The length and width of 15 random pustules per leaf were measured using a microscope (magnification 10X) with a micrometer, and the means calculated.

In the field (experiment 6-7), rust severity (Fig. 6), based on the modified Cobb's scale (Peterson et al, 1948) was recorded for individual plants at three different dates, at ten day intervals, starting from growth stage 45-47 (Fig. 7) or boot swollen (Zadoks et al, 1974). Assessments for any block were recorded within a short period (2.5 days) at each date.

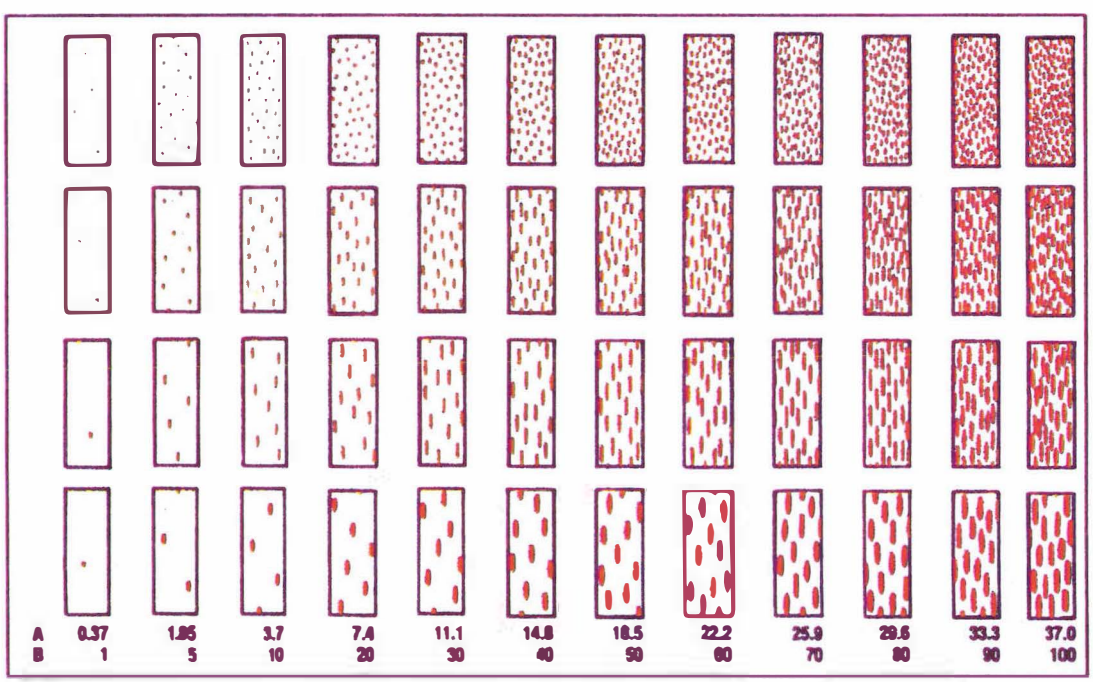
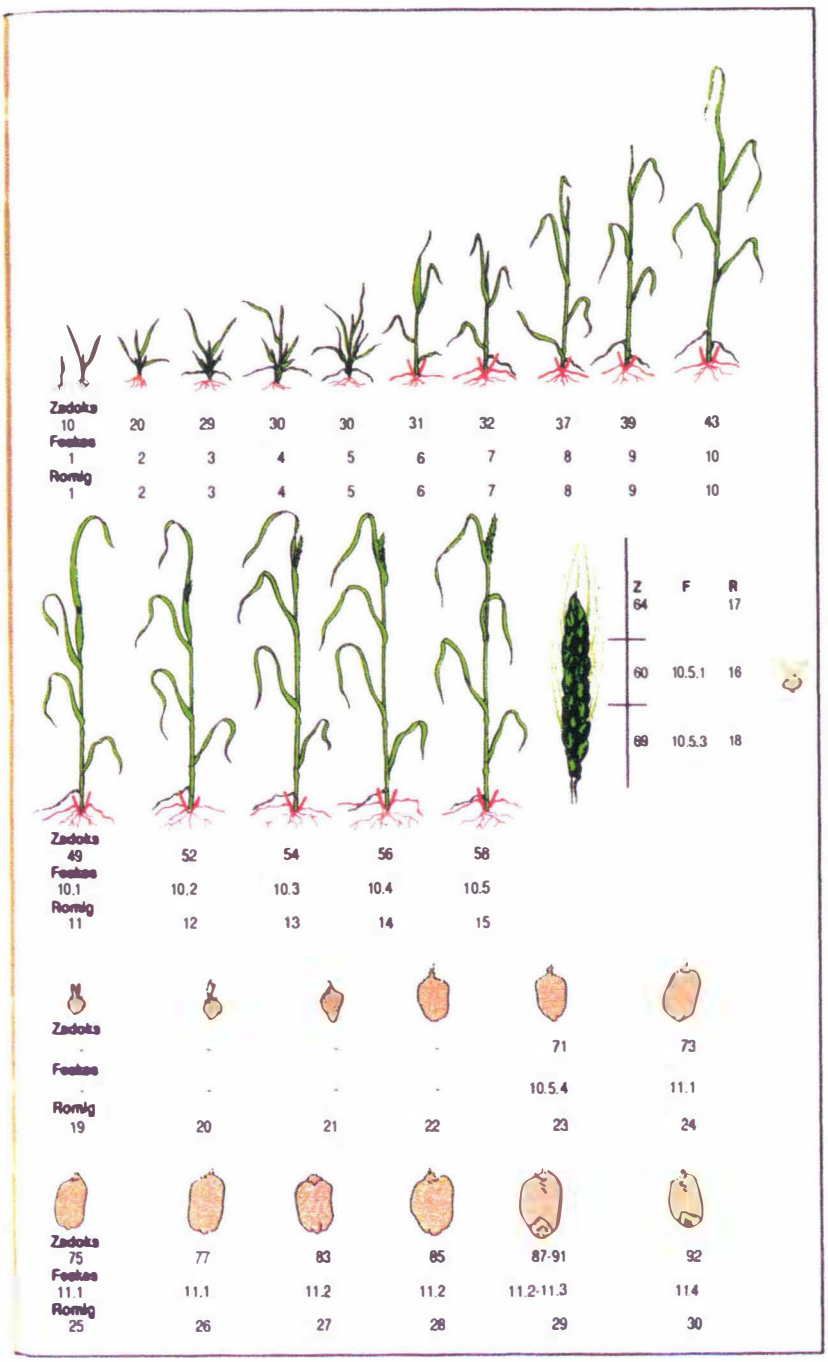


Fig. 6. The modified Cobb scale: A, actual percentage occupied by rust uredinia; B, rust severities of the modified Cobb scale after Peterson *et al.*

Fig. 7. Descriptions of the growth stages of wheat after Roelfs *et al.* (1992)



Description	Growth scale Zadoks	Feekes	Romig
Dry seed	00	—	—
Start of imbibition	01	—	—
Leaf just at coleoptile	09	1	—
First leaf through coleoptile	10	1	1
First leaf unfolded	11	—	1
Two leaves unfolded	12	—	—
One or more leaves unfolded	19	—	—
Main shoot only	20	2	—
Main shoot and 1 tiller	21	3	2
Main shoot and 2 tillers	22	3	2
Main shoot and 9 or more tillers	29	3	3
Pseudo-stem	30	4-5	4-5
1st node detectable	31	6	6
2nd node detectable	32	7	7
6th node detectable	36	—	—
Flag leaf just visible	37	8	8
Flag leaf ligule/collar just visible	39	9	9
Flag leaf sheath extending	41	10	—
Boot just visibly swollen	43	10	10
Boot swollen	45	10	10
Flag leaf sheath opening	47	10.1	—
First awns visible	49	10.1	11
1st spikelet of inflorescence just visible	50	10.1	—
1/4 of inflorescence emerged	52	10.2	12
1/2 of inflorescence emerged	54	10.3	13
3/4 of inflorescence emerged	56	10.4	14
Emergence of inflorescence completed	58	10.5	15
Beginning of anthesis	60	10.5.1	16
Anthesis half-way	64	—	17
Anthesis complete	69	10.5.3	18
Kernels near middle of head 1/8 formed	—	—	19
Kernels near middle of head 1/4 formed	—	—	20
Kernels near middle of head 1/2 formed	—	—	21
Kernels near middle of head 3/4 formed	—	—	22
Caryopsis watery ripe	71	10.5.4	23
Early milk	73	11.1	24
Medium milk	75	11.1	25
Late milk	77	11.1	26
Early dough	83	11.2	27
Soft dough	85	11.2	28
Hard dough	87	11.2	29
Caryopsis hard, 16% water	91	11.3	29
Caryopsis hard	92	11.4	30

3.4. Data analysis

In the glasshouse experiments 1-5, the latent period (days) and pustule size, mm^2x1000, (1/4*π*length*width) data were not transformed, because they are continuous data. Infection type (scale 0-9) data was subjected to a test of normality using correlation test for normality (Bliss,C.I., 1967) by MINITAB statistical package (Minitab Inc., 1989). This test supplies a normal probability plot, which is a useful supplement to histograms in checking for nonnormality. MINITAB plots the sample versus the values, and if the sample is from a normal population, the plot is approximately a straight line. It exhibited curvature if the population is not normal. The correlations of all tests were not significant from unity. The scale for pustule density (no.x100) data, which follows the poisson distribution, were changed using a square root transformation, $(X+0.5)^{1/2}$, (Steel and Torrie, 1980). The value of half was added because real zeroes were present in data.

When the progress of a rust epidemic has been measured at several times during its development, the area under the disease progress curve (AUDPC) can be calculated. In the field (experiments 6-7), since epidemics develop in a logistic fashion, which implies that there is a limit to their growth and that their increase is initially exponential. Therefore rust severity was transformed using a logit, ln[X/(100-X)]+10 (van der Plank 1963; Röbbelen and Sharp, 1978; Stubbs et al, 1986; Berger, 1988). A value of ten was added to obtain all positive numbers. Area under the disease progress curve was obtained by :

$$AUDPC = [\sum_{i}^{t} (X_{i+1} + X_i) / 2] * T_{i+1} - T_i$$

where X_i is severity (transformed) in time i_{th} and T_i is date of reading in time i_{th} (day).

3.5. Experimental design

1. Cultivars experiment. Three experiments were conducted one for each of three pathotypes. Each experiment consisted of two randomised complete blocks. Each pot (experimental unit) contained six plants. The model for a randomised complete block design is as follows:

$$x_{ijk} = \mu + \alpha_i + \beta_j + \varepsilon_{ij} + \phi_{(ij)k}$$

where x_{ijk} = the observed variable

i = 1...t where t is the number of genotypes

j = 1...b where b is the number of blocks

k = 1...s where s is the number of samples per plot

μ = the grand mean

α_i = the effect of the i_{th} cultivar

β_j = the effect of the j_{th} block

ε_{ij} = the ij_{th} residual (error) effect

$\phi_{(ij)k}$ = the ijk_{th} within plot effect

To obtain more information, the three experiments were pooled into a combined analysis. Pathotypes were analogous to environments, and genotype-pathotype interactions were estimated. The model for this pooled randomized complete block design was :

$$x_{ijkl} = \mu + \gamma_k + \beta_{j(k)} + \alpha_i + (\alpha\gamma)_{ik} + \varepsilon_{ijk} + \phi_{(ijk)l}$$

where x_{ijkl} = the observed value of the $ijkl_{th}$ plot

i = 1...t where t is the number of genotypes

j = 1...b where b is the number of blocks

k = 1...p where p is the number of pathotypes

l = 1...s where s is the number of samples per plot

μ = the grand mean

γ_k = the effect of the k_{th} pathotype

$\beta_{j(k)}$ = the effect of the j_{th} block nested within the k_{th} pathotype

α_i = the effect of the i_{th} cultivar

$(\alpha\gamma)_{ik}$ = the interaction of i_{th} cultivar and k_{th} pathotype

ε_{ijk} = the ijk_{th} residual (error) effect

$\phi_{(ijk)l}$ = the $ijkl_{th}$ within plot error

The source of variation, degrees of freedom, expectation of mean squares and F-ratio (Steel and Torrie, 1980) are presented in Table 1, using a random effect model.

Table 1. Sources of variation, degree of freedom, expectations of mean squares and F-ratios of pooled analysis of variance

S.O.V.	DF	MS	E(MS)	F-ratio*
Pathotype	p-1	MS_P	$\sigma^2+g\sigma^2_{B(P)}+b\sigma^2_{GP}+bg\sigma^2_P$	$(MS_e+MS_P)/(MS_B+MS_{GP})$
Block	p(b-1)	$MS_{B(P)}$	$\sigma^2+g\sigma^2_{B(P)}$	MS_B/MS_e
Cultivar	g-1	MS_G	$\sigma^2+g\sigma^2_{GP}+be\sigma^2_G$	MS_G/MS_{GP}
Path.*Cult.	(g-1)(p-1)	MS_{GP}	$\sigma^2+b\sigma^2_{GP}$	MS_{GP}/MS_e
Error	p(g-1)(b-1)	MS_e	σ^2	

* Random effects philosophy

The expectations of mean squares were obtained following Crump (1946). When the numerator or denominator is a linear function of mean squares, a complex F-test will be used (Satterthwaite, 1946; Crump, 1946). This has degrees of freedom as follows:

$$f' = [\sum_i^t (MS_i)]^2 / \sum_i^t [(MS_i)^2 / f_i]$$

where MS_i and f_i are the i_{th} mean square and degree of freedom (Satterthwaite, 1946). The computer programme THWAITE (Gordon pers. comm.) was used to estimate this complex F-test, and its degrees of freedom. This programme also estimated all variance components and their standard errors. The mean-squares and means comparison were first performed using SAS PROC GLM. To estimate full and restricted heritabilities along with their standard errors, the computer programme AOVTEMP was used (Gordon pers. comm.).

2. Specific diallel. Two experiments were conducted one for each two pathotypes (111E143A-, 232E137A-). Each experiment consisted of two randomised complete blocks. Each pot (experimental unit) contained six plants. The model for a randomised complete block design is as before. The analyses of variance were separately performed on all parents, F1,s and both.

3. Specific vs nonspecific factorial. The situation is as same as experiment two.

4. Nonspecific glasshouse diallel. Three experiments were conducted one for each three pathotypes (106E139A-, 111E143A- and 232E137A-). The experiment consisted of two randomised complete blocks for each pathotype 111E143A- and 232E137A- with six plants in each pot (experimental unit); but for pathotype 106E139A-, there were four blocks and five plants per pot. The model for a randomised complete block design is as before. The analyses of variance were separately carried out on all parents, F_1,s and both.

5. Nonspecific glasshouse generation mean. One experiment was conducted with pathotype 106E139A-. The experiment consisted of four randomised complete blocks with five plants in each pot (experimental unit). Plots were of different size for each generation, to allow for segregation. A weighted analysis was performed using inverse intra-plot variance as weight. The total size were : 5 plots for P_1, P_2 and F_1; 12 plots for each backcross and 45 plots for the F_2. The model for a randomised complete block design is as before.

6. Nonspecific field diallel. One experiment was conducted for pathotypes 106E139A-. The experiment consisted of five randomised complete blocks. Each row (experimental unit) was 2 m long with 45 cm spacing between rows and ten plants per row. The model for a randomised complete block design is as before. The analyses of variance were separately performed on all parents, F1,s and both.

7. Nonspecific field generation means. One experiment was conducted with pathotype 106E139A-. All generations were planted at Massey university, Palmerston North; the experiment consisted of five randomised complete blocks with ten plants in each plot (experimental unit). Plots (rows) were of different size for each generation, to allow for segregation. A weighted analysis using reciprocal of intra-plot variance, was used for the generation mean analysis. The total size were : 5 rows for P_1, P_2 and F_1; 10 rows for each backcross; 20 rows for the F_2; 100 rows for F_3; and 30 rows for each backcross selfing. The model for a randomised complete block design is as before. The extra generations (F_3 and backcrosses selfing) were provided so that both forms of the genetic analysis could be offered.

Analysis of all experiments used the procedure GLM of the SAS statistical

package (SAS Institute Inc., 1985) was used. Duncan's new multiple ranges were used where the genotype number was more than ten (Balaam, 1963); LSD otherwise.

3.6. Genetic analysis

Several mating designs were used to study types of gene action that condition the inheritance of stripe rust resistance as follows :

3.6.1. Diallel mating design

3.6.1.1. Graphical diallel

The F_1 and parental data were analyzed using the statistical and graphical techniques described by Hayman (1954b), Jinks (1954), Mather and Jinks (1977). Using this approach , it is possible to estimate parameters which provide information about the genetic system controlling quantitative traits. The parental array variances ($\bar{V}r$), the covariances of each array with the non-recurrent parent ($\bar{W}r$), variance of parents (V_p), and variance of parental array means ($V_{\bar{r}}$) were calculated. Their components (additive, D, dominance, H_1 and H_2, components and covariance of additive and dominance components, F) are as follows:

$$\bar{V}r = (1/4)D + (1/4)H_1 - (1/4)F$$

$$\bar{W}r = (1/2)D - (1/4)F$$

$$V_p = D$$

$$V_{\bar{r}} = (1/4)D + (1/4)H_1 - (1/4)H_2 - (1/4)F$$

The test of significance of W_r+V_r and W_r-V_r values of arrays, by analysis of variance was conducted and amongst array mean squares compared with their respective error mean squares. If dominance are present W_r+V_r must change from array to array. If there is non-allelic interaction, W_r-V_r will vary between arrays, although if only dominance is present, W_r-V_r will not vary more than expected from error variation. The W_r/V_r graphic analyses were performed on F_1 and F_2 data, using linear regression

analysis. The W_r/V_r graph provides : (1) it supplies a test of the adequacy of the model; in the absence of non-allelic interaction and with independent distribution of the genes among the parents W_r is related to V_r by a straight regression line of unit slope; (2) given that the model is adequate, a measure of the average level of dominance is provided by departure from the origin of the point where the regression line cuts the W_r axis (above the origin partial dominance, in the origin complete dominance, and below the origin overdominance); (3) the relative order of the points along the regression lines indicates the distribution of dominant and recessive genes among the parents (nearest to origin most dominant genes and furthest from the origin fewest dominant genes). Tests of significance of slope, (its deviation from unity) were conducted. If it deviated significantly from one, epistasis and/or disequilibrium was indicated.

Genetic components of variation, D (additive component), H_1 and H_2 (dominance components), and F (non-independent contributions of additive and dominance effects), were estimated. In fact those components of variation can be estimated by one or more combination of V_p (variance of parents), $\bar{V}r$ (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 $V_{\bar{r}}$ (variance of parental array means) as follows:

$$D = V_p - E_p$$

$$H_1 = 4\bar{V}r + V_p - 4\bar{W}r - \{[(4(n-1))/n]\, E_F + E_p\}$$

$$H_2 = 4\bar{V}r - 4V_{\bar{r}} - [(4(n-1)^2)/n^2]\, E_F - [(4(n-1))/n^2]\, E_p$$

$$F = 2V_p - 4\bar{W}r - [(2(n-2))/n]\, E_p$$

where n : number of parents

E_F : F_1 error mean square

E_P : parent error mean square

The equations of Mather and Jinks (1982) of estimating genetic components were modified to suit a half diallel. Because Mather and Jinks used a full diallel, therefore it is unsuitable in the present study, so by removing a half in all coefficients of the F_1 error

mean square (E_F) it would be suitable. Standard errors of genetical components were not estimated as "no worthwhile estimate of the errors of these component is available" (Mather and Jinks, 1982).

There are some statistics (Mather and Jinks, 1982) that may be calculated by those components of variation as follows :

1. $(H_1/D)^{1/2}$ measures average degree of dominance over all segregating loci. The statistic may describe three dominance types, namely, partial ($H_1<D$), complete ($H_1=D$), and overdominance ($H_1>D$).

2. $(0.5*F)/[D(H_1-H_2)]^{1/2}$ measures the extent to which the 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 $H_2/(4H_1)$ less than 0.25.

4. $[(4DH_1)^{1/2}+F]/[(4DH_1)^{1/2}-F]$ measures the proportion of dominant to recessive alleles over all parents.

5. Broadsense and narrowsense heritability estimates can be calculated from

$$h^2_{BS} = (0.5D+0.5H_1-0.25H_2-0.5F)/(0.5D+0.5H_1-0.25H_2-0.5F+E)$$

$$h^2_{NS} = (0.5D+0.5H_1-0.5H_2-0.5F)/(0.5D+0.5H_1-0.25H_2-0.5F+E)$$

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

6. $r_{(P,Wr+Vr)}$ is the correlation between W_r+V_r for each array and the mean of the its common parent. This statistic identifies whether the distribution of dominant to recessive alleles is correlated with common parent phenotype.

7. σ^2_{AR} / σ^2_{DR} (= 0.5 D / 0.25 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. The number of effective factors (K) can be estimated by using:

$$K = (M_{F1} - M_P)^2 / 0.25\ H_2$$

where M_{F1} and M_P are the overall means for F_1 progeny and parents, respectively (Jinks 1954).

3.6.1.2. Combining ability analysis

The data were analyzed to estimate general and specific combining ability effects (g.c.a. and s.c.a., respectively) (Griffing, 1956) using Method 2 (parents and one set of F_1's are included, but not reciprocal F_1's). The model I (fixed effects) was examined, by using SAS macro procedures. There are $p(p+1)/2$ genotypes, where p is number of parents. The model is as follow :

(Fixed) $$x_{ij} = u + g_i + g_j + s_{ij} + (1/bc)\sum_{k=1}\sum_{l=1} e_{ijkl}$$

i = 1...p where p is number of parents
j = 1...p where p is number of parents
k = 1...b where b is number of blocks
l = 1...c where c is number of samples per plot

where u is the population mean

g_i and g_j are the general combining ability effects for the i_{th} parent and j_{th} parent, respectively

s_{ij} is the specific combining ability effect of the cross between the i_{th} and j_{th} parents

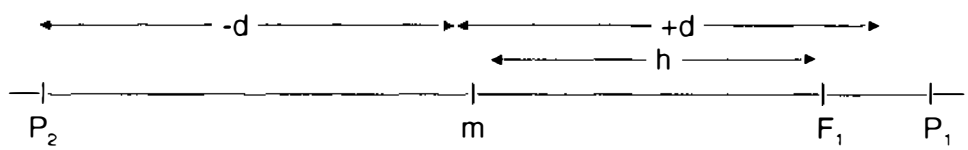
e_{ijkl} is the effect associated with $ijkl_{th}$ individual observation

Because no genetic variation was expected within the pure line parents or the F_1's, plots mean were used for all characters. In this model to test the differences among g.c.a. effect $F_{[(p-1),m]} = Mg / M_e$' was used and to test the differences among s.c.a. $F_{[p(p-}$

$_{1)/2,m]} = M_s / M_e'$ was used, where *p* is the number of parents, m is the degree of freedom associated with the error mean squares, and M_g, M_s and Me' are the g.c.a., s.c.a. and error mean squares (Griffing, 1956). The relative importance of g.c.a. and s.c.a. can be estimated by calculating ratios of relevant mean squares, $2 M_g / 2 M_g + M_s$, as suggested by Baker (1978).

3.6.2. Generation mean analysis

The generation mean analyses were carried out for experiments five and seven. The model which was proposed for generation mean analysis (Mather and Jinks, 1982) can show the relationship of the additive (d) and dominance (h) components of gene action in the parental, F_1 and midparent (m) means when susceptibility (the parent with the higher score) is partially dominant (as well as for any other condition of dominance).



These analyses were executed if the weighted analysis of variance indicated significance for generations. The genetic component was further partitioned into six components to estimate gene effects, as in the model :

$$Y = m + \alpha d + \beta h + \alpha^2 i + 2\alpha\beta j + \beta^2 l$$

where Y : the mean of a generation
m : the mean of all generations in a cross
[d] : pooled additive effect
[h] : pooled dominance effect
[i] : pooled interaction between additive effects
[j] : pooled interaction between additive and dominance effects
[l] : pooled interaction between dominance effects
α, β, α^2, $2\alpha\beta$, β^2 : products of genetic parameters

The coefficients of genetic components in some generations used are listed here (Mather and Jinks, 1982):

	m	d	h	i	j	l
P_1	1	1	0	1	0	0
P_2	1	-1	0	1	0	0
F_1	1	0	1	0	0	1
F_2	1	0	1/2	0	0	1/4
F_3	1	0	1/4	0	0	1/16
BC_1	1	1/2	1/2	1/4	1/4	1/4
BC_2	1	-1/2	1/2	1/4	-1/4	1/4
BCS_1	1	1/2	1/4	1/4	1/8	1/16
BCS_2	1	-1/2	1/4	1/4	-1/8	1/16

The standard procedure consisted of estimating those gene effects from the means of the available types of generations, followed by a comparison of the observed generation means with expected values derived from the estimates of the six parameters. If there are six generations it is not possible to conduct a goodness of fit test. The estimates of the six or less parameters were obtained by using weighted least square, because the number of individuals and variances differed in each generation, weighted generation means were used by taking weights as the reciprocals of squared standard errors of each mean (Mather and Jinks, 1982). In this study all six generations were examined with two, three, four, five and six parameters to see if a parsimonious model could explain the observed means as efficiently as the full model. These parsimonious models were tested for goodness of fit to the observed mean by the chi-square test with four, three, two and one degree of freedom. These have been called scaling test by Cavalli (1952), Hayman (1958a) and Mather and Jinks (1982). Rowe and Alexander (1980) expanded the procedure to more generations in matrix notation as **E G = O** where the **O** is column vector of generation means, and **G** vector of genetic parameters to be estimated by least squares and **E** matrix depends upon the genetic model and can consist of the genetic expectations of the six generations in terms of two, three, four, five and six parameters model.

The solution for the parameter estimates obtained by the inversion of matrix **E**,

and post multiplication by vector **O**, expressed in matrix form as $\mathbf{G = E^{-1} O}$. The inversion and multiplication of the matrix was carried by the MINITAB statistical software (1989). All operations can be stated as follows:

$\mathbf{W}$ = Diagonal $[1/\sigma^2_{xi}]$ (as weights)

$\mathbf{E\ G = O}$ or $\mathbf{(E'W)E\ G = (E'W)\ O}$

$\mathbf{G = E^{-1}\ O}$ or $\mathbf{G = (E'W\ E)^{-1}\ E'W\ O}$

$\mathbf{E'}$= transpose of $\mathbf{E}$

and for standard error of components of mean as follows:

$\mathbf{V_G = E'V_O\ E}$

$\mathbf{V_O}$ = Diagonal $[\sigma^2_{xi}]$ or $\mathbf{W = V_O^{-1}}$

$\mathbf{V_G = (E'W\ E)^{-1}}$

3.6.3. Generation variance analysis

According to Mather and Jinks (1977), components of variation from all generations were calculated. Their expectations are as follows:

$V_{P1} = E_w$

$V_{P2} = E_w$

$V_{F1} = E_w$

$V_{F2} = 1/2D + 1/4H + E_w$

$V_{BC1} = 1/4D + 1/4H - 1/2F + E_w$

$V_{BC2} = 1/4D + 1/4H + 1/2F + E_w$

where

E_w : non-heritable component of variation

D : additive component of variation

H : dominance component of variation

F : dependent contribution of d and h over all loci

And to calculate them, we have as follows:

$$E_w = 1/4\ (V_{P1} + V_{P2} + 2V_{F1})$$
$$D = 4V_{F2} - 2\ (V_{BC1} + V_{BC2})$$
$$H = 4\ (V_{BC1} + V_{BC2} - V_{F2} - E_w)$$
$$F = V_{BC1} - V_{BC2}$$

and also we can estimate the dominance ratio as $\sqrt{}(H/D)$; and $F/\sqrt{}(D*H)$ which can provide evidence of dominance deviations at different loci.

Having only four equations for the estimation of four parameters we must obtain a prefect fit solution to them, and we can neither calculate the standard deviation of the estimates of D, H, E, and F, nor indeed can we test the goodness of fit of the additive-dominance model as a whole (Mather and Jinks, 1982).

3.6.4. Factorial mating designs

This mating design was used for the experiment three (seedling versus adult plant resistant cultivars). The design II mating design or factorial mating design was described by Comstock and Robinson (1948, 1952). In this design sources of variation for male, female and their interaction, plus replication and male-female combinations x replicates (as error) are available. The model can be presented as :

$$Y_{hijk} = \mu + \alpha_i + \beta_j + (\alpha\beta)_{ij} + R_h + e_{hijk}$$

Where

Y_{hijk} : the observation of the K_{th} full sib progeny in a plot in the h_{th} replication of the i_{th} paternal plant and the j_{th} maternal plant

μ : grand mean

α_i : the effect of the i_{th} paternal plant

β_j : the effect of the j_{th} maternal plant

$(\alpha\beta)_{ij}$: the interaction of the paternal and maternal plants

R_h : the effect of the h_{th} replication

e_{hijk} : the environmental effect and remainder of genetic effect between full sibs on the same plot

The genetic interpretation of the variance components was reviewed in chapter 2.

Crosses between inbred lines were made in the greenhouse according to factorial mating design of Comstock and Robinson (1948, 1952). Adult plant resistant cultivars were used as males while seedling resistant cultivars were used as females. Appropriate F-tests can be made to test for the differences among males and among females and for their interactions, i.e. error term was adult-seedling resistance combination x replicates. According to Hallauer and Miranda (1981) the genetic information is similar to a diallel, but different sets of parents are used as males and females for the factorial mating design. Because a diallel is only a factorial mating design where the number of male and female parents are equal.

3.6.5. Heritability

For experiments five and seven (generation means analysis in glasshouse and field), broadsenes and narrowsense heritabilities estimates based on variances of populations were calculated by different methods as follows:

$$\sigma^2_{F2} = V_A + V_D + V_E$$

$$\sigma^2_{BC1} + \sigma^2_{BC2} = V_A + 2\,V_D + 2\,V_E$$

$$V_A = 2\,\sigma^2_{F2} - (\sigma^2_{BC1} + \sigma^2_{BC2})$$

$$V_D = \sigma^2_{F2} - V_A - V_E$$

if

$$V_E = \sigma^2_{P1} + \sigma^2_{P2} + 2\,\sigma^2_{F1} \qquad \text{(Mather and Jinks, 1977)}$$

$$V_E = \sigma^2_{P1} + \sigma^2_{P2} + \sigma^2_{F1} \qquad \text{(Allard, 1960; Simmonds, 1979)}$$

then

$$h^2_{BS} = (V_A + V_{D)} / (V_A + V_D + V_E)$$

$$h^2_{NS} = V_A / (V_A + V_D + V_E) \qquad \text{(Warner, 1952)}$$

or broad sense heritability can be calculated by :

$$h^2_{BS} = (\sigma^2_{F2} - V_E) / \sigma^2_{F2}$$

where

$V_E = \sigma^2_{F1}$ (Burton, 1951)

$V_E = (\sigma^2_{P1} + \sigma^2_{P2}) / 2$ (Allard, 1960)

$V_E = (\sigma^2_{P1} + \sigma^2_{P2} + \sigma^2_{F1}) / 3$ (Kelly and Bliss, 1975; Burton, 1952)

$V_E = \sqrt{(\sigma^2_{P1} * \sigma^2_{P2})}$ (Mahmud and Kramer, 1951; Kelly and Bliss, 1975)

For their standard error of them using the variance of a ratio e.g. Z=X/Y as follows:

$$\sigma^2_z = \{\mu^2_y \sigma^2_x + \mu^2_x \sigma^2_y - 2\mu_x \mu_y \text{Cov}(x,y)\}/\mu^4 y$$

where

$\mu_x = E(x)$, $\mu_y = E(y)$; σ^2_x, σ^2_y, σ^2_z are variances of x, y and z, respectively

A standard error for h^2_{NS} was used from Ketata *et al.* (1976) as follow:

$$S.E._{h2Ns} = \{2 \{[(V_{B1} + V_{B2})^2 / df_{F2}] + (V^2_{B1} / df_{B1}) + (V^2_{B2} / df_{B2})\} / V^2_{F2}\}^{1/2}$$

where df_{F2}, df_{B1}, and df_{B2} refer to the degrees of freedom associated with V_{F2}, V_{B1}, and V_{B2}, respectively. Significance of h^2_{NS} was also evaluated noting that the ratio $2V_{F2} / (V_{B1} + V_{B2})$ approximately follows an F distribution with n_1 and n_2 degrees of freedom

where $n1 = df_{F2}$

$n2 = (V_{B1} + V_{B2})^2 / [(V^2_{B1} / df_{B1}) + (V^2_{B2} / df_{B2})]$

For broad sense heritability, Kelly and Bliss (1975); Burton (1952) i.e. $V_E = (\sigma^2_{P1} + \sigma^2_{P2} + \sigma^2_{F1}) / 3$, standard error can be approximated from van Ginkel and Scharen (1987) as follows :

$$S.E.h^2_{BS} = \{ [1/9]*[2/V_{F2})^2] * [(V_{P1}+V_{P2}+V_{F1})^2/df_{F2} + (V_{P1})^2/df_{P1} + (V_{P2})^2/df_{P2} + (V_{F1})^2/df_{F1}] \}^{1/2}$$

where df_{P1}, df_{P2} df_{F1} df_{F2} are equal to degree of freedom of the P_1, P_2, F_1 and F_2

populations, respectively.

The heritability based on sources of variations in a randomised complete block design (experiment five) can be estimated as follows :

$$\sigma^2_G = (TMS - \sigma^2_e) / r \qquad h^2_{BS} = \sigma^2_G / (\sigma^2_G + \sigma^2_e)$$

where

TMS : the variance of treatment (populations)
σ^2_G : the genotypic variance
σ^2_e : the error variance
r : the number of replications

and for a pooled randomised complete block design (experiment one) it can be express in two forms, I. full and II. restricted heritability (Gordon *et al.*, 1972) :

I. $h^2_{BS} = \sigma^2_G / \sigma^2_{P1}$
II. $h^2_{BS} = \sigma^2_G / \sigma^2_{P2}$

where σ^2_{P1} (full phenotypic variance) and σ^2_{P2} (restricted phenotypic variance) were defined as follows :

$$\sigma^2_{P1} = \sigma^2_E + \sigma^2_G + \sigma^2_{GE} + \sigma^2_B + \sigma^2_e$$
$$\sigma^2_{P2} = \sigma^2_G + \sigma^2_{GE} + \sigma^2_e$$

where

σ^2_E : macro environment variance
σ^2_G : genotypic variance
σ^2_{GE} : genotype environment interaction variance
σ^2_B : block variance (meso environment)
σ^2_e : error variance

The standard error of heritability estimates are determined according to Gordon *et al.* (1972) by computer programme AOVTEMP (Gordon pers. comm.).

In diallel cross (experiment two, four and six) whether Hayman's or Griffing's method, heritability is estimable, respectively, as follows :

$$h^2_{BS} = (1/2\ D + 1/2\ H_1 - 1/4\ H_2 - 1/2\ F) / (1/2\ D + 1/2\ H_1 - 1/4\ H_2 - 1/2\ F + E)$$

$$h^2_{NS} = (1/2\ D + 1/2\ H_1 - 1/2\ H_2 - 1/2\ F) / (1/2\ D + 1/2\ H_1 - 1/4\ H_2 - 1/2\ F + E)$$

where E is the pooled error mean square from analysis of variance of parents and F_1's.

$$h^2_{BS} = (2\ \sigma^2_{gca} + \sigma^2_{sca}) / (2\ \sigma^2_{gca} + \sigma^2_{sca} + \sigma^2_e)$$

$$h^2_{NS} = 2\ \sigma^2_{gca} / (2\ \sigma^2_{gca} + \sigma^2_{sca} + \sigma^2_e)$$

3.6.6. Number of genes

For experiments five and seven, the minimum number of genes or effective factors was estimated by following formulas (Lande, 1981):

$$n = (\mu_{P2}-\mu_{P1})^2/8V_S$$

V_S can be calculated by several methods,

$V_S = V_{F2} - V_{F1}$ (Castle, 1921)

$V_S = V_{F2} - (0.5\ V_{F1} + 0.25\ V_{P1} + 0.25\ V_{P2})$ (Wright, 1968)

$V_S = 2V_{F2} - V_{BC1} - V_{BC2}$ (Wright, 1968)

$V_S = V_{BC1} + V_{BC2} - (V_{F1} + 0.5\ V_{P1} + 0.5\ V_{P2})$

where

μ_{P2}, μ_{P1} are mean phenotype of parent one and two ($P_2 > P_1$)

V_S is variance segregating

V_{P1}, V_{P2}, V_{F1}, V_{F2}, V_{BC1} and VBC2 are variance of first, second,

F_1, F_2, first and second backcrosses

and their standard error are :

$$S.E.\ n = \{n^2\ [\ 4*(V_{P1}/N_{P1} + VP2/N_{P2})/(\mu_{P2}-\mu_{P1})^2 + Var(V_S)/(V_S)^2\ ]\ \}^{1/2}$$

and $Var(V_S)$ can be estimated in four ways :

$$Var(V_S) = [2*(V_{F2})^2/N_{F2}] + [2*(V_{F1})^2/N_{F1}]$$

$$Var(V_S) = [2*(V_{F2})^2/N_{F2}] + [0.5*(V_{F1})^2/N_{F1}] + [0.125*(V_{P1})^2/N_{P1}] + [0.125*(V_{P2})^2/N_{P2}]$$

$$Var(V_S) = [8*(V_{F2})^2/N_{F2}] + [2*(V_{BC1})^2/N_{BC1}] + [2*(V_{BC2})^2/N_{BC2}]$$

$$Var(V_S) = [2*(V_{BC1})^2/N_{BC1}] + [2*(V_{BC2})^2/N_{BC2}] + [2*(V_{F1})^2/N_{F1}] + [0.5*(V_{P1})^2/N_{P1}] + [0.5*(V_{P2})^2/N_{P2}]$$

The number of genes or effective factors (for experiments five and seven) may be calculated by the following equations as well :

$$n = (\mu_{F1}-\mu_{P1})^2/[4*(V_{BC1}-0.5*(V_{F1}+V_{P1}))]$$

$$n = (\mu_{P2}-\mu_{F1})^2/[4*(V_{BC2}-0.5*(V_{F1}+V_{P2}))]$$

where μ_{F1} is mean phenotypic of F_1

For experiment seven, the Wright's formula (1968) can be changed for F_3 population which is:

$$n = (\mu_{P2}-\mu_{P1})^2/[5.33*(V_{F3}-(V_{P2}+V_{P1})/2)]$$

3.6.7. Genetic correlation

Genetic correlations was estimated for experiment one. Because two or more traits may be correlated phenotypically, but we are seeking to know whether such a

correlation is a manifestation of an underlying genetical correlation or reflects environmental factors. Genetic correlation, r_A, is the correlation of breeding values for two traits within individuals in the population, whereas an environmental correlation, r_E, is the correlation of environmental deviations (possibly including non-additive genetical). It may immediately be thought of as containing most of the remaining information in phenotypic correlation, r_P, which is the correlation of phenotypic values (Falconer, 1981):

$$r_P = COV_P/(V_{PX}*V_{PY})^{1/2} = (COV_A+COV_E/(V_{PX}*V_{PY})^{1/2}$$

$$r_G = COV_A/(V_{AX}*V_{AY})^{1/2}$$

3.7. Multivariate analysis

3.7.1. Discriminant analysis

Experiment one was subjected to MANOVA. Because MANOVA is the statistical technique concerned with analysing the variance of multiple measurements on several populations, and discriminant analysis is the technique for centroid comparison between those populations (Manly, 1986).

In plant breeding, the criteria for assessing resistance involve a number of correlated attributes of resistance, hence the MANOVA and discriminant analysis will assist the evaluation of genotypes.

Discriminant function analysis can be used to separate two or more groups of individuals given measurements for these individuals on several variables. The data for a discriminant function analysis do not need to be standardised to have zero means and unit variances prior to the start of the analysis, as is usual with principle component and factor analysis. This is because the outcome of a discriminant function analysis is not affected in any important way by the scaling of individual variables.

A SAS PROC MANOVA of general linear model (GLM) was used in this analysis. Following the discriminant functions, the four components of resistance for each genotype were changed to one set of score for each pathotype. This led to a new data set involving fifteen rows (Cultivars) and three columns (pathotypes). So each genotype's

response was described by three scores. In order to identify the response of each genotype over all pathotypes and classify them, these scores were subjected to a cluster analysis.

3.7.2. Cluster analysis

The aim of clustering was together the most similar entities into the same cluster, and to segregate the dissimilar entities into different clusters, thus "reducing" the number of entities. A similarity matrix is constructed first, based often on Minkouski matrix. The second Minkowski leads to the well-known Euclidean distance (Anderberg, 1973). In Ward's minimum-variance (or sum-of-square) method, the distance between two cultivars is the pooled amongst-cluster sum of squares of the similarity scores. At each cluster stage, the corresponding increment in the within-cluster sum-squares is minimized after considering all possible cluster merges from the current stage. The sum of squares are easier to interpret when they are divided by the total sum of squares to give proportions of variance. As clustering proceeds and clusters merge, the internal homogeneity of clusters decreases. The sacrifice of the internal homogeneity is unavoidable as the number of clusters is reduced. Ward's method joins clusters to maximize the likelihood at each level of the hierarchy under the following assumptions (SAS Institute Inc., 1990):

- multivariate normal mixture
- equal spherical covariance matrices (homogeneity of dispersions)
- equal sampling probabilities

The PROC CLUSTER from SAS programme was used to calculate cluster analysis. To determine a truncation point for the dendrogram (to define the number of clusters), the method proposed by Gordon (pers. comm.) was used. This is based on the most significant F value for the ratio "amongst cluster mean squares" / "within cluster mean squares". This technique can use either the similarity mean of square, or a parallel MANOVA partitioning of the original variates. The latter was used in this case.

The stage of clustering with the most significant F was used as the dendrogram function point to defining the optimum value of cluster. This concept reflects the fundamental philosophy behind the efficient definition of groups : that the amongst-group variability should be minimum relative to the within-group variability (Gordon, pers.

comm.). In this way, by clustering multiple discriminant scores combining components of resistance for each pathotype, the reaction of the wheat lines were discriminated.

CHAPTER 4

RESULTS

4.1. Cultivar experiment

Fifteen cultivars of wheat were tested against three pathotypes of stripe rust at three different times, measuring four components of resistance: infection type, latent period, pustule size and pustule density.

4.1.1. Univariate analysis

4.1.1.1. ANOVA and means

A pooled analysis of variance was used to estimate means of cultivars over three pathotypes, means of pathotypes over fifteen cultivars, and their interactions (Table 2). The pathotype (race), cultivar, and their interaction were highly significant for all characters.

The mean infection type for cultivars in response to each pathotype, and the mean over three pathotypes, are presented in Table 3. For pathotype 232E137A-, Tiritea and Batten had high infection type values whereas Oroua and Sapphire had infection types of less than 4 and other cultivars were intermediate (infection type 4-7). For the pathotype 111E143A-, Tiritea, Pegasus and Sapphire had high infection type whereas Batten had low infection type. For pathotype 106E139A-, Tiritea had high infection type whereas Pegasus, Batten and Sapphire had infection types of less than 4. The comparison of means pooled over the three pathotypes indicated that Tiritea generally had a high infection type (susceptible) whereas Batten with a low infection type generally was resistant. Some cultivars such as Tiritea, Takahe, Otane, Yeoman and Ruapuna showed consistent infection types in response to the three pathotypes, whereas the infection types of Batten, Pegasus, Sapphire, Oroua and Karamu were pathotype-specific. Other cultivars such as Kotare, Flanders, Elite Lepeuple, Domino and Briscard showed some variation in reaction to the three pathotype. It can be concluded that in response to the pathotypes, considerable variation in cultivar reaction come from interaction of gene(s) in the hosts and those in pathotypes.

Latent period as days from inoculation to rupture of the first pustule was measured for fifteen cultivars in response to three pathotypes (Table 4). Pustules were formed on all cultivars in response to pathotype 232E137A-, allowing the measurement of latent period. Latent period ranged from 13.00 days for Sapphire to 11.00 days for Tiritea. For pathotype 111E143A-, Batten had a hypersensitive response with the absence of pustules. For this pathotype, apart from Batten, the longest latent period was for Karamu, with 14.25 days, whereas Tiritea with 11.67 days had shortest latent period. Pustules did not form on either Batten or Pegasus in response to pathotype 106E139A-, the cultivars showing hypersensitivity (i.e. presence of flecks) and immunity (no visible sign of disease) respectively. Removing Batten and Pegasus from consideration, Sapphire and Tiritea had the longest and shortest latent period with 15.75 and 11.60 days, respectively. The comparison of means of fifteen cultivars pooled over three pathotype indicated that there was no difference between Sapphire, Ruapuna and Karamu which had the longest latent period (apart from Batten and Pegasus) while Tiritea had the shortest latent period.

The results of pustule size measurement are presented in Table 5. In response to pathotype 232E137A-, the range of pustule size was from 39.75 to 29.57 for Batten and Elite Lepeuple, respectively. In the case of pathotype 111E143A- which was not virulent on Batten, the pustule size for Batten was zero. Due to the adding and multiplying of a constant value to all data (discussed in Materials and Methods) the value of 10.00 was used for Batten. Apart from Batten the range of pustule size was from 55.41 to 32.12 for Sapphire and Kotare, respectively. In response to pathotype 106E139A-, Batten and Pegasus did not form pustules. For the other cultivars, the maximum pustule size was recorded as 41.05 the for Takahe and the minimum was 29.82 for Kotare. Pooling the results of pustule size over the three pathotypes showed that Tiritea had the largest and Kotare the smallest size of pustule.

The results of pustule density are presented in Table 6. Tiritea had the highest number of pustules in response to all pathotypes. For pathotype 232E137A-, apart from Oroua and Sapphire which were resistant to this pathotype, Batten and Elite Lepeuple had significantly lower pustule density than the others, although this pathotype was virulent on Batten and expected to give high pustule density. For pathotype 111E143A-,

Batten did not form any pustules, since this pathotype was not virulent on it, but Kotare and Elite Lepeuple had lower pustule densities than the others. For pathotype 106E139A-, Batten and Pegasus did not have any pustules, but Sapphire and Kotare had lower pustule densities than the others. By pooling the mean of cultivars over three pathotypes, Tiritea had the highest and Kotare the lowest pustule density.

The analysis of variance showed that race or pathotype was significant (Table 2). To understand which pathotype is the most aggressive to the range of cultivars chosen, the mean of each pathotype over the fifteen cultivar was compared for each character (Table 7). For all characters, there was no overlapping for the means. In case of infection type, although the mean of the pathotype of 232E137A- was more aggressive on some cultivars than the others, it was close to the mean of pathotype 111E143A-. The mean of pathotype 106E139A- was less than the others. This order among the pathotype means was observed also for latent period. The most aggressive pathotype 232E137A- had the shortest mean latent period and the least aggressive pathotype 106E139A- had the longest mean latent period. For pustule size the pathotype 111E143A- was more aggressive than the others since it found more pustules than pathotype 232E137A- and the smallest pustules size belonged to pathotype 106E139A-. For pustule density the order was similar to that of infection type and latent period.

4.1.1.2. Variance components and heritability

Estimates of variance components and heritability (full and restricted) are presented in Table 8. The variance component of pathotype x cultivar interaction was the most important component for all characters. Both full and restricted heritabilities were highly significant and restricted heritability was higher than full heritability. Latent period and pustule density had the highest and lowest heritabilities (Table 8), respectively. Using t-test there was no difference between both heritabilities for each character because variance of block was small.

4.1.1.3. Phenotypic and genotypic correlation

Phenotypic and genotypic correlations for all components of resistance in

response to three pathotypes are presented in Table 9. All correlations, whether phenotypic or genotypic, were highly significant, except the phenotypic correlation between latent period and pustule density. Genotypic correlation was calculated by removal of environmental effects from the phenotypic correlations. Generally, genotypic correlation was greater than phenotypic correlation. Latent period was a negatively correlated with other characters. All correlations for pathotype 106E139A- were high and followed by pathotypes 111E143A- and 232E137A-. In response to pathotype 106E139A-, a high correlation was obtained between latent period and pustule density, followed by pustule size and pustule density, latent period and pustule density, infection type and pustule density, infection type and pustule size, and infection type and latent period. For pathotype 111E143A-, the descending order of correlations was between pustule size and number, latent period and pustule density, latent period and pustule size, infection type and pustule size, infection type and pustule density, and infection type and latent period. In the case of pathotype 232E137A-, the descending order of correlations was between infection and pustule size, infection type and pustule density, infection type and latent period, pustule size and pustule density, latent period and pustule size, and latent period and pustule density.

To estimate the phenotypic correlation between components (as sets) of each pathotype, PROC CANCORR was used. These correlations are presented in Table 10. A high correlation was obtained between latent period, pustule size and pustule density of pathotype 106E139A- with latent period and pustule density of pathotype 111E143A-. It can be expected that there is no correlation between components of resistance of different pathotype which can be seen in Table 10.

4.1.2. Multivariate analysis

4.1.2.1. Multiple discriminant analysis

Multivariate analysis of variance with a randomised complete block design was performed for each of three pathotypes across all characters. The purposes were to discriminate cultivars with all components of resistance simultaneously for each pathotype separately and to look for patterns amongst these discriminants to identify cultivar type. Table 11 shows the descriptive statistics of the cultivar discriminant for

each pathotype, where Wilk's Lambda was transformed as an F approximation. Strong significant differences were detected between all groups (cultivars over four components of resistance). Table 11 also shows the discriminant function, eigenvalue, proportion of total discriminant power accounted for by each multiple discriminant function, and cumulative amount of discriminant power of functions. In this structure, all original variables can contribute with the dependent variable in each linear function; therefore not only we can determine the relationship between function and the original variables, but one can also determine the relationship between the original variables in their own right. Variables which have high negative coefficients negatively contribute with those variables having positive coefficients. The magnitude of the coefficients shows the relative contribution of original variables to each function.

In response to pathotype 232E137A-, the first function accounts for 90% of total discriminant power and is followed by 6%, 3% and 1% for second, third and fourth functions (Table 11). Since the first function accounts for most of the discriminant power, the rest only accounts for a small part of the discriminant power and is of no value in this analysis. Therefore, the first function was retained for further analysis. Function one was mainly associated with infection type and pustule density and negatively associated with latent period (Table 12). The first function was too strong to use in grouping cultivars and by using a standardised canonical coefficient it may be written as follows:

$$Z_1 = 2.84\ IT - 0.05\ LP - 0.12\ PS + 1.55\ PN$$

In this function, according to structure and standardised coefficient it can be interpreted that the relationship of infection type and pustule density to score was the prime positive determinor while it was pseudo for latent period and pustule density.

In the case of pathotype 111E143A-, the Wilk's Lambda F test was highly significant for the first three functions and for the fourth function it was significant at the 5% probability level (Table 11). The eigenvalue explained 88% and 9% of the ratio of the between cultivar variation to pooled within cultivars variation for the first and the second canonical variables. The first canonical variable structure (Table 11) showed that the first canonical variable was most strongly influenced and negatively correlated with the

characteristic of latent period, pustule density and pustule size in response to this pathotype. Here again the first function was too strong to use in the grouping of cultivars and by using standardised canonical coefficient it may be written as follows:

$$Z_1 = -0.96\ IT - 5.44\ LP + 0.79\ PS + 1.09\ PN$$

This can be interpreted as meaning that latent period and pustule density (in response to this pathotype for these fifteen cultivars) with high coefficients and correlations on the first canonical variable tended to produce a susceptible reaction with short latent period and high pustule density. The relationship of latent period to score was the prime negative determinor while it was prime positive determinor for pustule size and pustule density; and it was reverser (suppressor) for infection type.

In response to pathotype 106E139A-, the Wilk's Lambda F-test was highly significant (Table 11) and the first two functions account for 93% and 6% of total discriminant power, respectively. By accepting the first function, the first canonical structure shows that the first canonical variable was strongly influenced by latent period, pustule size, pustule density and infection type respectively (Table 12). The first function can be written as follows :

$$Z_1 = 0.68\ IT - 0.02\ LP + 0.13\ PS + 0.26\ PN$$

From the coefficients in the equation for Z_1 it can be seen that this variable will tend to be large when there are high values in everything,specially in infection type, except latent period. The interpretation of the relationship of all characters to the score is similar with interpretation for pathotype 111E143A-.

The score, **S**, for each cultivar on the based of first function was obtained by multiplying the standardised cultivar means, **Z**, by standardised canonical coefficients, **U**, (resulted from first canonical discriminant function), i.e. **S = U'Z** by SCOREST programme (Gordon's unpublished). Table 13 displays the score of each cultivar in response to three pathotypes. Thus, four original variables (infection type, latent period, pustule size and pustule density) were reduced to one set of scores for each pathotype.

Not only was the number of comparisons between cultivars reduced, but the validity of these comparisons was enhanced, since the first function accounted for most of the total discriminant power. As it can be seen from Table 12, susceptibility to one pathotype was shown by positive and resistance with negative values. For example, in reaction to pathotype 232E137A- the most susceptible (over four components) and resistant cultivars were Tiritea and Oroua with score values 47.40 and -69.06, respectively. For pathotype 111E143A-, Tiritea and Batten were the most susceptible and resistant cultivars and in case of pathotype 106E139A-, Takahe and Batten were the most susceptible and resistant cultivars.

The scores on these three pathotype can be plotted to show differences among cultivars in three dimensions, and then the differences can be readily visualized. Fig. 9 shows the cultivars based on the first functions which ploted on three pathotypes as three axes. The symbols of cultivar were explained in Table 13. The cultivars Batten and Pegasus were placed apart from of the other cultivars. To see the differences among fifteen cultivars based on those scores (which were made from four components of resistance of each pathotype), a cluster analysis was used.

4.1.2.2. Canonical discriminant analysis

The four variables (components of resistance) across the fifteen cultivars were analyzed by canonical discriminant analysis (SAS programme), to discriminate among pathotypes. Table 11 shows the Wilk's Lambda and other statistics. Both functions were highly significant and the functions account for 79% and 21% of total discriminant power (Table 11). Since it is worthwhile to plot the first few significant canonical variables to illustrate which of these variables have the most discriminating power in separating groups (pathotypes), the separation of these pathotypes can be seen in Fig. 8.

4.1.2.3. Cluster analysis

In order to classify these fifteen cultivars (discriminated over four components of resistance for each of pathotypes), the discriminant scores (three for each cultivar) were clustered by Ward's method. The resulting dendrogram (Fig. 10) shows that the sum-of-

squares between clusters at each stage of clustering, and the groups formed at that stage. The probabilities of the MANOVA F-test over the original variable of several sequential clustering stages are presented in Table 14. The minimum probability was for the cluster eight. The Fig. 10 shows that the truncation point of the dendrogram was determined at the cluster level 8. Eight groups involving cultivar(s) were formed by truncating as follows:

- group 1 : Tiritea, Takahe
- group 2 : Otane
- group 3 : Ruapuna, Briscard, Domino, Yeoman, Elite Lepeuple, Karamu
- group 4 : Oroua
- group 5 : Kotare, Flanders
- group 6 : Sapphire
- group 7 : Pegasus
- group 8 : Batten

Table 2. Pooled analysis of variance for four components of resistance in fifteen cultivars with three pathotypes of stripe rust

S.O.V.	D.F.	MS			
		Infection Type	Latent Period	Pustule Size	Pustule Number
Race	2	203.05 **	924.20 **	3440.57 **	49497.46 **
Block(Race)	3	0.14 ns	0.25 ns	6.26 ns	627.78 ns
Cultivar	14	153.34 **	550.81 **	2102.23 **	23609.57 **
Race X Cultivar	28	33.90 **	118.53 **	610.55 **	7987.82 **
Error	42	0.38	0.41	5.87	494.49

ns and ** : not-significant and significant at 1% probability level, respectively

The complex-F, (Race + Error) / (Block + Race*Cultivar), was significant at P=0.001 level

Table 3. Differences amongst fifteen cultivars with three pathotypes of stripe rust for infection type (scale 0-9) using Duncan's multiple ranges

232E137A-		111E143A-		106E139A-		over three pathotypes	
Tiritea	8.9 a	Tiritea	9.0 a	Tiritea	8.8 a	Tiritea	8.9 a
Batten	8.5 ab	Pegasus	8.8 a	Otane	7.3 b	Otane	7.6 b
Takahe	7.9 bc	Sapphire	8.1 b	Oroua	6.9 b	Takahe	7.1 c
Briscard	7.7 cd	Otane	7.7 bc	Takahe	6.2 c	Briscard	6.2 d
Otane	7.6 d	Takahe	7.5 bcd	Yeoman	4.4 d	Domino	6.0 de
Domino	7.6 d	Oroua	7.5 d	Domino	4.3 de	Ruapuna	5.9 de
Elite L.	6.8 e	Briscard	6.7 e	Karamu	4.3 de	Oroua	5.7 ef
Yeoman	6.8 e	Ruapuna	6.4 ef	Ruapuna	4.3 de	Yeoman	5.6 ef
Ruapuna	6.7 ef	Domino	6.2 f	Flanders	3.6 ef	Sapphire	5.4 gf
Kotare	6.7 ef	Yeoman	5.4 g	Briscard	3.6 f	Elite L.	5.3 g
Karamu	6.3 f	Flanders	5.1 g	Kotare	3.5 f	Pegasus	5.0 h
Pegasus	5.4 g	Elite L.	5.2 g	Elite L.	3.4 f	Kotare	4.9 h
Flanders	5.3 g	Kotare	4.9 g	Sapphire	3.0 f	Flanders	4.9 h
Sapphire	3.4 h	Karamu	5.5 h	Batten	2.0 g	Karamu	4.8 h
Oroua	2.7 h	Batten	1.9 i	Pegasus	0.0 h	Batten	3.5 i

Means with the same letter within columns are not significantly different (P=0.05)

Table 4. Differences amongst fifteen cultivars with three pathotypes of stripe rust for latent period (days) using Duncan's multiple ranges

232E137A-		111E143A-		106E139A-		over three pathotypes	
Sapphire	13.0 a	Batten	30.0 a	Pegasus	30.0 a	Batten	25.5 a
Oroua	12.7 a	Karamu	14.3 b	Batten	30.0 a	Pegasus	17.6 b
Ruapuna	12.4 ab	Ruapuna	13.5 bc	Sapphire	15.8 b	Sapphire	13.4 c
Karamu	12.3 abc	Briscard	13.4 c	Elite L.	15.0 bc	Ruapuna	13.4 c
Flanders	12.2 abcd	Elite L.	13.1 cd	Flanders	14.6 cd	Karamu	13.4 c
Briscard	12.1 abcd	Domino	13.0 cd	Ruapuna	14.4 cd	Briscard	13.2 cd
Pegasus	11.8 bcde	Kotare	12.7 cde	Kotare	14.2 cde	Elite L.	13.1 cde
Batten	11.8 bcde	Oroua	12.6 de	Briscard	14.2 cde	Kotare	13.0 def
Kotare	11.7 bcde	Sapphire	12.5 def	Karamu	13.8 def	Oroua	12.8 efg
Elite L.	11.7 bcde	Flanders	12.3 defg	Yeoman	13.4 ef	Flanders	12.7 fg
Yeoman	11.6 cde	Otane	12.2 efg	Oroua	13.3 fg	Domino	12.6 gh
Domino	11.6 cde	Takahe	12.1 efg	Domino	13.1 fg	Yeoman	12.4 hi
Otane	11.6 cde	Yeoman	12.1 efg	Otane	12.5 gh	Otane	12.1 ij
Takahe	11.3 de	Pegasus	11.8 fg	Takahe	12.1 hi	Takahe	11.9 j
Tiritea	11.0 e	Tiritea	11.7 g	Tiritea	11.6 i	Tiritea	11.4 k

Means with the same letter within columns are not significantly different (P=0.05)

Table 5. Differences amongst fifteen cultivars with three pathotypes of stripe rust for pustule size (mm^2 x 1000) using Duncan's multiple ranges

232E137A-		111E143A-		106E139A-		over three pathotypes	
Batten	39.8 a	Sapphire	55.4 a	Takahe	41.1 a	Tiritea	40.7 a
Tiritea	37.8 a	Tiritea	44.0 b	Tiritea	40.3 ab	Sapphire	39.9 a
Takahe	36.1 ab	Pegasus	43.0 b	Otane	37.2 bc	Takahe	38.5 b
Otane	33.7 bc	Otane	42.3 bc	Oroua	36.3 cd	Otane	37.8 b
Briscard	33.0 bcd	Briscard	38.7 cd	Elite L.	34.8 cd	Briscard	35.4 c
Flanders	32.8 bcd	Oroua	38.5 d	Briscard	34.3 cd	Oroua	35.0 c
Ruapuna	32.7 bcd	Takahe	37.5 de	Karamu	33.8 cd	Ruapuna	34.5 cd
Yeoman	32.5 bcd	Ruapuna	37.3 de	Domino	33.8 cd	Domino	34.3 cde
Domino	32.0 bcd	Domino	36.9 def	Ruapuna	33.6 cd	Flanders	33.3 def
Pegasus	31.5 cd	Karamu	34.5 efg	Flanders	33.5 cde	Karamu	33.1 ef
Karamu	31.4 cd	Flanders	33.4 fg	Sapphire	33.0 de	Yeoman	32.5 fg
Kotare	31.2 cd	Yeoman	32.2 g	Yeoman	32.8 de	Elite L.	32.2 fg
Sapphire	30.2 d	Elite L.	32.2 g	Kotare	29.8 e	Kotare	31.2 g
Oroua	30.0 d	Kotare	32.1 g	Pegasus	10.0 f	Pegasus	29.2 h
Elite L.	29.6 d	Batten	10.0 h	Batten	10.0 f	Batten	20.5 i

Means with the same letter within columns are not significantly different (P=0.05)

Table 6. Differences amongst fifteen cultivars with three pathotypes of stripe rust for pustule density (x 100)/mm^2 using Duncan's multiple ranges

232E137A-		111E143A-		106E139A-		Pooled over	
Tiritea	228.1 a	Tiritea	231.1 a	Tiritea	230.3 a	Tiritea	229.8 a
Otane	204.5 b	Sapphire	219.9 a	Otane	208.5 b	Otane	199.2 b
Ruapuna	203.5 b	Karamu	195.5 b	Takahe	198.4 bc	Yeoman	193.1 b
Yeoman	201.6 b	Oroua	191.8 b	Yeoman	190.0 cd	Takahe	191.1 b
Karamu	195.8 bc	Pegasus	188.1 bc	Oroua	187.7 cde	Karamu	189.8 bc
Takahe	191.7 bcd	Otane	186.0 bc	Elite L.	187.6 cde	Ruapuna	189.0 bc
Flanders	185.4 cde	Yeoman	185.5 bcd	Ruapuna	178.9 def	Flanders	178.9 cd
Kotare	182.2 cde	Ruapuna	183.1 bcd	Karamu	177.0 defg	Elite L.	176.3 d
Pegasus	180.7 de	Takahe	182.9 bcde	Briscard	175.0 efg	Domino	174.8 d
Domino	178.9 de	Domino	177.6 cde	Flanders	173.7 efg	Briscard	174.4 d
Briscard	175.4 de	Flanders	175.7 cde	Domino	166.6 fg	Oroua	174.0 d
Elite L.	173.7 e	Briscard	172.6 def	Kotare	164.2 g	Sapphire	173.8 d
Batten	172.2 e	Elite L.	169.2 ef	Sapphire	137.5 h	Kotare	168.0 d
Sapphire	158.0 f	Kotare	160.1 f	Pegasus	70.7 i	Pegasus	150.9 e
Oroua	141.8 g	Batten	70.7 g	Batten	70.7 i	Batten	106.5 f

Means with the same letter within columns are not significantly different (P=0.05)

Table 7. Mean differences of four components of resistance with three pathotypes of stripe rust races, pooled over fifteen cultivars

Pathotype	Infection Type (scale 0-9)	Latent Period (days)	Pustule Size (mm^2 x 1000)	Pustule Number (x100)/mm^2
232E137A-	6.5563 a	11.8389 a	32.9535 a	185.0830 a
111E143A-	6.3252 b	13.9448 b	36.7194 b	179.0970 b
106E139A-	4.4266 c	15.9021 c	31.5644 c	167.6440 c
$LSD_{0.05}$	0.1386	0.1449	0.5369	4.9169

Means with the same letter within columns are not significantly different

Table 8. The estimations of variance components, their standard errors, heritabilities and their standard errors for pooled analysis of variance of fifteen cultivars in response to three pathotypes of stripe rust

S.O.V.	Variance Components			
	Infection Type	Latent Period	Pustule Size	Pustule Density
Race	1.129±1.355	5.372± 6.165	18.86± 22.96	275.84± 330.31
Block	0.003±0.002	0.002± 0.003	0.01± 0.07	1.78± 6.98
Cultivar	19.907±9.777	72.047±35.097	248.61±135.19	2603.63±1529.23
Race x Cultivar	16.760±4.530	59.060±15.839	302.34± 81.59	3746.67±1068.78
Error	0.380±0.083	0.410±0.090	5.87± 1.28	494.49± 107.91
Full h^2	0.52±0.06 **	0.53±0.03 **	0.43±0.02 **	0.37±0.01 **
Restricted h^2	0.54±0.06 **	0.55±0.03 **	0.45±0.02 **	0.38±0.01 **

** : significant at p=0.01

Table 9. Phenotypic and genotypic correlations among four components of resistance (IT: Infection Type, LP: Latent Period, PS: Pustule Size, and PD: Pustule Density) in reaction to three pathotypes of stripe rust

Character	Pathotype	LP		PS		PD	
		Phenotypic	Genotypic	Phenotypic	Genotypic	Phenotypic	Genotypic
IT	106E139A-	-0.71 **	-0.75 **	0.77 **	0.80 **	0.78 **	0.87 **
	111E143A-	-0.67 **	-0.71 **	0.76 **	0.85 **	0.65 **	0.76 **
	232E137A-	-0.42 **	-0.78 **	0.53 **	0.66 **	0.38 **	0.71 **
LP	106E139A-			-0.96 **	-0.98 **	-0.87 **	-0.94 **
	111E143A-			-0.75 **	-0.81 **	-0.78 **	-0.88 **
	232E137A-			-0.22 **	-0.62 **	-0.14 ns	-0.59 **
PS	106E139A-					0.88 **	0.96 **
	111E143A-					0.74 **	0.91 **
	232E137A-					0.33 **	0.53 **

Table 10. Correlations among components of resistance from three pathotypes in fifteen cultivars of wheat

		Infection Type (IT)		Latent Period (LP)		Pustule Size (PS)		Pustule Density (PD)	
		111E143A-	232E137A-	111E143A-	232E137A-	111E143A-	232E137A-	111E143A-	232E137A-
IT	106E139A-	0.34	0.21	-0.33	-0.03	0.25	0.04	0.45	0.19
	111E143A-		-0.20		-0.04		-0.18		0.11
LP	106E139A-	-0.24	-0.22	0.63	-0.09	-0.41	0.05	-0.59	-0.10
	111E143A-		0.29		-0.15		0.60		-0.19
PS	106E139A-	0.32	0.22	-0.62	0.27	0.45	-0.21	0.62	-0.09
	111E143A-		-0.40		0.26		-0.47		0.04
PD	106E139A-	0.28	0.40	-0.59	-0.26	0.33	0.25	0.57	0.44
	111E143A-		-0.31		0.17		-0.43		0.26

Table 11. Discriminatory power and related statistics for cultivar discriminant, for each pathotype in fifteen cultivars of wheat

Pathotype	Func.	Eigenvalue	Proportion	Cumulative	Wilks' Lambda			
					Approx F	Num DF	Den DF	Pr > F
232E137A-	1	117.85	0.90	0.90	7.69	56	45.0	0.0001
	2	8.03	0.06	0.96	3.17	39	36.3	0.0003
	3	4.40	0.03	0.99	2.16	24	26.0	0.0288
	4	0.66	0.01	1.00	0.84	11	14.0	0.6062
111E143A-	1	440.34	0.88	0.88	25.12	56	45.0	0.0001
	2	46.54	0.09	0.97	10.49	39	36.3	0.0001
	3	10.18	0.02	0.99	5.36	24	26.0	0.0001
	4	2.16	0.01	1.00	2.75	11	14.0	0.0391
106E139A-	1	1886.58	0.93	0.93	52.19	56	45.0	0.0001
	2	127.39	0.06	0.99	16.95	39	36.3	0.0001
	3	10.05	0.01	1.00	6.53	24	26.0	0.0001
	4	3.47	0.00	1.00	4.41	11	14.0	0.0055
All three	1	0.63	0.79	0.79	41.69	8	882	0.0001
	2	0.17	0.21	1.00	24.71	3	442	0.0001

Table 12. Canonical structure and coefficient of two first function for four components of resistance of three pathotypes

Pathotype	Character	Between Canonical Structure		Standardized Canonical Coefficients	
		Discriminant 1	Discriminant 2	Discriminant 1	Discriminant 2
232E137A-	Infection Type	0.9786	0.1429	2.8395	0.0085
	Latent Period	-0.7727	-0.2069	-0.0544	-0.1444
	Pustule Size	0.6713	0.6285	-0.1221	1.1171
	Pustule Density	0.8381	-0.3251	1.5475	-1.3140
111E143A-	Infection Type	0.6824	0.7185	-0.9619	2.7890
	Latent Period	-0.9940	-0.0173	-5.4363	2.2402
	Pustule Size	0.8312	0.4468	0.7855	0.2076
	Pustule Density	0.9137	0.2470	1.0882	0.2817
106E139A-	Infection Type	0.7284	0.6790	-1.9941	5.4653
	Latent Period	-0.9952	-0.0178	-7.4215	4.3359
	Pustule Size	0.9877	0.1275	6.5797	0.6786
	Pustule Density	0.9422	0.2605	1.4649	-0.2747
All three	Infection Type	-0.6797	0.7335	-0.8258	1.0323
	Latent Period	0.9285	-0.3714	1.5230	0.4737
	Pustule Size	0.1503	0.9886	1.7516	0.8374
	Pustule Density	-0.8670	0.4984	0.0771	-0.6338

Fig. 8. Plot of canonical scores of fifteen cultivars of wheat on discriminant functions 1 and 2 (B: Batten, O: Oroua, P: Pegasus races)

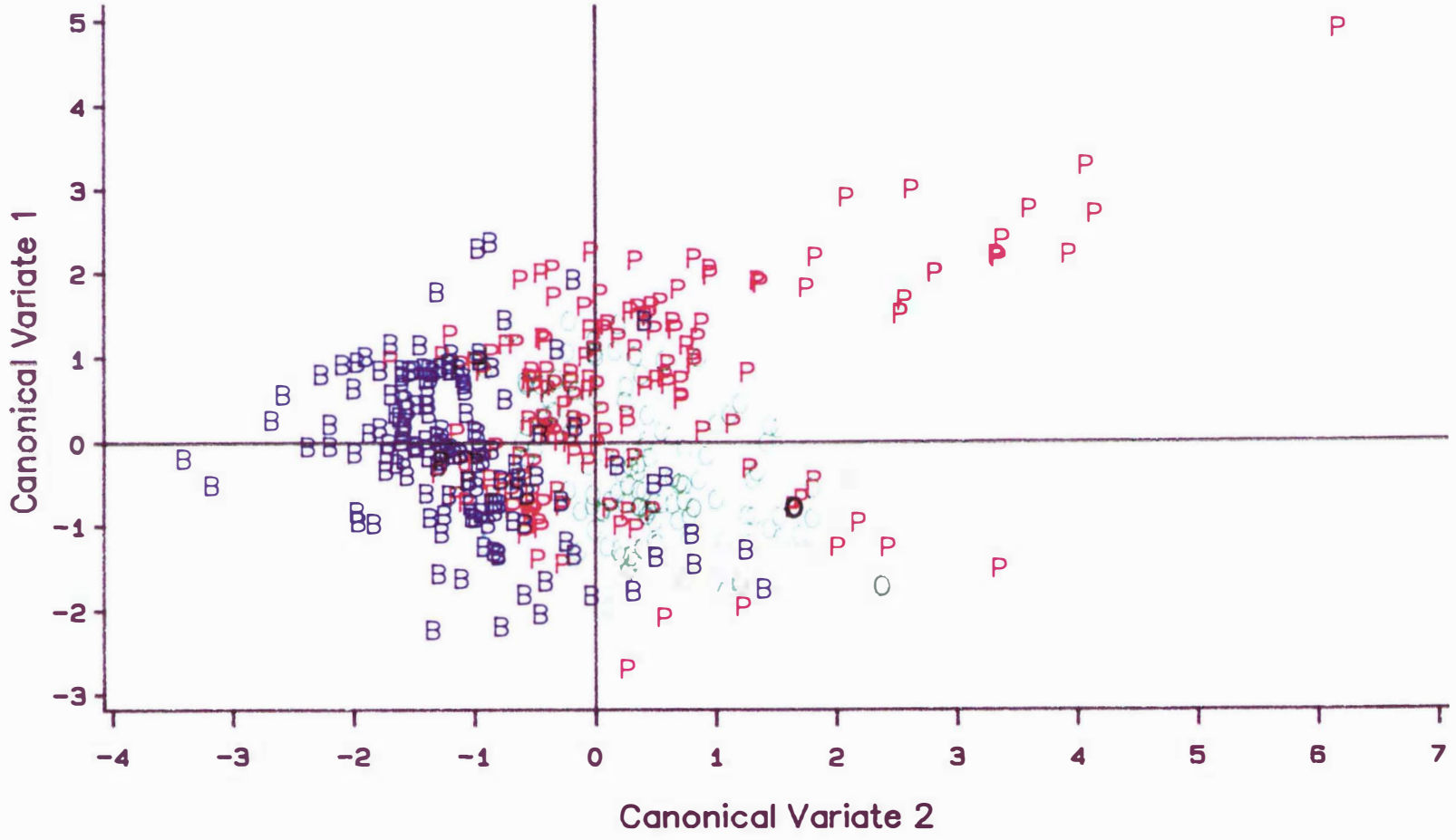
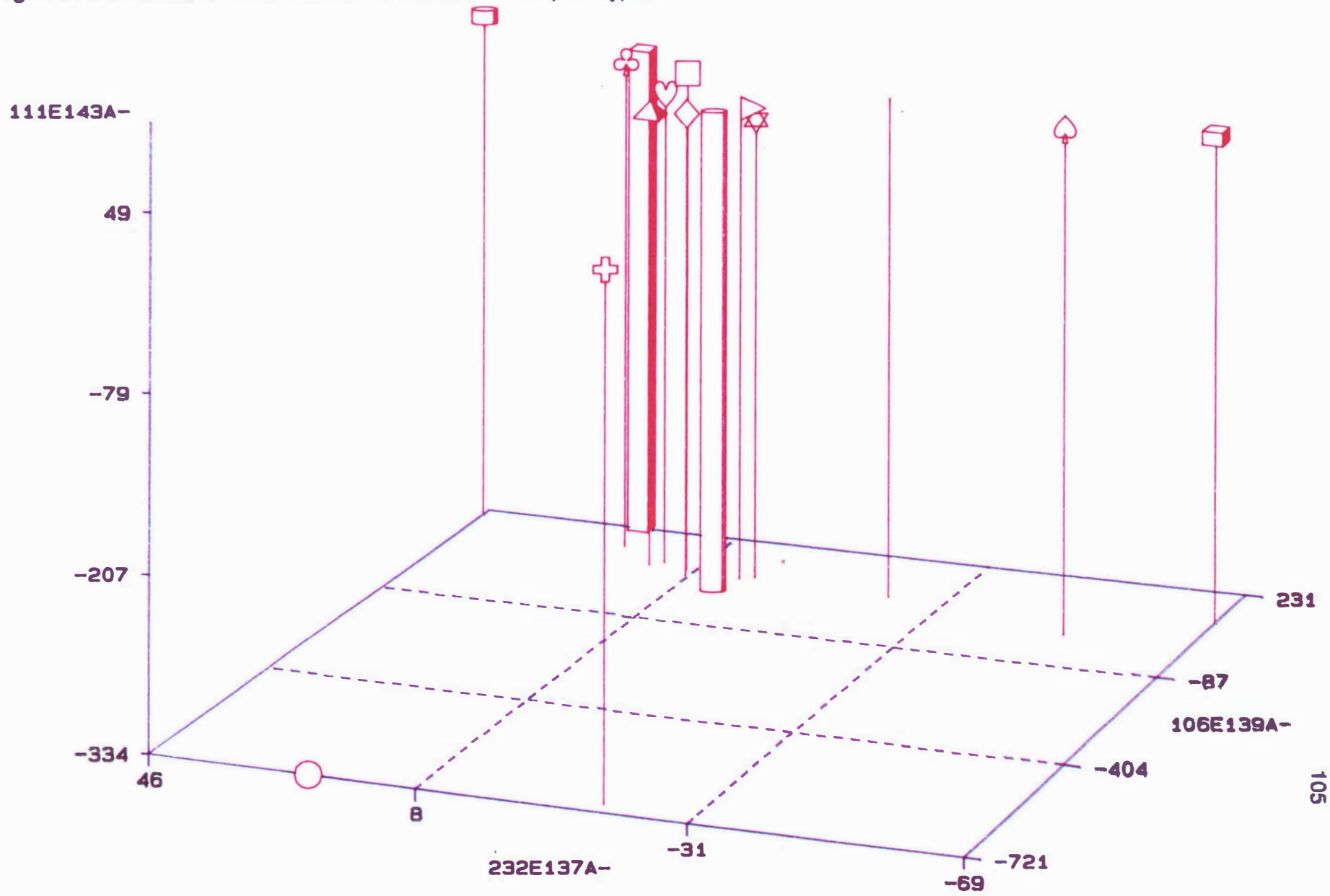
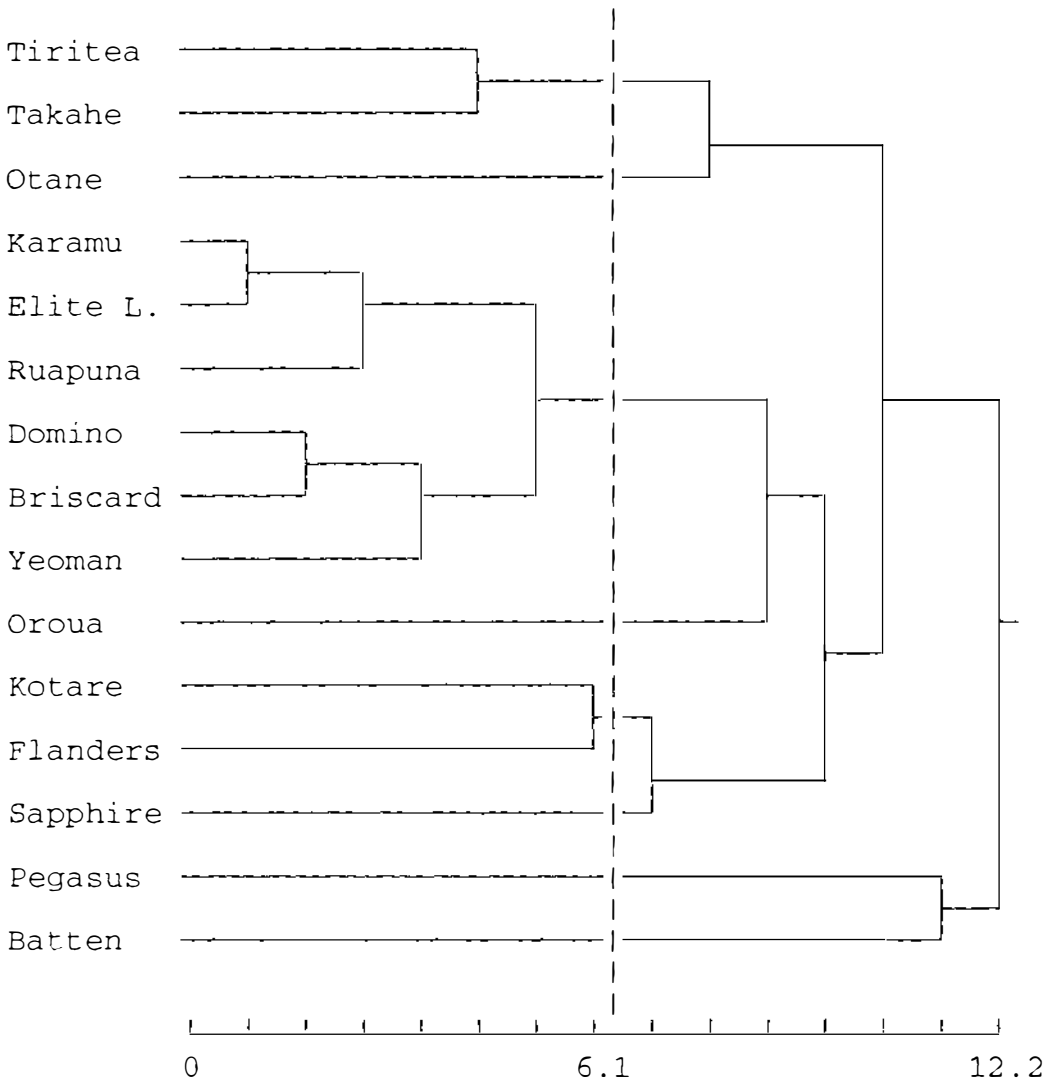


Table 13. Discriminant scores for fifteen cultivars for three pathotypes of stripe rust

Cultivar	Pathotype			Symbol
	232E137A-	111E143A-	106E139A-	
Oroua	-69.06	25.67	118.10	Cube
Pegasus	-17.62	37.90	-695.84	Cross
Sapphire	-51.13	38.13	16.99	Spade
Batten	23.19	-333.98	-721.26	Ballon
Karamu	-1.21	9.20	98.78	Star
Briscard	15.05	10.09	101.53	Pyramid
Domino	13.43	18.97	116.04	Heart
Otane	21.50	33.54	157.08	Club
Ruapuna	8.38	12.31	79.13	Diamond
Tiritea	47.40	48.52	214.41	Cylinder
Kotare	2.28	24.03	38.87	Pillar
Takahe	22.49	32.35	230.94	Prism
Elite Lep.	0.71	18.25	90.44	Flag
Yeoman	9.31	39.35	100.62	Square
Flanders	-22.07	37.69	81.49	Point

Fig. 9. Plot of scores of fifteen cultivars of wheat on three pathotypes





(Sum of Squares Between Clusters x 10^5)

Fig. 10. Ward's minimum variance cluster analysis of four components of resistance in response to three pathotypes between fifteen cultivars of wheat

Table 14. The probability of MANOVA cluster membership at each stage of clustering

Number of	df		Wilks' Lambda	Probability
stage	Num	Den	F ratio	
3	33	3.7	187.85	0.000737996771932
4	30	9.6	157.73	0.000030321140002
5	27	12.4	106.25	0.000008915696526
6	24	16.2	87.51	0.000000162909566
7	21	18.9	88.07	0.000000034419969
8	18	21.5	97.35	0.000000021467201
9	15	23.7	101.92	0.000000054910501
10	12	25.5	108.51	0.000000210904994
11	9	26.1	119.36	0.000000527880498
12	6	24.0	131.16	0.000000941134843
13	3	15.0	139.58	0.000002781479225

4.2. Specific diallel

To study inheritance of resistance in race-specific cultivars, five seedling resistant cultivars involving Oroua (Yr7), Pegasus (Yr1,6), Sapphire (6,7), Batten (Yr9), Karamu (YrA) and one susceptible cultivar, Tiritea, and their progenies were tested with two pathotypes in two diallel mating designs.

4.2.1. Graphical (Hayman) analysis

The analyses of variance for both pathotypes 111E143A- and 232E137A- are presented in Tables 15-22. All genotypes (parents, F_1,s and pooled), with exception of parents for latent period to pathotype 232E137A-, were found to be significantly different. This suggests that high genetic variability existed for the all components of resistance in the genotypes studied.

The genotypic means of all characters for both pathotypes are presented in Tables 23 & 24. In case of pathotype 111E143A-, which is virulent on Yr 1,6 and Yr7 (Cromey, pers. comm.), Pegasus, Sapphire and Oroua showed a high infection type as well as Tiritea which is susceptible to all pathotypes. Batten and its crosses showed a lower infection types, indicating the pathotype lacks allele(s) for virulence which one matched with those for resistance in Batten. For latent period, despite a large range of 2.52 days (Tiritea with 11.67 days and Karamu with 14.18 days) there were no significant differences for latent period (because of a large standard error). Since this pathotype is not virulent on Batten and its crosses, there were no pustules formation on some individual plants to record a latent period. These were arbitrarily given the value of 40 days (the end of experiment) for the purpose of a diallel analysis. On that basis, Batten and its crosses with Tiritea, Karamu and Oroua were in different classes. For pustule size, Sapphire was highly susceptible and then Tiritea and Pegasus, whereas, for pustule density, Tiritea had the most pustules, followed by Sapphire, Oroua and Pegasus. For all characters measured Batten and its crosses showed resistance.

For pathotype 232E137A-, which is virulent on Yr9 (Cromey, pers.comm.), Batten reacted as a highly susceptible cultivar and Karamu as a moderately susceptible cultivar,

whereas Oroua and Sapphire reacted as resistant and Pegasus reacted as a moderately resistant cultivar (Table 24). For latent period, Tiritea and its crosses with Karamu and Oroua, had the shortest latent period, whereas Oroua x Karamu and Oroua x Batten had the longest latent period. Apparently this pathotype is less virulent on Oroua compared with Pegasus and Sapphire. Batten had the biggest pustules whereas Oroua x Batten had the smallest. For pustule density Tiritea was a susceptible genotype and Sapphire x Batten and Oroua x Batten were resistant.

The mean estimates of array variance ($\bar{V}_r$), array common parent-offspring covariance ($\bar{W}_r$), variance of array means ($V_{\bar{r}}$) and variance of parent lines (V_p) are presented in Table 25 as well as the regression equations for W_r/V_r. For both pathotypes, the regression equations of all components of resistance were significant, except for latent period in response to pathotype 232E137A-, indicating the regression slope was significantly different from zero.

Except for pustule size in response to pathotype 111E143A- and latent period in response to pathotype 232E137A-, the W_r+V_r for both pathotypes were found to be heterogeneous (Tables 26 & 27), indicating that dominance was present.

The W_r-V_r for latent period for pathotype 111E143A- and all components of resistance in response to pathotype 232E137A- were significant. Non-significance of W_r-V_r implies a lack of any non-allelic interaction, and significance of that implies that there is non-allelic interaction.

The deviation of regression slope from unity was not significant, except for latent period and pustule density in response to pathotype 232E137A-. The deviation from unity indicates graphical distortion was due to one or more of epistasis, correlated gene distributions and environment (Mather and Jinks, 1982).

Components of variation and other genetic statistics are presented in Tables 26 & 27. The additive genetic variance (D) was greater than dominance genetic variance (H_1 and H_2) for infection type (both pathotypes), latent period, pustule size and pustule

density in the case of pathotype 111E143A-, indicating a preponderance of additive genetic variance. The H_1 and H_2 were positive in all cases. The F value was positive, except for latent period and pustule size in response to pathotype 232E137A-. The positive sign of the F value indicated that dominance alleles were more frequent than the recessive alleles.

The average degree of dominance, $(H_1/D)^{1/2}$, was less than unity for all components of resistance to pathotype (both pathotypes), except for latent period, pustule size and number in response to pathotype 232E137A- which was more than unity. If the average degree of dominance is less than one it indicates partial dominance and greater than one indicates over dominance is present.

The positive and negative alleles as shown by uv values were less than 0.25, except for pustule density to pathotype 232E137A- where it was close to 0.25. A uv of less than 0.25, thus suggests inequality of distribution of increasing and decreasing alleles.

The estimates of the consistency of expression of the degree of dominance across all segregating loci were between zero and one, except for latent period in response to 232E137A-, which was more than unity. The absolute value of the statistic varies from 0 to 1, where 1 indicates a constant dominance level over all loci.

The ratio of dominant to recessive alleles varied for all components of resistance to both pathotypes.

Estimates of narrow sense and broad sense heritability are presented in Tables 26 & 27 for all characters (both pathotypes). Except for latent period and pustule size to 232E137A- which had a moderate narrowsense heritability, all characters showed a high narrowsense heritability. This indicated that the additive genetic variance is more important than dominance, which was found previously from the genetic statistics.

The correlations between common parent mean and (W_r+V_r) were positive, high, moderate to low, except for pustule size (111E143A-) and pustule density (both

pathotypes) which were negative.

The ratio of σ^2_A/σ^2_D varied from component to component. This ratio indicated that there may be an excess of genes with additive effects in the parents for the all components except for latent period, pustule size and pustule density in response to pathotype 232E137A-. These agreed with genetic statistics which was found previously.

An estimate of effective factors for infection type (both pathotypes), and pustule density (232E137A-) was unable to be interpreted as it was below the value one. However for other characters it was estimated between 2-4 genes.

Ranking of arrays with respect to the proportion of dominant alleles in the parents may be obtained from W_r/V_r plots (Fig. 11-14). For infection type (Fig. 11), Batten was close to the origin, indicating that it had the greatest proportion of dominant alleles. In decreasing order of dominance, the other parents were Sapphire, Oroua, Pegasus and Tiritea. Karamu had the greatest proportion of recessive alleles in response to pathotype 111E143A-. With respect to pathotype 232E137A-, Batten had greatest proportion of the recessive alleles and in increasing order of dominance, Pegasus, Sapphire, Oroua and Karamu had lower dominant alleles than Tiritea. For latent period (Fig. 12), Batten had the highest proportion of recessive genes in response to both pathotypes. Pegasus and Sapphire had the highest proportion of dominant alleles to pathotype 111E143A-, whereas Tiritea had highest proportion of dominant alleles in response to pathotype 232E137A-. For pustule size (Fig. 13) to pathotype 111E143A-, Batten, Tiritea and Oroua showed the highest proportion of recessive alleles, where as Pegasus had the highest the dominant alleles, and the other cultivars were in the middle. In the case of pathotype 232E137A-, Batten kept its recessive position, but the other cultivars were grouped close to the origin, indicating that they had highest proportion of dominant alleles. For pustule density (Fig. 14), Batten had the highest proportion of recessive alleles in response to all pathotypes, whereas Karamu and Tiritea (111E143A-) and Tiritea (232E137A-) showed highest the recessive alleles.

4.2.2. Griffing combining ability analysis

The analysis of variance for general combining ability (GCA) and specific combining ability (SCA) showed that both combining abilities values were highly significant (Tables 28 & 29). Although SCA was highly significant, the ratio $2MS_{GCA}/(2MS_{GCA} + 2MS_{SCA})$ for all components was more than 0.93, except for latent period and pustule density (232E137A-) which was more than 0.80. This indicted the relatively higher magnitude of the GCA variance over SCA variance.

All estimates of GCA and SCA effects for components of resistance (both pathotypes) are presented in Tables 30-37. In response to pathotype 111E143A- (Tables 30-33), Batten had the highest GCA for lower infection type, for longest latent period, smaller pustule size and number than other cultivars, followed by Karamu. It should be noted that resistance in terms of combining ability is negative for infection type, pustule size and number, but is positive for latent period. In the case of SCA, Batten x Tiritea for infection type, Batten x Oroua for latent period, Batten x Tiritea for pustule size and Karamu x Tiritea for pustule density had the highest SCA, indicating dominance for those hybrids for higher resistance.

In response to pathotype 232E137A- (Tables 34-37), Oroua for infection type, Tiritea for latent period, and Oroua for pustule size and number had the highest values, which indicate as the best combining ability and the presence of additive gene action involved in the inheritance of resistance. The greatest SCA occurred in : Oroua x Batten for infection type, latent period and pustule size, Sapphire x Batten and Oroua x Batten for pustule density, suggesting that nonadditive gene action, such as dominance and epistasis, effects were involved in inheritance of stripe rust resistance in these crosses.

The ranking of cultivars according to their performance (Tables 23 & 24) was almost same as their GCA (Tables 30-37) for all characters in response to those two pathotypes. Narrowsense and broadsense heritabilities were very high (>0.79).

Table 15. Analysis of variance for infection type of a 6 x 6 diallel inoculated with stripe rust pathotype 111E143A-

S.O.V.	Parents		F1,s		Pooled	
	df	MS	df	MS	df	MS
Block	1	0.31 ns	1	0.95 **	1	0.26 ns
Genotypes	5	17.71 **	14	9.31 **	20	11.02 **
Error	5	0.07	14	0.09	20	0.13

Table 16. Analysis of variance for latent period of a 6 x 6 diallel inoculated with stripe rust pathotype 111E143A-

S.O.V.	Parents		F1,s		Pooled	
	df	MS	df	MS	df	MS
Block	1	0.28 ns	1	1.38 **	1	0.53 ns
Genotypes	5	1.93 *	14	0.68 **	20	0.91 **
Error	5	0.23	14	0.15	20	0.28

Table 17. Analysis of variance for pustule size of a 6 x 6 diallel inoculated with stripe rust pathotype 111E143A-

S.O.V.	Parents		F1,s		Pooled	
	df	MS	df	MS	df	MS
Block	1	6.6 ns	1	0.02 ns	1	2.27 ns
Genotypes	5	122.2 *	14	24.42 **	20	45.30 **
Error	5	20.2	14	3.04	20	6.72

Table 18. Analysis of variance for pustule density of a 6 x 6 diallel inoculated with stripe rust pathotype 111E143A-

S.O.V.	Parents		F1,s		Pooled	
	df	MS	df	MS	df	MS
Block	1	226.3 ns	1	20.5 ns	1	18.0 ns
Genotypes	5	830.4 *	14	842.9 **	20	798.2 **
Error	5	79.5	14	86.9	20	92.8

ns, * & ** : not significant, significant at P = 0.05 & P = 0.01, respectively

Table 19. Analysis of variance for infection type of a 6 x 6 diallel inoculated with stripe rust pathotype 232E137A-

S.O.V.	Parents		F1,s		Pooled	
	df	MS	df	MS	df	MS
Block	1	0.07 ns	1	0.08 ns	1	0.01 ns
Genotypes	5	10.27 *	14	4.61 **	20	5.81 **
Error	5	0.10	14	0.10	20	0.11

Table 20. Analysis of variance for latent period of a 6 x 6 diallel inoculated with stripe rust pathotype 232E137A-

S.O.V.	Parents		F1,s		Pooled	
	df	MS	df	MS	df	MS
Block	1	0.23 ns	1	0.004 ns	1	0.12 ns
Genotypes	5	0.73 ns	14	0.989 **	20	0.87 **
Error	5	0.39	14	0.204	20	0.25

Table 21. Analysis of variance for pustule size of a 6 x 6 diallel inoculated with stripe rust pathotype 232E137A-

S.O.V.	Parents		F1,s		Pooled	
	df	MS	df	MS	df	MS
Block	1	0.01 ns	1	0.93 *	1	0.57 ns
Genotypes	5	35.40 **	14	20.83 **	20	28.13 **
Error	5	1.72	14	0.16	20	0.56

Table 22. Analysis of variance for pustule density of a 6 x 6 diallel inoculated with stripe rust pathotype 232E137A-

S.O.V.	Parents		F1,s		Pooled	
	df	MS	df	MS	df	MS
Block	1	101.8 ns	1	54.4 ns	1	136.3 ns
Genotypes	5	1777.8 **	14	1386.0 **	20	1414.6 **
Error	5	91.4	14	121.4	20	108.85

ns, * & ** : not significant, significant at P = 0.05 & P = 0.01, respectively

Table 23. Duncan's multiple ranges of six cultivars and their progenies for four components of resistance inoculated with stripe rust pathotype 111E143A

Cultivar	Infection Type (scale 0-9)	Latent Period (days)	Pustule Size (mm^2 x 1000)	Pustule density (x100)/mm^2
Tiritea	9.00 a	11.67 a	44.04 bc	231.1 a
Pegasus	8.83 ab	11.73 a	42.98 bc	188.1 efgh
Tiritea/Pegasus	8.42 abc	11.92 a	45.38 bc	224.95 ab
Pegasus/Karamu	8.33 abcd	11.83 a	42.59 bc	220.3 ab
Sapphire	8.08 bcde	12.50 a	55.41 a	219.9 ab
Oroua/Pegasus	7.75 cdef	12.00 a	44.98 bc	224.5 ab
Tiritea/Sapphire	7.58 defg	12.50 a	44.36 bc	222.8 ab
Tiritea/Karamu	7.56 defg	11.56 a	39.81 bcd	205.6 bcdef
Pegasus/Sapphire	7.50 defg	12.00 a	45.82 b	218.6 abc
Oroua	7.50 defg	12.58 a	38.50 cde	191.8 defgh
Tiritea/Oroua	7.33 efg	12.17 a	42.23 bc	232.0 a
Sapphire/Oroua	7.25 efg	12.25 a	41.60 bc	214.3 abcd
Sapphire/Karamu	7.00 fg	12.00 a	41.65 bc	212.0 abcde
Oroua/Karamu	6.75 g	12.00 a	38.55 cde	213.1 abcd
Batten/Pegasus	3.58 h	13.00 a	33.49 ed	156.5 i
Karamu	3.55 h	14.18 a	34.37 ed	194.9 cdefg
Sapphire/Batten	3.29 hi	13.50 a	32.89 e	170.1 hi
Tiritea/Batten	2.83 hi	24.50 b	22.25 f	186.4 fgh
Karamu/Batten	2.83 hi	24.08 b	24.30 f	177.5 ghi
Batten/Oroua	2.58 ij	28.67 b	22.61 f	174.2 ghi
Batten	1.92 j	40.00 c	10.00 g	70.7 j

Means with the same letter within column are not significantly different (P=0.05)

Table 24. Duncan's multiple ranges of six cultivars and their progenies for four components of resistance inoculated with stripe rust pathotype 232E137A-

Cultivar	Infection Type (scale 0-9)	Latent Period (days)	Pustule Size (mm^2 x 1000)	Pustule density (x100)/mm^2
Tiritea	8.92 a	11.00 a	37.81 b	228.10 a
Batten	8.43 ab	11.43 abc	39.75 a	172.15 efg
Tiritea/Batten	8.27 ab	11.27 abc	36.34 b	208.20 abc
Tiritea/Karamu	8.13 ab	11.00 a	30.59 ef	214.08 ab
Karamu/Batten	7.75 bc	12.00 abcd	28.76 gh	168.81 fg
Sapphire/Karamu	7.22 cd	12.33 abcd	28.77 gh	175.69 efg
Tiirtea/Sapphire	7.08 cde	11.50 abcd	32.43 cd	195.23 bcde
Tiritea/Pegasus	6.73 de	11.18 ab	33.38 c	208.91 abc
Batten/Pegasus	6.70 de	12.20 abcd	28.81 gh	195.88 bcde
Tiritea/Oroua	6.67 de	11.08 a	32.60 cd	201.57 bcd
Pegasus/Karamu	6.42 def	12.08 abcd	32.34 cde	175.33 efg
Karamu	6.25 ef	12.25 abcd	31.43 def	195.77 bcde
Pegasus/Sapphire	5.70 fg	12.20 abcd	31.06 def	188.66 bcdef
Pegasus	5.30 gh	11.70 abcd	31.51 def	180.65 defg
Oroua/Karamu	5.00 gh	12.86 d	28.20 h	164.04 fgh
Sapphire/Oroua	4.67 h	11.78 abcd	28.76 gh	187.60 cdef
Sapphire/Batten	3.67 i	12.17 abcd	24.81 i	132.22 i
Sapphire	3.57 i	12.50 abcd	30.21 fg	157.97 gh
Oroua/Pegasus	3.00 ij	12.86 d	28.53 gh	140.54 ih
Oroua	2.73 j	12.64 cd	29.91 fgh	142.08 ih
Batten/Oroua	2.56 j	14.29 e	21.68 j	120.15 i

Means with the same letter within column are not significantly different (P=0.05)

Table 25. W_r/V_r regression equations and basic array statistics for stripe rust resistance in six cultivars of wheat

character[1]	pathotype	regression equation	F-test	V_p	$\bar{V}_r$	$\bar{W}_r$	$V_{\bar{r}}$
IT	111E143A-	$W_r = 0.89 + 1.03\ V_r$	74.52 **	8.98	3.42	1.05	2.79
	232E137A-	$W_r = 0.86 + 0.72\ V_r$	9.92 *	6.44	2.59	0.18	1.51
LP	111EA43A-	$W_r = 20.80 + 0.82\ V_r$	13.60 **	126.31	32.85	15.12	19.20
	232E137A-	$W_r = 0.23 + 0.04\ V_r$	0.07 ns	0.40	0.42	-0.16	0.20
PS	111E143A-	$W_r = 47.70 + 1.01\ V_r$	125.85 **	228.58	51.09	51.52	47.20
	232E137A-	$W_r = -3.97 + 0.64\ V_r$	10.36 *	17.45	13.16	-8.55	3.57
PD	111E143A-	$W_r = 577.00 + 1.00\ V_r$	145.91 **	3262.78	619.01	620.83	469.72
	232E137A-	$W_r = 237.00 + 0.41\ V_r$	4.75 (*)	858.13	609.42	-100.31	309.90

[1] Infection Type, Latent Period, Pustule Size and Pustule density respectively

ns, * & ** : not significant, significant at P = 0.05 & P = 0.01, respectively

Table 26. Genetic statistics for stripe rust resistance in six cultivars of wheat

	111E143A-			
statistics	Infection Type	Latent Period	Pustule Size	Pustule density
$W_r + V_r$	**	**	ns	**
$W_r - V_r$	ns	**	ns	ns
t-test(β - 1)	0.25 ns	0.81 ns	0.11 ns	0.00 ns
D	8.97	126.22	221.78	3229.69
H_1	4.74	65.52	8.99	642.28
H_2	2.45	54.52	7.11	466.26
F	0.10	60.71	26.55	1553.04
$\sqrt{(H_1/D)}$	0.73	0.72	0.21	0.45
uv (H_2 / $4H_1$)	0.13	0.21	0.20	0.18
$0.5F / \sqrt{[D(H_1 - H_2)]}$	0.01	0.81	0.67	1.03
dom./rec. genes	1.02	2.00	1.87	3.34
h^2_{BS}	98%	95%	93%	92%
h^2_{NS}	88%	74%	62%	82%
r (P_r , $W_r + V_r$)	0.43	0.78	-0.35	-0.93
σ^2_A / σ^2_D	7.34	4.65	59.60	13.85
K (effective factors)	0.31	1.63	2.72	3.70
E_{pool}	0.13	0.28	6.71	92.83
$E_{parents}$	0.07	0.23	20.16	79.50
$E_{hybrids}$	0.09	0.23	3.04	86.93

ns, * & ** : not significant, significant at P = 0.05 & P = 0.01, respectively

Table 27. Genetic statistics for stripe rust resistance in six cultivars of wheat

	232E137A-			
Statistics	Infection Type	Latent Period	Pustule Size	Pustule density
$W_r + V_r$	**	ns	**	*
$W_r - V_r$	**	*	**	**
t-test(β - 1)	1.22 ns	6.00 **	1.80 ns	3.11 **
D	6.40	0.14	16.01	821.16
H_1	5.56	0.59	50.78	1035.33
H_2	4.17	0.50	37.89	991.42
F	1.80	-0.50	14.74	-333.20
$\sqrt{(H_1/D)}$	0.93	2.09	1.78	1.12
uv (H_2 / $4H_1$)	0.19	0.21	0.19	0.24
$0.5F / \sqrt{[D(H_1 - H_2)]}$	0.30	-2.17	0.51	-0.88
dom./rec. genes	1.36	0.06	1.70	0.69
h^2_{BS}	98%	66%	97%	89%
h^2_{NS}	72%	49%	41%	63%
r (P_r , $W_r + V_r$)	0.37	0.29	0.84	-0.56
σ^2_A / σ^2_D	3.07	0.55	0.85	1.66
K (effective factors)	0.01	2.24	1.39	0.01
E_{pool}	0.11	0.25	0.56	108.85
$E_{parents}$	0.10	0.39	1.72	91.40
$E_{hybrids}$	0.10	0.20	0.16	121.42

ns, * & ** : not significant, significant at P = 0.05 & P = 0.01, respectively

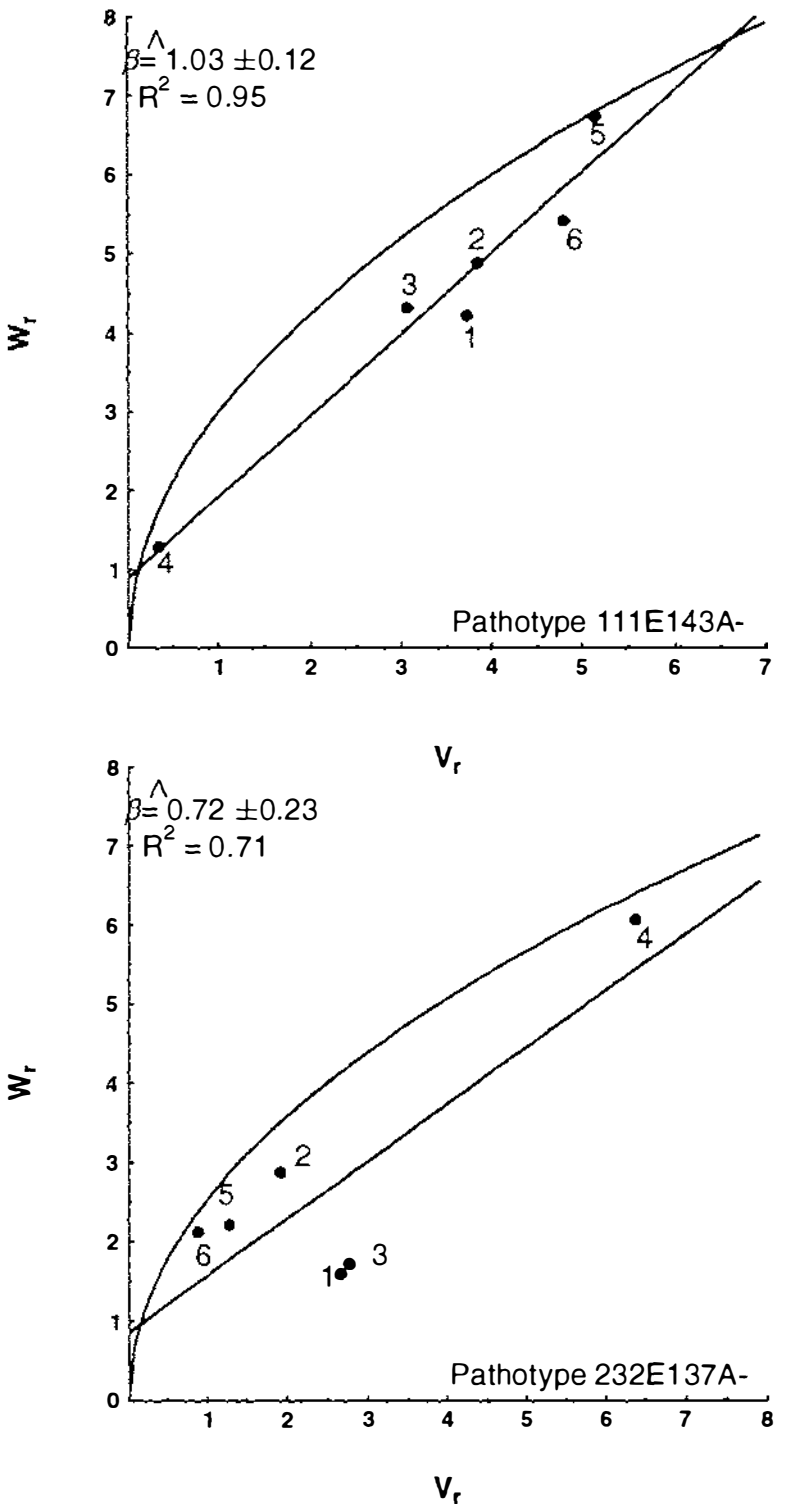


Fig. 11. The W_r/V_r graphs of infection type from crosses of six cultivars of wheat (1= Oroua, 2= Pegasus, 3= Sapphire, 4= Batten and 5= Karamu, 6= Tiritea) in response to two pathotypes of stripe rust

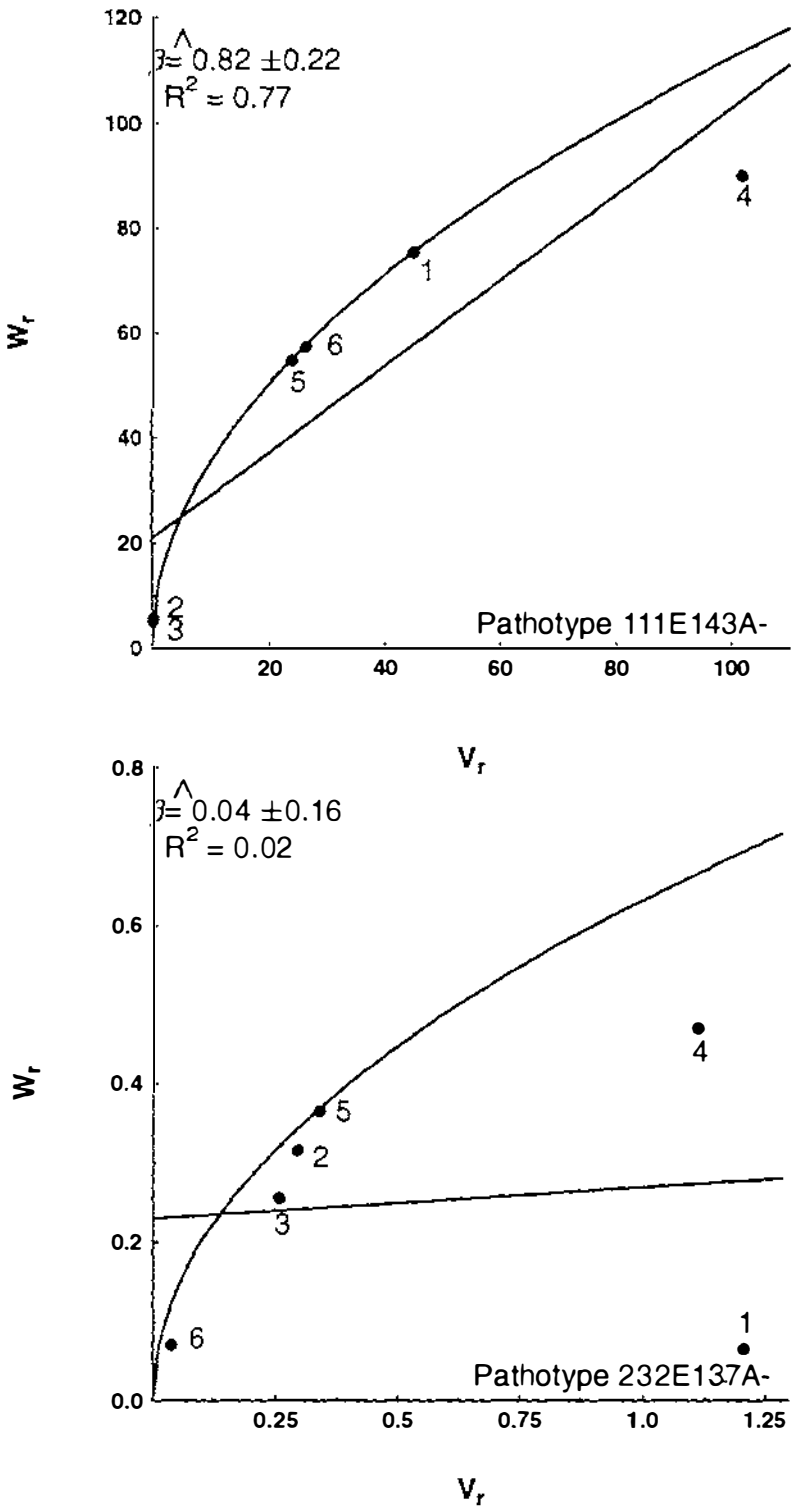


Fig. 12. The W_r/V_r graphs of latent period from crosses of six cultivars of wheat (1= Oroua, 2= Pegasus, 3= Sapphire, 4= Batten and 5= Karamu, 6= Tiritea) in response to two pathotypes of stripe rust

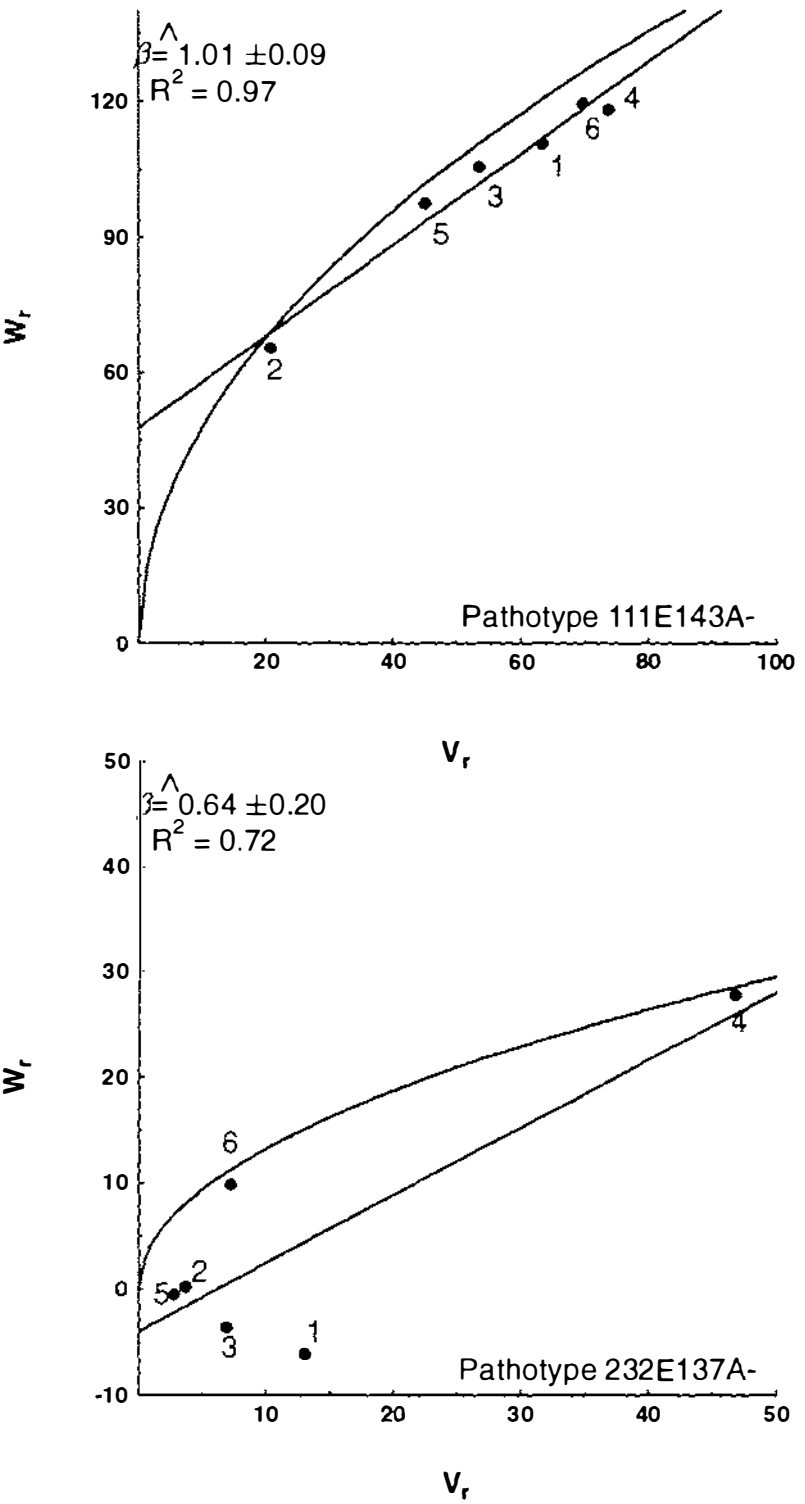


Fig. 13. The W_r/V_r graphs of pustule size from crosses of six cultivars of wheat (1= Oroua, 2= Pegasus, 3= Sapphire, 4= Batten and 5= Karamu, 6= Tiritea) in response to two pathotypes of stripe rust

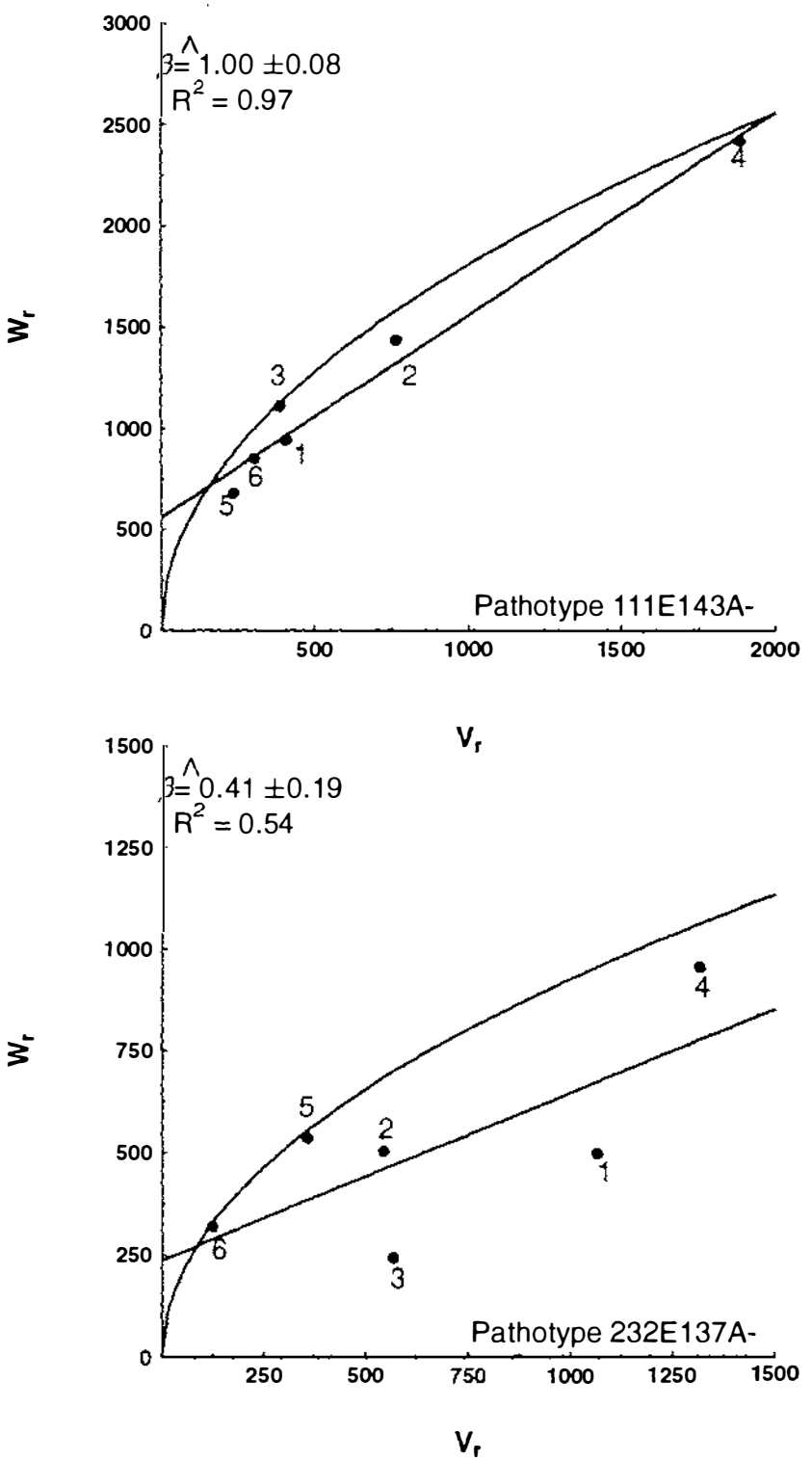


Fig. 14. The W_r/V_r graphs of pustule density from crosses of six cultivars of wheat (1= Oroua, 2= Pegasus, 3= Sapphire, 4= Batten and 5= Karamu, 6= Tiritea) in response to two pathotypes of stripe rust

Table 28. Mean squares of general and specific combining ability for four components of resistance to stripe rust pathotype 111E143A-

S.O.V.	df	MS[1]			
		IT	LP	PS	PD
GCA[2]	5	20.4 **	171.1 **	403.8 **	4497.6 **
SCA_3	15	1.0 **	16.1 **	8.7 **	261.5 **
Error	199	0.5	2.5	3.6	32.1
Ratio[4]		0.98	0.96	0.99	0.97
h^2_{BS}[5]		0.99	0.99	0.99	0.99
h^2_{NS}[6]		0.96	0.95	0.98	0.97

Table 29. Mean squares of general and specific combining ability for four components of resistance to stripe rust pathotype 232E137A-

S.O.V.	df	MS[1]			
		IT	LP	PS	PD
GCA[2]	5	11.6 **	1.1 **	25.6 **	2280.6 **
SCA[3]	15	1.6 **	0.4 **	13.1 **	347.4 **
Error	199	0.5	0.04	0.2	47.3
Ratio[4]		0.94	0.85	0.80	0.93
h^2_{BS}[5]		0.98	0.99	0.99	0.99
h^2_{NS}[6]		0.92	0.83	0.79	0.92

[1] Mean Squares of Infection Type, Latent Period, Pustule Size and Pustule density
[2] General Combining Ability
[3] Specific Combining Ability
[4] $2MS_{GCA}/(2MS_{GCA} + MS_{SCA})$
[5] Broad sense heritability
[6] Narrow sense heritability
** significant at P = 0.01

Table 30. Estimates of general combining ability (diagonal) and specific combining ability (above diagonal) for infection type to stripe rust pathotype 111E143A-

	Oroua	Pegasus	Sapphire	Batten	Karamu	Tiritea
Oroua	0.44**	-0.11ns	-0.06ns	-1.00**	0.60**	-0.34**
Pegasus		1.26**	-0.63**	-0.82**	1.35**	-0.08ns
Sapphire			0.70**	-0.55**	0.58**	-0.36**
Batten				-3.03**	0.14ns	-1.38**
Karamu					-0.45**	0.77**
Tiritea						1.07**

$S.E._{GCA}$= 0.0340 $S.E._{SCA}$=0.0934

Table 31. Estimates of general combining ability (diagonal) and specific combining ability (above diagonal) for latent period to stripe rust pathotype 111E143A-

	Oroua	Pegasus	Sapphire	Batten	Karamu	Tiritea
Oroua	-0.91**	0.18ns	0.30ns	4.65**	-1.77**	-0.87**
Pegasus		-2.92**	2.06**	-9.01**	2.07**	0.88**
Sapphire			-2.79**	-8.64**	0.10ns	1.33**
Batten				9.28**	0.12ns	1.27**
Karamu					-0.96**	-1.43**
Tiritea						-1.70**

$S.E._{GCA}$= 0.0490 $S.E._{SCA}$=0.1371

Table 32. Estimates of general combining ability (diagonal) and specific combining ability (above diagonal) for pustule size to stripe rust pathotype 111E143A-

	Oroua	Pegasus	Sapphire	Batten	Karamu	Tiritea
Oroua	0.55 *	2.47**	-3.28**	-2.07**	1.36ns	1.73 *
Pegasus		4.45**	-2.96**	4.91**	1.50 *	0.97ns
Sapphire			6.82**	1.95**	-1.82 *	-2.41**
Batten				-13.38**	1.04ns	-4.32**
Karamu					-0.87**	0.73ns
Tiritea						2.44**

$S.E._{GCA}$= 0.2420 $S.E._{SCA}$=0.6717

Table 33. Estimates of general combining ability (diagonal) and specific combining ability (above diagonal) for pustule density to stripe rust pathotype 111E143A-

	Oroua	Pegasus	Sapphire	Batten	Karamu	Tiritea
Oroua	7.33**	14.84**	-2.43ns	16.41**	3.77ns	8.26ns
Pegasus		4.74**	4.46ns	1.31ns	13.56**	3.76 *
Sapphire			11.81**	7.83**	-1.81ns	-5.47**
Batten				-47.12**	22.63**	17.11**
Karamu					4.40**	-15.26**
Tiritea						18.85

$S.E._{GCA}$= 0.8980 $S.E._{SCA}$= 2.4959

Table 34. Estimates of general combining ability (diagonal) and specific combining ability (above diagonal) for infection type to stripe rust pathotype 232E137A-

	Oroua	Pegasus	Sapphire	Batten	Karamu	Tiritea
Oroua	-1.66**	-0.99**	1.14**	-2.26**	0.03ns	2.01**
Pegasus		-0.37**	0.88**	0.59**	0.16ns	-0.68**
Sapphire			-0.83**	-1.98**	1.42**	0.13ns
Batten				0.47**	0.66**	0.30ns
Karamu					0.62**	-0.26ns
Tiritea						1.76**

$S.E._{GCA}$= 0.0310 $S.E._{SCA}$= 0.0859

Table 35. Estimates of general combining ability (diagonal) and specific combining ability (above diagonal) for latent period to stripe rust pathotype 232E137A-

	Oroua	Pegasus	Sapphire	Batten	Karamu	Tiritea
Oroua	0.46**	0.40 *	-0.81**	1.60**	0.29ns	-0.77**
Pegasus		-0.07ns	0.14ns	0.03ns	0.04ns	-0.14ns
Sapphire			0.07ns	-0.13ns	0.16ns	0.05ns
Batten				0.17**	-0.27ns	0.72ns
Karamu					0.04ns	-0.43 *
Tiritea						-0.68**

$S.E._{GCA}$= 0.0470 $S.E._{SCA}$= 0.1296

Table 36. Estimates of general combining ability (diagonal) and specific combining ability (above diagonal) for pustule size to stripe rust pathotype 232E137A-

	Oroua	Pegasus	Sapphire	Batten	Karamu	Tiritea
Oroua	-2.04**	-0.43 *	1.16**	-7.63**	-0.06ns	0.66**
Pegasus		0.16**	1.27**	-2.69**	1.89**	-0.75**
Sapphire			-1.21**	-5.33**	-0.32ns	-0.34ns
Batten				0.50**	-2.04**	1.86**
Karamu					-0.55**	-2.84**
Tiritea						3.13**

$S.E._{GCA}$= 0.0700 $S.E._{SCA}$= 0.1939

Table 37. Estimates of general combining ability (diagonal) and specific combining ability (above diagonal) for pustule density to stripe rust pathotype 232E137A-

	Oroua	Pegasus	Sapphire	Batten	Karamu	Tiritea
Oroua	-19.39**	-21.27**	35.19**	-29.77**	-0.13ns	12.31**
Pegasus		2.18 *	14.68**	24.39**	-10.41**	-1.92ns
Sapphire			-7.22**	-29.87**	-0.65ns	-6.11 *
Batten				-9.72**	-5.04ns	15.15**
Karamu					4.54**	0.90ns
Tiritea						29.62**

$S.E._{GCA}$= 0.9720 $S.E._{SCA}$= 2.7032

4.3. Specific vs nonspecific factorial mating design

To understand the interaction of adult plant resistance factors and seedling resistance factors, four cultivars with adult plant resistance and five cultivars with seedling resistance to stripe rust were intercrossed in a factorial mating fashion.

The analyses of variance for both pathotypes are presented in Table 38 & 39. For pathotype 111E143A-, the analysis of variance indicated the presence of highly significant variation among cultivars with the adult plant resistance for pustule density only whereas all characters were highly significant for cultivars with the seedling resistance. The interactions between cultivars with seedlinh resistance and adult plant resistance were significant for pustule size ($P < 0.01$) and pustule density ($P < 0.05$) only. For pathotype 232E137A-, the analysis of variance showed that cultivars with the adult plant resistance , cultivars with the seedling resistance and their interactions, were significant for all characters.

Componenets of resistance to stripe rust of all hybrids for all characters in response to pathotype 111E143A- are presented in Table 40. The lowest infection type was observed in F_1's which had Batten as a parent and the highest was observed in those F_1's which had Pegasus as a parent. Apart from the crosses with Batten which had a hypersensitive reaction and were given an arbitrary value of latent period, there were no differences between hybrids for latent period. Ignoring the crosses of Batten, the smallest pustule size was observed in Oroua x Ruapuna and the largest was observed in Pegasus x Otane; and the lowest and highest pustule densities were observed in Karamu x Ruapuna and Sapphire x Briscard, respectively.

Componenets of resistance to stripe rust of all F_1's in response to pathotype 232E137A- are presented in Table 41. Hybrids with Batten were generally the most susceptible genotypes, although the Batten x Domino as a moderately susceptible. Sapphire x Domino had the lowest infection type, it was followed by Oroua x Domino. The genotype Karamu x Otane had the shortest latent period and Sapphire x Domino had the longest latent period. All crosses with Batten, except that with Domino, had the largest pustule size and Sapphire x Domino had the smallest pustule size. The lowest

pustule density was observed in the cross Sapphire x Briscard whereas the highest one was observed in cross Karamu x Otane.

For both pathotypes, estimates of components of variance and their standard errors for all characters are presented in Table 42. In response to pathotype 111E143A-, for cultivars with the adult plant resistance the additive genetic variance was negative for all characters except for pustule density. The additive genetic variance for cultivars with the seedling resistance was positive and high in comparison with the additive genetic variance of cultivars with the adult plant resistance. Dominance genetic variance for all characters was smaller than additive genetic variance. In response to pathotype 232E137A-, the additive genetic variance of the seedling resistant cultivars was larger than the additive genetic variance of adult plant resistant cultivars for all characters, except for pustule density for which they were similar. The dominance variance was smaller than the additive part.

In the case of infection type, the degree of dominance for each cross was estimated (Table 43 & 44). In response to pathotype 111E143A-, the degree of dominance was negative, except for all crosses with Karamu, indicating dominance for the lower infection type. Dominance was for higher infection type for crosses with Karamu. The degree of dominance was similar for crosses with Batten and Pegasus. In response to pathotype 232E137A-, dominance was for lower infection type for crosses with Oroua and Pegasus whereas for Sapphire, Batten and Karamu, dominance was different (for lower or higher infection type) depending on the cross.

Table 38. North Carolina II analysis of variance for four components of resistance in response to stripe rust pathotype 111E143A-

S.O.V.	df	MS			
		IT	LP	PS	PD
Block	1	14.70 **	9.87 ns	0.02 ns	56.22 ns
Adult plant cultivars	3	1.18 ns	28.30 ns	28.65 ns	3802.41 **
Seedling cultivars	4	197.25 **	2232.82 **	3511.83 **	65443.38 **
Adult x Seedling	12	2.11 ns	35.32 ns	138.69 **	1472.38 *
Adult - Seedling x Block	19	1.35	34.88	40.04	465.90

Table 39. North Carolina II analysis of variance for four components of resistance in response to stripe rust pathotype 232E137A-

S.O.V.	df	MS			
		IT	LP	PS	PD
Block	1	0.01 ns	0.10 ns	2.43 ns	64.66 ns
Adult plant cultivars	3	16.28 **	29.36 **	27.24 **	10789.76 **
Seedling cultivars	4	40.28 **	41.80 **	121.60 **	9484.92 **
Adult x Seedling	12	3.88 **	18.26 **	20.30 **	2064.13 *
Adult - Seedling x Block	19	0.46	1.24	4.82	761.00

ns : not significant, * and ** : significant at P = 0.05 and 0.01, respectively

Table 40. Components of resistance to stripe rust pathotype 232E137A- of twenty F_1 hybrids among cultivars differing in resistance

Hybrid	Infection Type	Latent Period	Pustule Size	Pustule Density
Pegasus/Otane	8.17 a	11.33 c	46.48 a	197.9 abcde
Sapphire/Briscard	7.75 abc	12.50 c	45.46 ab	218.2 a
Pegasus/Briscard	7.75 abc	11.50 c	41.99 abcd	211.9 ab
Pegasus/Ruapuna	7.58 abcd	12.75 c	42.13 abcd	192.9 bcdef
Sapphire/Otane	7.40 abcde	12.20 c	37.85 cdef	198.1 abcde
Pegasus/Domino	7.33 abcde	11.42 c	38.99 bcdef	207.7 abc
Oroua/Otane	7.33 abcde	12.00 c	41.53 abcdef	193.4 bcdef
Oroua/Briscard	7.17 abcdef	12.33 c	41.26 abcdef	207.8 abc
Sapphire/Ruapuna	7.09 abcdef	12.55 c	44.49 abcd	182.0 ef
Sapphire/Domino	6.91 abcdefg	12.55 c	44.77 abc	181.5 ef
Oroua/Ruapuna	6.83 bcdefg	13.33 c	36.57 ef	185.0 def
Karamu/Domino	6.50 cdefgh	12.00 c	45.23 abg	183.5 ef
Oroua/Domino	6.00 fgh	13.25 c	39.73 abcdef	205.2 abcd
Karamu/Otane	5.82 gh	12.46 c	39.66 abcdef	192.2 bcdef
Karamu/Briscard	5.50 h	11.67 c	37.10 ef	196.2 bcde
Karamu/Ruapuna	5.50 h	12.70 c	42.23 abcd	177.0 ef
Batten/Ruapuna	2.75 i	26.83 b	24.28 g	120.5 g
Batten/Domino	2.50 i	24.83 b	24.31 g	120.2 g
Batten/Briscard	2.40 i	26.90 b	23.82 g	122.2 g
Batten/Otane	2.25 i	33.17 a	16.05 h	79.8 h

Means with the same letter within the columns are not significantly different (Duncan's multiple ranges $P<0.05$)

Table 41. Components of resistance to stripe rust pathotype 232E137A- of twenty F_1 hybrids among cultivars differing in resistance

Hybrid	Infection Type	Latent Period	Pustule Size	Pustule Density
Batten/Briscard	8.00 a	12.25 cd	33.47 a	145.3 ij
Batten/Ruapuna	7.75 a	11.75 d	33.44 a	184.3 abcdef
Batten/Otane	7.50 a	11.83 cd	33.38 a	175.2 defg
Karamu/Domino	6.71 b	11.67 d	32.06 abcd	177.8 defg
Pegasus/Ruapuna	6.67 b	11.58 d	33.28 ab	197.4 abcd
Karamu/Briscard	6.58 bc	12.25 cd	32.03 abcd	171.4 efgh
Batten/Domino	6.56 bc	11.78 d	32.21 abcd	171.7 efgh
Karamu/Ruapuna	6.50 bc	11.63 d	30.96 bcde	189.6 abcde
Sapphire/Otane	6.50 bc	12.50 bcd	29.03 ef	160.3 fghij
Sapphire/Ruapuna	6.46 bc	12.00 cd	31.77 abcd	176.2 defg
Karamu/Otane	6.40 bc	11.50 d	32.78 abc	206.5 a
Pegasus/Briscard	6.00 bcd	12.75 bcd	31.61 abcd	141.6 ijk
Pegasus/Otane	5.88 cd	11.75 d	32.01 abcd	189.6 abcde
Pegasus/Domino	5.88 cd	12.00 cd	30.75 cde	198.2 abcd
Oroua/Ruapuna	5.36 de	12.08 cd	29.92 de	158.9 ghij
Oroua/Otane	5.10 e	11.90 cd	31.94 abcd	164.4 fghi
Oroua/Briscard	5.00 e	12.20 cd	31.70 abcd	163.9 fghi
Sapphire/Briscard	4.00 f	13.80 b	27.16 fg	120.3 k
Oroua/Domino	3.82 fg	13.18 bc	30.82 cde	148.2 hij
Sapphire/Domino	3.22 gh	15.22 a	25.72 g	136.3 jk

Means with the same letter in the columns are not significantly different (Duncan's multiple ranges $P<0.05$)

Table 42. Estimates of components of variance and heritability for four components of resistance inoculated with two pathotypes of stripe rust based on the factorial mating design

Parameter[2]	111E143A-				232E137A-			
	IT[1]	LP	PS	PD	IT	LP	PS	PD
$V_{A(adult)}$	-0.4±0.1	-2.8±2.2	-44.0±5.5	932.0±246.8	5.0±10	4.4±2.0	2.8±1.9	3490.3±686.9
$V_{A(seedling)}$	97.6±14.2	1098.8±161.2	1686.6±253.5	31985.5±4723.5	18.2±2.9	11.8±3.1	50.6±8.8	3710.4±691.4
V_D	1.5±0.45	0.9±8.6	197.3±26.9	2013.0±287.1	6.8±0.7	34.0±3.5	31.0±3.9	2606.3±407.4
σ^2_{wp}	0.48	38.51	43.84	611.51	0.54	8.33	7.63	847.80

[1]IT:infection type (scale 0-9), LP:latent period (days), PS:pustule size (mm^2x1000) and PD:pustule density ((x100)/mm^2)

[2]$V_{A(adult)} = 4Cov_{HS}$ (additive variance for Adult)

$V_{A(seedling)} = 4Cov_{HS}$ (additive variance for Seedling)

$V_D = 4Cov_{FS} - [4Cov_{HSadult} + 4Cov_{HSseedling}]$ (dominance variance)

$\sigma^2_{wp} = \sigma^2_{within\ plot}$ (variance of environment)

Table 43. Degree of dominance *(h/d)*[1] of infection type for each of twenty crosses in response to pathotype 111E143A-

Cultivar		Oroua	Pegasus	Sapphire	Batten	Karamu
	Mean	7.50	8.83	8.08	1.92	3.55
Briscard	7.00	-0.32	-0.19	-0.07	-0.81	0.13
Domino	6.18	-1.27	-0.14	-0.23	-0.73	1.24
Otane	7.67	-2.89	-0.14	-2.29	-0.89	0.10
Ruapuna	6.42	-0.24	-0.04	-0.19	-0.63	0.36

[1] h = the deviation of the F_1 from the midparent (negative value means deviation towards resistance & positive value means deviation towards susceptibility)
d = the departure of the susceptible (or resistance) parent from the midparent

Table 44. Degree of dominance *(h/d)*[1] of infection type for each of twenty crosses in response to pathotype 232E137A-

Cultivar		Oroua	Pegasus	Sapphire	Batten	Karamu
	Mean	2.73	5.30	3.57	8.43	6.25
Briscard	7.73	-0.09	-0.43	-0.79	-0.23	-0.55
Domino	7.56	-0.55	-0.49	-1.18	-3.27	-0.30
Otane	7.64	-0.04	-0.50	0.44	-1.35	-0.79
Ruapuna	6.73	-0.32	-0.92	0.83	0.20	0.04

[1] h = the deviation of the F_1 from the midparent (negative value means deviation towards resistance & positive value means deviation towards susceptibility)
d = the departure of the susceptible (or resistance) parent from the midparent

4.4. Nonspecific glasshouse diallel

To study inheritance of components of resistance involving infection type, latent period, pustule size and pustule density, four adult plant resistant cultivars and one susceptible were intercrossed in a diallel fashion and tested with three pathotypes in three diallel mating designs.

4.4.1. Infection Type

Analyses of variance of infection type for pathotypes 106E139A-, 111E143A- and 232E137A- are presented in Tables 45-47, respectively. In all three pathotypes, the source of variation due to genotypes (whether parents, F_1's or pooled) was highly significant, indicating that at least one type of genetic variation was present. Duncan's multiple ranges for genotype means of each pathotype are presented in Tables 57-59. The most susceptible genotype to all pathotypes of stripe rust was Tiritea. Almost all crosses involving Tiritea showed a susceptible reaction. The mean of the resistant genotype to these pathotypes was 3.65, 5.67 and 6.44 for 106E139A-, 111E143A- and 232E137A-respectively, indicating pathotype 232E137A- is more aggressive on these cultivars than the others. The following crosses, Ruapuna x Domino for pathotype 106E139A-; Briscard x Domino, Briscard x Ruapuna, and Ruapuna x Domino for pathotype 111E143A-; and Briscard x Domino for pathotype 232E137A- showed more resistance than either of their parents.

4.4.1.1. Graphical (Hayman) analysis

Estimates of basic array statistics V_p, $\bar{Wr}$, $\bar{Vr}$ and $V_{\bar{r}}$ for all pathotypes are presented in Table 60. Also included are the W_r/V_r regression equations and their F-tests. The regression slope was significantly different from zero for pathotypes 106E139A- and 111E143A-, but not for pathotype 232E137A- (Table 60). It was therefore appropriate to conduct t-test on the departure of slope from unity (Mather and Jinks, 1982). The results are presented in Tables 61-63.

The W_r/V_r regression plot for infection type for the three pathotypes is presented

in Fig. 15. The coefficient of determination and regression slope plus its standard error are included. Analysis of variance of W_r+V_r and W_r-V_r for arrays was carried out and showed only W_r+V_r for pathotype 106E139A- was significant (Table 61-63). Significance of W_r-V_r indicated that the data may be adequately accounted for by an additive-dominance model while no significant difference between estimates of W_r+V_r denoted that dominance was trivial. The deviation of regression slope from unity (β - 1) was significant for pathotype 111E143A- and not significant for pathotypes 106E139A- and 232E137A- (Tables 61-63). The deviation from unity indicates graphical distortion due to one or more of epistasis, correlated gene distributions and environment effects (Mather and Jinks, 1982). It should be mentioned that the slope of regression for pathotype 232E137A- was significant, despite the high value of its standard error. This probably was due to the position of Domino which was far from other parents (Fig. 15). Coefficient of determination was high for pathotypes 106E139A- and 111E143A-, but low for pathotype 232E137A- (Fig. 15). Jinks (1954) proposed the removal of one array if the slope is different from unity. Using this procedure, it was hoped to remove major disturbances causing non-conformance to an additive-dominance model. But, by removing one or more parents, we were indirectly sampling, whereas our purpose was to study a character which needs all parents to be randomly selected. On this basis, no parent was removed.

Genetical components of variation plus other relevant statistics (Mather and Jinks, 1982) are presented in Tables 61-63. The large amount of D component can be seen in the response to pathotypes 106E139A- and 111E143A-, indicating a preponderance of additive genetic variance. In some cases, the negative estimate of H_1 and H_2, suggested that dominance was trivial, since these components have positive expectations as seen from formulae, $H_1 = 4uvh^2$ and $H_2 = 16u^2v_2h^2$ (Mather and Jinks, 1982); and consequently the estimate of $\sqrt{(H_1/D)}$, uv, $0.5F/\sqrt{[D(H_1-H_2)]}$ and ratio of dominance to recessive genes, were also regarded as trivial. For pathotypes 232E137A-, H_1 was positive indicating the presence of dominance and for the other pathotype due to it being negative, it was not calculated. The estimate of F was the negative for pathotype 111E143A- and the positive for other pathotypes; where negative, suggesting inequality of gene frequencies with an excess of recessive over dominant alleles, and where positive, case suggesting the inequality of gene frequencies with an excess of

dominant over recessive alleles.

For pathotype 232E137A-, the degree of dominance was 1.20, indicating overdominance.

The gene frequency was 0.20 for pathotype 232E137A-, suggesting inequality of increasing and decreasing genes; when gene frequencies are equal (u = v = 0.5), the maximum value of 0.25 occurs.

For pathotypes 232E137A-, the ratio $0.5F/\sqrt{[D(H_1-H_2)]}$ was 0.65, indicating the dominance level over the majority of loci. A constant dominance level over all show a value one for this ratio. And the proportion of dominant to recessive alleles over all parents was 1.78.

Broad sense and narrow sense heritabilities for each pathotype are presented in Tables 61-63. The narrow sense heritability was never bigger than the broad sense heritability. For two pathotypes 106E139A- and 111E143A-, the narrow sense heritability was high whereas for pathotype 232E137A- it was half of that, indicating the effect of dominance on fixable (additive) variation.

Correlations between common parent mean and W_r+V_r for pathotypes are presented in Tables 61-63. The correlations for pathotypes 106E139A- and 232E137A- were significant, indicating that distribution of dominant to recessive alleles was significantly correlated with common parent phenotype. That correlation was not significant for pathotype 111E143A-, indicating that the distribution of dominant to recessive alleles was only slightly correlated with common parent phenotype, and that the amount of dominance was trivial.

The σ^2_A/σ^2_D ratio shows the relative importance of these two genetic variances and (Tables 61-63). Effective factors in response to two pathotypes was less than unity whereas for the other pathotype it was high, suggesting due to its assumptions, it is not valid.

A ranking of parental arrays with respect to proportion of dominant alleles may be obtained from the W_r/V_r plots (Fig. 15). The smallest (W_r,V_r) points, closest to the origin, correspond to parental arrays with the greatest proportion of dominant alleles. In this study, for pathotype 106E139A-, the coefficient of determination was quite acceptable. The regression showed that the highly susceptible cultivar, Tiritea, had the highest proportion of dominant alleles, whereas resistant cultivars, Domino and Briscard, had least dominant alleles. The F_2 W_r/V_r graphical analysis was also performed for infection type in response to pathotype 106E139A- (Fig. 19). The intercept, coefficient of determination and slope of F2 regression line were similar with those of the F_1 regression line. Even the distribution of the points in the F_2 was similar to that of the F_1. Domino had the most recessive alleles in the F_1 whereas in the F_2 it tended to be more intermediate. In fact, dominance should be more difficult to detect in the F_2, because of reduced heterozygosity. On that basis all other cultivars other than Domino, moved very slightly toward the middle or additive position. For pathotype 111E143A-, Domino had the highest proportion of dominant alleles, and Briscard again had the least dominant alleles, as for the pathotype 106E139A-. For pathotype 232E137A-, coefficient of determination was low. Otane and Ruapuna coincided with each other and had the highest the proportion of dominant alleles while Briscard and Tiritea had the fewest dominant alleles. The position of Domino was somewhat far from the others. 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, 1977). It should also be noted that when coefficient of determination 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. 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 time-consuming to obtain, and valid only when coefficient of determination equals one and the t-test (β-1) is nonsignificant. A better approach is the correlation of parental phenotypic means against (W_r+V_r) (Mather and Jinks, 1977). Also for all three pathotypes the intercepts were above the origin, indicating the presence of partial dominance.

In summary, stripe rust resistance, in terms of infection type, against pathotypes 106E139A- and 111E143A-, was controlled by genes acting additively, and for pathotype 232E137A- was controlled by additive and other components of variation. Dominance and epistasis and/or correlated gene distributions were of relatively minor importance, for first two pathotypes, but could be important for the third pathotype.

4.4.1.2. Griffing combining ability analysis

The analysis of variance for combining ability for each pathotypes, (Tables 64-66) showed that both general (GCA) and specific (SCA) combining ability variances were highly significant. The GCA variance was larger than the SCA variance, the ratio $2MS_{GCA}/(2MS_{GCA} + MS_{SCA})$ being 0.99, 0.97 and 0.88 for pathotypes 106E139A-, 111E143A-and 232E137A-, respectively. The closer this ratio is to unity, the greater the predictability based on general combining ability alone (Baker, 1978). The relatively higher magnitude of the GCA variance than that of the SCA variance indicates the predominance of additive (fixable) gene effects in the genetic control of the stripe rust among parents used in this diallel cross. On the other hand, the significant SCA variance suggests a considerable amount of non-additive variance among the parents used for diallel crossing was present. Narrow sense and broadsense heritabilities ranged from 0.86 to 0.99 for all pathotypes, respectively.

The comparison of GCA performance of individual varieties for the stripe rust resistance for each pathotype (Tables 67-69) show that Tiritea and Otane for all three pathotypes and Briscard for pathotype 232E137A- had positive GCA effects indicating susceptibility as the infection type, whereas the others had negative GCA effects, indicating good combining ability for lower infection type. The ranking of the varieties according to their GCA variance was similar to that based on performance *per se* (Tables 45-47), indicating again the preponderance of the additive action of genes.

The estimates of SCA effects for infection type (Tables 67-69) show that out of the total of 10 cross combinations, for each pathotype, 5, 3 and 5 of them had negative and the rest positive SCA effects for pathotypes 106E139A-, 111E143A- and 232E137A- respectively. The highest negative SCA was shown by the combinations Briscard x

Domino for pathotypes 106E139A- and 232E137A- and Briscard x Ruapuna for pathotype 111E143A-, indicating dominance for those hybrids to lower infection type.

4.4.2. Latent Period

The analyses of variance of latent period for the three pathotypes are presented in Tables 48-50. There were significant differences amongst genotypes (except for F_1's to pathotype 111E143A-) in response to all three pathotypes. These differences suggest that much genetic variability was present for latent period in the genotypes studied, and diallel analysis was warranted for it. The genotypic means for latent period for each pathotypes are presented in Tables 57-59. For all three pathotypes, Tiritea showed a shorter latent period than the others whereas longer latent period were recorded in Briscard for pathotype 106139A- and Ruapuna for pathotypes 111E143A- and 232E137A-. This indicated that the shortest latent period is the most susceptible cultivar and *vice versa*. Because the means of cultivars with a longer latent period were 15.10, 13.50 and 12.37 days for pathotypes 106E139A-, 111E143A- and 232E137A- respectively, it suggests that pathotype 232E137A- was more virulent on these cultivars. For pathotype 106E139A-, a longer latent period was observed in Briscard x Ruapuna than in its parents. For other pathotypes, some hybrids had shorter latent period than their parents.

4.4.2.1. Graphical (Hayman) analysis

The estimates of the basic array statistic, W_r/V_r regression equation and their F-test are presented in Table 60. The regression slope was significantly different from zero for all pathotypes.

All the diallel statistics for latent period for the three pathotypes are presented in Tables 61-63. The W_r+V_r was highly significant only for pathotype 106E139A-, indicating the presence of dominance; for other pathotypes it was not significant. The W_r-V_r for all three pathotypes was not significant. The regression coefficients (β) were not significant for pathotypes 106E139A- and 232E137A-, whereas it was significant for pathotype 111E143A-. An overall view suggested that non-allelic interaction was probably present

for the last pathotype.

Additive genetic variance (D) was greater than the dominance genetic variance (H_1 and H_2) for pathotypes 106E139A- and 111E143A-. In the case of pathotype 232E137A-, dominance variance (H_1) was greater. Estimates of F were positive for all three pathotypes, indicating the inequality of gene frequencies with an excess of dominant over recessive alleles (Tables 61-63).

The degree of dominance, $\sqrt{(H_1/D)}$, indicated that partial dominance for pathotype 111E143A- was present and for pathotype 232E137A- overdominance was observed. It was not calculated in the case of pathotype 106E139A- due to its negative value.

The gene frequencies were 0.21 and 0.15 for pathotype 111E139A- respectively, indicating inequality of increasing and decreasing genes for latent period.

The proportions of positive to negative alleles were 1.11 and 0.59 for pathotypes 111E143A- and 232E137A-. In the case of the first pathotype the $0.5F/\sqrt{[D(H_1-H_2)]}$ ratio was found be approximately equal to 1, suggesting that the observed dominance was consistent for all the loci, rather than variable degrees of dominance at different loci.

The proportion of dominant to recessive alleles over all parents were 2.63 and 2.17 for pathotypes 111E143A- and 232E137A-. Estimates of $0.5F/\sqrt{[D(H_1-H_2)]}$ and the proportion of dominant to recessive genes were trivial for pathotype 106E139A-, due to the unimportance of dominance.

Heritability estimates, both broad sense and narrow sense, are presented in Tables 61-63. In all cases narrow sense heritability values were less than broad sense. The ranges of broad sense and narrow sense heritability for latent period in response to the three pathotypes were 60-73 and 30-52, respectively. It can be concluded that environment affects latent period more than infection type.

The correlations between common parent means and W_r+V_r for each array were significant and are presented in Tables 61-63. This indicated that the distribution of

dominant to recessive alleles was correlated with common parent phenotype for all three pathotypes.

The effective factors for pathotypes 106E139A- and 111E143A- were 1.56 an 3.90, but less than one for pathotype 232E137A-. And to show the importance of additive to dominance variance, the σ^2_A/σ^2_D could be used to estimate the relative importance of genetic variances.

The graphic analysis (Fig. 16) for pathotype 106E139A- shows that Briscard and Ruapuna contained most recessive alleles, while Otane contained most dominant alleles. Tiritea and Domino were intermediate. The relationship between W_r and V_r for latent period of F_1 and F_2 populations (Fig. 3) inoculated with 106E139A- indicate that the intercept, coefficient of determination and slope of regression lines F_1 and F_2 were the same. The distribution of cultivars on regression lines was slightly different. Briscard in the F_2 showed more recessive alleles than in the F_1, whereas Domino showed more dominant alleles than in the F_1. Other points in the F_2 were intermediate compared with F_1. For pathotype 111E143A-, the order of parents the same as for the first pathotype, but Ruapuna, Domino and Tiritea contained less recessive alleles. For pathotype 232E137A-, Ruapuna contained the highest proportion of recessive alleles whereas Tiritea had the highest proportion of dominant alleles. The rest of parents were intermediate them. The coefficient of determination was 98.2, 79.2 and 94.0 for pathotypes 106E139A-, 111E143A-and 232E137A- respectively. The intercept of the regression line for pathotypes 106E139A- and 111E143A- was above the origin and for pathotype 232E137A- was below the origin, indicating partial dominance and overdominance, respectively. These results were in agreement with the previous results.

4.4.2.2. Griffing combining ability analysis

In the analysis of latent period for each pathotype, the mean squares for GCA are larger than those for SCA (Tables 64-66). Both GCA and SCA are highly significant. The ratio, $2MS_{GCA}/(2MS_{GCA} + MS_{SCA})$, was 0.97, 0.83 and 0.89 for pathotypes 106E139A-, 111E143A- and 232E137A- respectively, indicating the relative importance of additive to nonadditive effects. Narrowsense and broadsense heritabilities ranged from 0.82 to

0.99 for all pathotypes, respectively.

For the three pathotypes, the GCA for Briscard, Ruapuna and Domino were positive, suggesting they are suitable parents for obtaining a longer latent period; whereas for Otane and Tiritea they were negative (Tables 70-72). The ranking of cultivars according to their GCA and performance (Tables 48-50) was the same.

The SCA effects for latent period for pathotypes 106E139A-, 111E143A- and 232E137A- showed that 3, 3 and 4 combinations had positive value and the rest were negative (Tables 70-72). The highest SCA was shown by the combinations Otane x Tiritea for pathotype 106E139A-, Ruapuna x Tiritea for pathotype 111E143A- and Briscard x Otane for pathotype 232E137A-, suggesting the presence of dominance for longer latent period in those hybrids.

4.4.3. Pustule Size

The analysis of variance for pustule size showed highly significant differences amongst the genotypes (parents, hybrids and pooled), indicating that a diallel analysis might validly be performed (Tables 51-53). The pustule size on different parents exhibited a large variation from 33.69 (mm^2 x 1000), for Tiritea, to 40.17, for Ruapuna, in response to pathotype 106E139A- (Table 51). The range of pustule size in response to pathotype 111E143A- was from 36.85 to 44.40 (mm^2 x 1000) for Domino and Tiritea, respectively (Table 52). For pathotype 232E137A-, Tiritea with a mean pustule size of 37.81 and Domino with mean pustule size of 32.03 were the most susceptible and resistant parents, respectively (Table 53). For pathotype 106E139A-, Otane x Tiritea, Briscard x Ruapuna and Briscard x Domino; for pathotype 111E143A-, Briscard x Tiritea and Otane x Tiritea and for pathotype 232E137A-, Briscard x Domino, Briscard x Ruapuna, Ruapuna x Domino and Ruapuna x Tiritea had smaller pustules than their parents.

4.4.3.1. Graphical (Hayman) analysis

Basic array statistics and the W_r/V_r regression equation for the three pathotypes

are shown in Table 60. All three regression equations are highly significant.

Estimates of variance and covariances of arrays and their sums and differences for each pathotype are presented in Tables 61-63. The W_r+V_r was highly significant for pathotypes 106E139A- and 232E137A- and was significant at the 10% probability level for pathotype 111E143A-, indicating the presence of dominance. The W_r-V_r was not significant for pathotype 106E139A-, but was highly significant for the other pathotypes, indicating the presence of non allelic interaction.

There was not a significant deviation of β from unity, indicating again the presence of dominance (Tables 61-63). Hence, both analyses indicated that an additive-dominance model was adequate to account for the this diallel.

Genetic components and other appropriate statistics (Mather and Jinks, 1982) are given in Tables 61-63.

The D component was positive and relatively large for two pathotypes (106E139A- and 111E143A-), indicating additivity was important; D for pathotype 232E137A- was less than the H_1 and H_2 components. Negative estimates of H_1 and H_2 for pathotype 111E143A- suggest that dominance was trivial. This is supported by the results of W_r+V_r. For other pathotypes the values of H_1 and H_2 were positive and notable. Positive estimates of F for all three pathotypes indicated an excess of dominant over recessive alleles.

The degree of dominance could not be estimated for pathotype 111E143A-, whereas $\sqrt{(H_1/D)}$ was estimated 0.62 and 1.30 for pathotypes 106E139A- and 232E137A- respectively, indicating partial and over dominance for them.

The estimate of uv was 0.25, 0.10 and 0.19 for pathotypes 106E139A-, 111E143A- and 232E137A-, respectively. Gene symmetry occurred for pathotype 106E139A- only, gene asymmetry ($uv < 0.25$) occurred for the other pathotypes.

Estimates of $0.5F / \sqrt{[D(H_1-H_2)]}$ were not calculated for pathotype 111E143A- and

it was larger than one for pathotype 106E139A-, but it was 0.70 for pathotype 232E137A-.

The proportion of dominant to recessive alleles over all parents was estimated at 2.46 and 2.07 for pathotype 106E139A- and 232E137A-. It could not be estimated for pathotype 111E143A-.

The estimates of broad sense and narrow sense heritability are presented in Tables 61-63. Usually narrow sense heritability was smaller than broad sense heritability, but in response to pathotype 111E143A- the narrow sense heritability was greater than broad sense heritability. It should be 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.

The correlations between the common parent mean and W_r+V_r for each array, were 0.98, 0.65 and 0.96 for pathotypes 106E139A-, 111E143A- and 232E137A-. All three correlations were significant, this indicated that the distribution of dominant to recessive alleles was correlated with the common parent phenotype.

The number of effective factors (K) was less than one for pathotype 232E137A-, because dominance was indicated as trivial. For pathotypes 106E139A- and 111E143A- the effective factors were 2.84 and 2.41, respectively.

The ratio of additive to dominance genetic variance was negative for pathotype 111E143A-, because dominance was negative. However, it was 5.26 and 1.57 for pathotypes 106E139A- and 232E137A-, respectively, indicating the importance of the additive part, especially in the response to pathotype 106E139A-.

The W_r/V_r graphic analysis (Hayman, 1954b) was performed to rank arrays with respect to the proportion of dominant alleles in the parents for pustule size (Fig. 17), and the coefficient of determination for three pathotypes are presented. None of the regression lines was tangential to the limiting parabola, $W_r^2 = V_r * V_p$ (where W_r is the

covariance of all the offspring in each parental array with the nonrecurring parent, V_r is the variance of all the offspring of each parent, and V_p is the variance of parents). The intercepts of regression lines for pathotypes 106E139A- and 111E143A- were above the origin, indicating partial dominance; for pathotype 232E137A- it was below the origin, suggesting overdominance, which is supported by previous results. In Fig. 17 for pathotype 106E139A-, Tiritea was located apart from the other parents and it was indicated that Tiritea had the highest proportion of recessive alleles. Briscard, Ruapuna and Domino, on the other hand, were located close to origin, suggesting they had the greatest number dominant alleles, and for Otane which was into mediate, suggesting the presence of additive component. Using another pathotypes, 111E143A-, Tiritea, Otane and Briscard (in recessive, middle and dominant areas, respectively) maintained their positions on the regression line. But Ruapuna and Domino moved toward the recessiveness. With respect to pathotype, 232E137A-, Tiritea displayed the highest proportion of dominant alleles, as for other pathotypes. Otane and Domino were close to origin and Briscard and Ruapuna were a little further from the origin, suggesting the presence of dominant alleles in these cultivars.

4.4.3.2. Griffing combining Ability analysis

Significant GCA and SCA effects for three pathotypes indicated that additive and nonadditive gene effects were important for pustule size in these cultivars (Tables 64-66). Variation for pustule size was attributed mainly to GCA effects rather than SCA effects. The relative importance of GCA and SCA, calculated according to Baker (1978), was 0.94, 0.95 and 0.80, for pathotypes 106E139A-, 111E143A- and 232E137A- respectively. These ratios were close to unity, suggesting that additive effects were more important than nonadditive effects for pustule size. Narrowsense and broadsense heritabilities ranged from 0.80 to 0.99 for all pathotypes, respectively.

Estimates of GCA and SCA effects for all three pathotypes are presented in Tables 73-75. For the three pathotypes, Tiritea and Otane had positive values, whereas the rest of parents had negative values. Negative GCA effects indicate a contribution to greater resistance, while positive GCA effects indicate a contribution to greater susceptibility. The ranking of cultivars according to their GCA and performance had

similar patterns (Tables 51-53).

The positive values for SCA were 1, 4 and 4 for pathotype 106E139A-, 111E143A- and 232E137A- respectively. The crosses Ruapuna x Domino in response to pathotype 106E139A-; Briscard x Ruapuna, Briscard x Domino and Ruapuna x Tiritea in response to pathotype 111E143A- and Ruapuna x Otane in response to pathotype 232E137A- had a high SCA values, suggesting that nonadditive gene action, such as dominance and epistasis effects, were involved in the inheritance of stripe rust resistance in these crosses.

4.4.4. Pustule Density

The analysis of variance for pustule density from all genotypes (parents, F_1's and pooled) for three pathotypes are presented in Tables 54-56. In all cases differences between genotypes were highly significant. Genotype means and Duncan's comparison are showed in Tables 57-59. Tiritea was the most susceptible to all three pathotype, whereas Domino x Briscard in response to pathotype 106E139A-, and Briscard in response to pathotypes 111E143A- and 232E137A- were the most resistant genotypes. Briscard x Domino showed more resistance than its parents to pathotype 106E139A-.

4.4.4.1. Graphical (Hayman) analysis

Basic array statistics and W_r/V_r regression equations for the three pathotypes are shown in Table 60. The regression slope for pathotype 106E139A- and 232E137A- were markedly different from zero, whereas it was not different for pathotype 111E143A-.

The array values for W_r+V_r were heterogeneous across parental arrays for pathotype 106E139A- and 232E137A- ($P = 0.01$ and 0.10, respectively), whereas it was homogeneous for pathotype 111E143A- (Tables 61-63). Heterogeneity indicates the presence of dominance. The array values for W_r-V_r were homogeneous, indicating that epistasis was not detected and an additive-dominance model was applicable (Tables 61-63).

Regression slopes (W_r/V_r) deviated from unity at P = 0.10 and 0.05 for pathotypes 106E139A- and 111E143A- respectively, indicating variation due to environment and/or non-additivity other than dominance, whereas it was not significant for pathotype 232E137A-, however indicating the presence of dominance (Tables 61-63).

Genetic components of variation plus other relevant statistics for the three pathotypes (Mather and Jinks, 1982) are given in Tables 61-63. The estimates of D for all three pathotypes were greater than H_1 and H_2. The H components were not negative. Hence, additivity was of major importance specially in the case of pathotype 232E137A-. The F value was negative for pathotype 106E139A-, indicating a greater proportion of recessive to dominant alleles in the parents, whereas it was positive for pathotype 111E143A- and 232E137A-, indicating a preponderance of dominant to recessive alleles in the parents.

The $\sqrt{(H_1/D)}$ measures mean degree of dominance over all loci. The value was less than one, 0.81, 0.74 and 0.33, for pathotypes 106E139A-, 111E143A- and 232E137A-, respectively, indicating partial dominance.

The quantity $(H_2/4H_1)$ estimates the relative average frequency of increasing and decreasing alleles in the parental lines. In response to pathotype 111E143A-, the value was 0.17 indicate apparent dominance asymmetry of increasing and decreasing alleles, but for other pathotypes that value was more than 0.25.

The ratio $0.5F/\sqrt{[D(H_1-H_2)]}$ was 0.60 for pathotype 111E143A-, indicating that the observed dominance was variable at different loci. For the other pathotypes it could not be calculated, because the value of H_2 was greater than H_1.

The proportion of dominant to recessive alleles over all parents was 0.72, 2.04 and 4.90, for pathotypes 106E139A-, 111E143A- and 232E137A-, respectively.

With regard to heritability estimates, broad sense was greater than narrow sense heritability. For three pathotypes, a moderate heritability was estimated.

Correlation between common parent mean and W_r+V_r were -0.53, -0.39 and 0.11 for pathotypes 106E139A-, 111E143A- and 232E137A-, respectively. These indicated that the distribution of dominant to recessive alleles was correlated with the common parent phenotype.

The relative importance of additive to dominance variances for three pathotypes is presented in Tables 61-63.

The effective factors for pathotype 111E143A- was 2.96, whereas this value was less than unity for other pathotypes.

The positive W_r intercept of both the regression lines for pathotypes 106E139A- and 111E143A- (Fig. 18) indicated partial dominance, whereas the negative W_r intercept for pathotype 232E137A- (Fig. 18) indicates overdominance. It should be noted that the slope of the line was not different from unity, which means dominance can be the acceptable interpretation rather than overdominace. The distribution of the (W_r,V_r) points indicates the direction of dominance. For the pathotype 106E139A-, Domino and Briscard had a recessive position whereas Ruapuna showed dominance. The coefficient of determination was 92.8. Briscard maintained its position to pathotype 111E143A-, whereas Domino had more dominant alleles than the others. Otane showed an anomalous position. The coefficient of determination was 14.2. For pathotype 232E137A-, Briscard, Otane and Tiritea had most recessive alleles whereas Ruapuna had most dominant alleles. The coefficient of determination was 78.4.

4.4.4.2. Griffing combining ability analysis

In the Griffing analysis of pustule density, the GCA and SCA were highly significant for two pathotypes 106E139A- and 111E143A-. However for pathotype 232E137A-, only GCA was significant (Tables 64-66). The GCA mean squares were larger than SCA mean squares. The ratio of relative importance of additive to nonadditive effects for the three pathotypes was greater than 0.90, suggesting a major contribution of additive component.

Narrowsense and broadsense heritabilities ranged from 0.89 to 0.99.

In response to pathotype 106E139A-, the GCA for Tiritea and Otane, for pathotype 111E143A-, the GCA of Tiritea and Ruapuna and for pathotype 232E137A-, the GCA of Tiritea, Otane and Ruapuna were positive (Tables 76-78), indicating susceptibility for greater pustule density. The ranking of cultivars for their GCA and performance were almost similar to each other.

The most resistant hybrid was observed from SCA of Briscard x Domino which was -19.59 in response to pathotype 106E139A-. It can be seen for Ruapuna x Otane and Domino x Tiritea in response to pathotype 111E143A- and 232E137A-, respectively. Out of 10 cross combinations for pathotypes 106E139A-, 111E143A- and 232E137A- 5, 4 and 3 of them had negative value, suggesting that nonadditive gene action for lower pustule density was involved for the inheritance of this character.

Table 45. Analysis of variance for infection type of a 5 x 5 diallel inoculated with stripe rust pathotype 106E139A-

S.O.V	Parents		F1,s		Pooled	
	df	MS	df	MS	df	MS
Block	3	0.9 **	3	0.2 ns	3	0.1 ns
Treatment	4	20.5 **	9	7.6 **	14	10.8 **
Error	12	0.11	27	0.31	42	0.26

Table 46. Analysis of variance for infection type of a 5 x 5 diallel inoculated with stripe rust pathotype 111E143A-

S.O.V	Parents		F1,s		Pooled	
	df	MS	df	MS	df	MS
Block	1	0.03 ns	1	1.10 *	1	0.57 ns
Treatment	4	2.51 **	9	1.75 **	14	1.91 **
Error	4	0.06	9	0.18	14	0.17

Table 47. Analysis of variance for infection type of a 5 x 5 diallel inoculated with stripe rust pathotype 232E137A-

S.O.V	Parents		F1,s		Pooled	
	df	MS	df	MS	df	MS
Block	1	0.003 ns	1	0.059 ns	1	0.029 ns
Treatment	4	1.181 **	9	0.438 **	14	0.642 **
Error	4	0.027	9	0.060	14	0.048

Table 48. Analysis of variance for latent period of a 5 x 5 diallel inoculated with stripe rust pathotype 106E139A-

S.O.V	Parents		F1,s		Pooled	
	df	MS	df	MS	df	MS
Block	3	0.4 ns	3	1.0 *	3	1.3 **
Treatment	4	8.6 **	9	1.6 **	14	3.6 **
Error	12	0.17	27	0.24	42	0.22

ns, * & ** : not significant, significant at P= 0.05 & 0.01 level, respectively

Table 49. Analysis of variance for latent period of a 5 x 5 diallel inoculated with stripe rust pathotype 111E143A-

S.O.V	Parents		F1,s		Pooled	
	df	MS	df	MS	df	MS
Block	1	0.51 ns	1	0.02 ns	1	0.10 ns
Treatment	4	1.19 **	9	0.09 ns	14	0.53 **
Error	4	0.17	9	0.04	14	0.11

Table 50. Analysis of variance for latent period of a 5 x 5 diallel inoculated with stripe rust pathotype 232E137A-

S.O.V	Parents		F1,s		Pooled	
	df	MS	df	MS	df	MS
Block	1	0.016 ns	1	0.002 ns	1	0.002 ns
Treatment	4	0.537 **	9	0.296 **	14	0.357 **
Error	4	0.024	9	0.065	14	0.050

Table 51. Analysis of variance for pustule size of a 5 x 5 diallel inoculated with stripe rust pathotype 106E139A-

S.O.V	Parents		F1,s		Pooled	
	df	MS	df	MS	df	MS
Block	3	1.1 ns	3	0.1 ns	3	0.6 ns
Treatment	4	32.0 **	9	4.5 **	14	13.8 **
Error	12	0.40	27	0.20	42	0.29

Table 52. Analysis of variance for pustule size of a 5 x 5 diallel inoculated with stripe rust pathotype 111E143A-

S.O.V	Parents		F1,s		Pooled	
	df	MS	df	MS	df	MS
Block	1	0.28 ns	1	1.32 ns	1	0.40 ns
Treatment	4	19.55 **	9	3.40 **	14	7.90 **
Error	4	3.37	9	1.04	14	1.72

ns, * & ** : not significant, significant at P= 0.05 & 0.01 level, respectively

Table 53. Analysis of variance for pustule size of a 5 x 5 diallel inoculated with stripe rust pathotype 232E137A-

S.O.V	Parents		F1,s		Pooled	
	df	MS	df	MS	df	MS
Block	1	0.258 ns	1	0.0003 ns	1	0.078 ns
Treatment	4	10.568 **	9	3.350 **	14	6.774 **
Error	4	0.069	9	0.233	14	0.182

Table 54. Analysis of variance for pustule density of a 5 x 5 diallel inoculated with stripe rust pathotype 106E139A-

S.O.V	Parents		F1,s		Pooled	
	df	MS	df	MS	df	MS
Block	3	28.6 ns	3	145.7 ns	3	67.6 ns
Treatment	4	3070.7 **	9	2160.5 **	14	2294.5 **
Error	12	66.31	27	81.66	42	79.06

Table 55. Analysis of variance for pustule density of a 5 x 5 diallel inoculated with stripe rust pathotype 111E143A-

S.O.V	Parents		F1,s		Pooled	
	df	MS	df	MS	df	MS
Block	1	20.2 ns	1	3.53 ns	1	1.19 ns
Treatment	4	1093.7 **	9	347.24 **	14	587.48 **
Error	4	53.3	9	75.76	14	65.54

Table 56. Analysis of variance for pustule density of a 5 x 5 diallel inoculated with stripe rust pathotype 232E137A-

S.O.V	Parents		F1,s		Pooled	
	df	MS	df	MS	df	MS
Block	1	166.59 ns	1	15.68 ns	1	114.17 ns
Treatment	4	927.13 **	9	262.69 **	14	442.28 **
Error	4	33.57	9	85.54	14	69.45

ns, * & ** : not significant, significant at P= 0.05 & 0.01 level, respectively

Table 57. Duncan's multiple ranges of five cultivars and their progenies for four components of resistance inoculated with stripe rust pathotype 106E139A-

Cultivar	Infection Type (scale 0-9)	Latent Period (days)	Pustule Size (mm^2 x 1000)	Pustule Density (x100)/mm^2
Tiritea	8.75 a	11.70 a	40.17 a	231.0 a
Domino/Tiritea	7.65 b	12.10 ab	34.95 cd	221.3 a
Otane/Tiritea	7.63 b	12.47 bc	36.82 b	230.2 a
Otane	7.42 b	12.45 bc	37.41 b	207.5 b
Briscard/Tiritea	7.00 bc	13.21 cd	34.24 cde	200.2 bc
Ruapuna/Tiritea	6.40 cd	12.95 cd	35.11 c	193.3 c
Domino/Otane	6.10 de	12.80 bcd	34.87 cd	197.4 bc
Ruapuna/Otane	5.58 ef	13.32 de	33.95 ef	191.2 c
Briscard/Otane	5.26 f	13.05 cd	34.57 cde	221.2 a
Ruapuna/Domino	4.85 fg	13.07 cd	34.40 cde	171.6 d
Domino	4.25 gh	13.45 de	33.85 efg	168.6 d
Ruapuna	4.17 gh	14.83 gh	33.69 efg	177.3 d
Briscard/Ruapuna	4.10 h	14.30 fg	33.16 fg	190.9 c
Briscard	3.65 h	15.10 h	34.20 de	167.5 d
Briscard/Domino	3.65 h	13.95 ef	33.06 g	152.8 e

Means with the same letter within the column are not significantly different (P=0.05)

Table 58. Duncan's multiple ranges of five cultivars and their progenies for four components of resistance inoculated with stripe rust pathotypes 111E143A-

Cultivar	Infection Type (scale 0-9)	Latent Period (days)	Pustule Size (mm^2 x 1000)	Pustule Density (x100)/mm^2
Tiritea	9.00 a	11.67 a	44.40 a	231.10 a
Briscard/Tiritea	8.17 ab	11.75 a	38.44 de	215.89 ab
Otane/Tiritea	8.11 ab	11.89 a	42.18 abc	212.16 abc
Otane	7.67 bc	12.17 a	42.27 ab	186.03 defg
Briscard/Otane	7.50 bcd	12.40 ab	39.65 bcde	221.94 a
Ruapuna/Tiritea	7.42 bcde	12.42 ab	41.02 abcd	212.71 abc
Ruapuna/Otane	7.17 bcdef	12.27 ab	39.37 bcde	187.28 defg
Domino/Tiritea	7.08 cdef	12.00 a	38.40 de	201.39 bcd
Briscard	7.00 cdef	13.27 c	38.92 cde	172.79 g
Domino/Otane	6.50 defg	12.25 ab	39.32 bcde	190.64 defg
Ruapuna	6.42 efg	13.50 c	37.33 e	182.35 defg
Domino	6.18 fg	13.00 bc	36.85 e	177.62 fg
Briscard/Domino	5.75 g	12.17 a	38.95 cde	195.15 cdef
Ruapuna/Domino	5.75 g	12.33 ab	37.30 e	180.36 efg
Briscard/Ruapuna	5.67 g	12.25 ab	39.29 bcde	199.29 bcde

Means with the same letter within the column are not significantly different (P=0.05)

Table 59. Duncan's multiple ranges of five cultivars and their progenies for four components of resistance inoculated with stripe rust pathotype 232E137A-

Cultivar	Infection Type (scale 0-9)	Latent Period (days)	Pustule Size (mm^2 x 1000)	Pustule Density (x100)/mm^2
Tiritea	8.92 a	11.00 a	37.81 a	228.10 a
Briscard/Tiritea	8.25 b	11.25 abc	33.64 bc	213.82 abc
Otane/Tiritea	8.09 bc	11.18 ab	33.72 bc	221.62 ab
Briscard	7.73 cd	12.09 defg	33.02 bcde	175.38 g
Briscard/Otane	7.67 cde	12.17 efg	33.25 bcd	185.37 efg
Otane	7.64 cde	11.55 abcd	33.71 b	204.49 bcde
Briscard/Ruapuna	7.58 cde	11.75 cdef	31.67 fgh	196.78 cdef
Domino	7.56 cde	11.56 abcd	32.03 efg	178.90 fg
Ruapuna/Domino	7.46 de	11.27 abc	30.57 ij	210.49 abcd
Ruapuna/Tiritea	7.46 de	11.09 ab	29.97 j	208.45 bcd
Domino/Otane	7.36 de	11.64 bcde	32.36 def	194.72 cdefg
Ruapuna/Otane	7.25 de	11.25 abc	32.96 bcde	206.08 bcd
Domino/Tiritea	7.11 ef	11.60 bcd	31.27 ghi	191.71 efg
Ruapuna	6.73 fg	12.37 g	32.67 cde	203.46 bcde
Briscard/Domino	6.44 g	12.22 fg	31.3 hi	193.92 cdefg

Means with the same letter within the column are not significantly different (P=0.05)

Table 60. W_r/V_r regression equations and basic array statistics for stripe rust resistance in five cultivars of wheat

character[1]	isolate	regression equation	F-test	V_p	$\bar{V}r$	$\bar{W}r$	$V_{\bar{r}}$
IT	106E139A-	$W_r = 1.10 + 0.98\ V_r$	75.73 **[2]	5.13	1.49	2.55	1.28
	111E143A-	$W_r = 0.37 + 0.67\ V_r$	73.05 **	1.20	0.54	0.78	0.48
	232E137A-	$W_r = 0.10 + 0.34\ V_r$	0.45 ns	0.59	0.29	0.20	0.09
LP	106E139A-	$W_r = 0.37 + 1.06\ V_r$	164.58 **	2.14	0.43	0.87	0.35
	111EA43A-	$W_r = 0.09 + 0.48\ V_r$	11.40 **	0.59	0.13	0.17	0.04
	232E137A-	$W_r = -0.10 + 1.27\ V_r$	46.99 **	0.27	0.12	0.08	0.05
PS	106E139A-	$W_r = 1.03 + 1.00\ V_r$	246.88 **	8.10	1.84	2.93	1.10
	111E143A-	$W_r = 0.87 + 1.10\ V_r$	12.58 **	9.23	1.48	3.27	1.06
	232E137A-	$W_r = -0.39 + 0.73\ V_r$	10.02 **	5.19	2.44	1.43	0.69
PD	106E139A-	$W_r = 91.70 + 0.77\ V_r$	38.58 **	762.59	402.50	414.76	233.80
	111E143A-	$W_r = 148.00 + 0.24\ V_r$	0.50 ns	519.75	168.68	190.25	91.67
	232E137A-	$W_r = -14.50 + 1.28\ V_r$	10.88 **	428.81	112.72	167.85	60.11

[1] Infection Type, Latent Period, Pustule Size and Pustule Density, respectively

[2] ns : not significant ** : significant at P = 0.01

Table 61. Genetic statistics for stripe rust resistance in five cultivars of wheat

	106E139A-			
statistics	Infection Type	Latent Period	Pustule Size	Pustule Density
$W_r + V_r$	**	ns	**	**
$W_r - V_r$	ns	ns	ns	ns
t-test(β - 1)	4.13 **	3.71 **	0.32 ns	2.24 *
D	5.08	2.02	7.77	716.05
H_1	-0.25	-0.31	3.02	468.95
H_2	0.70	0.38	2.95	695.59
F	0.16	0.67	4.09	-189.69
$\sqrt{(H_1/D)}$			0.62	0.81
uv	-0.72	-0.30	0.25	0.37
$0.5F / \sqrt{[D(H_1 - H_2)]}$			2.92	
dom./rec. genes			2.46	0.72
h^2_{BS}	90%	67%	90%	87%
h^2_{NS}	83%	52%	65%	57%
r (P_r , $W_r + V_r$)	-0.71	0.82	0.98	-0.53
σ^2_A / σ^2_D	14.52	10.76	5.26	2.06
K (effective factors)	0.18	1.56	2.84	0.25
E_{pool}	0.26	0.21	0.29	79.06
$E_{parents}$	0.11	0.17	0.40	66.31
$E_{hybrids}$	0.11	0.23	0.20	81.66

ns : not significant ** : significant at P = 0.01

Table 62. Genetic statistics for stripe rust resistance in five cultivars of wheat

	111E143A-			
statistics	Infection Type	Latent Period	Pustule Size	Pustule Density
$W_r + V_r$	ns	ns	(*)	ns
$W_r - V_r$	ns	ns	**	ns
t-test(β - 1)	0.18 ns	0.75 ns	0.00 ns	1.92 (*)
D	1.23	0.47	6.71	499.22
H_1	-0.02	0.38	-1.04	275.36
H_2	0.01	0.31	-0.04	184.83
F	-0.67	0.38	2.35	253.85
$\sqrt{(H_1/D)}$		0.89		0.74
uv	-0.10	0.21	0.10	0.17
$0.5F / \sqrt{[D(H_1 - H_2)]}$		1.11		0.60
dom./rec. genes		2.63		2.04
h^2_{BS}	84%	60%	51%	77%
h^2_{NS}	84%	30%	53%	60%
r (P_r , $W_r + V_r$)	0.06	0.77	0.65	-0.39
σ^2_A / σ^2_D	400.03	3.00	-33.51	5.40
K (effective factors)	46.79	3.90	2.41	2.96
E_{pool}	0.17	0.11	1.72	65.54
$E_{parents}$	0.06	0.17	3.37	53.30
$E_{hybrids}$	0.18	0.04	1.04	75.76

ns : not significant (*) and ** : significant at P = 0.10 and P = 0.01, respectively

Table 63. Genetic statistics for stripe rust resistance in five cultivars of wheat

	232E137A-			
statistics	Infection Type	Latent Period	Pustule Size	Pustule Density
$W_r + V_r$	ns	ns	**	(*)
$W_r - V_r$	ns	ns	**	ns
t-test(β - 1)	1.29 ns	1.42 ns	1.17 ns	0.72 ns
D	0.59	0.27	5.22	429.97
H_1	0.84	0.31	8.82	46.85
H_2	0.69	0.19	6.64	81.10
F	0.39	0.21	4.71	187.62
$\sqrt{(H_1/D)}$	1.20	1.08	1.30	0.33
uv	0.20	0.15	0.19	0.43
$0.5F / \sqrt{[D(H_1 - H_2)]}$	0.65	0.59	0.70	
dom./rec. genes	1.78	2.17	2.07	4.90
h^2_{BS}	88%	73%	94%	64%
h^2_{NS}	44%	48%	42%	54%
r (P_r , $W_r + V_r$)	0.75	0.88	0.96	0.11
σ^2_A / σ^2_D	1.71	2.83	1.57	10.60
K (effective factors)	0.35	0.60	0.02	0.88
E_{pool}	0.05	0.05	0.18	69.45
$E_{parents}$	0.03	0.02	0.07	33.57
$E_{hybrids}$	0.60	0.06	0.23	85.54

ns : not significant (*), * and ** : significant at P = 0.10, 0.05 and 0.01, respectively

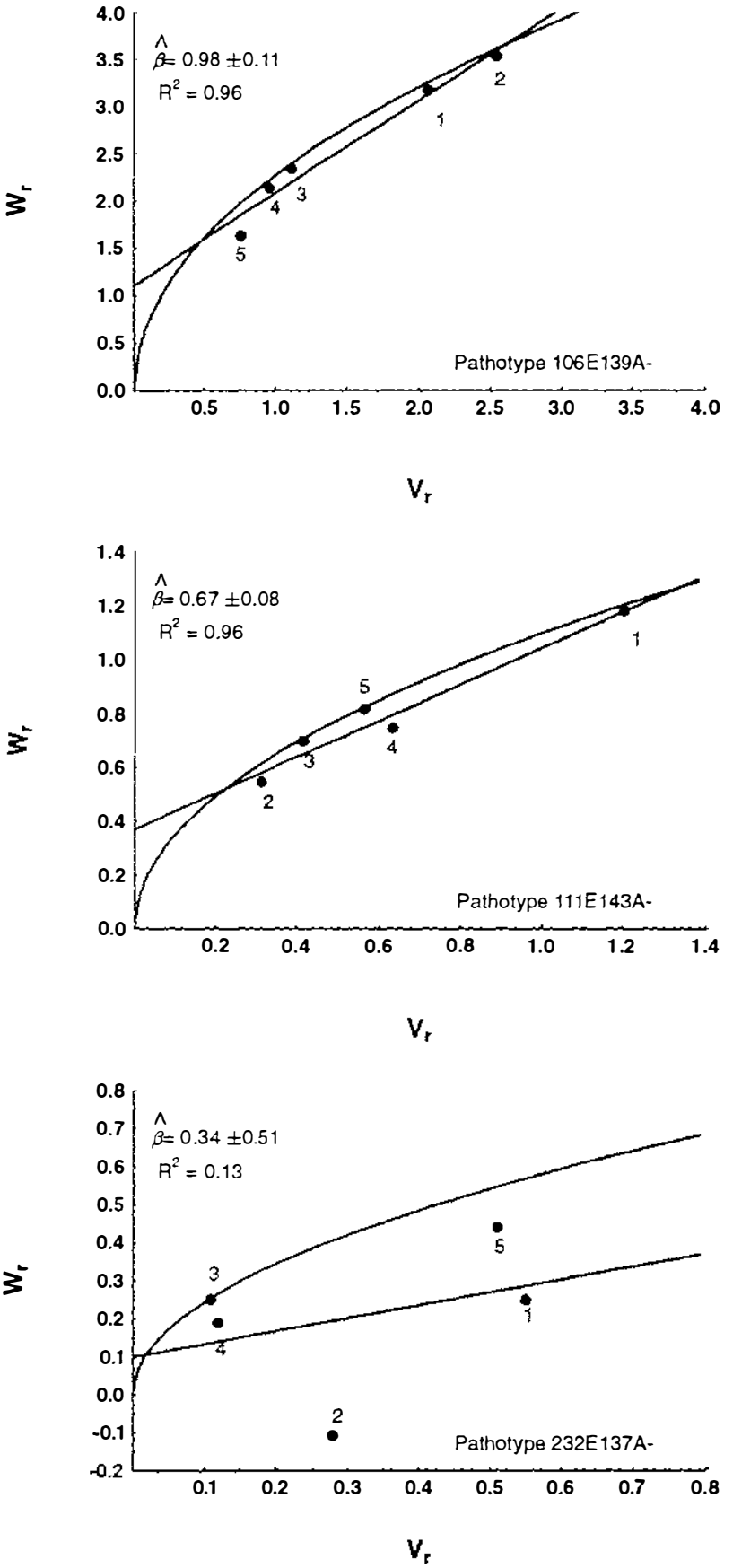


Fig. 15. The W_r/V_r graphs of infection type from crosses of five cultivars of wheat (1= Briscard, 2= Domino, 3= Otane, 4= Ruapuna and 5= Tiritea) in response to three pathotypes of stripe rust

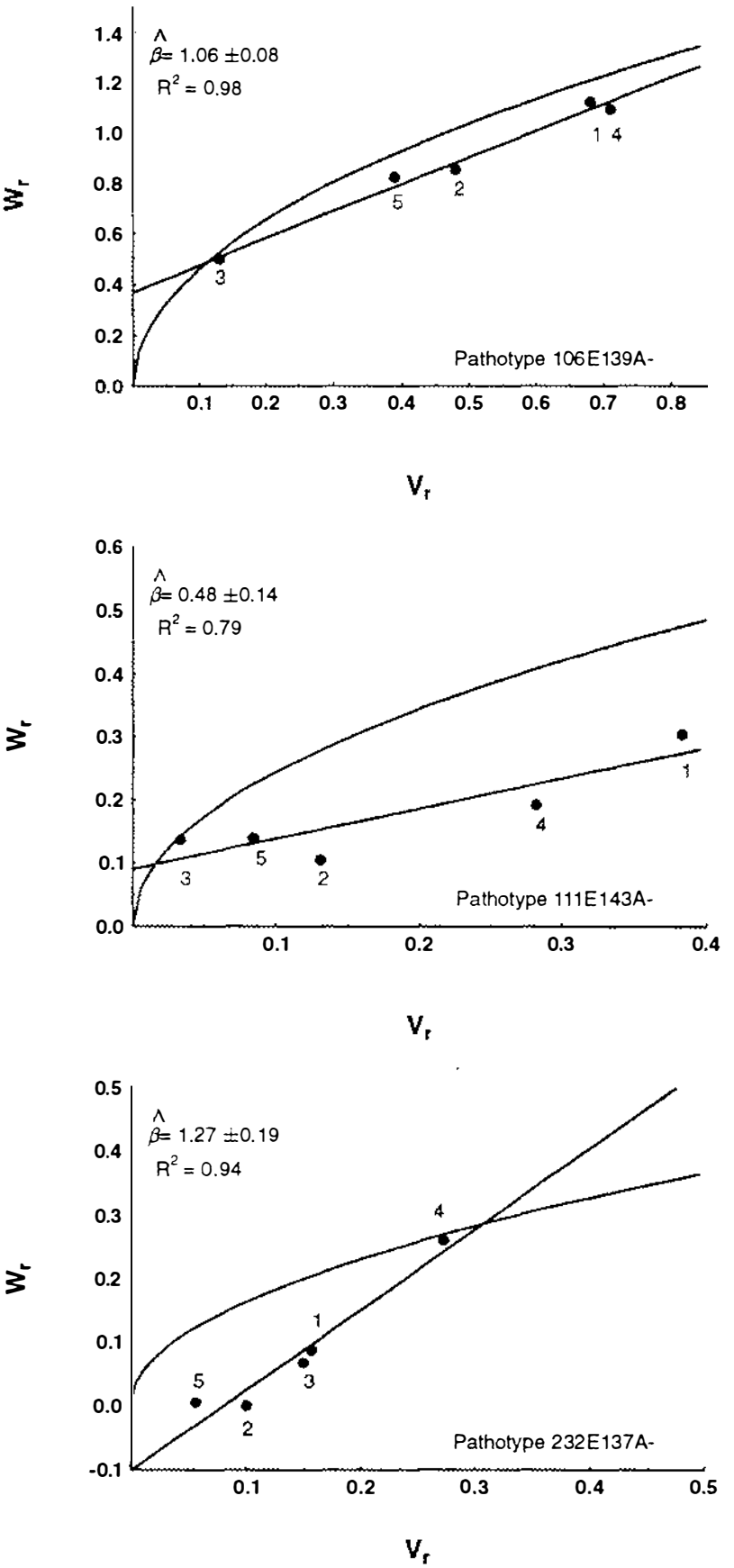


Fig. 16. The W_r/V_r graphs of latent period from crosses of five cultivars of wheat (1= Briscard, 2= Domino, 3= Otane, 4= Ruapuna and 5= Tiritea) in response to three pathotypes of stripe rust

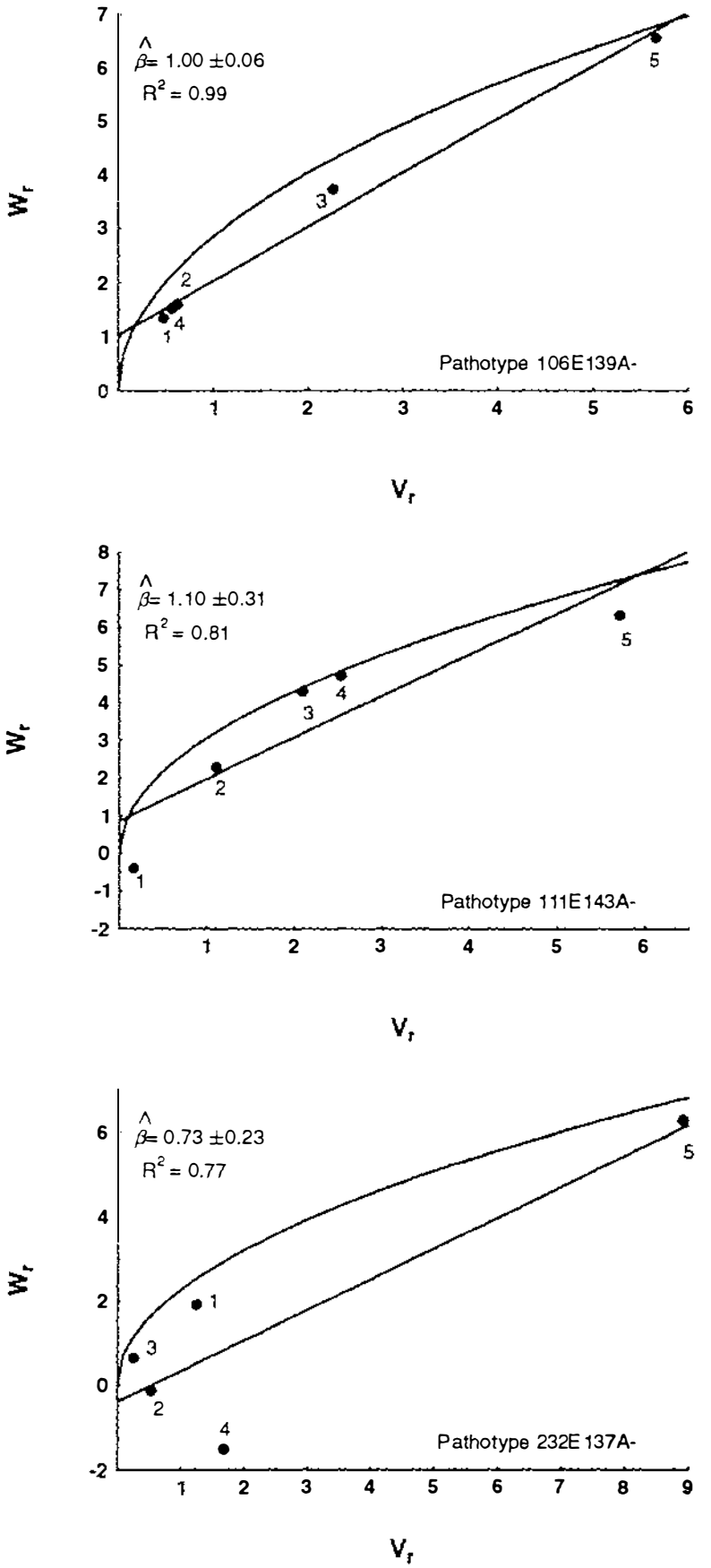


Fig. 17. The W_r/V_r graphs of pustule size from crosses of five cultivars of wheat (1= Briscard, 2= Domino, 3= Otane, 4= Ruapuna and 5= Tiritea) in response to three pathotypes of stripe rust

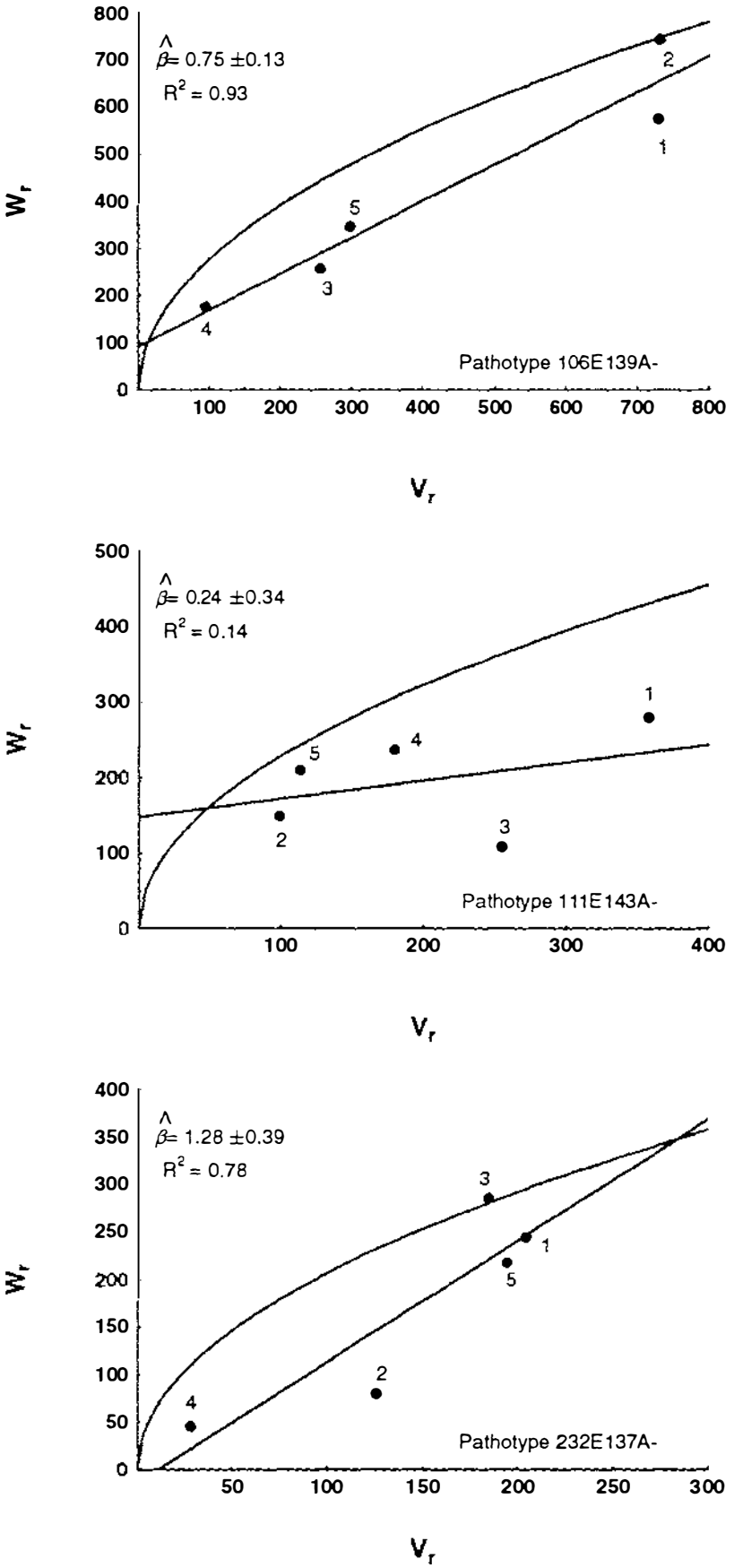


Fig. 18. The W_r/V_r graphs of pustule density from crosses of five cultivars of wheat (1= Briscard, 2= Domino, 3= Otane, 4= Ruapuna and 5= Tiritea) in response to three pathotypes of stripe rust

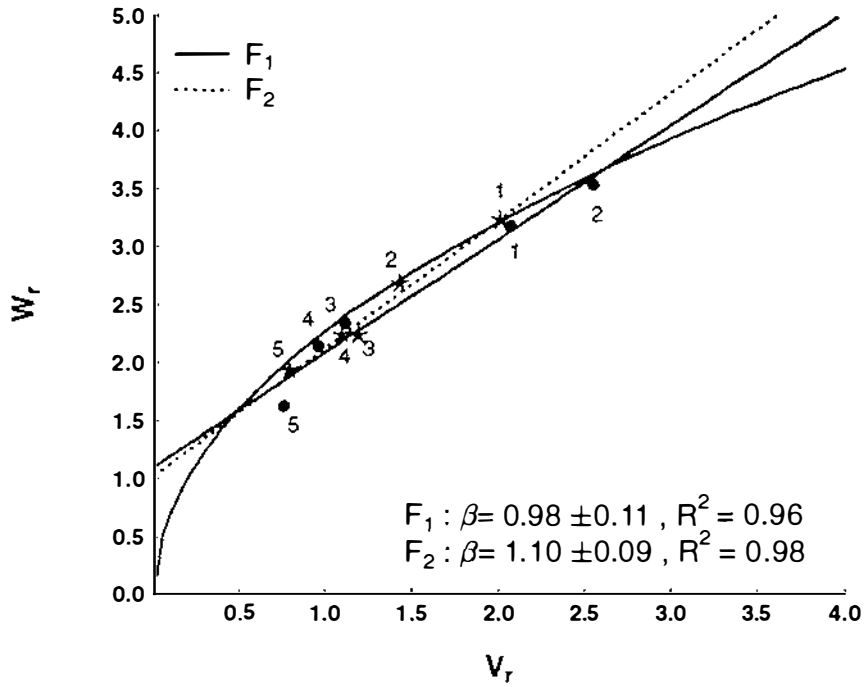


Fig. 19. W_r/V_r graph for infection type between parents comparing the F_1 (•) and F_2 (⋆) diallels of 5 x 5 diallel crosses (1= Briscard, 2= Domino, 3= Otane , 4= Ruapuna, 5= Tiritea), infected with stripe rust pathotype 106E139A-

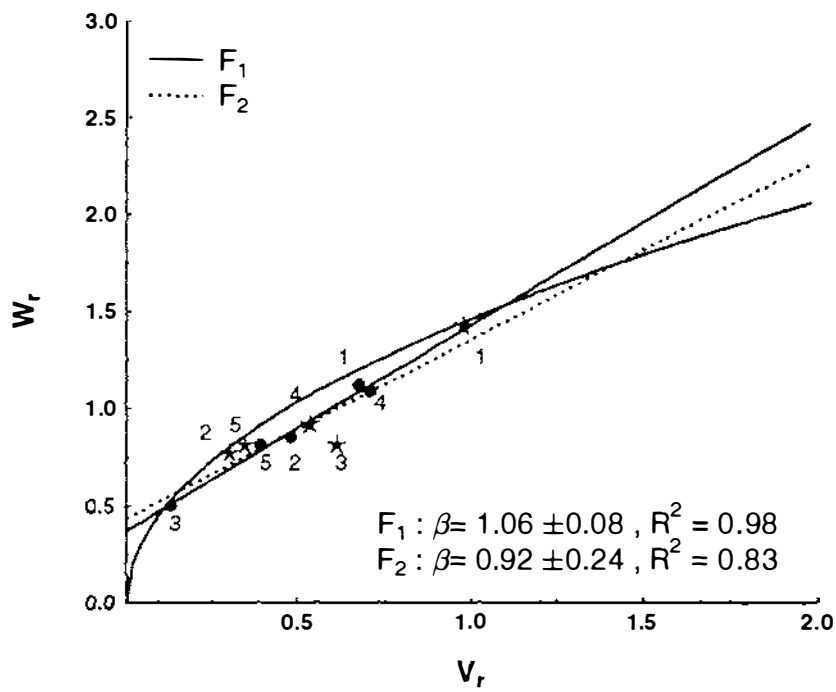


Fig. 20. W_r/V_r graph for latent period between parents comparing the F_1 (•) and F_2 (⋆) diallals of 5 x 5 diallel crosses (1= Briscard, 2= Domino, 3= Otane , 4= Ruapuna, 5= Tiritea), infected with stripe rust pathotype 106E139A-

Table 64. Mean squares of general and specific combining ability for four components of resistance to stripe rust pathotype 106E139A-

S.O.V.	df	MS[1]			
		IT	LP	PS	PD
GCA[2]	4	9.1 **	2.9 **	9.3 **	1555.8 **
SCA[3]	10	0.2 **	0.2 **	1.2 **	199.5 **
Error	233	0.03	0.03	0.1	12.6
Ratio[4]		0.99	0.97	0.94	0.94
h^2_{BS}[5]		0.99	0.99	0.99	0.99
h^2_{NS}[6]		0.98	0.96	0.94	0.94

Table 65. Mean squares of general and specific combining ability for four components of resistance to stripe rust pathotype 111E143A-

S.O.V.	df	MS[1]			
		IT	LP	PS	PD
GCA[2]	4	3.0 **	0.5 **	11.1 **	707.3 **
SCA_3	10	0.2 **	0.2 **	1.3 **	151.6 **
Error	150	0.03	0.02	0.4	16.4
Ratio[4]		0.97	0.83	0.95	0.90
h^2_{BS}[5]		0.99	0.98	0.98	0.99
h^2_{NS}[6]		0.96	0.82	0.93	0.89

Table 66. Mean squares of general and specific combining ability for four components of resistance to stripe rust pathotype 232E137A-

S.O.V.	df	MS[1]			
		IT	LP	PS	PD
GCA[2]	4	0.7 **	0.4 **	5.3 **	555.4 **
SCA[3]	10	0.2 **	0.1 **	2.6 **	87.4 ns
Error	150	0.03	0.03	0.1	48.7
Ratio[4]		0.88	0.89	0.80	0.93
h^2_{BS}[5]		0.98	0.97	0.99	0.96
h^2_{NS}[6]		0.86	0.86	0.80	0.89

[1] Mean Squares of Infection Type, Latent Period, Pustule Size and Pustule Density
[2] General Combining Ability
[3] Specific Combining Ability
[4] $2MS_{GCA}/(2MS_{GCA} + MS_{SCA})$
[5,6] broad- and narrow sense heritability, respectively

Table 67. Estimates of general combining ability (diagonal) and specific combining ability (above diagonal) for infection type to stripe rust pathotype 106E139A-

	Briscard	Domino	Otane	Ruapuna	Tiritea
Briscard	-1.04**	-0.53**	-0.16ns	0.14ns	0.62**
Domino		-0.55**	0.20ns	0.39**	0.78**
Otane			0.69**	-0.11ns	-0.48**
Ruapuna				-0.76**	-0.26*
Tiritea					1.66**

$S.E._{GCA}$=0.0390 $S.E._{SCA}$=0.1011

Table 68. Estimates of general combining ability (diagonal) and specific combining ability (above diagonal) for infection type to stripe rust pathotype 111E143A-

	Briscard	Domino	Otane	Ruapuna	Tiritea
Briscard	-0.15**	-0.45**	0.27*	-0.73**	0.35**
Domino		-0.67**	-0.20ns	-0.13ns	-0.22ns
Otane			0.35**	0.26*	-0.21ns
Ruapuna				-0.47**	-0.08ns
Tiritea					0.95**

$S.E._{GCA}$=0.0400 $S.E._{SCA}$=0.1055

Table 69. Estimates of general combining ability (diagonal) and specific combining ability (above diagonal) for infection type to stripe rust pathotype 232E137A-

	Briscard	Domino	Otane	Ruapuna	Tiritea
Briscard	0.02ns	-0.87**	0.06ns	0.32**	0.19**
Domino		-0.26**	0.02ns	0.46**	-0.67**
Otane			0.05*	-0.05ns	-0.00ns
Ruapuna				-0.30**	-0.28**
Tiritea					0.49**

$S.E._{GCA}$=0.0210 $S.E._{SCA}$=0.0561

Table 70. Estimates of general combining ability (diagonal) and specific combining ability (above diagonal) for latent period to stripe rust pathotype 106E139A-

	Briscard	Domino	Otane	Ruapuna	Tiritea
Briscard	0.74**	0.05ns	-0.52**	-0.24*	-0.02ns
Domino		-0.10**	0.07ns	-0.63**	-0.29**
Otane			-0.42**	-0.05ns	0.41**
Ruapuna				0.54**	-0.08ns
Tiritea					-0.77**

$S.E._{GCA}$=0.0350 $S.E._{SCA}$=0.0930

Table 71. Estimates of general combining ability (diagonal) and specific combining ability (above diagonal) for latent period to stripe rust pathotype 111E143A-

	Briscard	Domino	Otane	Ruapuna	Tiritea
Briscard	0.14**	-0.42**	0.05ns	-0.55**	-0.35**
Domino		0.09*	-0.05ns	-0.42**	-0.05ns
Otane			-0.14**	-0.25*	0.07ns
Ruapuna				0.31**	0.15ns
Tiritea					-0.39**

$S.E._{GCA}$=0.0320 $S.E._{SCA}$=0.0849

Table 72. Estimates of general combining ability (diagonal) and specific combining ability (above diagonal) for latent period to stripe rust pathotype 232E137A-

	Briscard	Domino	Otane	Ruapuna	Tiritea
Briscard	0.28**	0.30**	0.33**	-0.20**	-0.28**
Domino		0.04ns	0.04ns	-0.44**	0.32**
Otane			-0.04ns	-0.38**	-0.03ns
Ruapuna				0.07**	-0.23**
Tiritea					-0.35**

$S.E._{GCA}$=0.0220 $S.E._{SCA}$=0.0572

Table 73. Estimates of general combining ability (diagonal) and specific combining ability (above diagonal) for pustule size to stripe rust pathotype 106E139A-

	Briscard	Domino	Otane	Ruapuna	Tiritea
Briscard	-0.91**	-0.31**	-0.24*	-0.07ns	-1.49**
Domino		-0.68**	-0.16ns	0.95**	-0.97**
Otane			0.75**	-0.94**	-0.57**
Ruapuna				-0.83**	-0.70**
Tiritea					1.67**

$S.E._{GCA}$=0.0410 $S.E._{SCA}$=0.1067

Table 74. Estimates of general combining ability (diagonal) and specific combining ability (above diagonal) for pustule size to stripe rust pathotype 111E143A-

	Briscard	Domino	Otane	Ruapuna	Tiritea
Briscard	-0.45**	1.23**	-0.56ns	1.00**	-2.21**
Domino		-1.38**	0.04ns	-0.06ns	-1.32**
Otane			1.10**	-0.48ns	-0.02ns
Ruapuna				-0.81**	0.74ns
Tiritea					1.54**

$S.E._{GCA}$=0.1280 $S.E._{SCA}$=0.3356

Table 75. Estimates of general combining ability (diagonal) and specific combining ability (above diagonal) for pustule size to stripe rust pathotype 232E137A-

	Briscard	Domino	Otane	Ruapuna	Tiritea
Briscard	-0.02ns	-0.66**	0.12ns	-0.19ns	0.14ns
Domino		-0.92**	0.14ns	-0.38**	-1.60**
Otane			0.52**	0.56**	-0.90**
Ruapuna				-0.75**	-3.07**
Tiritea					1.17**

$S.E._{GCA}$=0.0420 $S.E._{SCA}$=0.1092

Table 76. Estimates of general combining ability (diagonal) and specific combining ability (above diagonal) for pustule density to stripe rust pathotype 106E139A-

	Briscard	Domino	Otane	Ruapuna	Tiritea
Briscard	-9.75**	-19.59**	23.80**	15.45**	-4.57*
Domino		-12.66**	2.94ns	-0.95ns	19.38**
Otane			12.31**	-6.28**	3.37ns
Ruapuna				-9.64**	-11.63**
Tiritea					19.73**

$S.E._{GCA}$=0.6720 $S.E._{SCA}$=1.7624

Table 77. Estimates of general combining ability (diagonal) and specific combining ability (above diagonal) for pustule density to stripe rust pathotype 111E143A-

	Briscard	Domino	Otane	Ruapuna	Tiritea
Briscard	-1.26ns	7.76**	25.80**	8.82**	2.56ns
Domino		-9.13**	2.36ns	-2.25ns	-4.07ns
Otane			-0.37ns	-4.08ns	-2.06ns
Ruapuna				6.05**	4.17ns
Tiritea					16.81**

$S.E._{GCA}$=0.7900 $S.E._{SCA}$=2.0716

Table 78. Estimates of general combining ability (diagonal) and specific combining ability (above diagonal) for pustule density to stripe rust pathotype 232E137A-

	Briscard	Domino	Otane	Ruapuna	Tiritea
Briscard	-9.24**	10.37**	-7.91**	1.79ns	9.82**
Domino		-8.10**	0.29ns	14.36**	-13.44**
Otane			1.64ns	0.21ns	6.74**
Ruapuna				3.35**	-8.13**
Tiritea					16.35**

$S.E._{GCA}$=0.8130 $S.E._{SCA}$=2.1325

4.5. Nonspecific glasshouse generation means

To study inheritance of infection type and latent period, the six generations, involving P_1, P_2, F_1, F_2, BC_1 and BC_2 of ten crosses were tested with common pathotype (106E139A-) in glasshouse. Pustule size and pustule density were difficult to measure because the number of plants were great, so only infection type and latent period are presented.

4.5.1. Infection type

4.5.1.1. Briscard x Tiritea

There were differences among generations (Table 79), with the mean of infection type for Briscard, Tiritea and the F_1 being 3.7, 8.8 and 7.0 respectively. (Table 79). The degree of dominance was 0.29 toward the susceptible parent, Tiritea. The frequency distribution of infection type for F_2 plants was continuous and skewed toward the susceptible parent. Transgressive segregation was observed in F_2 plants in both directions, also in the backcrosses, towards the recurrent parent (Fig. 21). Broad sense and narrow sense heritabilities were 0.80±0.08 and 0.52±0.03 respectively (Table 80). Genetic advance, assuming the top 1% of segregating F_2 plants were selected for resistance, from this cross was 2.20 (Table 80). The estimate of minimum number of genes controlling resistance was calculated with different formula (Table 81); the range of them was 1.2±0.30 - 1.9±0.28.

The results of generation mean analysis showed that the χ^2 of the three-parameter model is significant, suggesting the presence of epistasis in this cross (Table 82). To find the type of epistasis, the six-parameter model showed that the [i] and [j] are significant at 10% probability level. By using all possibles model, the five-parameter model involving m ,[d], [h], [j] and [l] fitted the best. The χ^2 of this model was not significant even at 5% probability level, indicating the adequacy of the model (Table 82). Two four-parameter models could be fitted but their χ^2 were highly significant (Table 82).

The components of variation were estimated from six generations (Table 83). The heritable variance comprises two parts, the D component depending on the d's which measure the departure of homozygote from the mid-parent and the H component which depends on the h's measuring the departure of heterozygote from the mid-parent

(Mather and Jinks, 1977). For this cross the values of D and H were 2.70 and 2.60, respectively. The dependant contribution of d and h over all loci (F) was +2.11. If the F value is positive, the genes from the higher scoring (susceptible) parent, are preponderantly dominant. In this cross the genes from the Tiritea were preponderantly dominant, as the comparison of F_1 mean with mid-parent had already suggested. The estimate of average dominance of the genes, $(D/H)^{1/2}$, was 0.98 which agrees with the relatively high level of dominance suggested by the analysis of the means. The ratio $F/(D*H)^{1/2}$ was 0.80. If h/d, the measure of dominance, is constant in both magnitude and sign for all the gene-pairs involved, the F value will be equal to $(D*H)^{1/2}$, and the ratio will be close to one. Variation in h/d results in F falling compared with $(D*H)^{1/2}$ and the greater the variation the greater the short fall (Mather and Jinks, 1982).

4.5.1.2. Ruapuna x Tiritea

The F-test showed that differences among generations are highly significant (Table 79). All generations, except F_1 and F_2 were in different groups (Table 79). The mean infection types for Ruapuna and Tiritea were 4.2 and 8.8, respectively and the mean infection type of the F_1 was 6.4 (Table 79). The degree of dominance was 0.04 for the resistant parent, indicating that the F_1 was in the midparent position. The F_2 distribution of infection type was continuous and skewed toward the susceptible parent (Fig. 21). The frequency distributions of both backcrosses were also continuous. Transgressive segregation was observed in the F_2 plants in both directions and toward the resistant parent for the backcross to the resistant parent. It can be concluded that the susceptible parent, Tiritea, may have gene(s) to contribute slightly to low infection type. Broad sense and narrow sense heritability for this cross were 0.84±0.10 and 0.43±0.08, respectively (Table 80). The genetic advance was 1.52 (Table 80). The number of gene was estimated from 1.3±0.18 to 12.1±3.6 (Table 81).

The result of the generation means revealed that only the additive part is significant and there was nothing else beyond the additive model (Table 82). All possible models were fitted and they agreed in this matter. The χ^2 was not significant for any of them, indicating the adequacy of the additive model. All components of variation are presented in Table 83. The D and H values were 1.54 and 2.97, respectively. The F value was negative, indicating those gene from the resistant cultivar, Ruapuna, were dominant which was supported by the deviation of the F_1 mean from the mid-parent. The

importance of dominance can be seen in the ratio $(H/D)^{1/2}$ which was 1.39. The ratio of $F/(D*H)^{1/2}$ was 0.39, indicating some genes in Ruapuna were recessive to their alleles from Tiritea.

4.5.1.3. Domino x Tiritea

The F-test showed that all generations had significant differences (Table 79). The degree of dominance was 0.51 toward the susceptible parent by using the means of infection type for Domino (4.3), Tiritea (8.8) and the F_1 (7.7). The distribution of F_2 plants for infection type was continuous and skewed toward Tiritea (Fig. 21). Transgressive segregation was observed in the F_2 distribution in both directions, and in each backcrosses towards the recurrent parent (Fig 21). The broad sense and narrow sense heritabilities were 0.86±0.21 and 0.66±0.18, respectively (Table 80). The genetic advance was relatively high (Table 80). The range of the number of genes was from 1.6±0.17 to 3±0.68 (Table 81).

The results of generation mean analysis for this cross showed that the χ^2 of the three-parameter model was significant, suggesting the presence of non-allelic interaction (Table 82). The six-parameter model was fitted, but neither [i], [j] and [l] components nor [h] was significant. Then other models were fitted and the best model for this cross was m, [d], [h] and [j]. The χ^2 of this model was not significant, showing the adequacy of the model. Also another five-parameter model - m, [d], [h], [i] and [j] - could be fitted but in contrast with the four parameter model had an increase in standard error (Table). Estimates of components of variation are presented in Table 83. The D value was more than the H value, then the average dominance 0.79, indicating importance of dominance. The F value was positive, indicating that genes in the Tiritea, susceptible parent, were dominant which had agreement with the deviation of F_1 from mid-parent. The ratio $F/(D*H)^{1/2}$ was 0.43, suggesting some genes in Domino must be recessive to their alleles from Domino.

4.5.1.4. Otane x Tiritea

The differences among generations are shown in Table 79. The mean of each parent was in a different groups. The mean infection types of Otane, Tiritea and the F_1 were 7.4, 8.8 and 7.6, respectively (Table 79). There was no statistical significant

difference between Otane and the F_1, suggesting full dominance for lower infection type, (Table 79). The frequency of the F_2 was skewed toward susceptibility (Fig. 21). Transgressive segregation was observed in F_2 plants and in the backcross with Otane towards resistance, indicating both parent have gene(s) for resistance; whereas for the backcross with Tiritea, it was towards susceptibility (Fig. 21). The broad and narrow sense heritabilities were 0.74±0.06 and 0.61±0.02, respectively (Table 80). The genetic advance was 1.55 (Table 80) and the number of genes was less one (Table 81).

The results of the generation means showed that the χ^2 of three-parameter model was significant, indicating the presence of digenic interactions. By using the six-parameter model, all interactions were significant, so the six-parameter as the best model to be fitted in this cross (Table 82). Components of variation are presented in Table 83. The additive part (D) was greater than dominance part (H). This can be seen in average dominance which was 0.65. The negative value of F indicated that genes in Otane were dominant and dominance was constant in sign and magnitude in different loci which was supported by the high value of the ratio $F/(D*H)^{1/2}$.

4.5.1.5. Briscard x Otane

The F-test was significant (Table 79). Briscard and its backcross were in the same group whereas other generations were in different groups (Table 79). The mean infection types of Briscard, Otane and the F_1 were 3.7, 7.4 and 5.3, respectively. The degree of dominance, according to the deviation of the F_1 from the midparent, was 0.14 towards the resistant parent. The F_2 distribution was skewed toward Otane and both backcrosses were skewed toward the their recurrent parents (Fig. 21). In the F_2 population, transgressive segregation was observed in both directions, indicating both parents contribute towards both resistance and susceptibility. The backcrosses showed transgressive segregation toward recurrent parents (Fig. 21). The heritability was 0.87±0.12 and 0.86±0.14 for broad and narrow sense, respectively (Table 80). The highest genetic advance was observed in this cross (Table 80). The number of genes were estimated from 0.4±0.97 to 2.5±2.1 (Table 81).

In generation mean analysis the χ_2 of the three-parameter model was significant, indicating the presence of digenic interaction (Table 82). The dominance, [h], and dominance x dominance interaction, [l] components were not significant in the six-

parameter model. Because [h] was significant in the three-parameter model, only [l] was omitted. The five-parameter model involving m, [d], [h], [i] and [j] was the best model and its χ^2 was not significant, indicating of the adequacy of this model, while the χ^2 of all four-parameter models and one five-parameter model - m, [d], [h], [j] and [l] - was significant (Table 82). The components of variation are presented in Table 83. The additive part was much greater than the dominance part and average dominance was 0.19 which is low. The F value was -0.19, indicating that genes from the resistant parent, Briscard, showed dominance but some genes in Briscard must be recessive to their alleles from Otane, due to low value of the ratio $F/(D*H)^{1/2}$.

4.5.1.6. Ruapuna x Otane

There were differences among generations (Table 79). The mean infection types of Ruapuna, Otane and the F_1 were 4.2, 7.4 and 5.6, respectively (Table 79). The degree of dominance was 0.03 toward Ruapuna, indicating no dominance. The frequency distribution of infection type for F_2 plants was continuous and normal (Fig. 21). Transgressive segregation was observed in both directions, indicating both parents have gene(s) for resistance and susceptibility. Transgressive segregation was also observed in both backcrosses toward their recurrent parents (Fig. 21). The broad sense and narrow sense heritabilities were 0.79±0.16 and 0.65±0.11 respectively (Table 80). The genetic advance was 2.64 which is relatively high (Table 80) and the number of genes was less than one (Table 81).

In generation mean analysis, the χ^2 of the three-parameter model was significant (Table 82), indicating the presence of digenic interaction. The six-parameter model showed that only the [i] is significant. By fitting all possible models, it was observed that the four-parameter model involving m, [d], [h] and [i] is the model that fitted best. The χ^2 of this model was not significant even at the 5% probability level, indicating the adequacy of the model, whereas the χ^2 of another four-parameter model - m, [d], [h] and [l] - was significant (Table 82). All components of variation are presented in Table 83. The D and H values were 3.06 and 1.28, respectively. The average dominance was 0.65, which show the importance of dominance. The F value was negative, indicating Ruapuna showed dominance which was supported by the deviation of the F_1 from the mid-parent. The ratio $F/(D*H)^{1/2}$ was 0.22, indicating dominance was not constant in sign and magnitude.

4.5.1.7. Domino x Otane

The F-test showed significant differences among generations (Table 79). The mean infection type for Domino, Otane and the F_1 were 4.3, 7.4 and 6.1, respectively (Table 79). The degree of dominance was 0.16 towards the susceptibility of Otane. The distribution of the F_2 and both backcrosses was continuous (Fig. 21). Transgressive segregation was observed in both directions in the distribution of F_2 and both backcrosses (Fig. 21). The broad and narrow sense heritabilities were 0.80±0.24 and 0.63±0.21, respectively (Table 80) and the genetic advance was 2.66 which is relatively high (Table 80). The range of number of genes was from 0.2±0.99 to 1.2±1.1 (Table 81).

In generation mean analysis, the χ^2 of the three-parameter model was not significant and only the additive component was significant (Table 82), which was also observed in six-parameter model. By fitting all possible models, two four-parameter models showed that they were the best. The first one involved m, [d], [h] and [i], and the second one involved m, [d], [h] and [l]. In both cases the χ^2 was not significant, indicating the adequacy of the models. The components of variation are presented in Table 83. The D, H values and $(H/D)^{1/2}$ were 1.32 0.40 and 0.55, respectively. This indicating that the additive component was more important than dominance component. The F value was negative which was not in agreement with the deviation of the F_1 from the mid-parent. This indicated that Domino had dominant alleles; and dominance was constant over majority of loci in sign and magnitude which was supported by the high value of the ratio $F/(D*H)^{1/2}$.

4.5.1.8. Briscard x Domino

Differences were observed among all generations (Table 79). The means infection types for Briscard, Domino and the F_1 were 3.7, 4.3 and 3.7, respectively (Table 79). There was full dominance for Briscard. The F_2 distribution was continuous and transgressive segregation was observed on both directions in the distribution of the F_2 and in both backcrosses (Fig. 21). The broad sense and narrow sense heritabilities were 0.72±0.15 and 0.85±0.22, respectively and genetic advance was 2.27 (Table 80). The number of genes was less than one (Table 81).

The results of the generation means showed that the χ^2 of the three-parameter

model was not significant, indicating adequacy of additive-dominance model (Table 82). By using the six-parameter model, only the additive part was significant, suggesting that the additive model is appropriate. This was confirmed by using the two parameter model. All components of variation are presented in Table 83. The additive value was greater than the H value and average dominance was 0.42. The negative value of F indicated Briscard showed dominance which is correspond with analysis of means, but it was not constant in sign and magnitude and some genes in Briscard must be recessive to their alleles in Domino. This was supported by the low value of $F/(D*H)^{1/2}$.

4.5.1.9. Ruapuna x Domino

The F-test showed no differences among generations (Table 79). The distributions of the F_2 and both backcrosses was continuous and normal (Fig. 21). Transgressive segregation was observed in both directions in the F_2 and backcrosses as well. Broad sense and narrow sense heritability were 0.41±0.11 and 0.37±0.09 respectively and the genetic advance was 1.10 (Table 80). The number of gene was less than one (Table 81).

In the generation mean analysis, the χ^2 of the three-parameter model and the additive component were not significant, indicating dominance model was adequate (Table 82). This was also seen in the six-parameter model. The components of variation are presented in Table 83. The additive part was greater than dominance part and $(H/D)^{1/2}$ was 0.49. The F was +0.09, indicating Domino showed dominance, but it was not constant in sign and magnitude. This was supported by low value of $F/(D*H)^{1/2}$.

4.5.1.10. Briscard x Ruapuna

The analysis of variance was significant at 5% level of probability, indicating at least differences between two generations (Table 79). Briscard and the backcross with Ruapuna were significantly different, but the other generations overlapped (Table 79). The mean infection types for Briscard, Ruapuna and the F_1 were 3.7, 4.2 and 4.1, respectively. The degree of dominance could not be estimated because there was no statistical differences between the parents and the F_1. The distributions of the F_2 and the both backcrosses was continuous (Fig. 21) and transgressive segregation was observed in the F_2 plants and the backcross with Briscard in both directions, whereas in the

backcross with Ruapuna it was toward susceptibility (Fig. 21). Broad sense and narrow sense heritabilities were 0.79±0.18 and 0.75±0.13, respectively and the genetic advance was high (Table 80) and the number of genes was almost zero (Table 81).

The result of the generation mean analysis for this cross showed that the χ^2 value for the three-parameter model was significant at the 5% probability level (Table 82), indicating the presence of digenic interaction. The result of the six-parameter model showed that [j] was not significant. By omitting of the nonsignificant component a five-parameter model involving m, [d], [h], [i] and [l] resulted. All components were significant. The χ^2 value was not significant for this cross, indicating no trigenic interaction. The five-parameter model had a reduction in standard error in comparison with six-parameter model. The components of variation are presented in Table 83. The D value was greater than H and the average dominance was 0.31 and the F value was +0.70, indicating Ruapuna showed dominance. The ratio $F/(D*H)^{1/2}$ was 0.68, indicating the measure of dominance is not constant in sign and magnitude and some alleles must be recessive to their alleles from Briscard.

Table 79. Differences among the mean values of six generations for the infection type of stripe rust pathotype 106E139A- in ten crosses of wheat

Gener-ation[1]	Cross[3]									
	1	2	3	4	5	6	7	8	9	10
P1	8.8 a[2]	8.8 a	8.8 a	8.8 a	7.4 a	7.4 a	7.4 a	4.3 a	4.3 a	4.2 ab
BC1	7.6 b	7.8 b	7.8 b	7.8 b	7.3 a	6.5 b	6.5 b	3.9 ab	4.7 a	4.6 a
F1	7.0 b	6.4 c	7.7 b	7.6 b	5.3 cc	5.6 c	6.1 bc	3.7 b	4.9 a	4.1 ab
F2	6.7 b	6.7 c	6.9 c	7.6 b	6.1 bd	5.0 cd	5.6 cd	4.0 ab	4.6 a	4.0 ab
BC2	4.5 c	5.5 d	5.9 d	6.7 c	4.3 d	4.5 de	4.8 de	3.6 b	4.9 a	4.0 ab
P2	3.7 c	4.2 e	4.3 e	7.4 b	3.7 e	4.2 e	4.3 e	3.7 b	4.2 a	3.7 b
1)F-test	**	**	**	**	**	**	**	*	ns	*
2)$LSD_{0.05}$:	1.01	0.88	0.62	0.53	0.59	0.82	0.82	0.47	0.81	0.74

*, ** and ns : Significant at 5 % , 1 % probability level and not significant, respectively
Least square means which have a same letter are not significantly different

3) 1:Briscard(P_2) × Tiritea(P_1) 2:Ruapuna(P_2) × Tiritea(P_1) 3:Domino(P_2) × Tiritea(P_1)
4:Otane(P_2) × Tiritea(P_1) 5:Briscard(P_2)× Otane(P_1) 6:Ruapuna(P_2) × Otane(P_1)
7:Domino(P_2) × Otane(P_1) 8:Briscard(P_2) × Domino(P_1) 9:Ruapuna(P_2) × Domino(P_1)
10:Briscard(P_2) × Ruapuna(P_1)

Fig. 21. Frequency distributions of the infection type of stripe rust pathotype 106E139A- in ten crosses of wheat

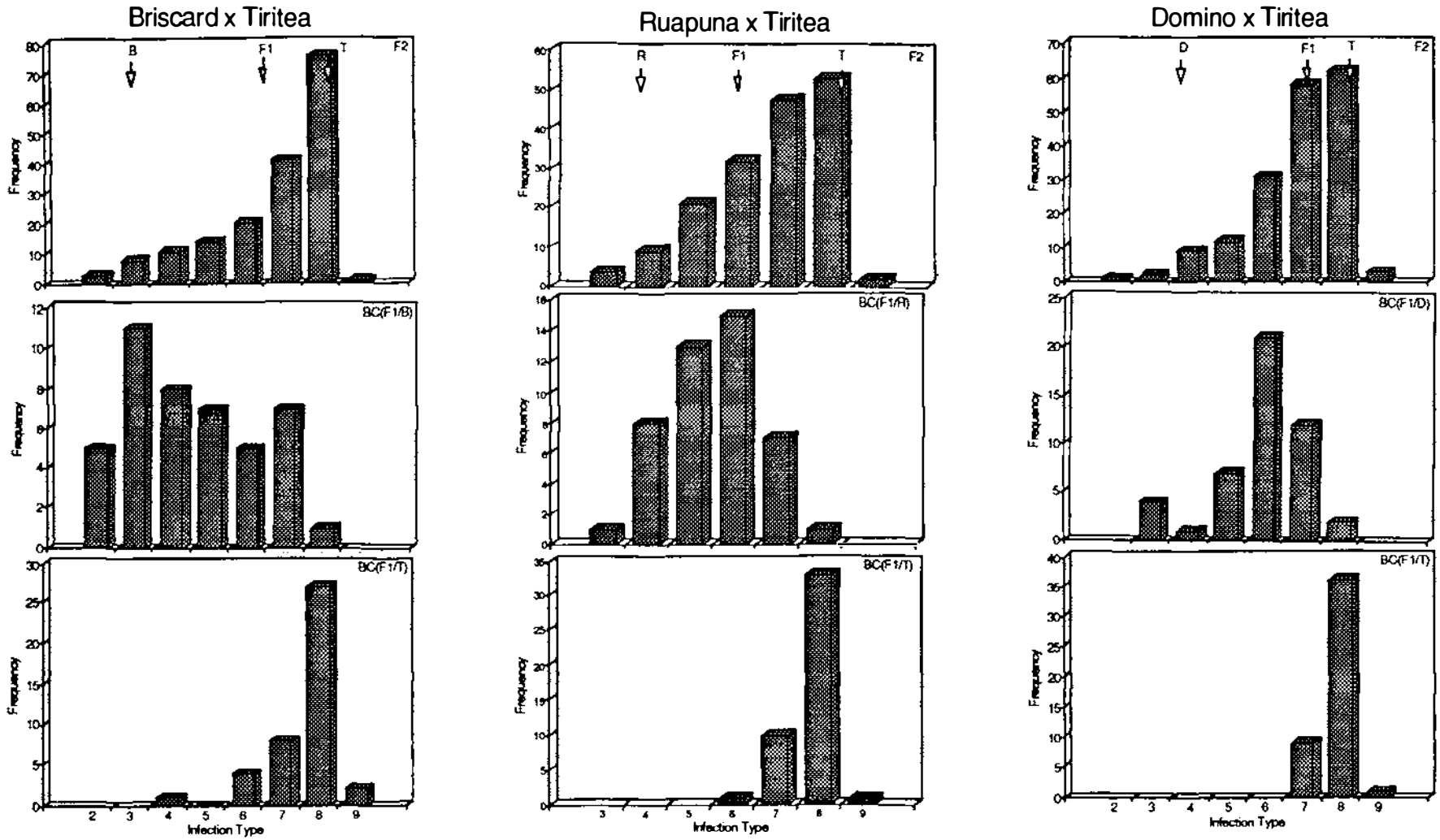


Fig. 21. Continued

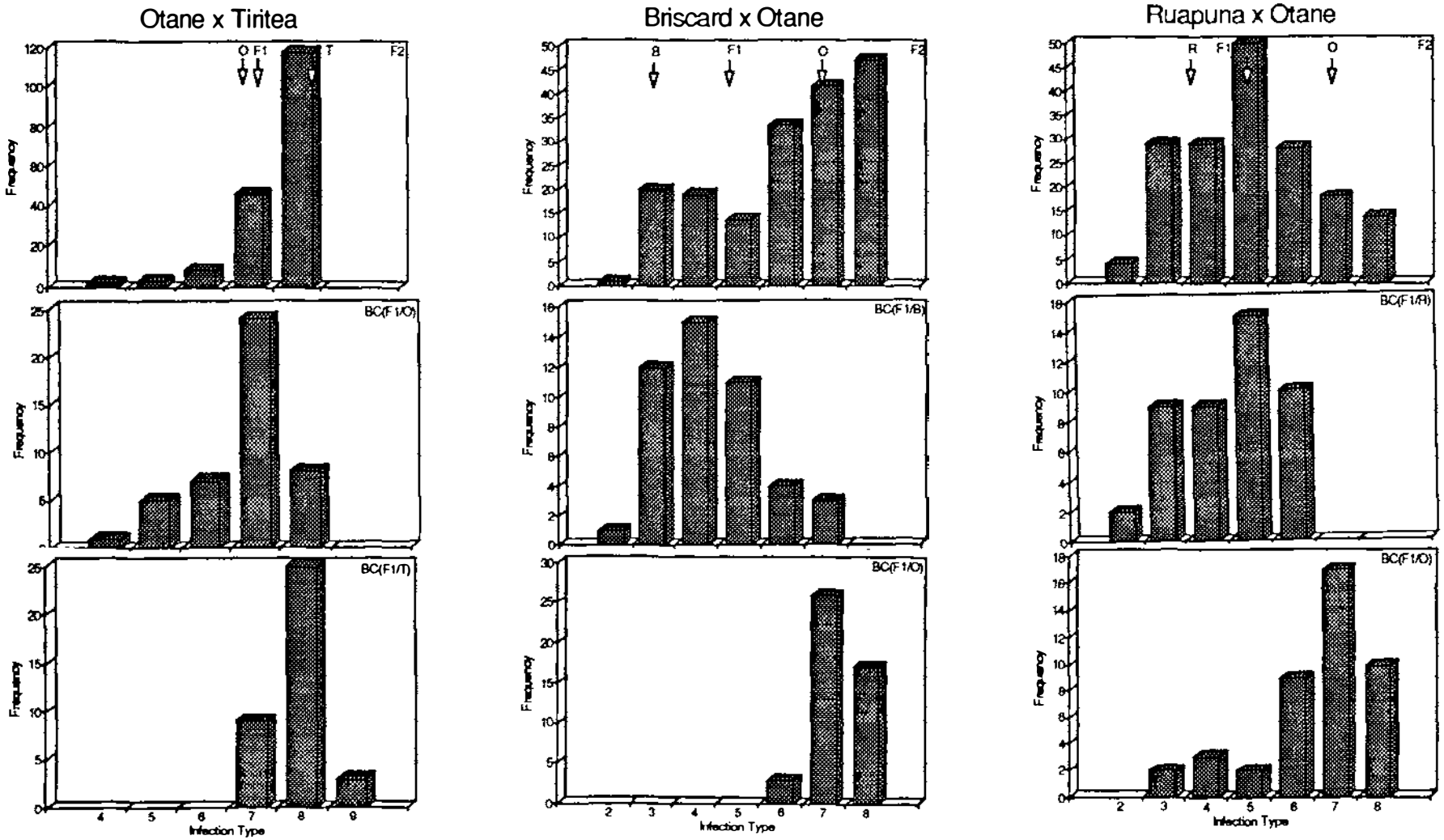


Fig. 21. Continued

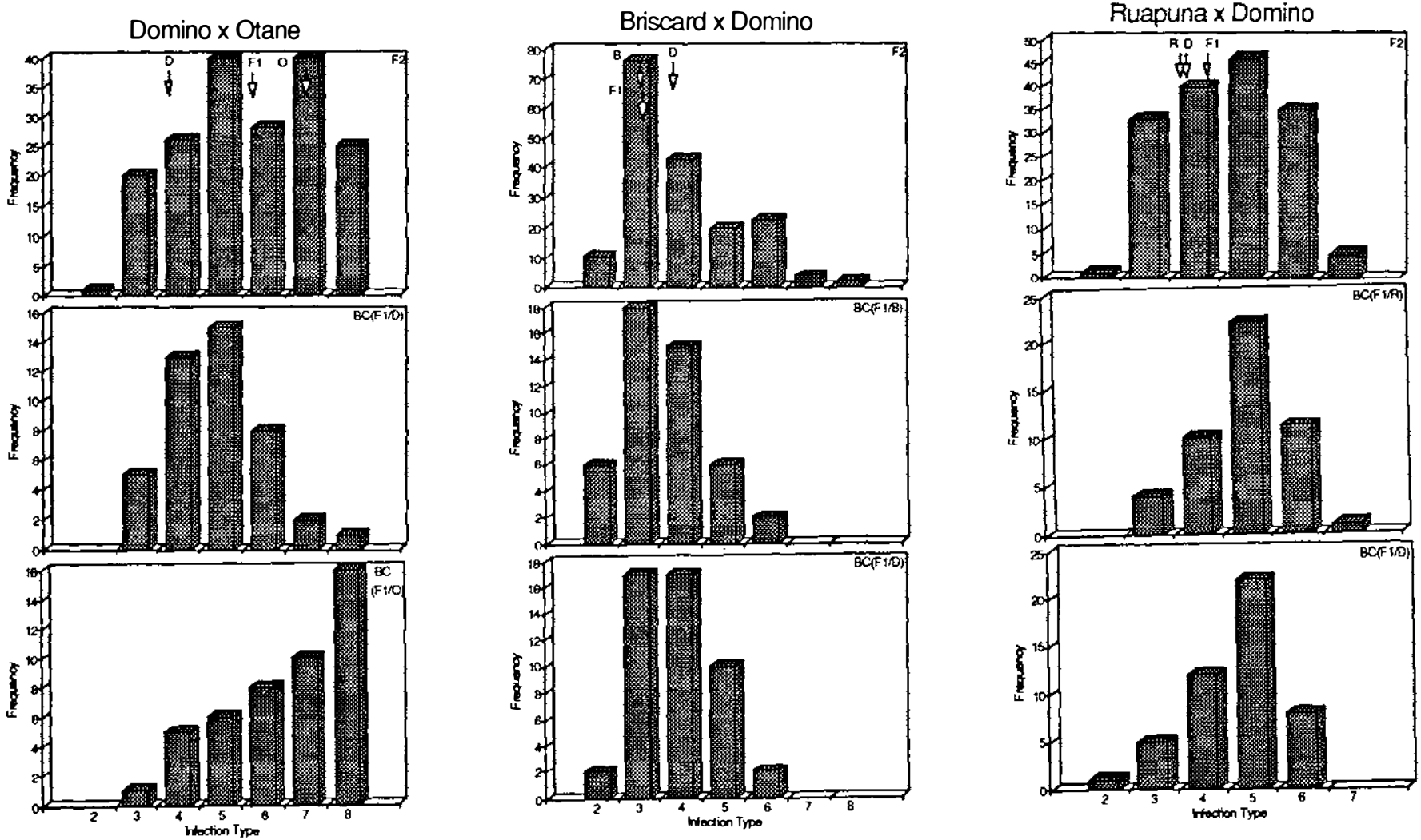


Fig. 21. Continued

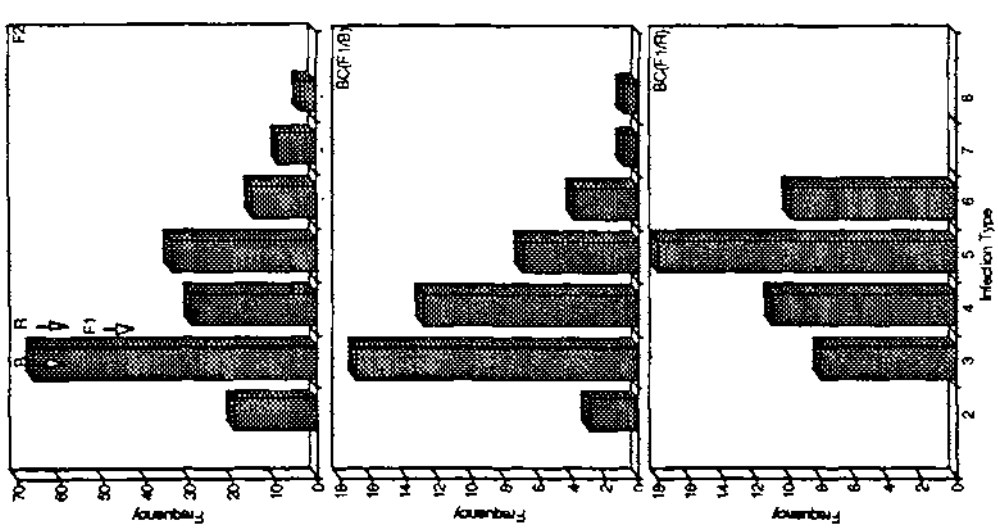


Table 80. Heritability estimates by different methods and genetic advance (GA) for the infection type in ten crosses of wheat inoculated with stripe rust pathotype 106E139A-

Cross	h^2_{BS}						h^2_{NS}	GA
	1	2	3	4	5	6		
Briscard/Tiritea	0.88	0.85	0.87	0.74	0.81	0.80	0.52	2.20
Ruapuna/Tiritea	0.86	0.84	0.85	0.84	0.84	0.84	0.43	1.52
Domino/Tiritea	0.92	0.88	0.88	0.85	0.86	0.86	0.66	2.22
Otane/Tiritea	0.76	0.75	0.76	0.73	0.75	0.74	0.61	1.55
Briscard/Otane	0.94	0.87	0.87	0.87	0.87	0.87	0.86	3.75
Ruapuna/Otane	0.83	0.87	0.87	0.71	0.81	0.79	0.65	2.64
Domino/Otane	0.78	0.81	0.83	0.80	0.80	0.80	0.63	2.66
Briscard/Domino	0.28	0.65	0.66	0.81	0.70	0.72	0.66	2.27
Ruapuna/Domino	0.03	0.50	0.53	0.31	0.44	0.41	0.37	1.10
Briscard/Ruapuna	0.23	0.81	0.81	0.76	0.79	0.79	0.75	2.93

h^2_{BS} : {(TMS-EMS)/r}/{[(TMS-EMS)/r]+EMS} (ANOVA method) used for 1. In this ratio $(V_{F2} - E_w) / V_{F2}$, environmental effect (E_w) is $(V_{P1}+V_{P2})/2$, $(V_{P1}+V_{P2})^{1/2}$, V_{F1}, $(V_{P1}+V_{P2}+V_{F1})/3$ and $(V_{P1}+V_{P2}+2V_{F1})/4$ for 2, 3, 4, 5 and 6, respectively.

h^2_{NS} : $[2V_{F2}-(V_{BC1}+V_{BC2})]/V_{F2}$

Table 81. Estimates of the number of segregating genes (effective factors) for infection type inoculated with stripe rust pathotype 106E139A-

Cross	Formula[1]						
	1	2	3	4	5	6	7
Briscard x Tiritea	1.7	1.6	2.5	1.2	1.7	1.2	1.9
Ruapuna x Tiritea	8.9	3.6	1.3	4.2	3.6	12.1	1.9
Domino x Tiritea	1.8	2.0	1.6	2.7	2.3	3.0	2.1
Otane x Tiritea	0.6	0.6	3.0	0.3	0.8	0.0	3.7
Briscard x Otane	1.4	0.9	0.4	2.5	0.9	1.6	1.9
Ruapuna x Otane	0.8	0.7	0.9	0.6	0.7	0.6	0.6
Domino x Otane	0.6	0.6	0.8	0.5	0.6	1.2	0.2
Briscard x Domino	0.0	0.0	0.0	0.0	0.0	0.0	0.3
Ruapuna x Domino	0.0	0.0	0.0	0.0	0.2	0.4	0.5
Briscard x Ruapuna	0.0	0.0	0.0	0.0	0.0	0.0	0.0

[1]1: $n = (\mu_{P2} - \mu_{P1})^2/[8(\sigma^2_{F2} - \sigma^2_{F1})]$

2: $n = (\mu_{P2} - \mu_{P1})^2/\{8[\sigma^2_{F2} - (0.5\sigma^2_{F1}+0.25\sigma^2_{P1}+0.25\sigma^2_{P2})]\}$

3: $n = (\mu_{P2} - \mu_{P1})^2/\{8[\sigma^2_{F2} - (\sigma^2_{BC1}+\sigma^2_{BC2})]\}$

4: $n = (\mu_{P2} - \mu_{P1})^2/\{8[(\sigma^2_{BC1} + \sigma^2_{BC2}) - (\sigma^2_{F1}+0.5\sigma^2_{P1}+0.5\sigma^2_{P2})]\}$

5: $n = \{(\mu_{P2} - \mu_{P1})^2[1.5-2h(1-h)]\}/\{8[\sigma^2_{F2} - 0.25(2\sigma^2_{F1}+\sigma^2_{P1}+\sigma^2_{P2})]\}$
where $h = (\mu F_1 - \mu P_1)/(\mu P_2 - \mu P_1)$

6: $n = (\mu_{F1} - \mu_{P1})^2/\{4[\sigma^2_{BC1} - 0.5(\sigma^2_{F1}+\sigma^2_{P1})]\}$

7: $n = (\mu_{P2} - \mu_{F1})^2/\{4[\sigma^2_{BC2} - 0.5(\sigma^2_{F1}+\sigma^2_{P2})]\}$
where $\mu P_1 < \mu P_2$

Table 82. Estimate of the genetic components of means , based on 2, 3, 4, 5, and 6 parameter models, for infection type in ten crosses of wheat

Briscard x Tiritea

m	6.13±0.06 **	5.98±0.09 **	4.43±0.30 **	5.99±0.09 **	6.13±0.10 **	4.40±0.30 **	4.38±0.68 **	6.18±0.10 **	4.93±0.74 **
[d]	2.64±0.09 **	2.70±0.09 **	2.63±0.09 **	2.67±0.10 **	2.67±0.09 **	2.58±0.10 **	2.63±0.09 **	2.58±1.10 **	2.58±0.10 **
[h]		0.41±0.18 *	2.58±0.44 **	0.36±0.19 (*)	-1.66±0.47 **	2.56±0.44 **	2.70±1.73 ns	-2.15±0.50 **	1.07±1.97 ns
[i]			1.72±0.31 **			1.77±0.32 **	1.76±0.68 **		1.25±0.74 (*)
[j]				0.54±0.52 ns		0.81±0.52 ns		1.44±0.55 **	1.04±0.60 (*)
[l]					2.53±0.53 **		-0.08±1.13 ns	2.98±0.55 **	1.01±1.29 ns
χ^2	38.05 **	33.08 **	23.81 **	32.01 **	9.87 **	0.61 ns	3.04 (*)	2.86 (*)	...

Ruapuna x Tiritea

m	6.57±0.05 **	6.48±0.08 **	6.82±0.35 **	6.48±0.08 **	6.46±0.08 **	6.82±0.35 **	6.58±0.50 **	6.46±0.09 **	6.55±0.54 **
[d]	2.28±0.08 **	2.29±0.08 **	2.30±0.08 **	2.28±0.09 **	2.28±0.08 **	2.29±0.09 **	2.29±0.08 **	2.29±0.09 **	2.29±0.09 **
[h]		0.24±0.18 ns	-0.28±0.56 ns	0.24±0.19 ns	0.72±0.45 ns	-0.30±0.57 ns	0.45±1.23 ns	0.75±0.48 ns	0.53±1.38 ns
[i]			-0.35±0.35 ns			-0.36±0.35 ns	-0.12±0.49 ns		-0.09±0.53 ns
[j]				0.02±0.36 ns		0.06±0.38 ns		-0.08±0.36 ns	-0.05±0.39 ns
[l]					-0.78±0.67 ns		-0.63±0.94 ns	-0.81±0.68 ns	-0.68±1.03 ns
χ^2	3.18 ns	1.45 ns	0.72 ns	1.45 ns	0.07 ns	0.44 ns	0.02 ns	0.03 ns	...

Table 82. Continued

Domino x Tiritea

m	6.39±0.05 **	6.39±0.09 **	5.88±0.21 **	6.40±0.09 **	6.54±0.10 **	6.03±0.22 **	7.00±0.48 **	6.50±0.11 **	6.45±0.76 **
[d]	1.49±0.09 **	2.17±0.09 **	2.12±0.10 **	2.31±0.10 **	2.18±0.09 **	2.26±0.11 **	2.22±0.10 **	2.25±0.11 **	2.25±0.11 **
[h]		1.01±0.15 **	1.69±0.29 **	1.15±0.15 **	-0.17±0.36 ns	1.61±0.29 **	-1.26±1.17 ns	0.26±0.47 ns	0.38±2.12 ns
[i]			0.64±0.23 **			0.46±0.24 (*)	-0.48±0.49 ns		0.05±0.75 ns
[j]				-1.16±0.35 **		-0.98±0.36 **		-0.60±0.45 ns	-0.64±0.68 ns
[l]					1.28±0.35 **		1.91±0.73 **	0.90±0.45 *	0.81±1.39 ns
χ^2	62.03 **	15.14 **	46.26 **	3.98 ns	1.83 ns	0.35 ns	0.87 ns	0.00 ns	...

Otane x Tiritea

m	7.63±0.04 **	7.92±0.07 **	7.46±0.16 **	7.92±0.07 **	8.08±0.08 **	7.46±0.16 **	8.77±0.37 **	8.09±0.08 **	9.15±0.40 **
[d]	0.73±0.07 **	0.71±0.07 **	0.70±0.07 **	0.69±0.08 **	0.72±0.07 **	0.67±0.08 **	0.74±0.07 **	0.67±0.08 **	0.67±0.08 **
[h]		-0.64±0.13 **	0.02±0.25 ns	-0.65±0.13 **	-1.93±0.31 **	0.02±0.25 ns	-3.75±1.00 **	-2.04±0.31 **	-4.87±1.09 **
[i]			0.55±0.18 **			0.55±0.18 **	-0.70±0.37 (*)		-1.06±0.39 **
[j]				0.22±0.32 ns		0.21±0.32 ns		0.52±0.33 ns	0.88±0.35 *
[l]					1.49±0.32 **		2.61±0.67 **	1.59±0.33 **	3.35±0.73 **
χ^2	36.94 **	31.43 **	51.52 **	30.96 **	9.85 **	21.05 **	6.17 (*)	7.31 **	...

Table 82. Continued

Briscard x Otane

m	5.86±0.06 **	5.58±0.09 **	6.87±0.38 **	5.62±0.09 **	5.48±0.09 **	6.95±0.38 **	5.61±0.61 **	5.54±0.09 **	6.77±0.65 **
[d]	2.09±0.08 **	2.11±0.08 **	2.13±0.08 **	1.86±0.09 **	2.09±0.08 **	1.88±0.09 **	2.10±0.08 **	1.89±0.09 **	1.86±0.09 **
[h]		0.78±0.20 **	-1.17±0.60 *	0.39±0.21 (*)	2.66±0.48 **	-1.64±0.60 **	2.38±1.45 (*)	1.80±0.51 **	-1.12±1.60 ns
[i]			-1.36±0.39 **			-1.41±0.39 **	-0.13±0.60 ns		-1.24±0.64 (*)
[j]				2.26±0.39 **		2.29±0.39 **		1.95±0.41 **	2.23±0.43 **
[l]					-2.88±0.66 **		-2.72±1.01 **	-2.07±0.68 **	-0.39±1.11 ns
χ^2	61.10 **	45.87 **	30.89 **	13.05 **	26.72 **	0.12 ns	26.67 **	3.72 (*)	...

Ruapuna x Otane

m	5.51±0.06 **	5.69±0.08 **	4.53±0.30 **	5.70±0.08 **	5.79±0.09 **	4.53±0.30 **	4.00±0.70 **	5.79±0.09 **	3.91±0.70 **
[d]	1.72±0.08 **	1.69±0.08 **	1.67±0.08 **	1.65±0.09 **	1.67±0.08 **	1.63±0.09 **	1.67±0.08 **	1.62±0.09 **	1.63±0.09 **
[h]		-0.54±0.18 **	1.10±0.44 *	-0.54±0.18 **	-1.98±0.48 **	1.11±0.44 *	2.58±1.83 ns	-1.97±0.48 **	2.86±1.83 ns
[i]			1.27±0.31 **			1.28±0.31 **	1.78±0.69 **		1.89±0.69 **
[j]				0.75±0.55 ns		0.78±0.55 ns		0.70±0.55 ns	0.84±0.55 ns
[l]					1.78±0.54 **		-1.00±1.20 ns	1.75±0.54 **	-1.19±1.21 ns
χ^2	26.68 **	19.88 **	5.19 (*)	17.97 **	9.03 *	0.96 ns	2.34 ns	7.41 *	...

Table 82. Continued

Domino x Otane

m	5.77±0.06 **	5.71±0.10 **	5.12±0.28 **	5.72±0.10 **	5.83±0.11 **	5.12±0.29 **	5.77±0.72 **	5.84±0.11 **	5.73±0.73 **
[d]	1.63±0.09 **	1.66±0.10 **	1.62±0.10 **	1.64±0.11 **	1.60±0.10 **	1.59±0.11 **	1.60±0.10 **	1.59±0.11 **	1.59±0.11 **
[h]		0.15±0.19 ns	0.94±0.40 *	0.14±0.19 ns	-1.03±0.51 *	0.94±0.40 *	-0.87±1.89 ns	-1.03±0.51 *	-0.76±1.91 ns
[i]			0.69±0.31 *			0.70±0.31 *	0.06±0.71 ns		0.10±0.72 ns
[j]				0.21±0.57 ns		0.28±0.58 ns		0.18±0.57 ns	0.19±0.58 ns
[l]					1.30±0.53 *		1.20±1.22 ns	1.29±0.53 *	1.13±1.24 ns
χ^2	6.75 ns	6.14 ns	2.00 ns	6.00 *	0.12 ns	0.83 ns	0.11 ns	0.02 ns	...

Briscard x Domino

m	3.82±0.06 **	3.96±0.10 **	4.16±0.23 **	3.79±0.10 **	3.95±0.12 **	4.16±0.23 **	4.83±0.57 **	3.95±0.12 **	4.83±0.57 **
[d]	0.27±0.10 *	0.29±0.10 **	0.28±0.10 **	0.30±0.12 *	0.29±0.10 **	0.29±0.12 *	0.30±0.10 **	0.30±0.12 *	0.30±0.12 *
[h]		-0.30±0.18 (*)	-0.56±0.33 (*)	-0.30±0.18 (*)	-0.19±0.48 ns	-0.56±0.33 (*)	-2.40±1.48 ns	-0.19±0.48 ns	-2.39±1.48 ns
[i]			-0.25±0.26 ns			-0.25±0.26 ns	-0.88±0.56 ns		-0.88±0.56 ns
[j]				-0.08±0.47 ns		-0.05±0.47 ns		-0.07±0.47 ns	-0.04±0.47 ns
[l]					-0.11±0.45 ns		1.22±0.96 ns	-0.11±0.45 ns	1.22±0.96 ns
χ^2	5.31 ns	2.56 ns	6.43 *	2.53 ns	2.49 ns	1.61 ns	0.01 ns	2.47 ns	...

Table 82. Continued

Ruapuna x Domino

m	4.55±0.05 **	4.25±0.09 **	4.32±0.26 **	4.25±0.09 **	4.21±0.10 **	4.32±0.26 **	3.52±0.52 **	4.21±0.10 **	3.53±0.52 **
[d]	-0.04±0.09 ns	-0.02±0.09 ns	-0.02±0.09 ns	0.04±0.10 ns	-0.02±0.09 ns	0.04±0.10 ns	-0.02±0.09 ns	0.04±0.10 ns	0.04±0.10 ns
[h]		0.77±0.18 **	0.68±0.42 ns	0.78±0.18 **	1.22±0.43 **	0.67±0.42 ns	3.01±1.39 *	1.23±0.43 **	2.98±1.39 *
[i]			-0.07±0.27 ns			-0.08±0.27 ns	0.70±0.52 ns		0.68±0.52 ns
[j]				-0.59±0.43 ns		-0.60±0.43 ns		-0.60±0.43 ns	-0.58±0.43 ns
[l]					-0.58±0.51 ns		-1.67±0.95 (*)	-0.59±0.51 ns	-1.65±0.95 (*)
χ^2	6.75 **	5.03 ns	41.76 **	3.11 ns	3.68 ns	3.03 (*)	1.85 ns	1.76 ns	...

Briscard x Ruapuna

m	4.06±0.06 **	3.96±0.09 **	4.04±0.27 **	3.96±0.09 **	3.90±0.11 **	4.02±0.27 **	2.57±0.65 **	3.91±0.11 **	2.74±0.66 **
[d]	0.33±0.09 **	0.34±0.09 **	0.34±0.09 **	0.25±0.11 *	0.34±0.09 **	0.26±0.11 *	0.32±0.09 **	0.26±0.11 *	0.26±0.11 *
[h]		0.29±0.18 ns	0.18±0.39 ns	0.26±0.18 ns	0.89±0.47 (*)	0.18±0.39 **	4.27±1.68 *	0.78±0.47 (*)	3.74±1.71 *
[i]			-0.09±0.29 ns			-0.07±0.29 **	1.34±0.64 *		1.17±0.65 (*)
[j]				1.03±0.51 *		1.03±0.51 *		0.96±0.51 (*)	0.80±0.52 ns
[l]					-0.70±0.50 ns		-2.74±1.09 *	-0.59±0.50 ns	-2.38±1.12 *
χ^2	11.28 *	8.75 *	10.96 **	4.61 (*)	6.78 *	4.55 *	2.40 ns	3.23 (*)	...

Table 83. The components of variation in six generations of wheat for infection type inoculated with stripe rust pathotype 106E139A-

Cross	D	H	F	E_w	$(H/D)^{1/2}$	$F/(D*H)^{1/2}$
Briscard x Tiritea	2.70	2.60	+2.11	0.53	0.98	0.80
Ruapuna x Tiritea	1.54	2.97	-0.83	0.29	1.39	0.39
Domino x Tiritea	2.14	1.32	+0.73	0.22	0.79	0.43
Otane x Tiritea	1.14	0.48	-0.67	0.24	0.65	0.91
Briscard x Otane	4.66	0.16	-0.19	0.35	0.19	0.22
Ruapuna x Otane	3.06	1.28	-0.43	0.52	0.65	0.22
Domino x Otane	1.32	0.40	-0.58	0.77	0.55	0.80
Briscard x Domino	2.44	0.40	-0.28	0.47	0.42	0.30
Ruapuna x Domino	0.82	0.20	+0.09	0.66	0.49	0.22
Briscard x Ruapuna	3.28	0.32	+0.70	0.47	0.31	0.68

4.5.2. Latent period

4.5.2.1. Briscard/Tiritea

The F-test showed all generations are significantly different (Table 84). The mean latent periods for Briscard and Tiritea were 15.1 and 11.7 respectively and the mean latent period of the F_1 was 13.2 (Table 84) which was less than midparent value in the direction of a shorter latent period (susceptible). The mean backcross values were significantly different from each other (Table 84). The degree of dominance was 0.10 for the short latent period. The frequency distributions of the latent period of the F_2 plants and both backcrosses were continuous, unimodal and skewed toward the shorter latent period (Fig. 22). Transgressive segregation was observed in F_2 plants toward long latent period. It can be concluded that the short latent period parent, Tiritea, may possess a gene or genes which contribute toward a longer latent period in this cross. The latent period of the backcross to Briscard was significantly longer than the latent period of the F_1 or the backcross to Tiritea. Heritability estimates for latent period are presented in Table 85. Heritability for this cross was different with different formulae, but broad and narrow sense heritabilities were 0.57±0.18 and 0.43±0.11, respectively (Table 85) and genetic advance was 1.41, assuming the top 1% of segregating plants were selected for resistance. The range of minimum number of genes was from 0.4±0.3 to 2.2±0.79 (Table 86).

The results of generation mean analysis for this cross showed that the χ^2 of the three-parameter model, - m, [d] and [h] was significant at the 10% probability level, suggesting the presence of digenic interactions (Table 87). The six-parameter model was fitted which showed [j] and [l] are not significant. Then other possible models were fitted, and the best model for this cross was m, [d], [h], [i] and [l]; and χ^2 also showed the adequacy of this model, with no presence of trigenic interactions. The m value in this model was almost the same as the m value in the six-parameter model and all standard errors were smaller than those in the six-parameter model, which demonstrates the precision of the model. This does not support the method of Mather and Jinks (1982), in which the removal of nonsignificant component(s) of the six-parameter model resulted the best model to be fitted with means, which were m, [d], [h], and [i] in this cross. But two components were not significant. The m value was 13.31 in this model whereas in the six-parameter model the m value was 11.92. The estimates of components of variation D and H were 1.63 and 0.46, respectively (Table 88), indicating the additive

component is about three times that of the dominance component. The F value was -0.04 which agreed with comparison of the F_1 mean with the mid-parent. This indicated that the genes from Tiritea are dominant. The estimate of average dominance of the gene was 0.53, indicating partial dominance. The ratio $F/(D*H)^{1/2}$ was 0.05, indicating the denominator was greater than the numerator and no evidence that the dominance deviations at different loci are not particularly consistent in sign or magnitude. There is good reason to believe that even though the genes from Tiritea for shorter latent period showed dominance, some of them must be recessive to their alleles from Briscard (Mather and Jinks, 1982).

4.5.2.2. Ruapuna/Tiritea

The F-test showed statistical differences between the generations (Table 84). The means of the latent period for Ruapuna, Tiritea and the F_1 were 14.8, 11.7 and 13.0 days respectively (Table 84). The frequency distribution of latent period for the F_2 plants was continuous and almost normal (Fig. 22) and there was transgressive segregation toward a longer latent period. The degree of dominance was 0.20 for a shorter latent period. The mean latent period of the F_1 backcross to Ruapuna was longer than that of the F_1 backcross to Tiritea which was 12.2 days (Table 84). Broad and narrow sense heritabilities were 0.73±0.09 and 0.64±0.08, respectively and the estimate of genetic advance was 1.91 (Table 85). The minimum number of genes controlling resistance was estimated from 0.9±0.31 to 11.7±3.2 (Table 86).

In generation analysis, the joint scaling test to verify the adequacy of the three-parameter model involving m, [d] and [h] revealed a lack of good fit ($\chi^2 = 9.48$) at the 5% probability level (Table 87), suggesting the presence of epistasis. Based on the findings of the test for epistasis, the six-parameter model was fitted to the observed family means. This indicated that the five components m, [d], [h], [i] and [l] were significant at the 1% probability level and [i] was significant at the 10% probability level respectively. Then all possible models were fitted to the observed means and all models, except the m, [d], [h], [i] and [l] model, had at least one nonsignificant component. This supported the method of Mather and Jinks (1982), in which the removal of a nonsignificant component such as [j] from the six-parameter model, and the fitting of the rest of the components as a model resulted in a better fit. The five-parameter model without [j] revealed that the standard error of all components was reduced and χ^2 gave a good fit

to the observed family means, (Table 87), suggesting the absence trigenic interactions in contributing to the differences among the generation means. The components of variation were 1.54 and 2.97 for D and H, respectively (Table 88). This agrees with relatively high level of dominance which has been already obtained from analysis of the means. The F value was negative (-0.83), indicating the genes from the susceptible parent, Tiritea, were dominant. The average of dominance was 1.39 and the ratio $F/(D*H)^{1/2}$ was 0.39, indicating little evidence that h/d at different loci are consistent in sign or magnitude.

4.5.2.3. Domino/Tiritea

The mean latent period of Domino, Tiritea and the F_1 were 13.5, 11.7 and 12.1 respectively (Table 84). The results of the F-test and least significant differences are presented in Table 84, which shows differences amongst means, but there is no significant difference between the F_1 and the shorter latent period parent. Based on the mean of the F_1 and both parents, the degree of dominance was 0.54 which showed the short latent period parent was partially dominant. The mean to the backcrosses were not significantly different (Table 84). The distribution of latent period of the F_2 plants was continuous, skewed toward shorter latent period (Fig. 22) and transgressive segregation was observed toward longer latent period. Estimated broadsense and narrowsense heritabilities for this cross were 0.70±0.18 and 0.49±0.14 and the genetic advance was 1.29 (Table 85) and the number of genes was less than one (Table 86).

In generation means, analysis of genetic components of the means in this cross suggests that there was non-allelic interactions, because χ^2 in the three-parameter model was significant (Table 87). The six-parameter model showed that [h] and [l] was not significant. But [h] was significant in the three-parameter model. Then from the fitting of possible models, only the five-parameter model involving m, [d], [h], [i] and [j], is significant and χ^2 which was not significant showed the adequacy of that model (Table 87). The reduction of the standard error in this model in comparison to the six-parameter model revealed the precision of this model as well (Table 87). All components of variation are presented in Table 88. The D and H values were close to each other. The negative F value agreed with the deviation of F_1 from the mid-parent, indicating the gene from the shorter latent period, Tiritea, was dominant. The average dominance of this cross was close to one, as was clear from D and H values. The ratio $F/(D*H)^{1/2}$ was 0.45,

indicating little evidence for consistency of dominance deviation in sign and magnitude.

4.5.2.4. Otane/Tiritea

All generations except the parent Tiritea were in a group with no differences in latent period between them (Table 84). The mean latent period of the F_1 plants was similar to Otane, indicating full dominance for longer latent period. The means and distributions of both backcrosses were the same (Fig. 22). The F_2 population was skewed toward a longer latent period (Fig. 22) and transgressive segregation was observed toward longer latent period. The broad and narrow sense heritabilities were 0.62±0.07 and 0.60±0.07, respectively and the genetic advance was 1.43 (Table 85). The number of genes was less than one (Table 86).

In generation mean analysis, the χ^2 value was significant at the 5% probability level for the three-parameter model, suggesting the effect of epistasis in this cross (Table 87). In the six-parameter model [i] and [j] were significant at the 10% level of probability, suggesting additive x additive (i) or additive x dominance (j) both of them are present in this cross. By fitting all possible models, the four-parameter model involving m, [d], [h] and [j] was the best. The [j] and χ^2 values were significant at the 10% probability level, which showed the adequacy of the model. The components of variation are presented in Table 88. The additive component was more important than the dominance component. The positive value of F was in agreement with the deviation of the F_1 mean from the mid-parent, indicating Otane the longer latent period parent, had dominant genes. The average dominance was low (0.20), indicating the importance of the additive component. The ratio $F/(D*H)^{1/2}$ was 0.90, indicating the measure of dominance is constant in both magnitude and sign for all the gene-pairs involved.

4.5.2.5. Briscard/Otane

Differences were observed amongst all generations (Table 84). Mean latent period of Briscard, Otane and the F_1 were 15.1, 12.5 and 13.1 days respectively (Table 84). The mean latent period of the backcross F_1 with Briscard (13.9) was significantly longer than the mean latent period of the backcross F_1 with Otane (12.5). The deviations of the F_1 from the midparent indicated a partial dominance (0.55) toward the short latent period parent. The frequency distribution of the latent period for the F_2 plants was

continuous and skewed toward a shorter latent period (Fig. 22) and there was transgressive segregation toward a longer latent period. The frequency distributions of both backcrosses were continuous and skewed toward a shorter latent period. The broad and narrow sense heritabilities were 0.45±0.13 and 0.26±0.07, respectively and genetic advance was 0.82 (Table 85), which is very low due to narrow sense heritability. The range of the minimum number of genes was from 0.2±0.09 to 1.4±0.08 (Table 86).

For generation mean analysis, the χ^2 value based on the three-parameter model was significant (Table 87). This indicated that there was epistasis in this cross, but when the six-parameter model was used not only were no interactions significant, but the [h] value also was not significant. By referring to other models which showed at least m, [d] and [h] were significant, it has been found that the four-parameter model involving m, [d], [h] and [l], is the best model to be fitted to these means. The χ^2 value for this model was not significant, indicating the adequacy of the model and also there was no trigenic interaction present. All components of variation are presented in Table 88. The dominance component (H) was greater than additive component (D). For that reason the average dominance was 1.19. The gene in Otane for shorter latent period, were dominant because the F value was negative. This can also be supported by deviation the F_1 from the mid-parent. The ratio $F/(D*H)^{1/2}$ was 0.88, indicating that h/d was constant in magnitude and sign.

4.5.2.6. Ruapuna/Otane

The F-test showed significant differences amongst generations (Table 84). The mean latent period of Ruapuna (14.8 days) was significantly longer than that of Otane (12.5 days). The mean latent period of the F_1 plants was 13.3 days and the degree of dominance was 0.27 toward shorter latent period (Table 84). The distribution of latent period of the F_2 population was continuous and almost normal (Fig. 22). There was transgressive segregation toward longer latent period; this also happened in the backcross with Ruapuna in which almost period of up to 19 days was observed. Transgressive segregation toward shorter latent period was not observed in either in the F_2 or in the backcross with Otane. The broad and narrow sense heritabilities were 0.67±0.16 and 0.65±0.16 respectively (Table 85) and the genetic advance was 2.27 which was relatively high (Table 85). The number of genes was less than one (Table 86).

Generation mean analysis under the three-parameter model suggested that there is non-allelic interaction in this cross. The χ^2 value was significant (Table 87). The six-parameter model showed that three- components involving m, [d] and [i] are significant (Table 87). Removing the nonsignificant components from the six-parameter model indicated that the standard error of all three components was reduced, 14.06±0.08 , 1.26±0.10 and -0.39±0.15 for m, [d] and [i] respectively. The χ^2 value (29.59) was significant, suggesting trigenic interaction for this cross. The components of variation are presented in Table 88. The D value was greater than the H value (2.26 and 0.20, respectively), therefore the average of dominance was not high. The negative F value indicated that genes from Otane, the susceptible cultivar, were dominant. Dominance was different in sign and magnitude due to the value of the ratio $F/(D*H)^{1/2}$.

4.5.2.7. Domino/Otane

There was no significant differences amongst generations (Table 84), although the difference between the two parents was one day. The frequency distributions of latent period for all generation are summarised in Fig. 22. In the F_2 distribution, transgressive segregation was observed for long latent period. Broad sense and narrow sense heritabilities were 0.50±0.11 and 0.23±0.0.07 respectively (Table 85). Because of the low narrow sense heritability, the genetic advance was low as well (Table 85). The number of genes was less than one (Table 86).

The results of generation mean analysis in this cross showed that three components were significant and the χ^2 indicated the presence of epistasis (Table 87). The six-parameter model showed [l] was significant at 10% probability level. By using all possible models, the two best models were recognised for this cross. One of them was the five-parameter model involving m, [d], [h], [j] and [l] and the second one was the four-parameter model m, [d], [h], and [l]. In the five-parameter model [j] was significant at 10% probability level. The χ^2 for both models, showed the adequacy of them. Both models had a reduction in their standard errors when contrasted with the six-parameter model. The value of components in the four-parameter model was almost the same as those in the six-parameter model, while there were differences between those values in the five-and six-parameter models. So the four-parameter model fitted better than the five-parameter model. The D and H values were 1.21 and 5.77, respectively (Table 88). This indicated dominance component was high and for that reason the average

dominance was 2.18. The F value was positive, indicating the excess of F1 over the mid-parent. The ratio $F/(D*H)^{1/2}$ was close to zero, suggesting that some genes are dominant and some recessive in each parent and also they may be different in their sign.

4.5.2.8. Briscard/Domino

The differences amongst generations are shown in Table 84. The mean latent periods of Briscard, Domino and the F_1 were 15.1, 13.5 and 14.0 days (Table 84). The degree of dominance was 0.38 and the frequency of the F_2 plants were almost normally distributed (Fig. 22). Transgressive segregation was observed toward both longer and shorter latent period. Broad and narrow sense heritabilities were 0.66±0.21 and 0.30±0.11 respectively and the genetic advance was 1.36 which is relatively high (Table 85). The number of genes was less than one (Table 86).

The results of the generation mean for this cross showed that the two components involving m and [d] played major roles in wheat for the expression of variability of the latent period to stripe rust (Table 87). All models had agreement on these two components. The χ^2 value showed the adequacy of the model (Table 87). All components of variation are presented in Table 88. The H value was high in comparison with D value, so the $(H/D)^{1/2}$ was more than unity, indicating overdominace. The F value was negative, indicating genes from Domino for shorter latent period, were dominant and it was supported by the deviation of the F_1 from the mid-parent. The $F/(D*H)^{1/2}$ was 0.98, indicating h/d was constant in sign and magnitude in different loci.

4.5.2.9. Ruapuna/Domino

In this cross, Ruapuna was significantly different from other generations (Table 84). The mean latent period of Ruapuna was 14.8 days whereas for Domino it was 13.5 days (Table 84) and the mean latent period for the F_1 was 13.1 days. There were no statistical difference between Domino and the F_1, it is suggested that there is complete dominance for shorter latent period, that the degree of dominance is 1. Transgressive segregation was observed for longer latent period (Fig. 22). The latent periods of the F_2 population were almost normally distributed. Broad and narrow sense heritabilities were 0.38 and 0.10, respectively and the genetic advance was low because of the narrow sense heritability (Table 85). The range of number of genes was from 0.5±0.09 to

2.0±0.8 (Table 86).

The result of generation mean analysis for this cross showed that the χ^2 value for the three-parameter model was significant, indicating the presence of digenic interaction (Table 87). The six-parameter model revealed that only [i] is significant. The removal of nonsignificant components resulted a four-parameter model involving m, [d], [h] and [i]. The χ^2 value of this model was not significant (Table 87), indicating the adequacy of this model. The components of variation are presented in Table 88. The dominance component was greater the than additive component, thus the average dominance was 2.30 which indicated the importance of dominance part. The F value was negative which agreed with F_1 deviation from the mid-parent, indicating the genes from Domino for shorter latent period, were dominant. The ratio $F/(D*H)^{1/2}$ was 0.65, suggesting that although the genes from Domino showed dominance, some of the must be recessive to their alleles from Ruapuna.

4.5.2.10. Briscard/Ruapuna

The two parents were not significantly different and other generations overlapped (Table 84). The means for Briscard, Ruapuna and the F_1 were 15.1, 14.8 and 14.3 respectively. The F_2 distribution was normal and transgressive segregation was observed for both shorter and longer period (Fig. 22). Transgressive segregation for longer latent period up to 19 days was also observed in the backcross to Briscard. Broad and narrow sense heritabilities were 0.72±0.14 and 0.66±0.11, respectively and the highest genetic advance comparable with other crosses was obtained in this cross (Table 85). The number of genes was less than one (Table 86).

In generation mean analysis, the χ^2 value was significant for the three-parameter model (Table 87). The [d] was not significant with this model which was in agreement with other models (Table 87). The six-parameter model showed that only [i] and [l] are significant. The best model which fitted with this cross was m, [h], [i] and [l]. This model was in fit to the five-parameter model m, [d], [h], [i] and [l] in which [d] was not significant. The χ^2 value showed the adequacy of this model. The similarity of the mean effect 'm' in this model with six-parameter model and also the reduction of standard errors of this model in comparison with the six-parameter model showed the precision of this model as well. The components of variation are presented in Table 88. The

additive component was much greater than the dominance component. The F value was negative in which it agreed with the deviation of the F_1 from the mid-parent, indicating that those genes in Ruapuna were dominant. The average dominance was 0.42 and the ratio $F/(D*H)^{1/2}$ showed that some genes in Ruapuna were recessive to their alleles from Briscard.

Table 84. Differences among the mean values of six generations for the latent period of stripe rust pathotype 106E139A- in ten crosses of wheat

Generation[1]	Cross[3]									
	1	2	3	4	5	6	7	8	9	10
P1	15.1 a[2]	14.8 a	13.5 a	11.7 a	15.1 a	14.8 a	13.5 a	15.1 a	14.8 a	15.1 a
BC1	14.2 ab	13.5 b	12.7 b	12.3 b	13.9 b	14.7 a	13.4 a	15.0 a	13.5 b	14.4 ab
F1	13.2 bc	13.0 b	12.1 cd	12.5 b	13.1 bc	13.3 b	12.8 a	14.0 b	13.1 b	14.3 ab
F2	13.1 c	13.2 b	12.7 b	12.5 b	13.0 c	14.4 a	13.4 a	14.1 ab	13.7 b	14.9 ab
BC2	12.7 c	12.2 c	12.3 bc	12.4 b	12.5 c	13.3 b	13.3 a	13.9 b	13.4 b	14.2 b
P2	11.7 d	11.7 c	11.7 d	12.5 b	12.5 c	12.5 c	12.5 a	13.5 b	13.5 b	14.8 ab
1)F-test	**	**	**	**	**	**	**	*	ns	*
2)$LSD_{0.05}$:	1.01	0.88	0.62	0.53	0.59	0.82	0.82	0.47	0.81	0.74

*, ** and ns : Significant at 5 % , 1 % probability level and not significant, respectively
Least square means which have a same letter are not significantly different

3) 1:Briscard(P_1) × Tiritea(P_2)
2:Ruapuna(P_1) × Tiritea(P_2)
3:Domino(P_1) × Tiritea(P_2)
4:Otane(P_1) × Tiritea(P_2)
5:Briscard(P_1)× Otane(P_2)
6:Ruapuna(P_{12}) × Otane(P_{21})
7:Domino(P_{12}) × Otane(P_{21})
8:Briscard(P_{12}) × Domino(P_{21})
9:Ruapuna(P_{12}) × Domino(P_2)
10:Briscard(P_1) × Ruapuna(P_2)

Fig. 22. Frequency distributions of the latent period of stripe rust pathotype 106E139A- in ten crosses of wheat

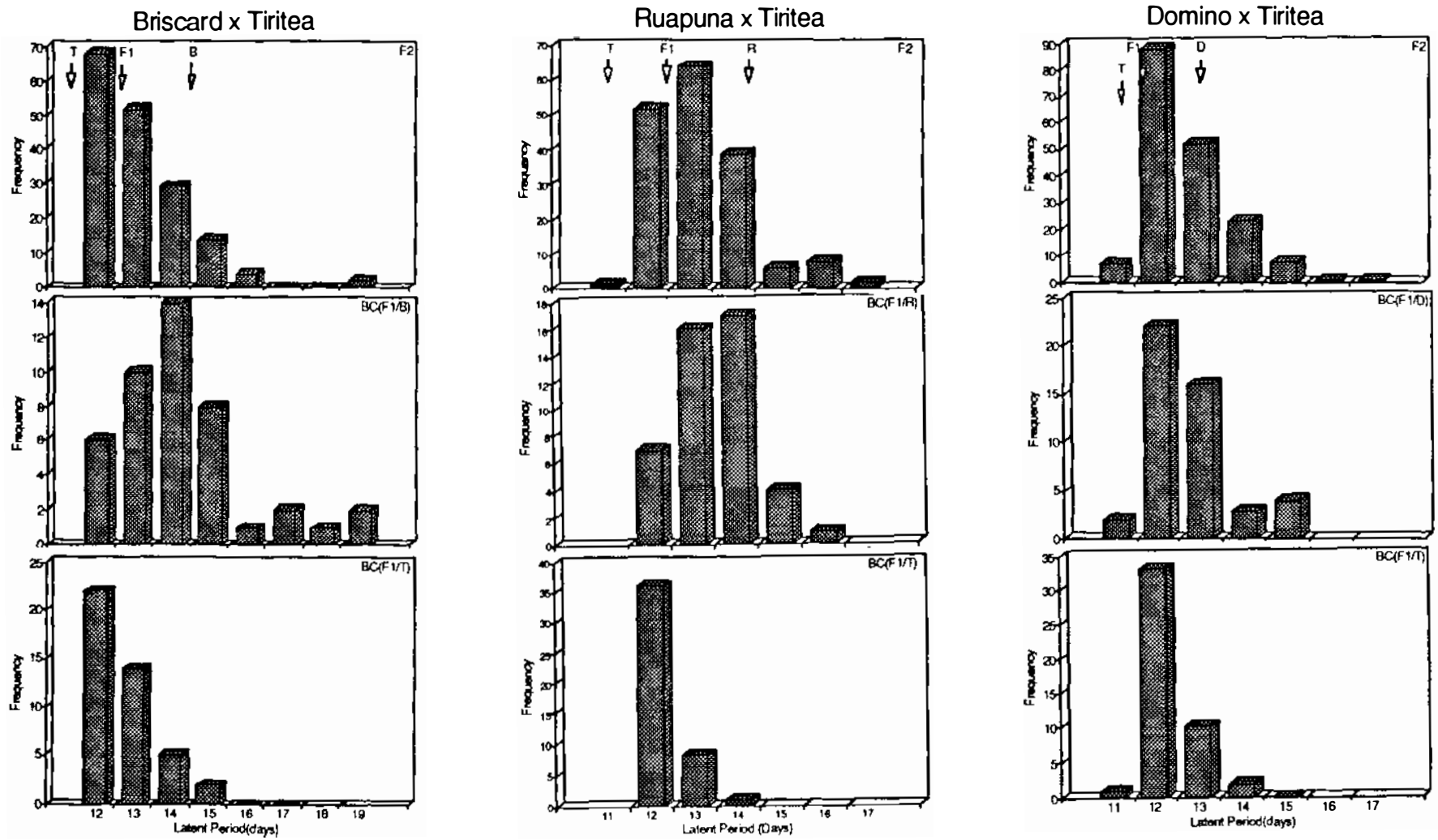


Fig. 22. Continued

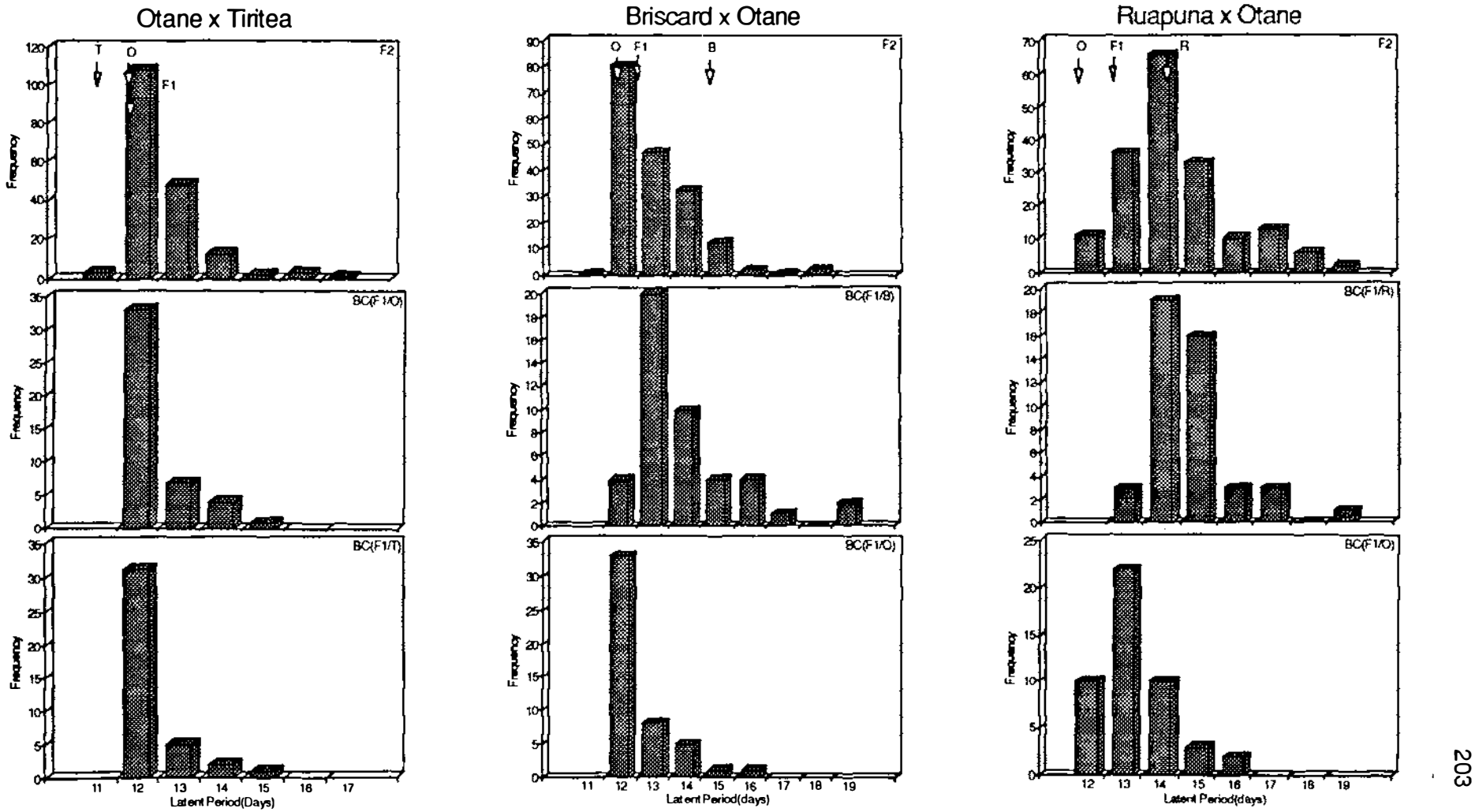
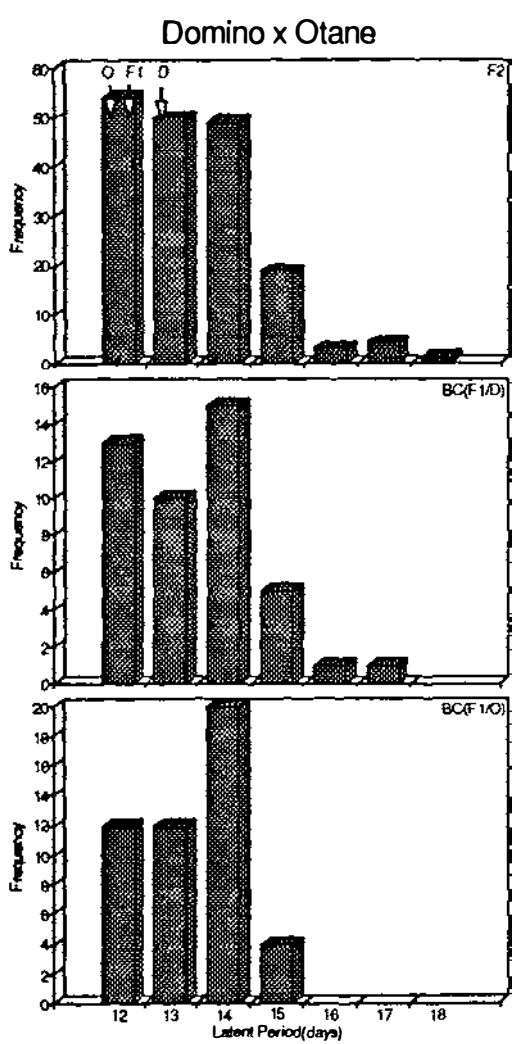
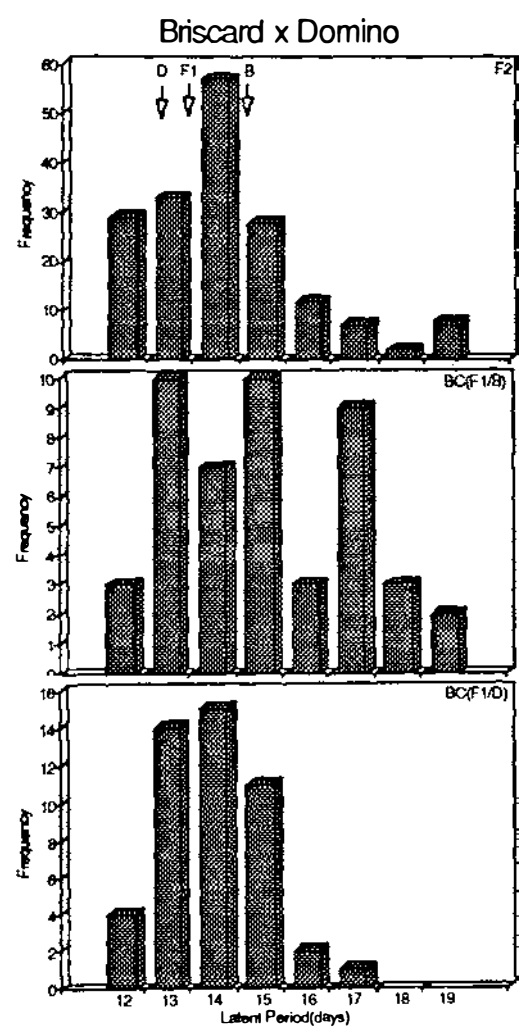


Fig. 22. Continued





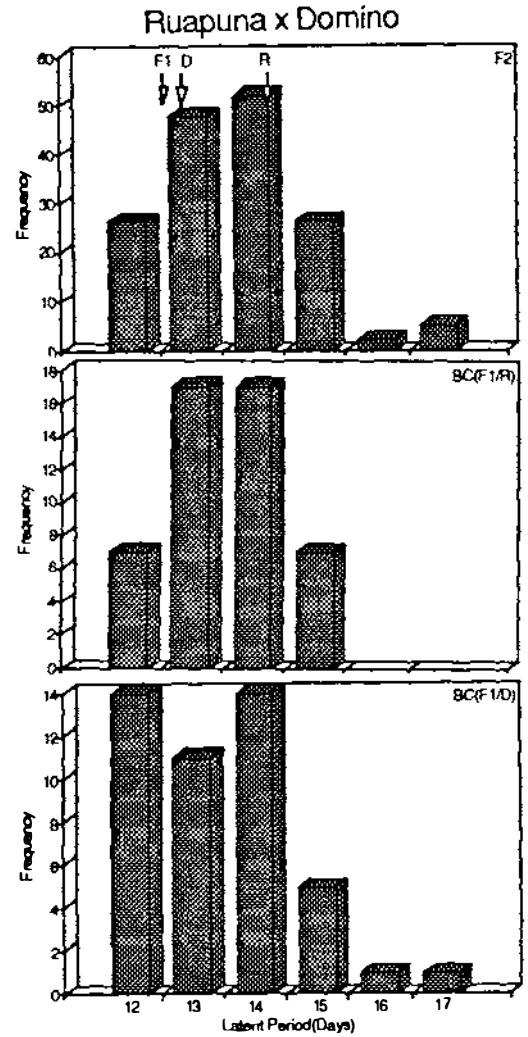


Fig. 22. Continued

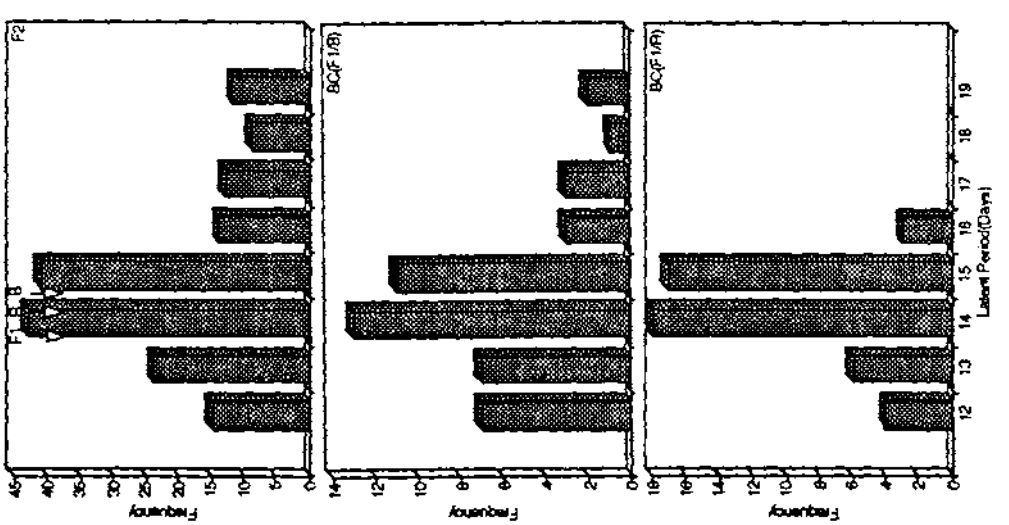


Table 85. Heritability estimates by different methods and genetic advance (GA) for the latent period in ten crosses of wheat inoculated with stripe rust pathotype 106E139A-

Cross	h^2_{BS}						h^2_{NS}	GA
	1	2	3	4	5	6		
Briscard/Tiritea	0.71	0.47	0.64	0.67	0.54	0.57	0.43	1.41
Ruapuna/Tiritea	0.86	0.83	0.83	0.63	0.76	0.73	0.64	1.91
Domino/Tiritea	0.70	0.50	0.58	0.91	0.63	0.70	0.49	1.29
Otane/Tiritea	0.39	0.71	0.70	0.54	0.65	0.62	0.60	1.43
Briscard/Otane	0.70	0.32	0.54	0.57	0.40	0.45	0.26	0.82
Ruapuna/Otane	0.87	0.68	0.73	0.68	0.68	0.67	0.65	2.27
Domino/Otane	0.07	0.66	0.70	0.34	0.55	0.50	0.23	0.75
Briscard/Domino	0.41	0.58	0.61	0.73	0.63	0.66	0.30	1.36
Ruapuna/Domino	0.20	0.39	0.39	0.37	0.39	0.38	0.10	0.31
Briscard/Ruapuna	0.30	0.74	0.74	0.70	0.73	0.72	0.66	3.30

h^2_{BS} : {(TMS-EMS)/r}/{[(TMS-EMS)/r]+EMS} (ANOVA method) used for 1. In this ratio $(V_{F2} - E_w) / V_{F2}$, environmental effect (E_w) is $(V_{P1}+V_{P2})/2$, $(V_{P1}+V_{P2})^{1/2}$, V_{F1}, $(V_{P1}+V_{P2}+V_{F1})/3$ and $(V_{P1}+V_{P2}+2V_{F1})/4$ for 2, 3, 4, 5 and 6, respectively.

h^2_{NS} : $[2V_{F2}-(V_{BC1}+V_{BC2})]/V_{F2}$

Table 86. Estimates of the number of segregating genes (effective factors) for latent period inoculated with stripe rust pathotype 106E139A-

Cross	Formula[1]						
	1	2	3	4	5	6	7
Briscard x Tiritea	1.4	1.6	2.2	0.6	1.6	1.5	0.4
Ruapuna x Tiritea	1.5	1.6	0.9	11.7	1.6	3.2	3.9
Domino x Tiritea	0.4	0.6	0.6	0.6	0.6	0.2	0.9
Otane x Tiritea	0.2	0.1	0.1	0.2	0.2	0.8	0.0
Briscard x Otane	1.1	1.4	1.4	0.5	1.6	0.2	0.7
Ruapuna x Otane	0.4	0.4	0.3	0.6	0.4	0.3	0.9
Domino x Otane	0.2	0.2	0.2	0.2	0.2	0.1	0.2
Briscard x Domino	0.2	0.2	0.4	0.1	0.2	0.1	0.1
Ruapuna x Domino	0.9	0.6	0.7	0.5	1.3	0.1	2.0
Briscard x Ruapuna	0.0	0.0	0.0	0.0	0.1	0.8	0.1

[1]1: $n = (\mu_{P2} - \mu_{P1})^2/[8(\sigma^2_{F2} - \sigma^2_{F1})]$

2: $n = (\mu_{P2} - \mu_{P1})^2/\{8[\sigma^2_{F2} - (0.5\sigma^2_{F1}+0.25\sigma^2_{P1}+0.25\sigma^2_{P2})]\}$

3: $n = (\mu_{P2} - \mu_{P1})^2/\{8[\sigma^2_{F2} - (\sigma^2_{BC1}+\sigma^2_{BC2})]\}$

4: $n = (\mu_{P2} - \mu_{P1})^2/\{8[(\sigma^2_{BC1} + \sigma^2_{BC2}) - (\sigma^2_{F1}+0.5\sigma^2_{P1}+0.5\sigma^2_{P2})]\}$

5: $n = \{(\mu_{P2} - \mu_{P1})^2[1.5-2h(1-h)]\}/\{8[\sigma^2_{F2} - 0.25(2\sigma^2_{F1}+\sigma^2_{P1}+\sigma^2_{P2})]\}$
where $h = (\mu F_1 - \mu P_1)/(\mu P_2 - \mu P_1)$

6: $n = (\mu_{F1} - \mu_{P1})^2/\{4[\sigma^2_{BC1} - 0.5(\sigma^2_{F1}+\sigma^2_{P1})]\}$

7: $n = (\mu_{P2} - \mu_{F1})^2/\{4[\sigma^2_{BC2} - 0.5(\sigma^2_{F1}+\sigma^2_{P2})]\}$
where $\mu P_1 < \mu P_2$

Table 87. Estimate of the genetic components of means , based on 2, 3, 4, 5, and 6 parameter models, for latent period in ten crosses of wheat

Briscard x Tiritea

m	13.24±0.06 **	13.28±0.12 **	12.98±0.24 **	13.31±0.12 **	13.28±0.13 **	12.97±0.24 **	11.71±0.59 **	13.38±0.14 **	11.92±0.69 **
[d]	1.52±0.10 **	1.54±0.12 **	1.60±0.13 **	1.63±0.13 **	1.55±0.12 **	1.71±0.14 **	1.64±0.13 **	1.68±0.14 **	1.68±0.14 **
[h]		-0.07±0.20 ns	0.34±0.37 ns	-0.18±0.21 ns	-0.12±0.49 ns	0.31±0.37 ns	3.96±1.57 *	-0.58±0.55 ns	3.33±1.98 (*)
[i]			0.38±0.28 ns			0.46±0.28 ns	1.63±0.60 **		1.45±0.67 *
[j]				-0.81±0.52 ns		-0.95±0.52 (*)		-0.99±0.56 (*)	-0.38±0.63 ns
[l]					0.05±0.49 ns		-2.46±1.04 *	0.42±0.53 ns	-2.04±1.25 ns
χ^2	7.96 (*)	7.83 (*)	5.67 (*)	5.33 (*)	7.82 (*)	2.66 ns	0.37 ns	4.70 *	...

Ruapuna x Tiritea

m	13.03±0.05 **	13.19±0.10 **	13.35±0.23 **	13.20±0.10 **	13.23±0.11 **	13.35±0.23 **	14.25±0.43 **	13.27±0.12 **	14.56±0.47 **
[d]	1.44±0.08 **	1.50±0.09 **	1.49±0.09 **	1.53±0.11 **	1.51±0.09 **	1.51±0.11 **	1.45±0.09 **	1.57±0.12 **	1.57±0.12 **
[h]		-0.34±0.15 (*)	-0.57±0.35 (*)	-0.36±0.19 (*)	-0.61±0.39 ns	-0.58±0.25 (*)	-3.03±1.06 **	-0.79±0.44 (*)	-3.97±1.20 **
[i]			-0.20±0.25 ns			-0.19±0.26 ns	-1.05±0.43 **		-1.30±0.45 **
[j]				-0.14±0.33 ns		-0.19±0.34 ns		-0.29±0.36 ns	-0.64±0.38 (*)
[l]					0.33±0.41 ns		1.72±0.70 **	0.47±0.45 ns	2.35±0.80 **
χ^2	13.17 **	9.48 *	23.28 **	9.29 **	8.82 *	8.76 **	2.82 (*)	8.18 **	...

Table 87. Continued

Domino x Tiritea

m	12.42±0.04 **	12.75±0.09 **	13.33±0.17 **	12.78±0.09 **	12.50±0.11 **	13.26±0.17 **	13.01±0.45 **	12.58±0.11 **	13.40±0.48 **
[d]	0.66±0.08 **	0.81±0.09 **	0.72±0.09 **	0.99±0.11 **	0.75±0.09 **	0.87±0.11 **	0.72±0.09 **	0.88±0.11 **	0.88±0.11 **
[h]		-0.53±0.12 **	-1.22±0.21 **	-0.60±0.12 **	0.87±0.38 *	-1.16±0.21 **	-0.37±1.13 ns	0.52±0.42 ns	-1.55±1.24 ns
[i]			-0.82±0.20 **			-0.70±0.21 **	-0.52±0.45 ns		-0.82±0.47 (*)
[j]				-1.19±0.35 **		-0.90±0.36 **		-0.76±0.38 *	-0.95±0.40 *
[l]					-1.27±0.32 **		-0.54±0.71 ns	-0.99±0.35 **	0.25±0.78 ns
χ^2	41.61 **	22.71 **	14.10 **	11.16 **	7.07 *	0.10 ns	5.73 *	3.17 (*)	...

Otane x Tiritea

m	12.35±0.04 **	12.13±0.07 **	12.58±0.19 **	12.14±0.07 **	12.07±0.08 **	12.58±0.19 **	12.73±0.41 **	12.08±0.08 **	12.77±0.41 **
[d]	-0.33±0.07 **	-0.32±0.07 **	-0.31±0.07 **	-0.38±0.08 **	-0.32±0.07 **	-0.37±0.08 **	-0.31±0.07 **	-0.38±0.08 **	-0.38±0.08 **
[h]		0.54±0.14 **	-0.13±0.30 ns	0.53±0.14 **	1.13±0.34 **	-0.14±0.30 ns	-0.56±1.07 ns	1.10±0.34 **	-0.68±1.07 ns
[i]			-0.52±0.21 *			-0.52±0.21 *	-0.66±0.40 (*)		-0.70±0.40 (*)
[j]				0.56±0.33 (*)		0.56±0.33 (*)		0.54±0.33 ns	0.57±0.33 (*)
[l]					-0.72±0.37 (*)		0.30±0.72 ns	-0.70±0.37 (*)	0.38±0.72 ns
χ^2	23.78 **	9.40 *	10.36 **	6.55 (*)	5.62 (*)	0.28 ns	2.88 (*)	3.05 *	...

Table 87. Continued

Briscard x Otane

m	13.16±0.06 **	13.57±0.13 **	12.90±0.29 **	13.55±0.13 **	13.76±0.14 **	12.90±0.25 **	12.86±0.59 **	13.78±0.16 **	12.80±0.66 **
[d]	0.96±0.11 **	1.20±0.13 **	1.35±0.13 **	1.16±0.14 **	1.30±0.13 **	1.33±0.15 **	1.35±0.14 **	1.33±0.16 **	1.33±0.16 **
[h]		-0.83±0.22 **	0.15±0.39 ns	-0.78±0.23 **	-2.08±0.52 **	0.16±0.39 ns	0.28±1.60 ns	-2.16±0.58 **	0.52±1.83 ns
[i]			0.89±0.29 **			0.88±0.29 **	0.93±0.60 ns		1.00±0.64 ns
[j]				0.37±0.54 ns		0.11±0.55 ns		-0.18±0.58 ns	0.17±0.62 ns
[l]					1.38±0.52 **		-0.08±1.07 ns	1.44±0.55 **	-0.25±1.22 ns
χ^2	23.86 **	9.64 *	42.04 **	9.18 *	2.49 ns	0.04 ns	0.08 ns	2.40 ns	...

Ruapuna x Otane

m	13.93±0.06 **	13.95±0.10 **	15.46±0.28 **	13.94±0.11 **	13.68±0.11 **	15.47±0.28 **	15.29±0.63 **	13.64±0.12 **	15.20±0.64 **
[d]	1.37±0.10 **	1.37±0.10 **	1.26±0.10 **	1.35±0.11 **	1.27±0.10 **	1.20±0.12 **	1.26±0.10 **	1.19±0.12 **	1.19±0.12 **
[h]		-0.04±0.19 ns	-2.13±0.40 **	-0.02±0.20 ns	2.36±0.49 **	-2.13±0.40 **	-1.67±1.63 ns	2.52±0.50 **	-1.39±1.65 ns
[i]			-1.78±0.30 **			-1.82±0.30 **	-1.62±0.63 **		-1.56±0.63 *
[j]				0.22±0.49 ns		0.54±0.50 ns		0.68±0.50 ns	0.57±0.50 ns
[l]					-2.72±0.51 **		-0.31±1.06 ns	-2.84±0.52 **	-0.50±1.07 ns
χ^2	36.74 **	36.69 **	12.14 **	36.49 **	8.02 *	0.22 ns	1.30 ns	6.19 *	...

Table 87. Continued

Domino x Otane

m	13.22±0.06 **	13.08±0.10 **	13.83±0.29 **	13.10±0.11 **	12.91±0.11 **	13.84±0.29 **	12.64±0.58 **	12.95±0.11 **	12.84±0.59 **
[d]	0.53±0.10 **	0.46±0.10 **	0.24±0.10 **	0.58±0.11 **	0.41±0.10 **	0.52±0.11 **	0.41±0.10 **	0.50±0.11 **	0.50±0.11 **
[h]		0.39±0.21 (*)	-0.80±0.47 (*)	0.29±0.22 ns	2.00±0.49 **	-0.86±0.47 (*)	2.67±1.54 (*)	1.80±0.50 **	2.08±1.58 ns
[i]			-0.88±0.31 **			-0.84±0.31 **	0.27±0.58 ns		0.11±0.58 ns
[j]				-1.10±0.49 *		-1.04±0.49 *		-0.84±0.49 (*)	-0.82±0.50 ns
[l]					-2.10±0.57 **		-2.52±1.06 *	-1.95±0.58 **	-2.12±1.09 (*)
χ^2	19.88 **	16.43 **	9.72 **	11.33 **	2.91 ns	3.79 (*)	2.69 ns	0.04 ns	...

Briscard x Domino

m	14.23±0.08 **	14.37±0.15 **	14.45±0.32 **	14.37±0.15 **	14.29±0.17 **	14.45±0.32 **	13.30±0.78 **	14.28±0.18 **	13.04±0.84 **
[d]	0.83±0.15 **	0.86±0.15 **	0.86±0.15 **	0.86±0.17 **	0.86±0.15 **	0.85±0.18 **	0.89±0.15 **	0.83±0.18 **	0.83±0.18 **
[h]		-0.30±0.26 ns	-0.41±0.47 ns	-0.30±0.26 ns	0.31±0.66 ns	-0.41±0.47 ns	2.84±2.06 ns	0.38±0.69 ns	3.61±2.25 ns
[i]			-0.10±0.37 ns			-0.10±0.37 ns	1.00±0.77 ns		1.24±0.82 ns
[j]				0.03±0.66 ns		0.05±0.67 ns		0.23±0.70 ns	0.61±0.73 ns
[l]					-0.65±0.64 ns		-2.19±1.35 ns	0.71±0.66 ns	-2.70±1.49 (*)
χ^2	4.71 ns	3.39 ns	5.03 (*)	3.39 ns	2.37 ns	3.31 (*)	0.68 ns	2.26 ns	...

Table 87. Continued

Ruapuna x Domino

m	13.62±0.06 **	14.11±0.13 **	14.32±0.29 **	14.13±0.13 **	14.13±0.14 **	14.30±0.29 **	15.22±0.57 **	14.14±0.14 **	15.02±0.58 **
[d]	0.47±0.12 **	0.50±0.12 **	0.50±0.12 **	0.69±0.14 **	0.50±0.12 **	0.69±0.14 **	0.53±0.12 **	0.69±0.14 **	0.69±0.14 **
[h]		-1.10±0.25 **	-1.44±0.47 **	-1.10±0.25 **	-1.28±0.56 *	-1.37±0.47 **	-4.12±1.54 **	-1.21±0.56 *	-3.50±1.57 *
[i]			-0.26±0.31 ns			-0.21±0.31 ns	-1.09±0.55 *		-0.88±0.56 ns
[j]				-1.28±0.52 *		-1.26±0.52 *		-1.27±0.52 *	-1.12±0.53 *
[l]					0.21±0.61 ns		1.97±1.08 (*)	0.13±0.61 ns	1.55±1.09 ns
χ^2	28.21 **	8.59 *	36.17 **	2.49 ns	8.47 *	2.02 ns	4.56 *	2.45 ns	...

Briscard x Ruapuna

m	14.62±0.08 **	14.96±0.15 **	15.43±0.36 **	14.95±0.15 **	15.00±0.18 **	15.36±0.36 **	17.36±0.79 **	14.97±0.18 **	17.29±0.84 **
[d]	0.17±0.15 (*)	0.25±0.15 ns	0.22±0.15 ns	0.13±0.18 ns	0.25±0.15 (*)	0.10±0.18 ns	0.16±0.15 ns	0.13±0.18 ns	0.13±0.18 ns
[h]		-0.74±0.28 **	-1.27±0.54 *	-0.68±0.29 *	-1.06±0.68 ns	-1.25±0.54 *	-6.87±2.01 **	-0.81±0.71 ns	-6.66±2.18 **
[i]			-0.47±0.41 ns			-0.51±0.41 ns	-2.38±0.78 **		-2.32±0.82 **
[j]				0.80±0.63 ns		0.86±0.63 ns		0.77±0.66 ns	0.17±0.69 ns
[l]					0.36±0.69 ns		3.81±1.32 **	0.14±0.71 ns	3.67±1.43 *
χ^2	16.72 **	9.75 *	30.59 **	8.15 *	9.48 **	6.59 *	0.06 ns	8.11 **	...

Table 88. The components of variation in six generations of wheat for latent period inoculated with stripe rust pathotype 106E139A-

Cross	D	H	F	E_w	$(H/D)^{1/2}$	$F/(D*H)^{1/2}$
Briscard x Tiritea	1.63	0.46	-0.04	0.35	0.53	0.05
Ruapuna x Tiritea	1.54	2.97	-0.83	0.29	1.39	0.39
Domino x Tiritea	0.98	0.84	-0.41	0.30	0.93	0.45
Otane x Tiritea	1.00	0.04	+0.18	0.31	0.20	0.90
Briscard x Otane	0.74	1.04	-0.77	0.79	1.19	0.88
Ruapuna x Otane	2.26	0.20	-0.33	0.57	0.30	0.49
Domino x Otane	1.21	5.77	+0.85	0.51	2.18	0.32
Briscard x Domino	1.92	4.04	-2.48	1.01	1.45	0.89
Ruapuna x Domino	0.28	1.48	-0.42	0.84	2.30	0.65
Briscard x Ruapuna	4.75	0.83	-1.46	1.01	0.42	0.74

4.6. Nonspecific field diallel

To study inheritance of stripe rust severity at adult stages in the field with common pathotype in N.Z., four adult plant resistant cultivars and one susceptible cultivar were chosen for diallel mating design and generation mean analysis. The stripe rust severity for these cultivars, based on the modified Cobb's scale for cereal rust (Fig. 6), in this trial is presented in Fig. 23. The susceptible parent, Tiritea, had a higher area under the disease progress curve, AUDPC, (for detail see Materials and Methods) than the resistant parents. For the resistant parents, stripe rust severity did not notably increase until the last data recording, but for the susceptible cultivar, the first data recording was about 30% severity and almost 100% at the last one . Among the resistant cultivars, the AUDPC for Otane was higher than for other cultivars. It was followed by Ruapuna, Domino and Briscard, in this order. A diallel mating design was used to determine the gene action of stripe rust resistance in Briscard, Domino, Otane and Ruapuna in terms of AUDPC.

4.6.1. Graphical (Hayman) analysis

The results from the analysis of variance carried out on plot means are presented in Table 89. Significant genotype differences at the 0.01% level were obtained in all cases; parents, F_1's and pooled. The means for each genotype, five parents and ten hybrids are presented in Table 90. Duncan's multiple ranges was used to compare parents and their hybrids. Tiritea and its crosses were the most susceptible genotypes, whereas Briscard and its crosses with Ruapuna and Domino were the most resistant. As the genotypic variances were significant for the area under the disease progress curve the Hayman-Jinks analysis was conducted.

The estimates of basic array statistics,W_r/V_r regression equation and their F-test and other relevant statistics are presented in Table 91. The regression slope was significantly different from zero for the area under the disease progress curve.

Analysis of variance was carried out to test the consistency of W_r-V_r over arrays; the test for the presence of non-additive genetic variation another analysis variance of W_r+V_r was carried out (Table 91). The array values for W_r+V_r were homogeneous, indicating that dominance was trivial, but array values for W_r-V_r were heterogenous,

indicating the presence of non-allelic interactions.

A two-tailed t-test was used to test the significance of the slope of the regression line from unity. It was not significant, indicating that epistasis, correlated gene distribution and the environment had less effect on graphical distortion (Mather and Jinks, 1982).

Additive genetic variance (D) was greater than the dominance genetic variance (H_1 and H_2). The F value was positive, indicating the inequality of gene frequencies with an excess of dominance over recessive alleles (Table 91). The degree of dominance, $(H_1/D)^{1/2}$ was less than unity, suggesting partial dominance.

The gene frequency were 0.15, indicating inequality of increasing (frequency of dominance or resistance) and decreasing (frequency of recessive or susceptibility) genes. The proportion of positive to negative alleles was 1.17, being almost equal to one. This suggested that dominance was consistent for all loci. The proportion of dominant to recessive alleles over all parents was 7.32.

High heritability, both broad and narrow sense, was obtained (92 and 88 percent) respectively (Table 91).

The correlations between common parent mean and W_r+V_r for each array was not different from zero. This indicated that distribution of dominant to recessive alleles was not correlated with the common parent phenotype.

The relative importance of additive to dominance genetic variances was shown by .the σ^2_A/σ^2_D ratio. It indicated that additive variance is much greater than dominance variance. The number of effective factors was estimated as 2.25, suggesting that the resistance is not controlled monogenically (Table 91).

The W_r/V_r graph and parabola, $W_r^2 = V_r * V_p$, for area under the disease progress curve is found in Fig. 24. Ranking of parental arrays with respect to the proportion of dominant alleles for the F_1 and F_2 can also be seen in Fig. 24. Those points which are close to the origin carry the greatest proportion of dominant alleles, whereas those points which are far from the origin correspond to parental arrays with the greatest proportion of recessive alleles. In the case of the F_1, Briscard and Otane had the highest proportion

of dominant alleles, whereas Tiritea, the susceptible cultivar, had the fewest dominant or most recessive alleles. Ruapuna and Domino were close to the origin but not as close as Briscard and Otane. In the case of the F_2, Ruapuna and Domino kept their positions, whereas Tiritea, Briscard and Otane moved toward the middle of the range. This is in agreement with the reducing of heterozygosity in F_2, and they tended to be placed in an additive position. The R-square for F_1 and F_2 were high (97.6 and 99.9 respectively). The slope of F_1 and F_2 regression lines were not significantly different from unity, indicating the presence of dominance. The intercepts of the F_1 and F_2 regression lines were close to each other and intersected the Y axis above the origin, indicating that partial dominance was present.

4.6.2. Griffing combining ability analysis

The analysis of variance for combining ability is presented in Table 92. Both general and specific combining abilities were highly significant. The ratio $2MS_{GCA}/(2MS_{GCA} + 2MS_{SCA})$ which shows the importance of GCA (Baker, 1978) was 0.98. The relatively higher magnitude of that ratio indicates the predominance of additive (fixable) gene effects in the genetic control of stripe rust resistance among the parents used in this diallel cross.

The comparison of GCA and SCA performance of cultivars for stripe rust resistance is presented in Table 93. On the diagonal, Tiritea had positive GCA effects, whereas the other cultivars had negative GCA effects. The positive values indicate susceptibility while the negative values indicate resistance. Briscard had the highest negative value , followed by Ruapuna, Domino and Otane. This ranking of cultivars according to GCA was similar to that based on their mean performance in the trial (Table 90), indicating again the preponderance of additive gene action. The SCA of each hybrid is in the above the diagonal. Out of the total of 10 cross combinations, only 4 values were significant, of which only one was positive. The highest negative SCA was shown by the combinations of Briscard x Tiritea, Otane x Tiritea and Ruapuna x Tiritea respectively. This indicates that dominance plays an important role for those hybrid to obtain a high level of stripe rust resistance to pathotype 106E139A-. It is noteworthy that Tiritea, which is susceptible, can be used to produce F_1 hybrid cultivars with higher of resistance than expected (but it was mostly at the more susceptible one).

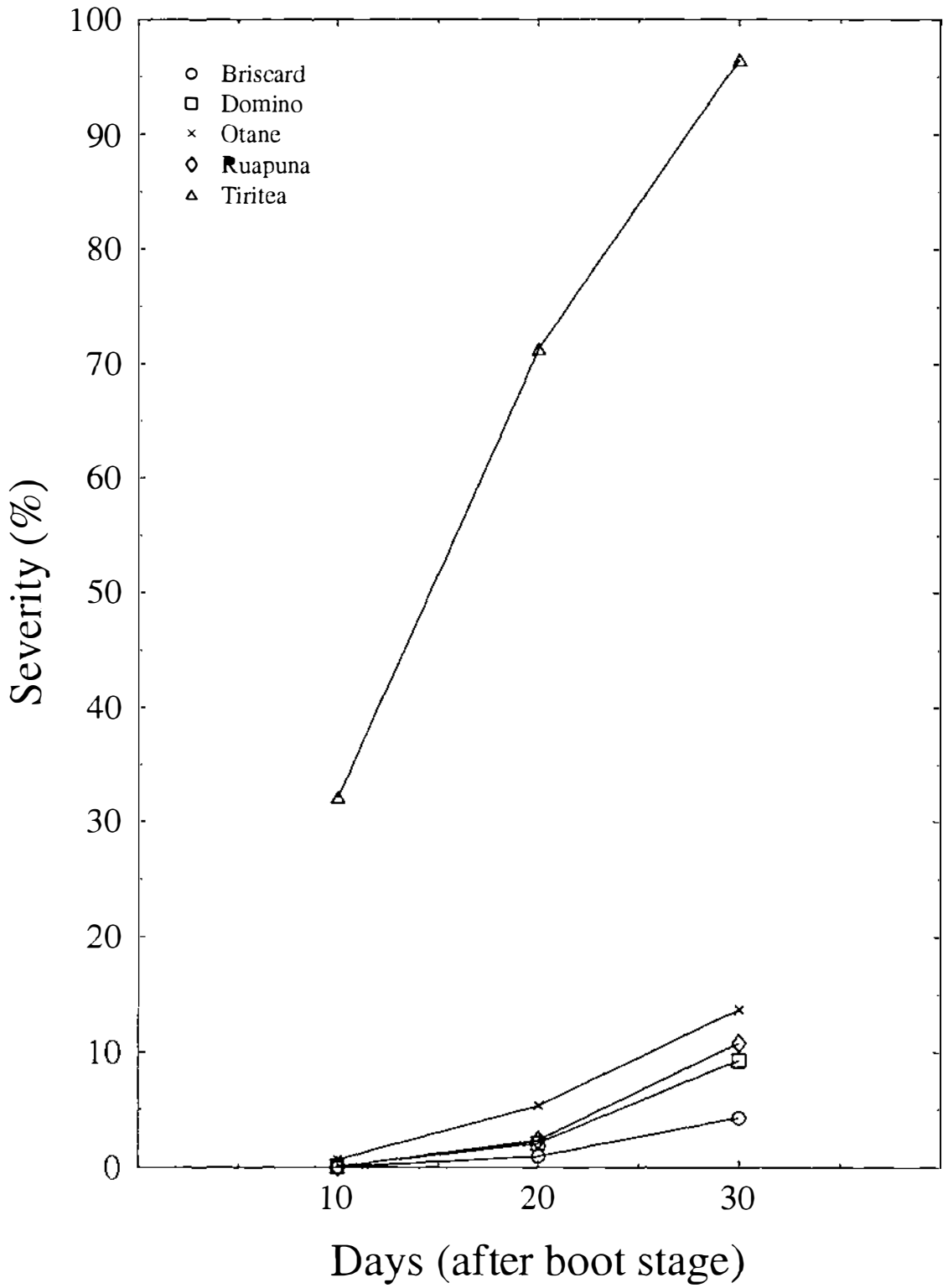


Fig. 23. Development of the disease progress curves for stripe rust on five wheat cultivars infected with pathotype 106E139A-

Table 89. Analysis of variance for the area under the disease progress curve of a 5 x 5 diallel inoculated with stripe rust pathotype 106E139A-

S.O.V.	Parents		F1,s		Pooled	
	df	EMS	df	EMS	df	EMS
Block	4	1143.4 **	4	995.0 **	4	9799.4 **
Treatment	4	19817.2 **	9	2852.8 **	14	76137.5 **
Error	16	71.9	36	118.0	56	985.4

** : significant at P = 0.01

Table 90. Means of five cultivars and their progenies for the area under the disease progress curve (AUDPC) inoculated with stripe rust pathotype 106E139A-

Genotype	AUDPC
Tiritea	243.42 a[1]
Tiritea/Domino	160.60 b
Tiirtea/Ruapuna	151.68 b
Tiritea/Otane	148.28 b
Tiritea/Briscard	127.33 c
Otane	121.32 cd
Otane/Ruapuna	117.33 cde
Otane/Domino	110.98 def
Otane/Briscard	108.87 def
Ruapuna/Domino	106.45 efg
Ruapuna	104.87 efg
Domino	103.19 efgh
Domino/Briscard	96.99 fgh
Ruapuna/Briscard	92.63 gh
Briscard	90.05 h

[1]Means with the same letter are not significantly different (P=0.05), using Duncan's multiple ranges

Table 91. Diallel genetic statistics, W_r/V_r regression equations and basic array statistics for stripe rust resistance in five cultivars of wheat inoculated with pathotype 106E139A-

statistics	Area Under the Disease Progress Curve		
$W_r + V_r$	ns	$W_r = 800 + 0.99\ V_r$	F-test 120.7 **
$W_r - V_r$	**	$V_p = 3942.17$	
t-test(β - 1)	0.11 ns	$\bar{V}_r = 710.52$	
D	3891.61	$\bar{W}_r = 1522.34$	
H_1	334.77	$V_{\bar{r}} = 592.00$	
H_2	194.16		
F	1734.30		
$\sqrt{(H_1/D)}$	0.29		
uv (H_2 / $4H_1$)	1.17		
$0.5F / \sqrt{[D(H_1 - H_2)]}$	0.15		
dom./rec. genes	7.32		
h^2_{BS}	92%		
h^2_{NS}	88%		
r (P_r , $W_r + V_r$)	0.18		
σ^2_A / σ^2_D	40.09		
K (effective factors)	2.25		
E_{pool}	985.40		
$E_{parents}$	71.86		
$E_{hybrids}$	118.03		

ns and ** : not significant and significant at P = 0.01, respectively

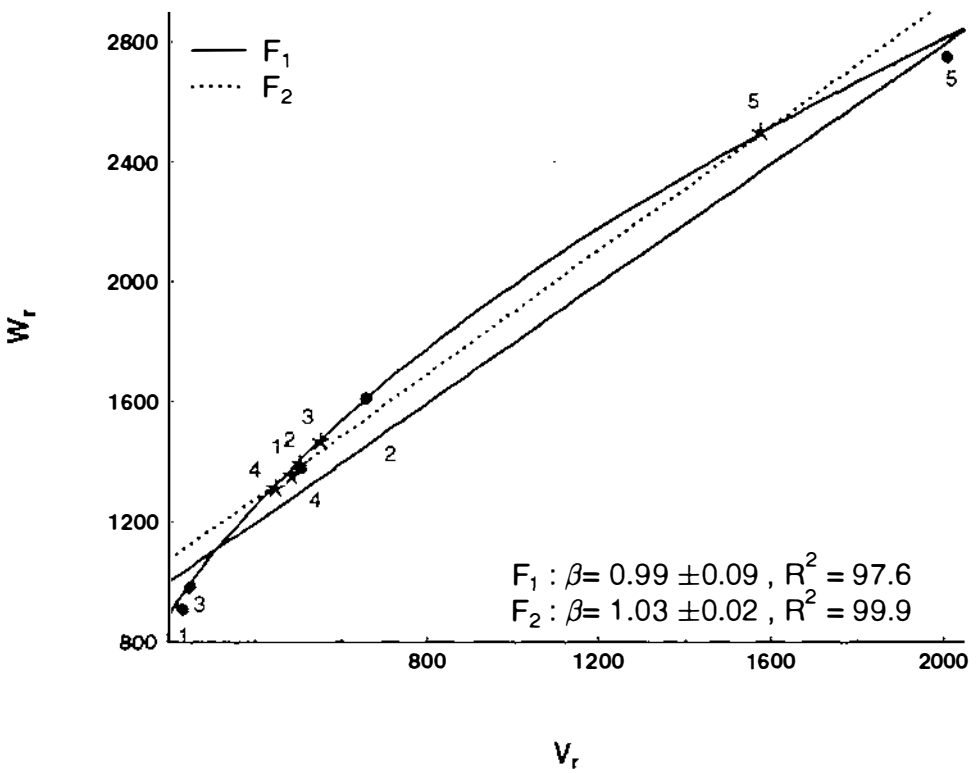


Fig. 24. W_r/V_r graph for area under the disease progress curve between parents in F_1 (•) and F_2 (⋆), below and above the line respectively, of a 5 x 5 diallel cross (1 = Briscard, 2 = Domino, 3 = Otane ,4 = Ruapuna, 5 = Tiritea), infected with stripe rust pathotype 106E139A-.

Table 92. Mean squares of general and specific combining ability for area under the disease progress curve inoculated with stripe rust pathotype 106E139A-

S.O.V.	df	MS
GCA[1]	4	4868.93 **
SCA_2	10	184.28 **
Error	675	5.06
Ratio[3]		0.98
h^2_{BS}		0.99
h^2_{NS}		0.98

[1] General Combining Ability
[2] Specific Combining Ability
[3] $2MS_{GCA}/(2MS_{GCA} + MS_{SCA})$
[4,5] Broad- and narrow sense heritability
** significant at P = 0.01

Table 93. Estimates of general combining ability (diagonal) and specific combining ability (above diagonal) for the area under the disease progress curve to stripe rust pathotype 106E139A-

	Briscard	Domino	Otane	Ruapuna	Tiritea
Briscard	-21.12**	2.68ns	8.03 *	-1.03ns	-23.01**
Domino		-10.20**	-0.79ns	1.87ns	-0.67ns
Otane			-3.67 *	6.22ns	-19.52**
Ruapuna				-10.85**	-8.94 *
Tiritea					45.84**

$S.E._{GCA}$=1.5010 $S.E._{SCA}$=3.9351

4.7. Nonspecific field generation means

The diallel mating design can not verify types of epistasis, although it gives us good genetic information. Therefore, quantitative genetic studies on stripe rust inheritance were continued with nine generations involving P_1, P_2, F_1, F_2, backcrosses (BC_1 and BC_2), F_3 lines, and backcross selfed lines (BCS_1 and BCS_2). Generation mean analysis was used to estimate the mode of gene action in detail. In generation mean analysis, the joint scaling test is more powerful than any of the other tests in detecting epistasis since it uses information from all populations. Generation mean analysis was used by three ways using different populations as followed:

1. P_1, P_2, F_1, F_2, BC_1 and BC_2
2. P_1, P_2, F_2, F_3, BCS_1 and BCS_2
3. P_1, P_2, F_1, F_2, F_3, BC_1, BC_2, BCS_1 and BCS_2

4.7.1. Briscard x Tiritea

Weighted analysis of variance indicated that highly significant differences existed among generations for the area under the disease progress curve (Table 94). Tiritea was the most susceptible genotype, with the highest AUDPC and was followed by both backcross and backcross selfed to Tiritea, and then by the F_2 and F_3 and finally by the F_1, backcross selfed and backcross to Briscard and Briscard (Fig. 25). In the last data recording, stripe rust severity increased less than 13% for Briscard, its backcross, backcross selfed and the F_1, whereas for the F_2 and F_3 lines up to 30%; for the backcross to Tiritea up to 60% and for Tiritea up to 100% was recorded. It is quite clear that rust development on the F_1, F_2, F_3, backcrosses and backcross selfed was intermediate to the development on the parents and it followed a theoretical trend. The mean AUDPC values of the backcross and backcross selfed with Tiritea were similar, but lower than Tiritea. The mean values for the F_2 and F_3 were also similar but were intermediate to both parents. The F_1, backcross to Briscard and backcross selfed to Briscard were in same group, whereas the backcross to Briscard, Briscard itself were similar, being the most resistant group (Table 94).

The mean AUDPC for Briscard, Tiritea and their F_1 was 95.4, 243.0 and 127.3, respectively. The degree of dominance according to the deviation of the F_1 the from mid-parent (Falconer, 1981) was estimated as 0.75, suggesting the occurrence of partial

dominance for resistance.

Broad sense and narrow sense heritability are presented in Table 95. There were 0.49±0.14 and 0.18±0.08, respectively. The genetic advance was 12.98, assuming that the top 1% of segregating plants were selected for resistance. Estimates of the minimum number of genes were calculated by different formulae for both AUDPC (Table 96). The range of the number for this cross was from 1.6±0.51 to 9.0±2.8.

The frequency distributions of transformed AUDPC of all generations is presented in Fig. 35. The distributions of the F_2 and F_3 were normal and the distribution of backcrosses and backcrosses selfed was skewed toward the their recurrent parents. The amount of skewness for backcrosses selfed was less than the for backcrosses. Transgressive segregation was observed in F_3 lines in both directions and also in the backcross with Briscard toward resistance and in the backcross with Tiritea toward susceptibility. The results suggest that the susceptible cultivar, Tiritea, can contribute resistance in terms of lower AUDPC.

The estimates of gene effects together with the scaling test and chi-square of two batch of six populations and nine populations for this cross were presented in Table 97. In the first case using P_1, P_2, F_1, F_2, BC_1 and BC_2, the chi-square of the three parameter model was not significant indicating the additive-dominance model, involving m, [d] and [h], for stripe rust resistance was the perfect fit and there was no epistasis. This was supported by using the six-parameter model in addition. In the second case using P_1, P_2, F_2, F_3 lines, BCS_1 and BCs_2 lines, the six-parameter model revealed a perfect fit. This analysis showed the presence of non-allelic interactions. In the third case using all nine generations; P_1, P_2, F_1, F_2, BC_1, BC_2, F_3 lines, BCS_1 and BCs_2 lines, the best model involving m, [d], [h] and [i] was fitted. The chi-square of this model was significant, suggesting that this model was not adequate and trigenic interaction or linkage or both were present. In all three cases, the [h] and [l] components possessed negative and positive signs, respectively, suggesting the predominance of a duplicate type of epistasis. It is obvious the [l] in the third case is very close to zero and not significant which was supposed to have and opposite sign of [h]. The negative sign of [h] indicated that partial dominance for low AUDPC or resistance was presented. All three cases gave a consistent value of additive gene effects [d], indicating the possibility of deriving highly resistant lines in later generations.

The components of variation, based on the basic six generations V_{P1}, V_{P2}, V_{F1}, V_{F2}, V_{BC1}, and V_{BC2} are presented in Table 107. The additive (D) and dominance (H) components were 274.46 and 926.4, respectively. The estimate of average dominance of the genes was 1.84, indicating overdominance for resistance. The F value was -481.23 which agreed with the deviation of the F_1 mean from the mid-parent toward the smaller parent, Briscard. This indicated that the genes from Briscard were mostly dominant. The ratio $F/(D*H)^{1/2}$ was 0.96, indicating the measure of dominance, h/d, is constant in both magnitude and sign for all gene-pairs involved. There is good reason to believe that almost all the genes from Briscard were dominant (Mather and Jinks, 1982).

4.7.2. Ruapuna x Tiritea

The analysis of variance together with the mean comparisons are presented in Table 94. The F-test was highly significant. There were five groups amongst means; Tiritea was the most susceptible genotype, then backcross and backcross selfed to Tiritea were in another group, followed by the F_1, F_2 and F_3 in a group, then both backcross and backcross selfed to Ruapuna in another group, and finally Ruapuna was the most resistant genotype (Table 94).

In the last data recording (Fig. 26), the stripe rust severity increased up to 20% for Ruapuna and their backcrosses, up to 40% for F_1, F_2 and F_3 and up to 70% for backcrosses to Tiritea, and about 100% for Tiritea. Rust development on all nine generations followed a theoretical trend. The mean values were 110.6, 234.2 and 151.7 for Ruapuna, Tiritea and the F_1, respectively. The estimate of degree of dominance was 0.58, indicating partial resistance for resistant parent, Ruapuna.

Heritability and genetic advance were estimated in different ways (Table 95). Broad sense and narrow sense heritabilities were 0.47±0.12 and 0.11±0.04, respectively. Genetic advance was 7.91, assuming 1% of the resistant plants in segregating generation were selected. The range of minimum number of genes was estimated from 4.2±0.5 to 12.2±2.3 (Table 96).

The frequency distributions of AUDPC of nine generation are presented in Fig. 36. The distribution of F_2 and F_3 lines was normal but it was close to the resistant parent,

Ruapuna, rather than Tiritea. Transgressive segregation was observed in F_2 and F_3 toward the resistant parent. Transgressive segregation was also observed in backcrosses and backcrosses selfed toward their recurrent parents. This indicated that Tiritea has a gene or genes for resistance and Ruapuna has a gene or genes for susceptibility.

The mode of gene action was determined by generations mean analysis (Table 98). In basic generations, using P_1, P_2, F_1, F_2, BC_1 and BC_2, chi-square of the three-parameter model was significant, indicating that the model was not adequate and there was the presence of non-allelic interactions. Then all possible models were fitted to the observed means, and all models had at least one nonsignificant component, except the m, [d], [h] and [l] model. It should be mentioned that in the six-parameter model only m and [d] were significant and standard error of all components in four-parameter model were less than six-parameter model, so it did not support the method of Mather and Jinks (1982). In the second case using P_1, P_2, F_2, F_3 lines, BCS_1 and BCs_2 lines, the chi-square of the three-parameter model was significant, indicating the presence of epistasis. In the six-parameter model, the [l] component was not significant and all possible models were fitted to the observed means. The best model was m, [d], [h], [j] and [l] which had increasing in the means and decrease in the standard errors in comparison with the six-parameter model. This one also did not follow the method of Mather and Jinks (1982). In the third case using all generations, P_1, P_2, F_1, F_2, BC_1, BC_2, F_3 lines, BCS_1 and BCs_2 lines, the model m, [d] and [i] was fitted and the chi-square of that was significant at 10% level, meaning that at the 5% probability level this model is adequate. In the first and second cases the [h] was negative whereas [l] was positive, indicating the duplicate type of epistasis. The negative [h] value indicated that partial dominance of low AUDPC was present which was confirmed by deviation of the F_1 from the mid-parent. In all cases the additive value was consistent, indicating the possibility of deriving highly resistant lines in generations.

Based on P_1, P_2, F_1, F_2, BC_1 and BC_2, the components of variation were estimated (Table 107). The D and H were 168.34 and 1067.76, respectively. The ratio $(H/D)^{1/2}$ was 2.52, indicating overdominace for resistance. This did not agree with analysis of the mean which show partial resistance. The F value was -538.93, indicating that the genes from the resistant parent, Ruapuna were dominant. This agrees with the deviation of the F_1 from the mid-parent. The ratio $F/(D*H)^{1/2}$ was close to unity, indicating

the h/d is constant in magnitude and sign for all gene-pairs involved.

4.7.3. Domino x Tiritea

The F-test showed statistical differences amongst the generations (Table 94). Tiritea was most susceptible genotype, and its backcross and backcross selfed were in a separate group (Table 94). The F_1, F_2 and F_3 were similar while backcross and backcross selfed to Domino were in a same group and finally Domino was in a separate group as the most resistant genotype. The degree of dominance was 0.25 towards resistant parent, Domino.

Rust development for nine generations is presented in Fig. 27. In the last data recording, stripe rust severity increased from less than 10% for Domino, less than 25% for backcross and backcross selfed to Domino, less than 40% for the F_1, F_2 and F_3, less than 60% for the backcross selfed to Tiritea, less than 70% for the backcross to Tiritea and about 100% for Tiritea. These generations also followed theoretical trend.

Broad sense and narrow sense heritability together with genetic advance are presented in Table 95. The heritability was 0.39±0.10 and 0.15±0.06 for broad sense and narrow sense respectively. Genetic advance was 11.09, assuming the top 1% of segregating plants were selected for resistance. The number of genes was estimated in different ways (Table 96). The range of minimum number of genes for this cross was 2.9±0.4 - 8.0±1.1.

The frequency distributions of nine generations for this cross are presented in Fig. 37. The frequency distributions of the AUDPC of F_2 plants and F_3 lines were normal. Transgressive segregation was found in the F_2 and F_3 toward the resistant parent, Domino, and transgressive segregation was observed in all backcrosses and backcrosses selfed toward the their recurrent parents. This is good evidence the even Tiritea which is susceptible can contribute to increased resistance.

The results of generations mean analysis are presented in Table 99. In the first case using P_1, P_2, F_1, F_2, BC_1 and BC_2, the additive-dominance model was not adequate since the chi-square was significant. Then other possible models were fitted and the best model for this cross was m, [d], [h] and [l]. It should be mentioned that in the six-

parameter model only m and [d] were significant whereas in the four-parameter model involving m [d], [h] and [l], all components were significant at the 1% level and they had an increase in their means and a decrease in their standard errors in comparison with the six-parameter model. Again this did not follow the method of Mather and Jinks (1982). In the second case using P_1, P_2, F_2, F_3 lines, BCS_1 and BCs_2 lines, the three-parameter model was not adequate since chi-square was significant. Then by using the six-parameter model in which only [i] was not significant and with the removal of [i], the five parameter model involving m [d], [h], [j] and [l] was the best model. The chi-square was not significant indicating there was not any tri-genic interaction. This followed the method of Mather and Jinks (1982). Other model were not adequate since all chi-squares were significant. In the third case using all nine generations, involving P_1, P_2, F_1, F_2, BC_1, BC_2, F_3 lines, BCS_1 and BCs_2 lines, the model involving m , [d], [i] and [l] was fitted but it was not adequate since the chi-square was significant, indicating presence of tri-genic interactions. In all cases, the [h] and [l] had opposite signs, suggesting the predominance of a duplicate type of epistasis. The [h] value was negative, indicating that partial dominance of resistance was present. This agrees with the deviation of the F_1 from the mid-parent and [d] in all cases was consistent.

In the estimates of the components of variation (Table 107), D and H were 241.8 and 751.04, respectively. The estimate of average dominance of the genes were 1.76, indicating over-dominance. This did not agree with partial dominance which already was obtained from the result of the means. The F value was negative, supporting that partial dominance existed for F_1 toward the resistant parent, Domino. The ratio $F/(D*H)^{1/2}$ was 0.58. This indicated that the dominance deviation at different loci are not constant in either magnitude or sign. There is good reason to believe that even though the genes from Domino showed a preponderance of dominance, some of them must be recessive to their alleles from Tiritea (Mather and Jinks, 1982).

4.7.4. Otane x Tiritea

Analysis of variance indicated that there were highly significant differences between genotypes (Table 94). Both parents were in different groups while the rest overlapped (Table 94). Rust development for all genotype is presented in Fig. 28. Otane and Tiritea were most resistant and susceptible genotypes respectively. Between the two parents the susceptibility in ascending order was F_1, F_3, F_2, backcross selfed and

backcross to Tiritea. In the last data recording, stripe rust severity increased up to 12% for Otane, up to 15% for the backcross selfed to Otane, up to 20% for the backcross to Otane, up to 22% for the F_1, up to 35% for the F_3, up to 39% for the F_2, up to 54% for the backcross selfed to Tiritea, up to 57% for the backcross to Tiritea and about 100% for Tiritea. The position of rust development of each genotype between the two parents followed theoretical trend.

The means AUDPC of Otane, Tiritea and the F_1 were 121.05, 248.18 and 148.28 respectively. The degree of dominance was estimated as 0.76, indicating partial dominance for the resistant parent.

Heritability was 0.62±0.9 and 0.46±0.6 for broad sense and narrow sense respectively (Table 95). The genetic advance according to narrow sense heritability, was estimated as 42.20, assuming the top 1% of segregating plants were selected for resistance. The range of minimum number of genes or effective factor (Table 96) was from 1.2±0.07 to 4.6±0.6.

The frequency distributions are presented in Fig. 38. The F_2 distribution was normal and showed transgressive segregation for both resistance and susceptibility. The F_3 distribution was similar to the F_2, but there was not any transgressive segregation for susceptibility. The backcross distributions to both parents demonstrate transgressive segregation towards the recurrent parent. There was slight transgressive segregation for the backcross selfing distributions.

The gene effects are presented in Table 100. In the first case using P_1, P_2, F_1, F_2, BC_1 and BC_2, the additive and dominance were highly significant and chi-square was not significant. This indicated that additive-dominance model was adequate and there were no digenic interactions. By using all possible models, only interactions in the six-parameter model were significant at the 10% probability level. In the second case using P_1, P_2, F_2, F_3 lines, BCS_1 and BCS_2 lines, the chi-square of the three-parameter model was significant, indicating that the additive-dominance model was not adequate. Then the six-parameter model was used and the additive x additive component was not significant, indicating model involving m, [d], [h], [j] and [l] was adequate. This matter was confirmed by fitting all possible models. In the third case using all nine generations involving P_1, P_2, F_1, F_2, BC_1, BC_2, F_3 lines, BCS_1 and BCs_2 lines, the model involving m,

[d], [i] and [l] was fitted but it was not adequate since the chi-square was highly significant, indicating presence of tri-genic interactions. In all cases the additive part was nearly constant and in the first and second cases the [h] was negative and of opposite sign to [l], indicating partial dominance of the resistant parent and the presence of duplicate type of epistasis.

By using the basic generations involving P_1, P_2, F_1, F_2, BC_1 and BC_2, the components of variation are presented in Table 107. The D and H values were 178.76 and 2615.64 respectively. The estimate of $(D/H)^{1/2}$ was 3.83, suggesting over dominance, which did not agree with the deviation of the F_1 mean from the mid-parent. The covariance of additive-dominance (F) was -704.67, indicating partial dominance for the resistant cultivar, Otane. The ratio $F/(D*H)^{1/2}$ was close to unity, indicating dominance in all loci was constant in sign and magnitude.

4.7.5. Briscard x Otane

The F-test showed that the differences among the mean of nine generations were significant at the 5% probability level (Table 94). The two parents were significantly different from each other and other genotypes overlapped (Table 94). The degree of dominance was 0.59 for the susceptible parent, Otane. This was estimated from the means of Briscard, Otane and the F_1 which were 87.68, 119.09 and 108.87 respectively.

The rust development (Fig. 29) increased up to 5% and 15% for Briscard and Otane, respectively. Other generations were intermediate to the development on the parents.

Broad sense and narrow sense heritabilities were estimated as 0.51±0.14 and 0.50±0.14 respectively and the genetic advance was 33.67, assuming that the top 1% of segregating plants were selected for resistance (Table 95). The minimum number of genes for the area under the disease progress curve was less than unity (Table 96).

The frequency distributions of all generations are presented in Fig. 39. Transgressive segregation was observed for low and high AUDPC in the F_2 and F_3 distributions. For the backcross to Briscard, transgressive segregation in both directions was observed whereas for the backcross to Otane it was observed towards

susceptibility. This situation was similar for the backcrosses selfed.

The estimates of gene effects and chi-square values are presented in Table 101. In the first case using P_1, P_2, F_1, F_2, BC_1 and BC_2, the additive-dominance model was not adequate since chi-square was highly significant. By using the six-parameter model, only m, [d] and [j] were significant (the first two at 1% and third one at the 10% probability level). This model m, [d] and [j] cannot be adequate since the [h] was already significant in the three parameter model whereas it was not in the six-parameter model. Then all possible models were fitted, two models showed that there were adequate. The chi-square of first model involving m, [d], [h], [j] and [l] was not significant, indicating adequacy of this model and the chi-square of the second one involving m, [d], [h] and [l] was also not significant. In the first model the [j] was significant at the 10% probability level which was the only difference between two models. In the second case using P_1, P_2, F_2, F_3 lines, BCS_1 and BCs_2 lines, the chi-square of the three-parameter model was significant, indicating the presence of non-allelic interactions. By fitting the six-parameter model, it was determined that [h] was not significant whereas in the three-parameter model it was. After fitting all possible models, it was found that the model involving m, [d], [h] and [i] was adequate and there was no interaction beyond additive x additive component. In the third case using all nine generation, involving P_1, P_2, F_1, F_2, BC_1, BC_2, F_3 lines, BCS_1 and BCs_2 lines, the model involving m , [d], and [i] was fitted and it was adequate since the chi-square was not significant, indicating nothing beyond digenic interactions.

The components of variation D and H were 654.52 and 18.44, respectively (Table 107). The average dominance was 0.17, indicating partial dominance that supported the degree of dominance which already estimated by means of the parents and the F_1. The covariance of additive-dominance (F) was +134.94. The positive sign of F suggested that there was partial dominance for susceptibility, which also supported the previous results. The ratio $F/(D*H)^{1/2}$ was close to unity, indicating the measure of dominance (h/d) was constant in sign and magnitude for all loci for Otane.

4.7.6. Ruapuna x Otane

The results from the analysis of variance are presented in Table 94. The F-test was significant at 5% probability level. Overlapping was observed in the comparison of

the means, the range of which was 102.3 - 124.7 for backcross selfed to Ruapuna and Otane respectively.

Rust development according to stripe rust severity did not increase more than 15% in all the nine generations (Fig. 30). That did not followed a theoretical trend.

The mean AUDPC for Ruapuna, Otane and the F_1 was 106.27, 124.65 and 115.46, respectively. Although the F_1 overlapped with both parents, the degree of dominance was 0.45, indicating partial dominance for the susceptible cultivar, Otane.

Heritability was estimated in different ways (Table 95). Broad sense and narrow sense heritabilities were 0.46±0.09 and 0.76±0.14 respectively. Narrow sense heritability was greater than broad sense heritability since the sampling variation the dominance variance was negative and reduced the numerator of broad sense heritability making smaller than narrow sense heritability.
Genetic advance was 53.95, assuming the top 1% of segregating plants were selected for resistance. The minimum number of genes was less than unity for the AUDPC (Table 96).

The frequency distributions for all generations are presented in Fig. 40. Transgressive segregation for both lower and higher AUDPC was observed in F_2, F_3 lines, backcrosses and backcrosses selfed to both parents.

The results of generation mean analysis are presented in Table 102. In the first case using P_1, P_2, F_1, F_2, BC_1 and BC_2, the chi-square of the three-parameter model was significant, indicating the presence of non-allelic interactions. By fitting the six-parameter model, it was determined that the model involving m, [d], [h], and [l] was the best; also it was confirmed by fitting all possible model. In the second case using P_1, P_2, F_2, F_3 lines, BCS_1 and BCs_2 lines, the three-parameter model was not adequate since it had a significant chi-square. The six-parameter model showed that the model involving m, [d], [h], [i] and [l] was the best, and it was confirmed by using all possible models. This followed the method of Mather and Jinks (1982). In the third case using all nine generations, involving P_1, P_2, F_1, F_2, BC_1, BC_2, F_3 lines, BCS_1 and BCs_2 lines, the [j] component was not significant and the model m, [d], [h], [i] and [l] was fitted, but since the chi-square was significant the model was not adequate and there was trigenic

interactions beyond that model.

The components of variation according to basic generations, P_1, P_2, F_1, F_2, BC_1 and BC_2 are presented in Table 107. The covariance of additive-dominance (F) value was +35.54, indicating presence of partial dominance for Otane, which agreed with deviation of the F_1 mean from the mid-parent. The additive component (D) was 1102.28 but the dominance component (H) was negative. This sometimes happens because of sampling deviation. Since H value was negative, $(H/D)^{1/2}$ and $F/(D*H)^{1/2}$ were not able to be estimated.

4.7.7. Domino x Otane

The F-test was significant at the 5% probability level, indicating there was at least one difference between them (Table 94). The range of the nine means was 100.9 for Domino to 129.3 for backcross selfed to Domino or Otane. Although the F_1 was not different from Domino, the degree of dominance according to the deviation of the F_1 from the mid-parent was 0.18 for the susceptible parent, Otane, indicating the presence of partial dominance.

The stripe rust severity for the nine generations is presented in Fig. 31. In the last data recording, severity increased up to a value of 20% for most susceptible one, but other genotypes were less than that value.

Broad sense and narrow sense heritabilities were 0.37±0.09 and 0.12±0.06 respectively, and genetic advance was 7.40, assuming the top 1% of segregating plants were selected for resistance (Table 95). The minimum number of genes for the AUDPC was from 0.0 to 3.1±3.9 (Table 96).

The frequency distributions are presented in Fig. 41. Transgressive segregation was observed in F_2 plants, backcrosses to Domino and Otane, and F_3 lines toward both lower and higher AUDPC, while for backcrosses selfed only towards higher AUDPC.

In generation mean analysis, in the first case using P_1, P_2, F_1, F_2, BC_1 and BC_2, the three-parameter model was significant (Table 103), indicating inadequacy of that model and the presence of non-allelic interactions. By fitting all possible models, two

found to be the best. These were the first model involving m, [d], [h], [i] and [j] and the second model involving m, [d], [h], [j] and [l], which had a not-significant chi-square. In the second case using P_1, P_2, F_2, F_3 lines, BCS_1 and BCs_2 lines, the chi-square of the three-parameter model was highly significant, indicating the presence of non-allelic interactions. Then six-parameter model showed that the m, [d], [h] and [l] was adequate, which was confirmed by fitting all possible models. This followed the method of Mather and Jinks (1982). In the third case involving P_1, P_2, F_1, F_2, BC_1, BC_2, F_3 lines, BCS_1 and BCs_2 lines, the chi-square was significant, indicating the presence of trigenic interactions. The m, [d] and [i] model was highly significant whereas [j] and [l] were significant at 10% probability level.

The components of variation D and H, according to the basic generations, P_1, P_2, F_1, F_2, BC_1 and BC_2, were 127.04 and 552.88, respectively (Table 107). The average dominance, $(D/H)^{1/2}$, was 2.09 indicating overdominace which did not agree with result of the deviation of the F_1 mean from the mid-parent. The F value was positive, indicating partial dominance for the susceptible parent, Otane. The ratio $F/(D*H)^{1/2}$ was 0.54, indicating that the measure of dominance was not constant in both magnitude and sign for all gene pairs involved. This means that some genes from Otane must be recessive to their alleles from Domino.

4.7.8. Briscard x Domino

The analysis of variance together with the comparisons of means are presented in Table 94. The F-test was significant at the 0.05% probability level. The range of means was from 90.6 to 105.8 for Briscard and the F_3 respectively. The mean AUDPC for Briscard, Domino and the F_1 were 90.58, 101.74 and 96.99 respectively (Table 94). Although the F_1 overlapped with both parents, the degree of dominance based on the deviation of the F_1 from the mid-parent was 0.39, indicating partial dominance for the susceptible parent, Domino.

The stripe rust progress in the field is presented in Fig. 32. The increase in severity for the last reading was less than 5% and 12% for Briscard and the F_3; other generations were between them.

Broad sense and narrow sense heritabilities were 0.29±0.15 and 0.11±0.11

respectively (Table 95). The genetic advance was 6.35, assuming the top 1% of segregating plants were selected for resistance. The minimum number of genes or effective factors was less than unity for AUDPC (Table 96).

The frequency distributions of AUDPC of all generations are presented in Table 41. Transgressive segregation for the F_2 plants, F_3, backcross and backcross selfed with Briscard was observed in both directions and for the backcross, and backcross selfed with Domino in the susceptible direction.

The estimates of gene effects using generation means analysis, in the first case using P_1, P_2, F_1, F_2, BC_1 and BC_2, the three-parameter model was significant at the 5% probability level, indicating the presence of non-allelic interactions (Table 104). By fitting a six-parameter model, only m, [d] and [i] were significant. By fitting all possible models it was determined that the model involving m, [d], [h] and [i] was the best and the chi-square was not significant, indicating the adequacy of the model. In the second case using P_1, P_2, F_2, F_3 lines, BCS_1 and BCs_2 lines, the best model was the six-parameter model. In the third case using all nine generations involving P_1, P_2, F_1, F_2, BC_1, BC_2, F_3 lines, BCS_1 and BCs_2 lines, the model m, [d], [h] and [i] was the best model and chi-square was highly significant, indicating there was trigenic interactions for this cross. In all cases, the sign of [h] was opposite of [l], indicating presence of duplicate type of epistasis.

By using basic generations, P_1, P_2, F_1, F_2, BC_1 and BC_2, the components of variation D and H were 105.90 and 344.40 respectively (Table 107). The average dominance, $(H/D)^{1/2}$, was 1.80, indicating overdominance for the susceptible parent, which did not agreed with the result of the deviation of the F_1 mean from the mid-parent. The F value was +37.53, indicating partial dominance for Domino, which agreed with the result of the analysis mean. The ratio $F/(D*H)^{1/2}$ was 0.20,indicating h/d was not constant in both magnitude and sign. That means some genes in Briscard were dominant as well.

4.7.9. Ruapuna x Domino

The analysis of variance was significant at the 10% probability level (Table 94). The range of means was from 103.7 for Domino to 113.2 for backcross to Domino respectively. The mean AUDPC for Domino, Ruapuna and F_1 were 103.66, 105.93 and

106.50 respectively. Although there was no statistical differences between the two parents and the F_1 (Table 3), the degree of dominance was estimated at 1.21.

In the last data recording, stripe rust severity increased less than 15% and 10% for backcross selfed to Ruapuna and Domino, respectively (Fig. 33); the other generation values were between them.

The heritability was estimated by different formulas (Table 95). Broad sense and narrow sense heritabilities were 0.26±0.08 and 0.10±0.06 respectively. The genetic advance was estimated at 6.25, assuming the top 1% of segregating plants were selected for resistance (Table 95). The minimum number of gene was less than unity for AUDPC (Table 96).

The frequency distributions are presented in Fig. 43. Transgressive segregation was observed for all segregating generations.

The estimates of gene effects using generation mean analysis, in the first case using P_1, P_2, F_1, F_2, BC_1 and BC_2, the three-parameter model was highly significant (Table 105). Then all possible model were fitted and in the six-parameter model only m was significant and other components were not. The model involving m, [d], [h], [l] was the best fit but the additive component was not significant as for all models. The chi-square was not significant, indicating the adequacy of the model. In the second case using P_1, P_2, F_2, F_3 lines, BCS_1 and BCs_2 lines, the three-parameter model was significant, indicating the presence of digeneic interactions. By fitting all possible models, in the six-parameter model only m and [i] were significant. The four parameter model involving m, [d], [h] and [l] was the best although the chi-square was significant. In the third case using all nine generations, involving P_1, P_2, F_1, F_2, BC_1, BC_2, F_3 lines, BCS_1 and BCs_2 lines, only the m and [i] were significant and the chi-square was not significant, indicating the adequacy of the model.

The components of variation, according to the basic six-generation V_{P1}, $V_{P2,}$, V_{F1}, V_{F2}, $V_{BC1,}$ and V_{BC2} are presented in Table 107. The additive (D) and dominance (H) components were 103.1 and 370.72, respectively. The average dominance was 1.90 and the F value was -74.37. This sign of the F was not in agreement with the deviation of the F_1 from the mid-point. The ratio $F/(D*H)^{1/2}$ was 0.38, indicating h/d was not constant in

sign and magnitude.

4.7.10. Briscard x Ruapuna

The F-test was significant at the 10% probability level (Table 94). The range of means was from 86.6 for Briscard to 101.7 for backcross selfed to Ruapuna, respectively. The means of Briscard, Ruapuna and F_1 were 86.56, 96.66 and 92.63 respectively. Although there was no difference between Ruapuna and the F_1, the degree of dominance was 0.45.

In the last data recording, stripe rust severity increased up to 5% and 10% for Briscard and backcross selfed to Briscard respectively (Fig. 34).

The heritability is presented in Table 95. Broad sense and narrow sense heritabilities were 0.61±0.16 and 0.84±0.22, respectively. Because the dominance component of variation was negative the narrow sense was greater than broad sense heritability. The genetic advance was 55.42, assuming the top 1% of segregating plants were selected for resistance (Table 95). The minimum number of gene or effective factor was less than unity for this cross (Table 96).

The frequency distributions for all generations are presented in Fig. 44. Transgressive segregation was observed for the F_2, F_3, and backcrosses in both directions, whereas for both backcrosses selfed it was towards the susceptibility.

The estimates of gene effects and chi-square value are presented in Table 106. In the first case using P_1, P_2, F_1, F_2, BC_1 and BC_2, the additive-dominance model was not adequate since chi-square was highly significant. By using the six-parameter model, only m, [d], [h] and [i] were significant and by using all possible models it was confirmed that the model involving m, [d], [h] and [i] was the best. In the second case using P_1, P_2, F_2, F_3 lines, BCS_1 and BCs_2 lines, the three-parameter model was not adequate. The six-parameter model showed that the model involving m, [d], [h], [i] and [l] was significant and this was confirmed by using all possible models. In the third case using all nine generations, involving P_1, P_2, F_1, F_2, BC_1, BC_2, F_3 lines, BCS_1 and BCs_2 lines, only m, [d] and [i] were significant and chi-square value was significant as well, indicating the presence of trigenic interactions.

By using the basic generations, P_1, P_2, F_1, F_2, BC_1 and BC_2, the components of variation D and H were 1049.32 and -586.84 respectively (Table 107). The average dominance, $(H/D)^{1/2}$, and $F/(D*H)^{1/2}$ were not estimated because H was negative. The F value was +67.68, indicating the presence of partial dominance for Ruapuna.

Table 94. Differences amongst means of nine generations for area under the disease curve in ten crosses of wheat

Generation[1]	Cross[3]									
	1	2	3	4	5	6	7	8	9	10
P_2	243.0 a[2]	234.2 a	248.0 a	248.2 a	119.1 ab	124.7 a	120.5 ab	101.7 ab	103.7 b	96.7 ab
BC_2	179.7 b	187.5 b	194.7 b	189.5 b	121.6 a	110.4 bc	117.2 ab	104.1 a	113.2 a	96.8 ab
BCS_2	175.0 b	182.1 b	182.0 c	183.6 bc	119.4 ab	113.2 abc	129.3 a	99.1 abc	108.2 ab	101.7 a
F_1	127.3 d	151.7 c	160.6 d	148.3 de	108.9 bcd	117.3 ab	111.0 c	97.0 abc	106.5 ab	92.6 a
F_2	147.0 c	153.0 c	157.4 d	168.0 cd	112.9 abc	112.0 abc	118.8 ab	103.1 ab	112.6 a	99.8 ab
F_3	150.3 c	149.9 c	156.4 d	159.1 d	117.5 abc	113.4 abc	120.9 ab	105.8 a	112.9 a	97.2 ab
BCS_1	114.7 d	126.4 d	126.2 e	134.0 ef	106.3 cd	102.3 c	129.3 a	100.1 ab	112.9 a	99.7 ab
BC_1	110.7 de	124.0 d	125.4 e	135.5 ef	99.7 d	106.9 bc	117.2 ab	94.4 bc	109.9 ab	89.8 ab
P_1	95.4 e	110.6 e	108.5 f	121.1 f	87.7 e	106.3 bc	100.9 c	90.6 c	105.9 ab	86.6 b
[1]F-test	**	**	**	**	*	*	*	*	(*)	*

[2](*), * and ** : significant at P = 10% ,5% and 1%, respectively
Means with the same letter are not significantly different

[3]1:Briscard(P_1) x Tiritea(P_2)
2:Ruapuna(P_1) x Tiritea(P_2)
3:Domino(P_1) x Tiritea(P_2)
4:Otane(P_1) x Tiritea(P_2)
5:Briscard(P_1) x Otane(P_2)
6:Ruapuna(P_1) x Otane(P_2)
7:Domino(P_1) x Otane(P_2)
8:Briscard(P_1) x Domino(P_2)
9:Ruapuna(P_1) x Domino(P_2)
10:Briscard(P_1) x Ruapuna(P_2)

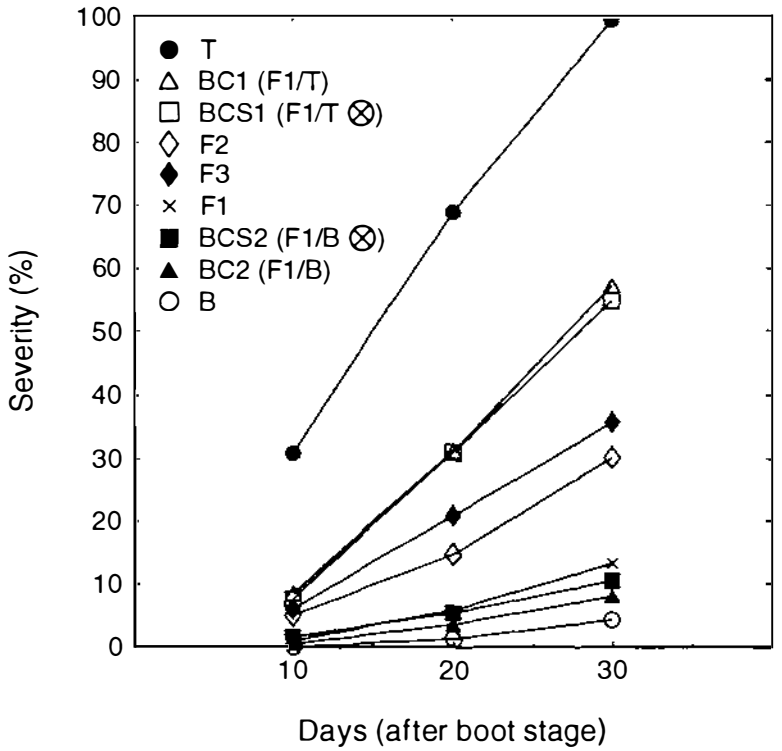


Fig. 25. Development of the disease progress curves for stripe rust on nine generations of Briscard x Tiritea infected with pathotype 106E139A-

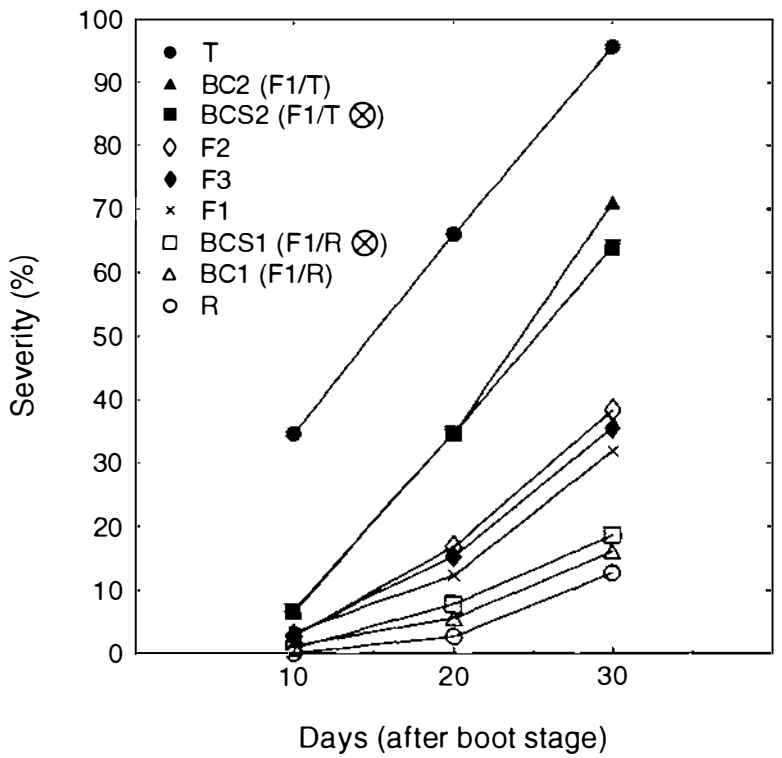


Fig. 26. Development of the disease progress curves for stripe rust on nine generations of Ruapuna x Tiritea infected with pathotype 106E139A-

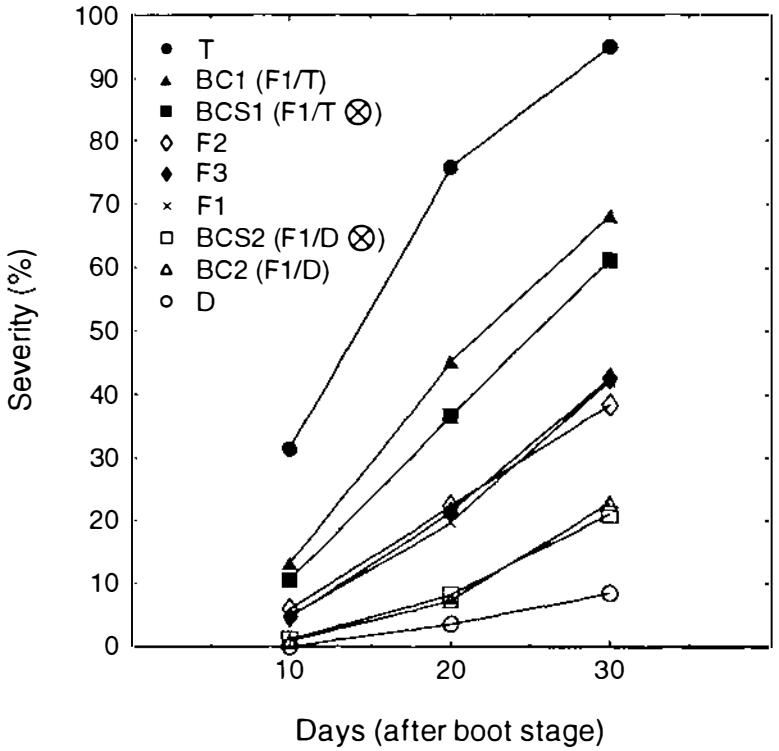


Fig. 27. Develpoment of the disease progress curves for stripe rust on nine generations of Domino x Tiritea infected with pathotype 106E139A-

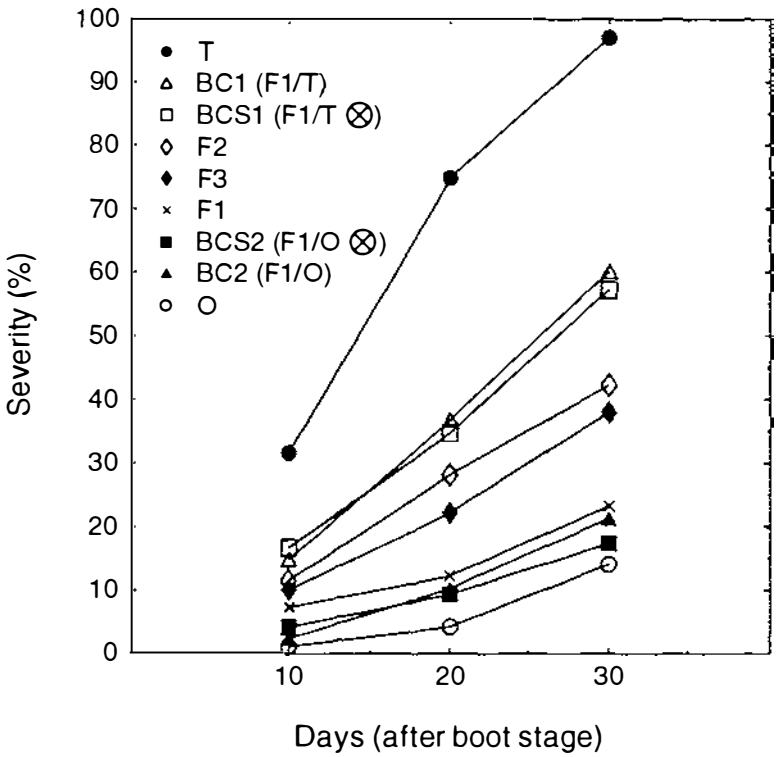


Fig. 28. Development of the disease progress curves for stripe rust on nine generations of Otane x Tiritea infected with pathotype 106E139A-

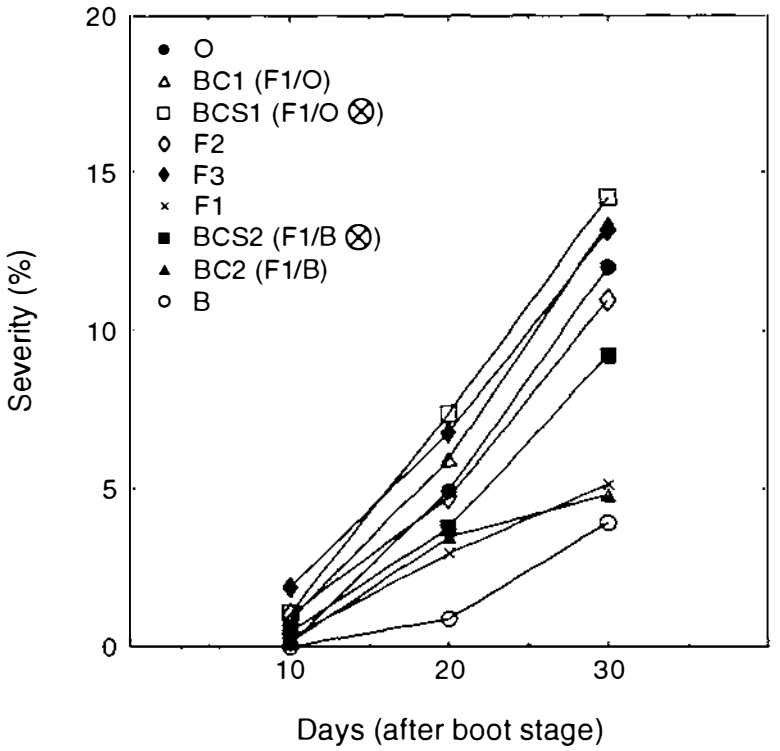


Fig. 29. Development of the disease progress curve for stripe rust on nine generations of Briscard x Otane infected with pathotype 106E139A-

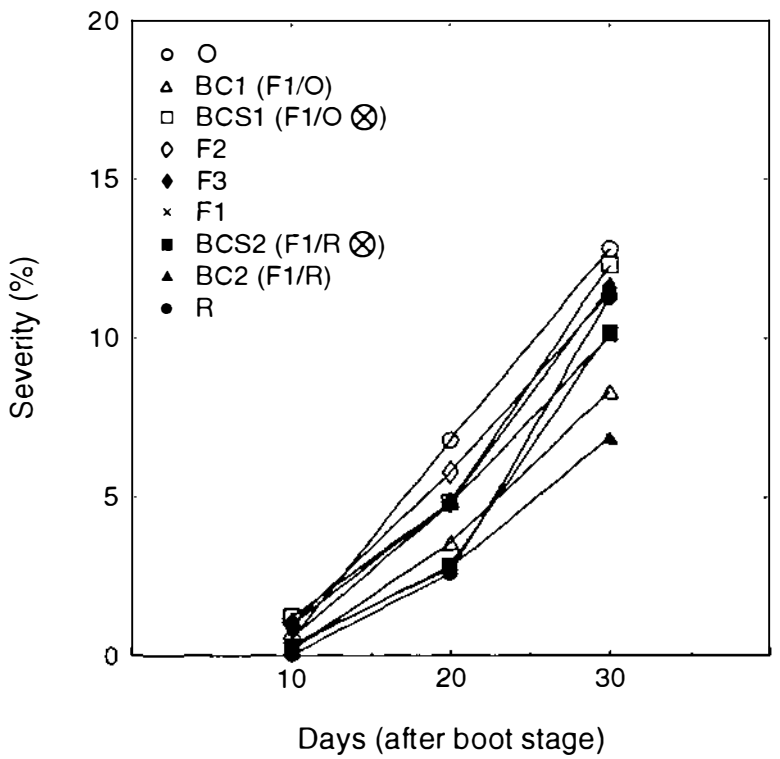


Fig. 30. Development of the disease progress curves for stripe rust on nine generations of Ruapuna x Otane infected with pathotype 106E139A-

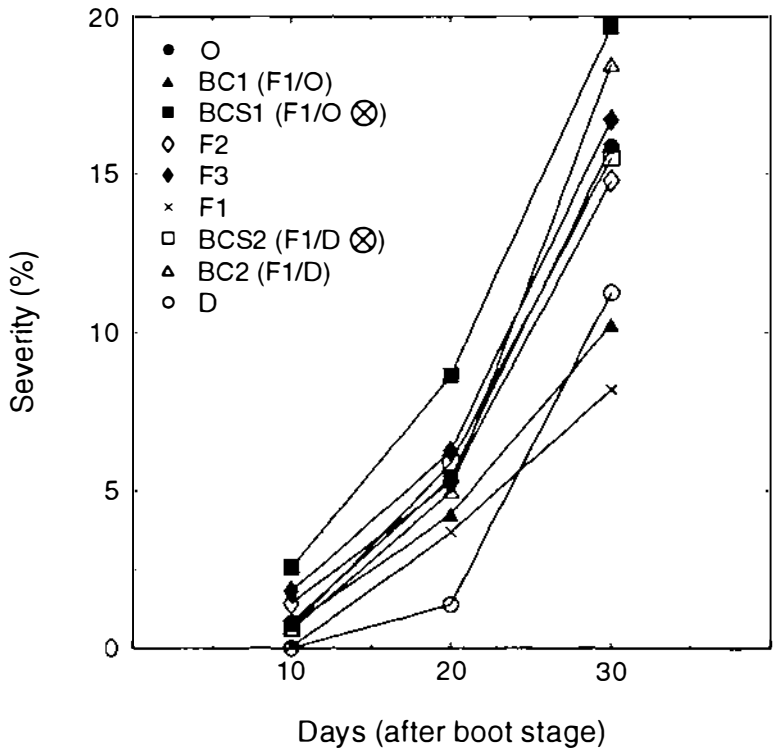


Fig. 31. Development of the disease progress curves for stripe rust on nine generations of Domino x Otane infected with pathotype 106E139A-

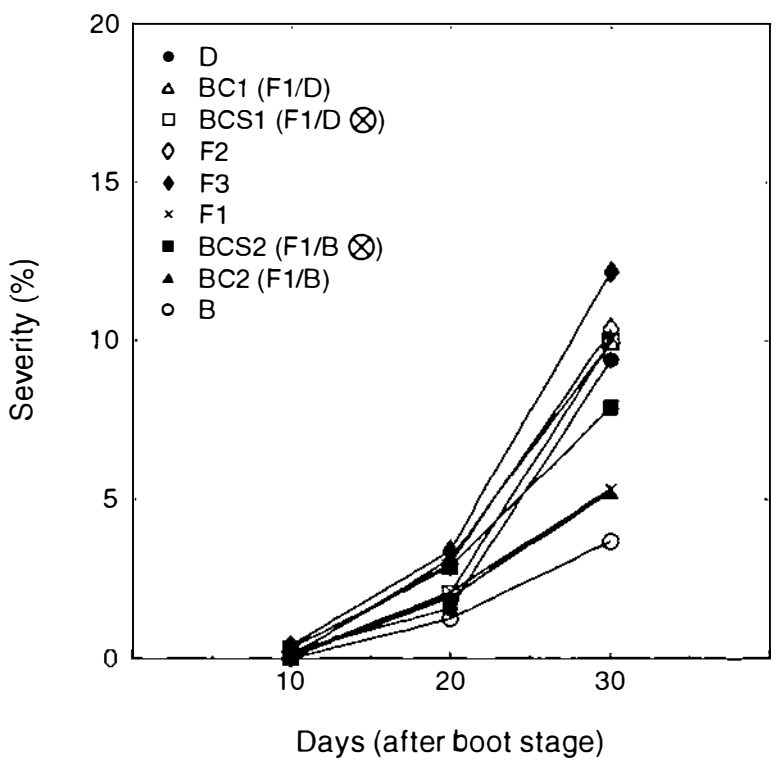


Fig. 32. Development of the disease progress curves for stripe rust on nine generations of Briscard x Domino infected with pathotype 106E139A-

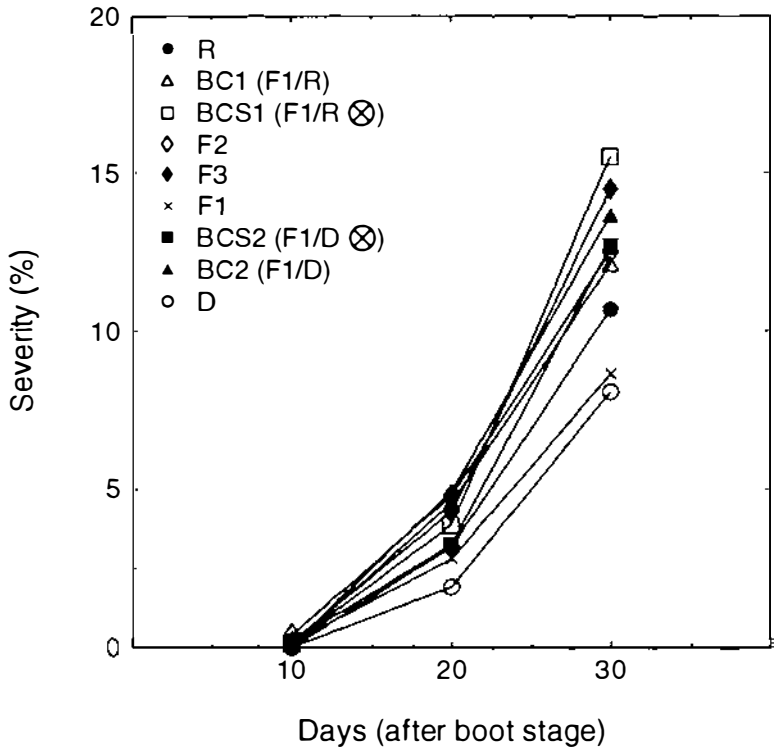


Fig. 33. Development of the disease progress curves for stripe rust on nine generations of Ruapuna x Domino infected with pathotype 106E139A-

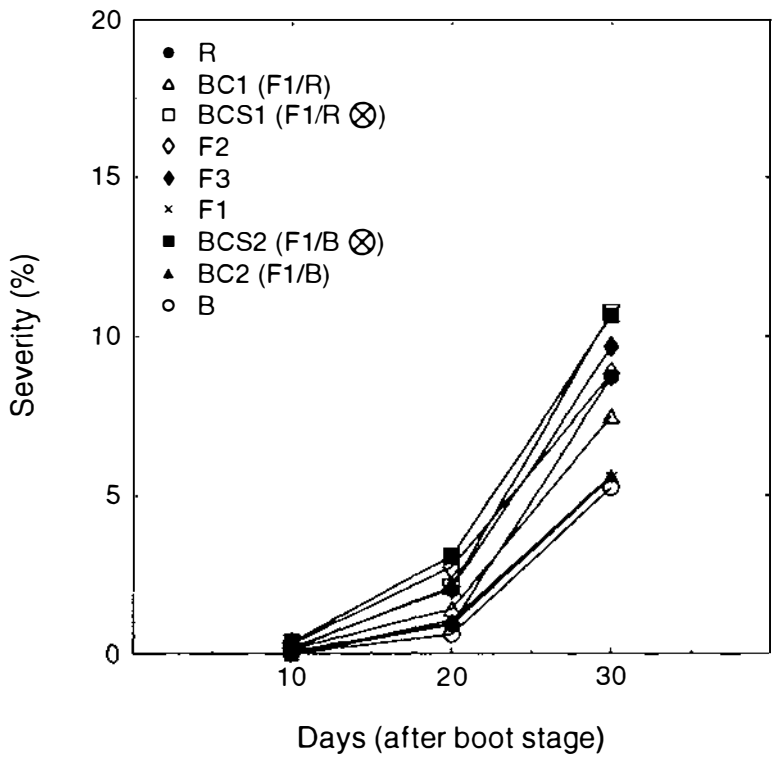


Fig. 34. Development of the disease progress curves for stripe rust on nine generations of Briscard x Ruapuna infected with pathotype 106E139A-

Table 95. Heritability estimates by different methods and genetic advance (GA) for the area under the disease progress curve (AUDPC) in ten crosses of wheat inoculated with stripe rust pathotype 106E139A-

Cross	h^2_{BS}					h^2_{NS}	GA
	1	2	3	4	5		
Briscard/Tiritea	0.94	0.96	0.91	0.93	0.93	0.79	57.0
Ruapuna/Tiritea	0.89	0.89	0.89	0.89	0.89	0.65	46.9
Domino/Tiritea	0.42	0.73	0.95	0.60	0.69	0.36	26.6
Otane/Tiritea	0.94	0.94	0.83	0.90	0.88	0.44	35.3
Briscard/Otane	0.76	0.82	0.89	0.81	0.83	0.30	20.2
Ruapuna/Otane	0.41	0.43	0.43	0.42	0.42	0.41	29.1
Domino/Otane	0.60	0.81	0.71	0.64	0.66	0.36	22.2
Briscard/Domino	0.81	0.82	0.73	0.78	0.77	0.62	35.8
Ruapuna/Domino	0.70	0.75	0.74	0.71	0.72	0.71	44.4
Briscard/Ruapuna	0.42	0.45	0.67	0.50	0.55	0.50	33.0

h^2_{BS} : In this ratio $(V_{F2} - E_W) / V_{F2}$, environmental effect (E_W) is $(V_{P1}+V_{P2})/2$, $(V_{P1}+V_{P2})^{1/2}$, V_{F1}, $(V_{P1}+V_{P2}+V_{F1})/3$ and $(V_{P1}+V_{P2}+2V_{F1})/4$ for 1, 2, 3, 4 and 5, respectively.

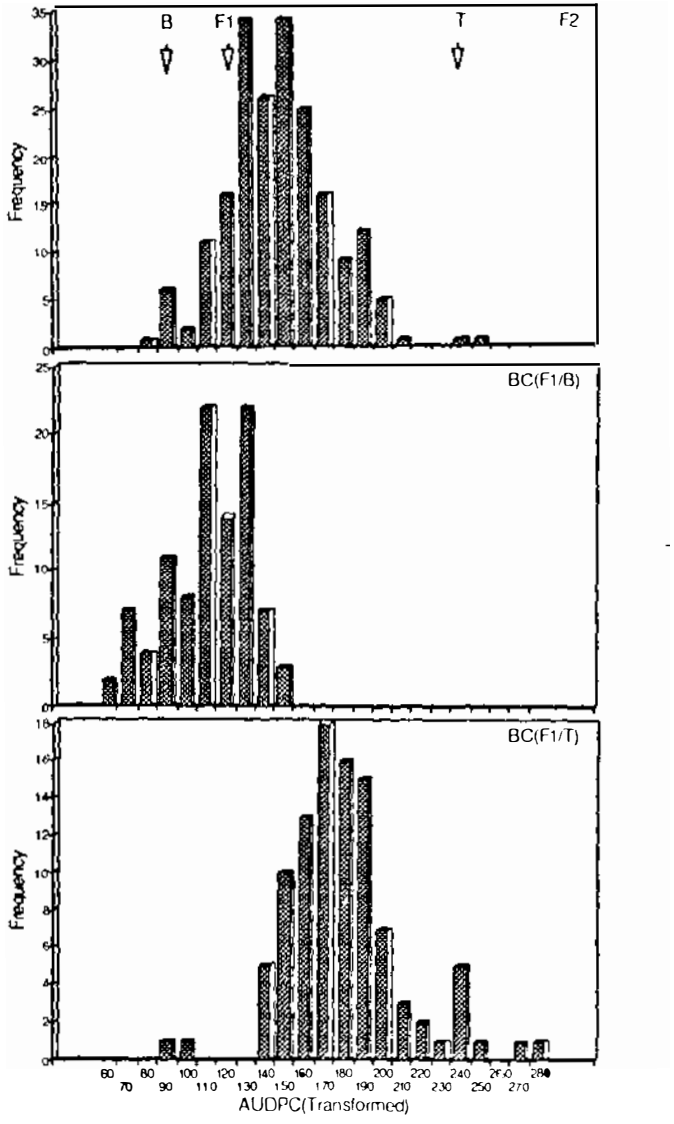
h^2_{NS} : $[2V_{F2}-(V_{BC1}+V_{BC2})]/V_{F2}$

Table 96. Estimates of the number of segregating genes or effective factors for the area under the disease progress curve (AUDPC) inoculated with stripe rust pathotype 106E139A-

Cross	Formula[1]							
	1	2	3	4	5	6	7	8
Briscard x Tiritea	3.6	4.6	9.0	3.1	6.3	1.6	5.5	2.4
Ruapuna x Tiritea	7.9	9.9	8.7	3.2	12.0	4.4	4.2	12.2
Domino x Tiritea	5.7	5.8	7.0	5.0	6.4	2.9	4.3	8.0
Otane x Tiritea	2.4	2.6	4.6	1.9	3.4	1.2	3.0	3.2
Briscard x Otane	0.1	0.1	0.3	0.1	0.1	0.1	0.1	0.1
Ruapuna x Otane	0.1	0.1	0.0	0.1	0.1	0.0	0.1	0.1
Domino x Otane	0.1	0.1	0.1	0.1	0.1	0.0	3.1	0.1
Briscard x Domino	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1
Ruapuna x Domino	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Briscard x Ruapuna	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0

[1]1: $n = (\mu_{P2} - \mu_{P1})^2/[8(\sigma^2_{F2} - \sigma^2_{F1})]$
2: $n = (\mu_{P2} - \mu_{P1})^2/\{8[\sigma^2_{F2} - (0.5\sigma^2_{F1}+0.25\sigma^2_{P1}+0.25\sigma^2_{P2})]\}$
3: $n = (\mu_{P2} - \mu_{P1})^2/\{8[\sigma^2_{F2} - (\sigma^2_{BC1}+\sigma^2_{BC2})]\}$
4: $n = (\mu_{P2} - \mu_{P1})^2/\{8[(\sigma^2_{BC1} + \sigma^2_{BC2}) - (\sigma^2_{F1}+0.5\sigma^2_{P1}+0.5\sigma^2_{P2})]\}$
5: $n = \{(\mu_{P2} - \mu_{P1})^2[1.5-2h(1-h)]\}/\{8[\sigma^2_{F2} - 0.25(2\sigma^2_{F1}+\sigma^2_{P1}+\sigma^2_{P2})]\}$
where $h = (\mu F_1 - \mu P_1)/(\mu P_2 - \mu P_1)$
6: $n = (\mu_{F1} - \mu_{P1})^2/\{4[\sigma^2_{BC1} - 0.5(\sigma^2_{F1}+\sigma^2_{P1})]\}$
7: $n = (\mu_{P2} - \mu_{F1})^2/\{4[\sigma^2_{BC2} - 0.5(\sigma^2_{F1}+\sigma^2_{P2})]\}$
where $\mu P_1 < \mu P_2$
8: $n = (\mu_{P2} - \mu_{P1})^2/\{5.33[\sigma^2_{F3} - (0.5\sigma^2_{P1}+0.5\sigma^2_{P2})]\}$

Fig. 35. Frequency distributions for the area under the disease progress curve (transformed) of six generations of Briscard x Tiritea infected with stripe rust pathotype 106E139A-



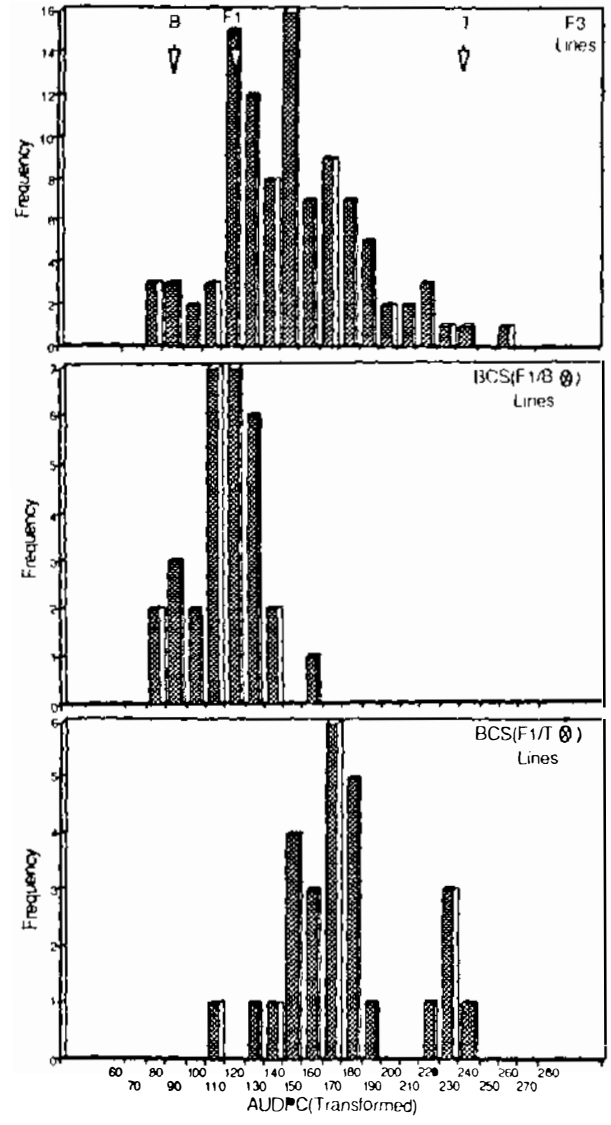


Fig. 36. Frequency distributions for the area under the disease progress curve (transformed) of six generations of Ruapuna x Tiritea infected with stripe rust pathotype 106E139A-

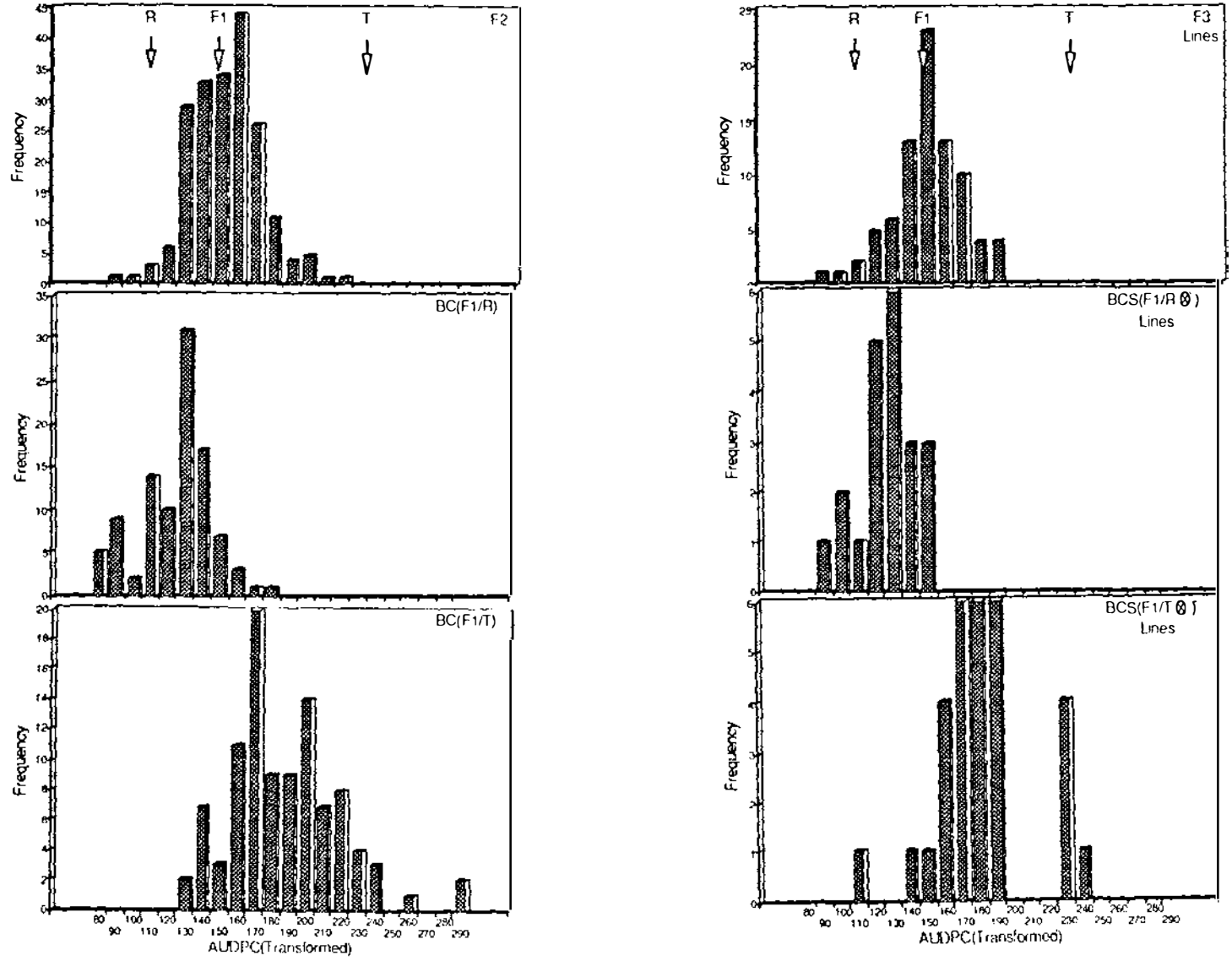
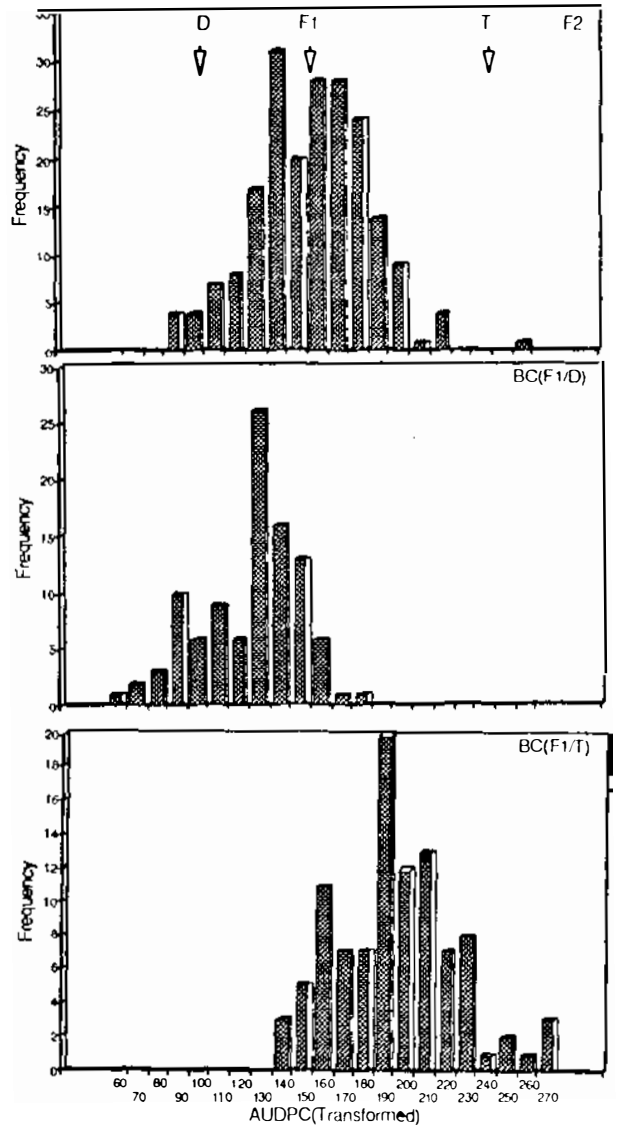


Fig. 37. Frequency distributions for the area under the disease progress curve (transformed) of six generations of Domino x Tiritea infected with stripe rust pathotype 106E139A-



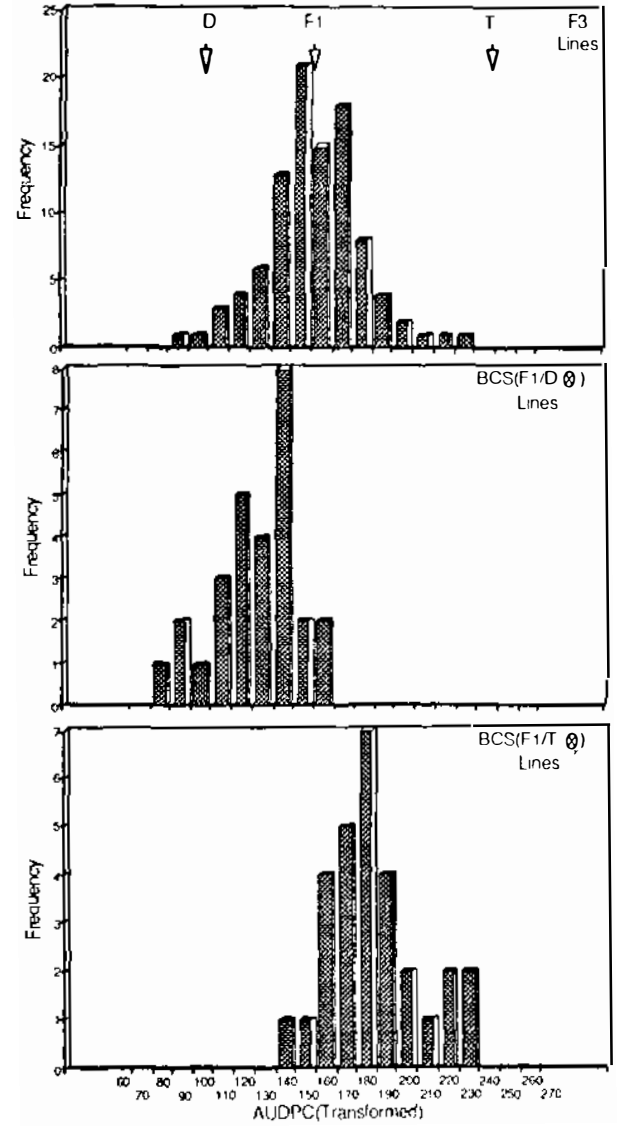
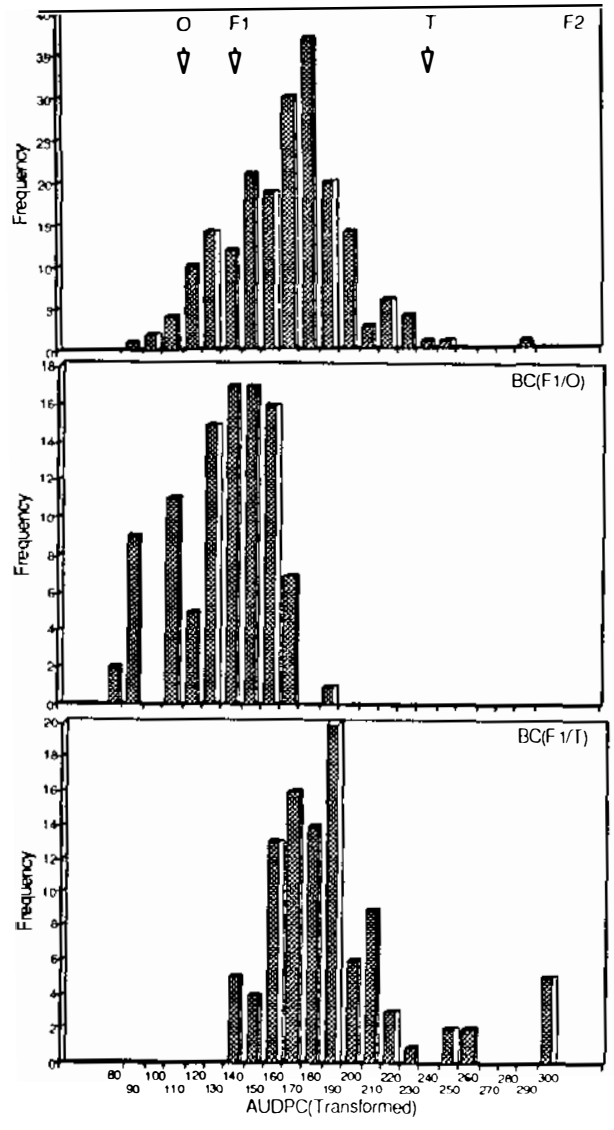


Fig. 38. Frequency distributions for the area under the disease progress curve (transformed) of six generations of Otane x Tiritea infected with stripe rust pathotype 106E139A-



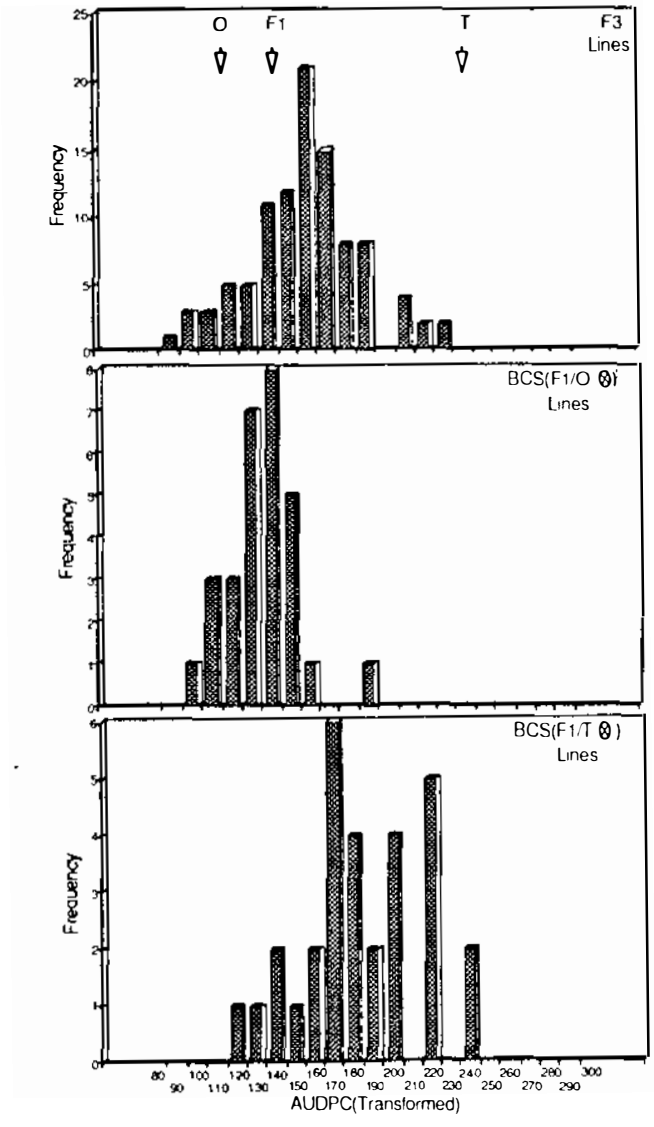
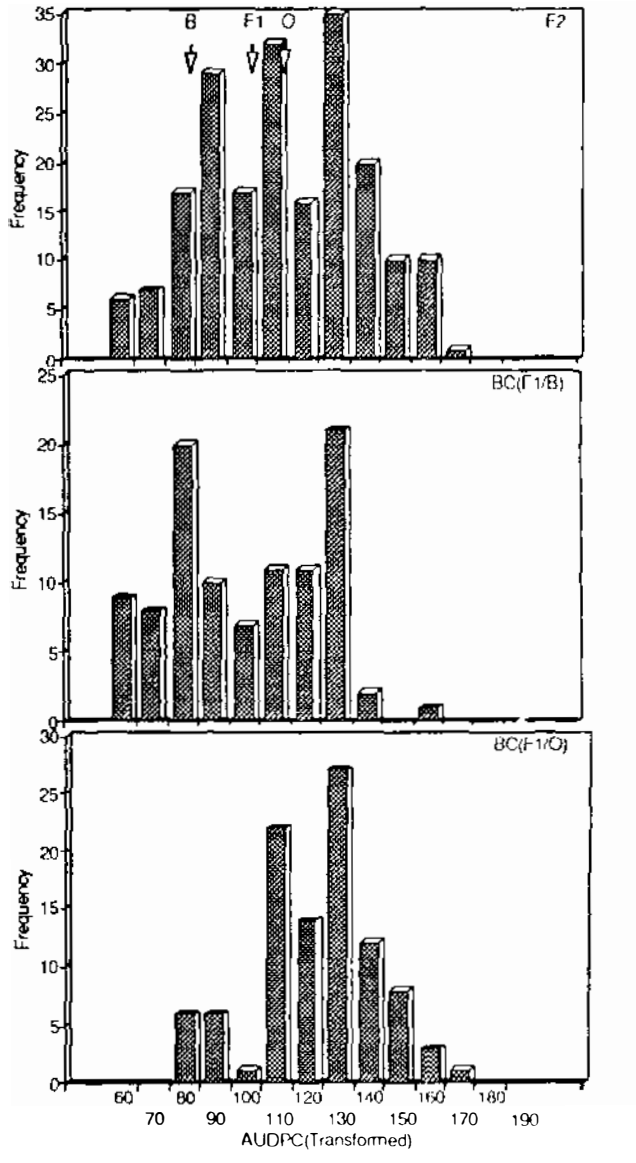


Fig. 39. Frequency distributions for the area under the disease progress curve (transformed) of six generations of Briscard x Otane infected with stripe rust pathotype 106E139A-



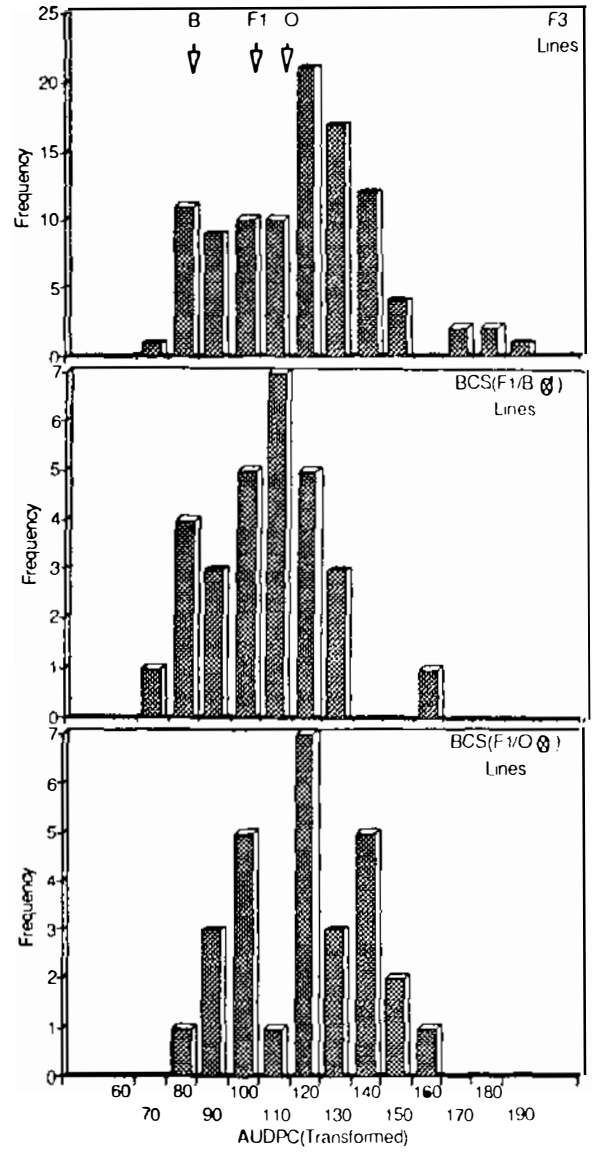


Fig. 40. Frequency distributions for the area under the disease progress curve (transformed) of six generations of Ruapuna x Otane infected with stripe rust pathotype 106E139A-

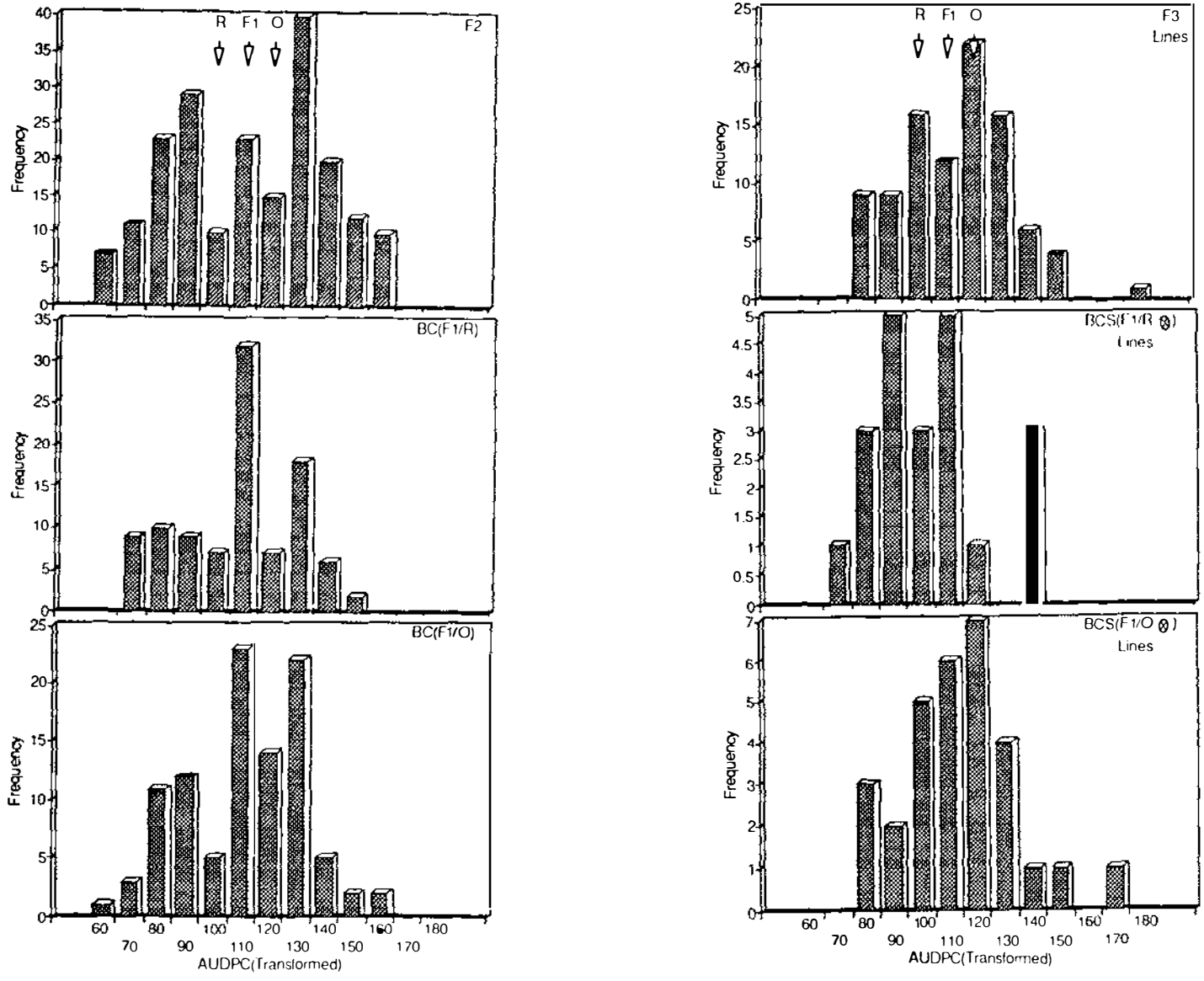
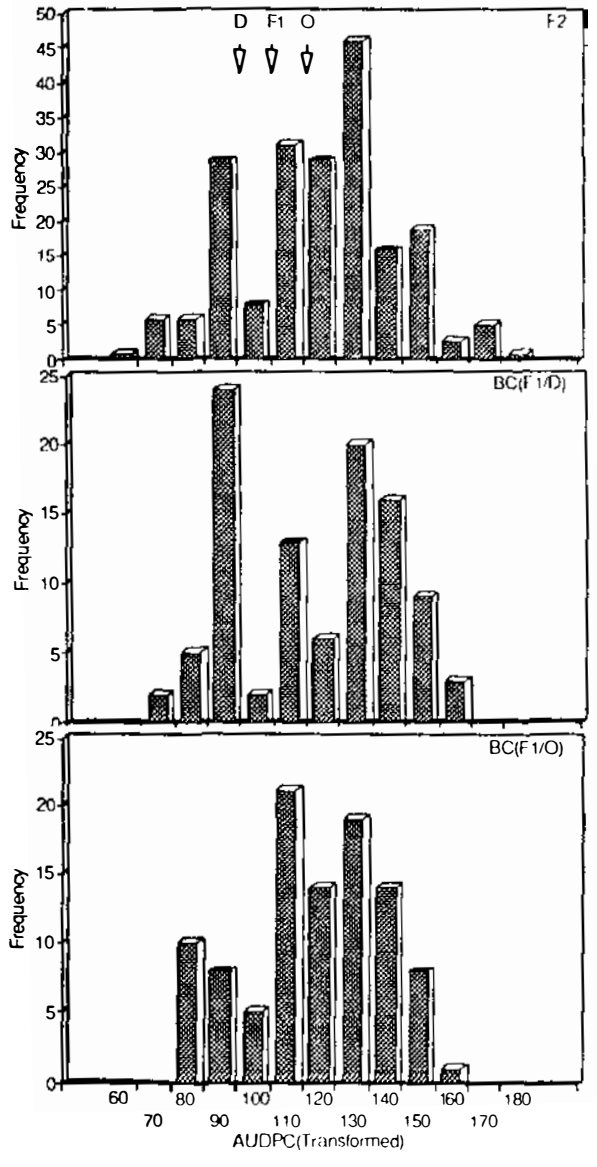


Fig. 41. Frequency distributions for the area under the disease progress curve (transformed) of six generations of Domino x Otane infected with stripe rust pathotype 106E139A-



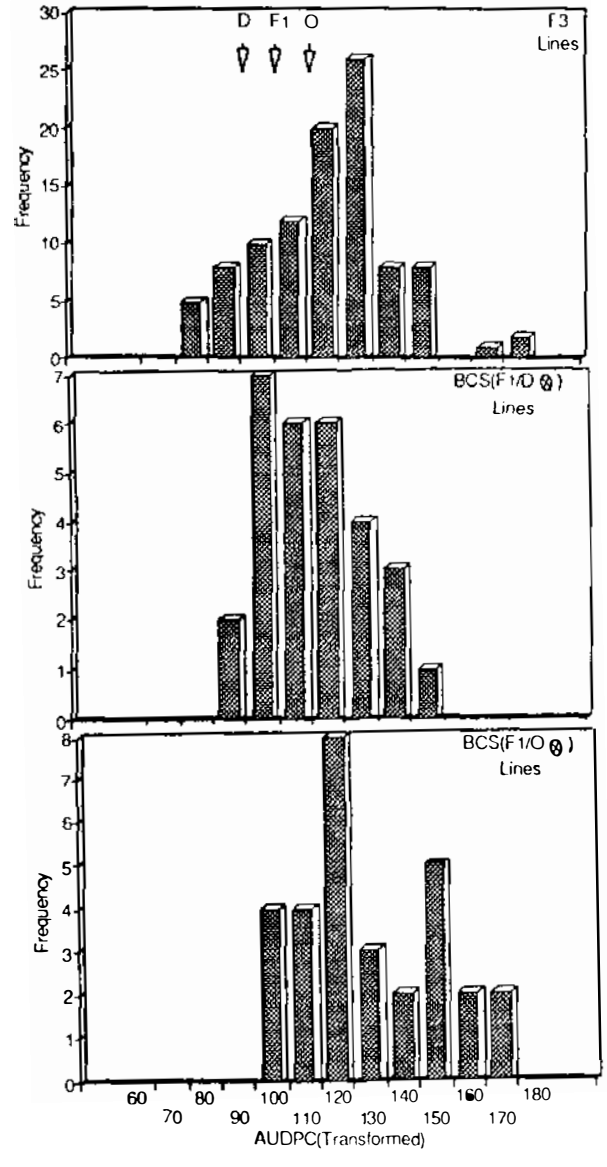


Fig. 42. Frequency distributions for the area under the disease progress curve (transformed) of six generations of Briscard x Domino infected with stripe rust pathotype 106E139A-

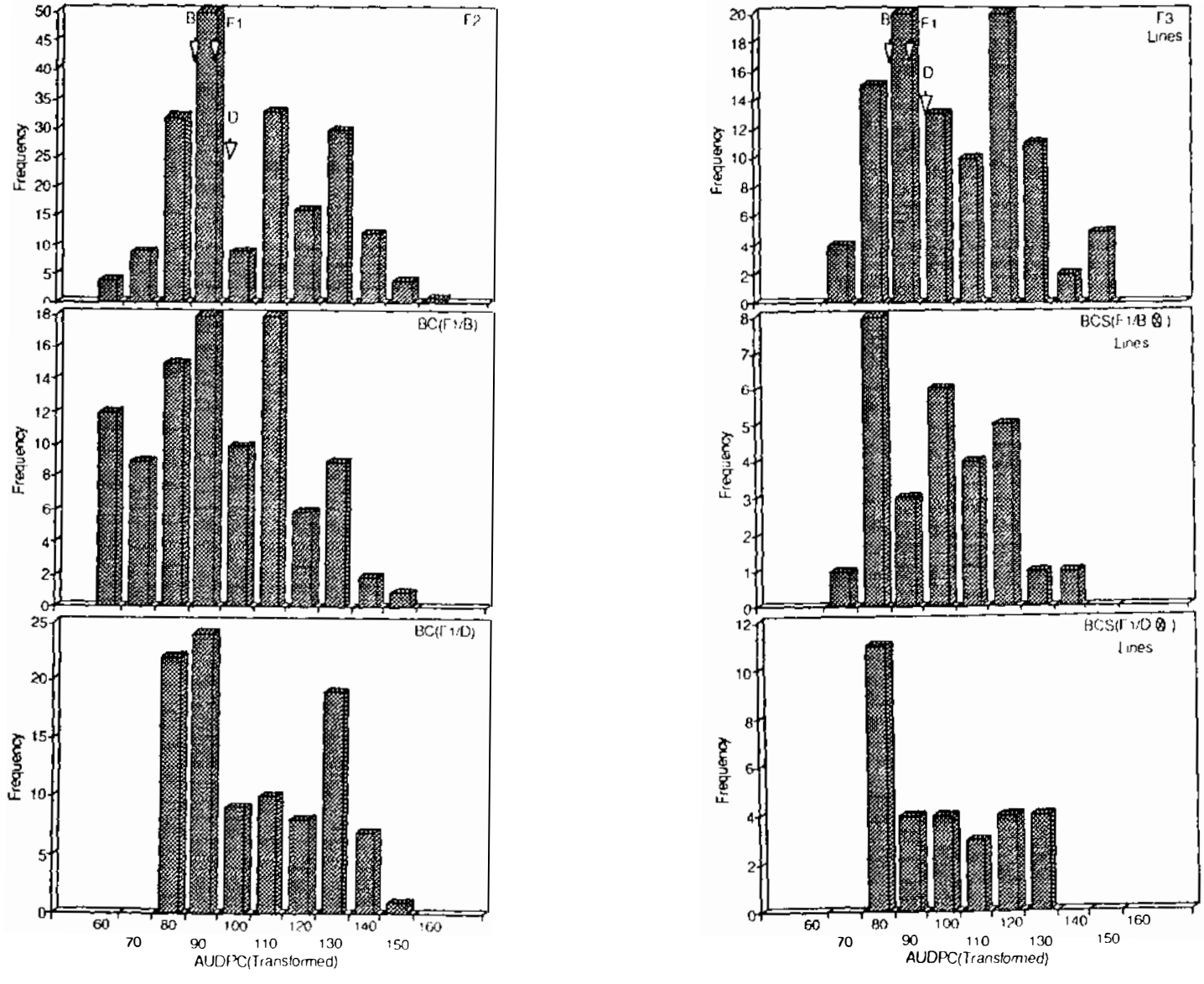
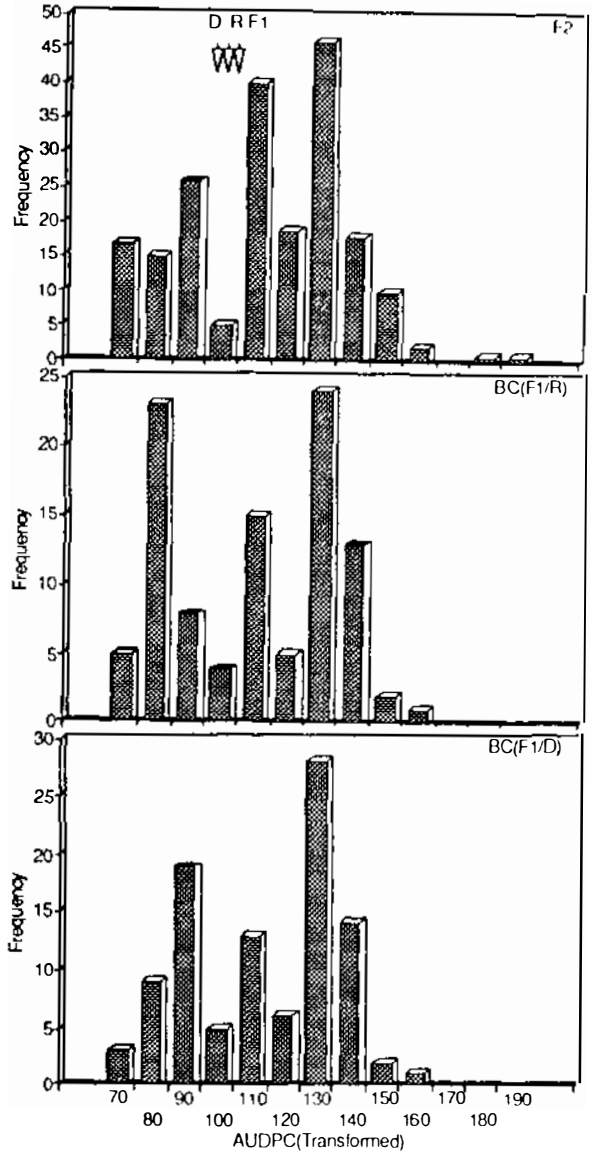


Fig. 43. Frequency distributions for the area under the disease progress curve (transformed) of six generations of Ruapuna x Domino infected with stripe rust pathotype 106E139A-



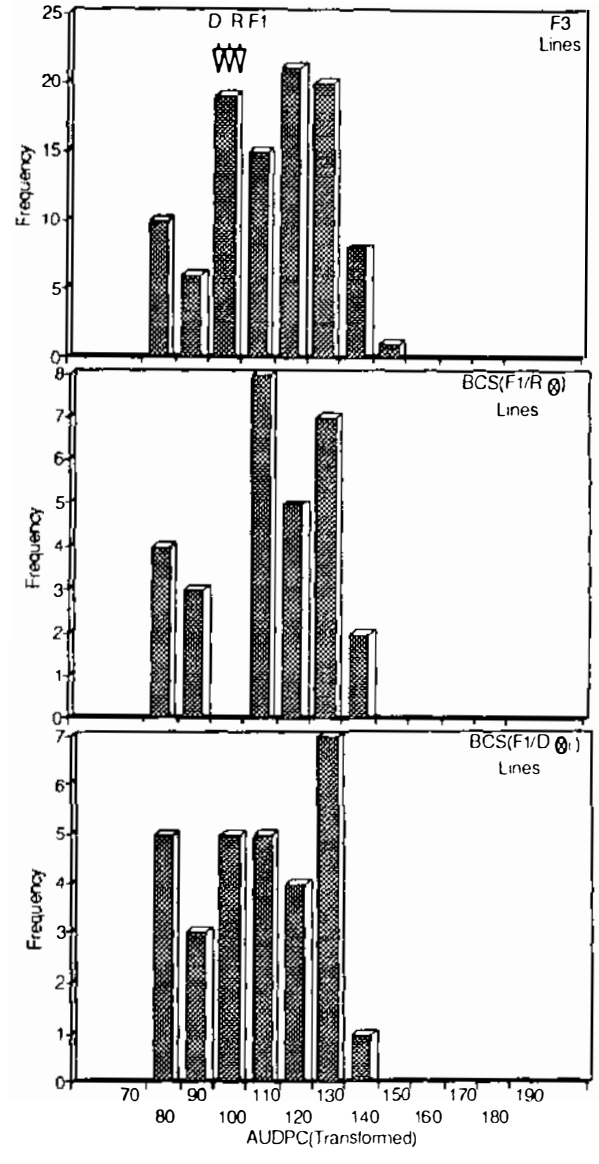
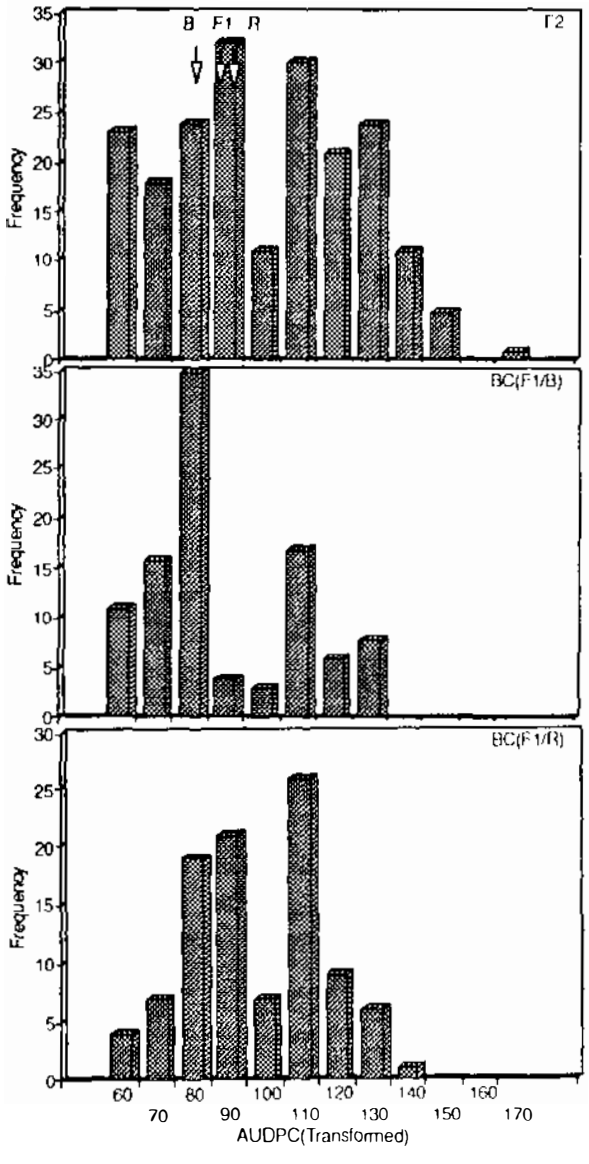


Fig. 44. Frequency distributions for the area under the disease progress curve (transformed) of six generations of Briscard x Ruapuna infected with stripe rust pathotype 106E139A-



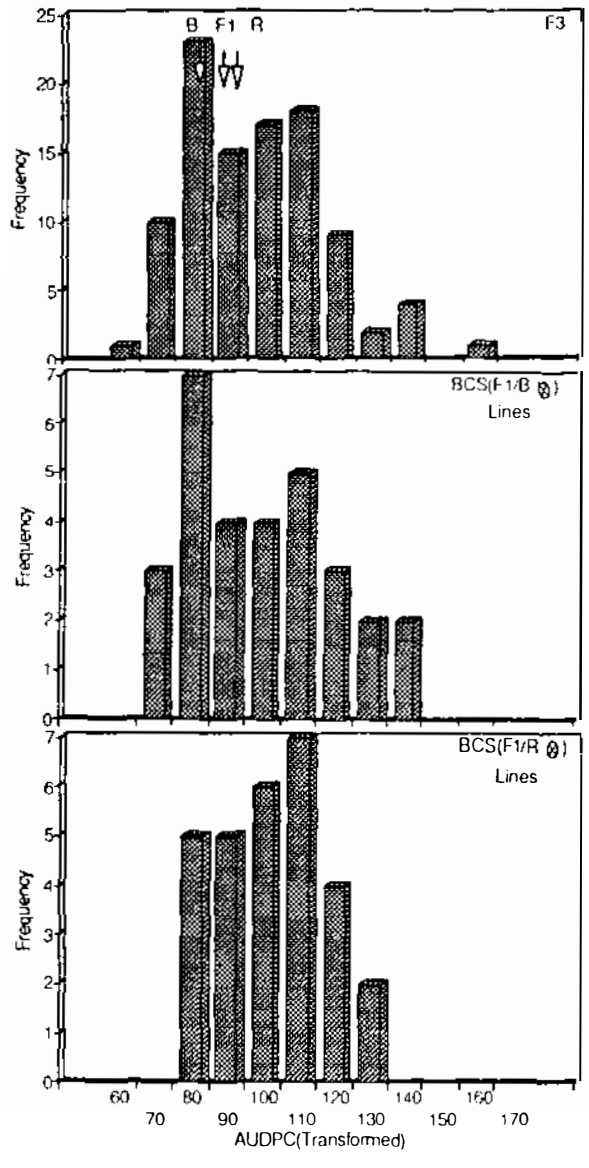


Table 97. Estimates of gene effects for AUDPC in cross Briscard x Tiritea

A) using P_1, P_2, F_1, F_2, BC_1 and BC_2

m	146.43±1.01**	167.78±1.90**	165.71±4.26**	167.84±1.90**	168.82±2.25**	165.41±4.27**	173.46±10.6**	169.20±2.28**	176.38±10.9**
[d]	67.77±1.89**	72.50±1.92**	72.61±1.93**	73.41±2.25**	72.58±1.92**	73.63±2.28**	72.46±1.94**	73.82±2.28**	73.82±2.28**
[h]		-41.45±3.12**	-38.82±5.76**	-41.72±3.14**	-48.35±8.64**	-38.66±5.76**	-59.72±26.8 *	-50.68±8.95**	-68.48±27.9 *
[i]			2.68±4.92ns			3.14±4.95ns	-4.68±10.4ns		-7.19±10.7ns
[j]				-6.45±8.24ns		-7.04±8.29ns		-8.58±8.58ns	-9.79±8.67ns
[l]					6.86±8.01ns		13.59±17.0ns	8.82±8.24ns	19.42±17.8ns
χ^2	178.57**	2.21ns	233.71**	1.60ns	1.48ns	1.19ns	1.28ns	0.45ns	...

B) using P_1, P_2, F_2, F_3, BCS_1 and BCS_2

m	150.18±0.78**	160.19±1.74**	151.30±3.22**	160.39±1.74**	167.68±2.24**	150.96±3.22**	183.41±7.54**	169.20±2.28**	191.15±7.80**
[d]	66.51±1.68**	68.37±1.70**	69.16±1.72**	71.17±2.24**	68.80±1.70**	72.48±2.27**	68.10±1.73**	73.82±2.28**	73.82±2.28**
[h]		-37.97±5.88**	-14.15±9.33ns	-39.38±5.92**	-114.01±15.5**	-14.28±9.33ns	-191.97±38.9**	-128.31±16.1**	-238.40±40.7**
[i]			12.97±3.95**			13.79±3.96**	-15.94±7.30 *		-21.96±7.46**
[j]				-25.48±13.3(*)		-29.83±13.4 *		-45.23±13.7**	-53.87±14.0**
[l]					145.30±27.4**		238.28±50.5**	167.83±28.2**	300.22±53.1**
χ^2	89.51**	47.75**	139.95**	44.08**	19.55**	31.97**	14.78**	8.66**	...

C) using P_1, P_2, F_1, F_2, BC_1, BC_2, F_3, BCS_1 and BCS_2

m	155.11±3.34**
[d]	71.30±2.15**
[h]	-26.55±12.8 *
[i]	9.82±3.67**
[j]	-12.27±8.19ns
[l]	-0.08±10.5ns
χ^2	37.07**

Table 98. Estimates of gene effects for AUDPC in cross Ruapuna x Tiritea

A) using P_1, P_2, F_1, F_2, BC_1 and BC_2

m	157.09±0.98**	168.35±1.92**	154.10±4.18**	168.08±1.93**	172.49±2.20**	154.15±4.18**	162.48±9.50**	172.43±2.23**	161.28±9.96**
[d]	57.47±1.83**	61.04±1.90**	62.42±1.93**	59.88±2.21**	61.94±1.91**	61.60±2.25**	62.28±1.94**	61.80±2.27**	61.80±2.27**
[h]		-23.93±3.49**	-3.37±6.40ns	-23.18±3.57**	-52.83±8.36**	-3.18±6.40ns	-27.20±25.1ns	-52.54±8.77**	-23.61±26.7ns
[i]			18.11±4.72**			17.82±4.74**	10.16±6.38ns		11.15±9.69ns
[j]				8.49±8.21ns		5.84±8.24ns		0.95±8.46ns	3.51±8.75ns
[l]					32.02±8.41**		16.40±16.7ns	31.79±8.67**	14.00±17.7ns
χ^2	62.77**	15.82**	88.04**	14.75**	1.34ns	0.62ns	0.16ns	1.32ns	...

B) using P_1, P_2, F_2, F_3, BCS_1 and BCS_2

m	153.09±0.64**	160.25±1.53**	146.60±2.36**	160.31±1.55**	170.89±2.16**	146.54±2.36**	151.93±6.39**	172.43±2.27**	154.92±6.61**
[d]	54.09±1.59**	56.05±1.64**	59.16±1.69**	56.43±2.15**	58.33±1.67**	61.29±2.24**	59.12±1.69**	61.80±2.27**	61.80±2.27**
[h]		-25.62±5.11**	12.03±7.11(*)	-25.86±5.19**	-119.20±14.4**	11.81±7.12(*)	-18.73±34.9ns	-129.98±15.1**	-36.63±36.4ns
[i]			24.01±3.16**			24.73±3.20**	19.30±6.13**		17.51±6.21**
[j]				-3.50±12.9ns		-19.04±13.1ns		-30.40±13.4 *	-24.17±13.6(*)
[l]					116.77±23.9**		41.65±46.4ns	182.18±24.9**	65.51±48.3ns
χ^2	86.91**	61.73**	25.62**	61.65**	13.08**	1.84ns	0.16ns	7.95**	...

C) using P_1, P_2, F_1, F_2, BC_1, BC_2, F_3, BCS_1 and BCS_2

m	148.75±2.83**
[d]	59.74±2.11**
[h]	5.33±11.8ns
[i]	21.88±3.15**
[j]	1.33±8.11ns
[l]	-1.83±10.6ns
χ^2	7.21(*)

Table 99. Estimates of gene effects for AUDPC in cross Domino x Tiritea

A) using P_1, P_2, F_1, F_2, BC_1 and BC_2

m	162.00±1.15**	171.31±2.35**	153.17±4.93**	170.51±2.46**	178.01±2.81**	153.21±4.93**	167.44±11.2**	178.25±3.08**	167.68±11.4**
[d]	-62.35±2.10**	-66.62±2.30**	-69.31±2.39**	-64.80±2.84**	-69.40±2.39**	-68.88±3.01**	-69.57±2.39**	-69.77±3.08**	-69.77±3.08**
[h]		-18.75±4.11**	6.04±7.21ns	-17.35±4.31**	-59.48±10.3**	6.06±7.21ns	-33.40±28.7ns	-60.31±11.2**	-34.10±29.4ns
[i]			23.94±5.72**			23.65±5.85**	10.67±10.9ns		10.57±11.0ns
[j]				-10.16±9.31ns		-2.22±9.52ns		1.88±9.75ns	1.01±9.79ns
[l]					42.07±9.76**		26.55±18.7ns	42.66±10.2**	27.01±19.2ns
χ^2	40.32**	19.55**	14.18**	18.35**	0.96ns	1.98ns	0.01ns	0.93ns	...

B) using P_1, P_2, F_2, F_3, BCS_1 and BCS_2

m	156.74±0.67**	162.49±1.81**	155.53±2.76**	163.15±1.92**	172.54±2.63**	155.56±2.76**	180.26±6.40**	178.25±3.08**	187.29±6.68**
[d]	-56.23±1.65**	-58.02±1.73**	-59.73±1.80**	-60.08±2.66**	-60.88±1.81**	-64.59±2.91**	-60.69±1.82**	-69.77±3.08**	-69.77±3.08**
[h]		-21.79±6.39**	-2.08±8.70ns	-24.03±6.76**	-105.58±17.1**	-3.03±8.71ns	-145.25±34.5**	-140.83±19.8**	-187.45±36.4**
[i]			12.16±3.64**			14.62±3.82**	-7.84±5.92ns		-9.04±5.93ns
[j]				14.04±13.8ns		30.78±14.5 *		54.50±15.2**	55.79±15.3**
[l]					150.54±28.6**		199.00±46.5**	198.21±31.5**	255.27±48.9**
χ^2	54.51**	42.86**	57.11**	41.83**	15.12**	27.20**	13.37**	2.33ns	...

C) using P_1, P_2, F_1, F_2, BC_1, BC_2, F_3, BCS_1 and BCS_2

m	158.42±3.08**
[d]	-61.64±2.66**
[h]	-18.56±13.0ns
[i]	11.35±3.47**
[j]	0.01±9.44ns
[l]	22.49±11.6(*)
χ^2	34.12**

Table 100. Estimates of gene effects for AUDPC in cross Otane x Tiritea

A) using P_1, P_2, F_1, F_2, BC_1 and BC_2

m	164.91±1.17**	183.29±2.23**	186.09±4.80**	183.82±2.26**	183.30±2.62**	185.52±4.82**	199.89±12.0**	184.62±2.76**	206.60±12.5**
[d]	53.01±2.09**	61.29±2.26**	60.93±2.33**	63.10±2.60**	61.30±2.30**	62.78±2.73**	60.80±2.33**	63.56±2.76**	63.56±2.76**
[h]		-35.02±3.61**	-38.61±6.54**	-36.18±3.70**	-35.08±9.76**	-38.33±6.54**	-76.03±30.4 *	-41.13±10.6**	-96.15±32.3**
[i]			-3.67±5.55ns			-2.25±5.66ns	-16.89±11.9ns		-21.98±12.2(*)
[j]				-13.15±9.42ns		-12.41±9.60ns		-14.80±9.98ns	-18.99±10.3(*)
[l]					0.07±9.06ns		24.41±19.4ns	4.79±9.61ns	37.83±20.7(*)
χ^2	99.71**	5.46ns	191.69**	3.51ns	5.46(*)	3.35(*)	3.43(*)	3.26(*)	...

B) using P_1, P_2, F_2, F_3, BCS_1 and BCS_2

m	161.95±0.75**	166.84±1.88**	149.95±3.08**	167.03±1.94**	180.19±2.49**	149.92±3.08**	176.69±6.94**	184.62±2.76**	185.62±7.35**
[d]	51.04±1.72**	52.65±1.81**	56.46±1.89**	53.32±2.51**	55.95±1.86**	59.99±2.68**	56.16±1.89**	63.57±2.77**	63.56±2.76**
[h]		-18.57±6.54**	29.67±9.55**	-19.32±6.82**	-141.55±16.5**	28.71±9.56**	-123.72±36.9**	-176.10±18.4**	-177.27±39.7**
[i]			27.03±3.90**			28.55±3.98**	3.61±6.70ns		-1.00±6.81ns
[j]				-5.33±13.8ns		-26.25±14.1(*)		-55.44±14.9**	-55.85±15.2**
[l]					234.30±28.8**		212.61±49.5**	277.67±31.1**	284.00±53.1**
χ^2	88.22**	80.15**	32.68**	80.01**	13.84**	28.57**	13.55**	0.02ns	...

C) using P_1, P_2, F_1, F_2, BC_1, BC_2, F_3, BCS_1 and BCS_2

m	155.31±3.38**
[d]	58.56±2.53**
[h]	17.79±13.9ns
[i]	22.33±3.71**
[j]	-12.65±9.51ns
[l]	-23.17±11.7 *
χ^2	44.56**

Table 101. Estimates of gene effects for AUDPC in cross Briscard x Otane

A) using P_1, P_2, F_1, F_2, BC_1 and BC_2

m	109.31±0.92**	106.01±1.62**	117.55±4.25**	105.90±1.62**	103.40±1.85**	117.38±4.25**	110.92±9.67**	103.39±1.85**	112.30±9.70**
[d]	17.52±1.59**	17.47±1.59**	17.42±1.59**	15.73±1.85**	17.28±1.59**	15.71±1.85**	17.35±1.59**	15.70±1.85**	15.70±1.85**
[h]		7.35±2.99 *	-8.10±6.05ns	7.30±2.99 *	28.01±7.66**	-8.07±6.05ns	9.80±24.2ns	27.29±7.68**	5.68±24.3ns
[i]			-13.81±4.70**			-13.74±4.70**	-7.52±9.49ns		-8.91±9.52ns
[j]				13.14±7.19(*)		12.97±7.19(*)		11.97±7.21(*)	12.54±7.23(*)
[l]					-22.55±7.70**		-11.85±15.5ns	-21.81±7.71**	-9.11±15.6ns
χ^2	18.27**	12.21**	6.09 *	8.87 *	3.64ns	0.34ns	3.01(*)	0.88ns	...

B) using P_1, P_2, F_2, F_3, BCS_1 and BCS_2

m	113.83±0.62**	109.20±1.44**	122.09±2.60**	109.20±1.44**	103.38±1.85**	122.09±2.60**	121.80±6.06**	103.39±1.85**	121.97±6.06**
[d]	14.74±1.41**	14.71±1.41**	14.64±1.41**	15.76±1.85**	14.46±1.41**	15.70±1.85**	14.64±1.42**	15.70±1.85**	15.70±1.85**
[h]		18.38±5.17**	-18.40±8.06 *	18.36±5.17**	78.21±13.0**	-18.43±8.06 *	-16.70±32.5ns	78.03±13.0**	-17.73±32.5ns
[i]			-18.68±3.14**			-18.69±3.14**	-18.42±5.77**		-18.58±5.77**
[j]				-10.05±11.5ns		-10.25±11.5ns		-9.08±11.5ns	-10.24±11.5ns
[l]					-118.49±23.6**		-2.36±43.4ns	-118.17±23.6**	-0.98±43.4ns
χ^2	48.80**	36.16**	0.90ns	35.39**	10.98**	0.00ns	0.80ns	10.36**	...

C) using P_1, P_2, F_1, F_2, BC_1, BC_2, F_3, BCS_1 and BCS_2

m	120.34±2.68**
[d]	14.47±1.75**
[h]	-12.61±10.8ns
[i]	-16.77±2.92**
[j]	8.55±6.88ns
[l]	1.03±9.48ns
χ^2	4.99ns

Table 102. Estimates of gene effects for AUDPC in cross Ruapuna x Otane

A) using P_1, P_2, F_1, F_2, BC_1 and BC_2

m	111.94±0.94**	112.45±1.79**	104.19±4.66**	112.28±1.79**	115.64±2.06**	104.17±4.66**	128.50±9.75**	115.46±2.06**	128.65±9.75**
[d]	7.56±1.67**	7.53±1.67**	7.42±1.67**	9.50±2.06**	7.27±1.67**	9.34±2.06**	7.23±1.67**	9.19±2.06**	9.19±2.06**
[h]		-1.13±3.38ns	10.21±6.81ns	-0.93±3.38ns	-24.66±8.20**	10.20±6.81ns	-55.04±24.0 *	-24.31±8.20**	-55.46±24.0 *
[i]			9.89±5.16ns			9.71±5.16(*)	-12.86±9.53ns		-13.19±9.53ns
[j]				11.60±7.07ns		-11.31±7.07ns		-11.33±7.07ns	-11.54±7.07ns
[l]					26.35±8.36**		43.87±15.5**	26.18±8.36**	44.15±15.5**
χ^2	14.53**	14.41**	10.77**	11.72 *	4.49ns	8.17**	2.67ns	1.91ns	...

B) using P_1, P_2, F_2, F_3, BCS_1 and BCS_2

m	111.84±0.60**	113.45±1.54**	115.49±2.50**	113.47±1.54**	115.41±2.06**	115.50±2.50**	137.67±5.93**	115.46±2.06**	137.91±5.95**
[d]	9.86±1.52**	9.78±1.52**	9.86±1.52**	9.38±2.06**	9.70±1.52**	9.50±2.06**	9.98±1.52**	9.19±2.06**	9.19±2.06**
[h]		-6.36±5.59ns	-12.45±8.11ns	-6.42±5.59ns	-24.70±13.9(*)	-12.49±8.11ns	-142.99±32.7**	-25.07±14.0(*)	-144.41±32.8**
[i]			-3.22±3.11ns			-3.21±3.11ns	-22.29±5.57**		-22.45±5.58**
[j]				3.45±12.2ns		3.19±12.2ns		4.49±12.2ns	6.97±12.2ns
[l]					35.56±24.8ns		183.11±44.4**	36.10±24.8ns	184.99±44.5**
χ^2	19.69**	18.40**	34.13**	18.31**	16.34**	17.25**	0.32ns	16.20**	...

C) using P_1, P_2, F_1, F_2, BC_1, BC_2, F_3, BCS_1 and BCS_2

m	120.54±2.49**
[d]	10.44±1.92**
[h]	-41.30±10.5**
[i]	-7.28±2.76**
[j]	-10.11±6.92ns
[l]	39.40±9.75**
χ^2	13.19**

Table 103. Estimates of gene effects for AUDPC in cross Domino x Otane

A) using P_1, P_2, F_1, F_2, BC_1 and BC_2

m	115.00±0.91**	113.65±1.68**	127.23±4.06**	114.31±1.70**	109.98±1.96**	127.32±4.06**	119.03±9.41**	110.68±1.98**	117.81±9.42**
[d]	-8.28±1.64**	-8.01±1.66**	-7.40±1.67**	-10.73±1.96**	-7.12±1.68**	-9.93±1.97**	-7.21±1.68**	-9.78±1.98**	-9.78±1.98**
[h]		2.95±3.08ns	-15.52±5.90**	2.21±3.10ns	29.58±7.88**	-15.50±5.90**	7.13±24.2ns	28.50±7.89**	10.77±24.2ns
[i]			-16.72±4.55**			-16.09±4.56**	-9.02±9.18ns		-7.13±9.21ns
[j]				19.70±7.51**		18.16±7.52 *		19.17±7.51 *	18.69±7.53 *
[l]					-28.59±7.79**		-15.18±15.7ns	-28.20±7.79**	-17.61±15.7ns
χ^2	21.51**	20.60**	6.61 *	13.71**	7.12 *	1.25ns	6.15 *	0.60ns	...

B) using P_1, P_2, F_2, F_3, BCS_1 and BCS_2

m	120.18±0.58**	117.05±1.43**	122.97±2.35**	116.95±1.44**	111.15±1.94**	122.97±2.35**	105.61±5.70**	110.68±1.98**	104.61±5.75**
[d]	-12.76±1.42**	-12.30±1.43**	-11.73±1.45**	-11.42±1.94**	-11.59±1.44**	-10.46±1.97**	-11.68±1.45**	-9.78±1.98**	-9.78±1.97**
[h]		12.07±5.05 *	-4.82±7.33ns	12.47±5.09 *	66.68±13.2**	-4.69±7.33ns	95.68±31.0**	69.88±13.4**	101.64±31.3**
[i]			-9.44±2.97**			-9.68±2.98**	5.57±5.39ns		6.07±5.40ns
[j]				-7.69±11.5ns		-10.90±11.5ns		-15.51±11.6ns	-16.36±11.6ns
[l]					-102.78±22.9**		-138.59±41.5**	-107.29±23.1**	-146.56±41.9**
χ^2	28.95**	23.24**	19.79**	22.79**	3.06ns	12.24**	1.99ns	1.26ns	...

C) using P_1, P_2, F_1, F_2, BC_1, BC_2, F_3, BCS_1 and BCS_2

m	121.13±2.57**
[d]	-12.21±1.85**
[h]	6.25±10.7ns
[i]	-8.19±2.84**
[j]	13.85±7.15(*)
[l]	-17.29±9.44(*)
χ^2	44.56**

Table 104. Estimates of gene effects for AUDPC in cross Briscard x Domino

A) using P_1, P_2, F_1, F_2, BC_1 and BC_2

m	99.61±0.89**	97.73±1.66**	109.19±4.46**	97.83±1.67**	96.03±1.84**	109.20±4.46**	111.26±8.81**	96.16±1.85**	111.63±8.82**
[d]	6.41±1.57**	6.56±1.57**	6.72±1.57**	5.39±1.84**	6.67±1.57**	5.60±1.85**	6.71±1.57**	5.58±1.85**	5.58±1.85**
[h]		4.45±3.33ns	-12.57±6.99(*)	4.24±3.34ns	19.39±7.71 *	-12.64±6.99ns	-18.45±22.8ns	18.89±7.72 *	-19.58±22.8ns
[i]			-13.28±4.80**			-13.18±4.80**	-15.23±8.62(*)		-15.48±8.63(*)
[j]				8.53±7.05ns		8.18±7.05ns		7.96±7.05ns	8.27±7.06ns
[l]					-18.43±8.57 *		4.18±15.4ns	-18.06±8.58 *	4.94±15.4ns
χ^2	10.90 *	9.12 *	1.26ns	7.65 *	4.49ns	0.10ns	1.38ns	3.22(*)	...

B) using P_1, P_2, F_2, F_3, BCS_1 and BCS_2

m	102.63±0.54**	99.73±1.35**	108.37±2.21**	99.62±1.35**	96.56±1.84**	108.37±2.21**	121.94±5.31**	96.16±1.85**	120.73±5.34**
[d]	1.72±1.33ns	1.91±1.34ns	2.33±1.34(*)	5.19±1.84**	2.01±1.34ns	5.78±1.84**	2.47±1.34(*)	5.58±1.85**	5.58±1.85**
[h]		11.19±4.75 *	-13.36±6.87(*)	11.78±4.76 *	40.29±12.5**	-13.09±6.87(*)	-91.82±28.8**	43.47±12.5**	-84.56±28.9**
[i]			-13.80±2.79**			-14.00±3.82**	-25.43±4.50**		-24.57±5.01**
[j]				-27.70±10.7**		-29.10±14.5**		-30.01±10.7**	-26.32±10.7 *
[l]					-54.49±21.5 *		108.20±38.5**	-59.26±21.6**	98.53±38.7 *
χ^2	43.88**	38.33**	14.12**	31.60**	31.93**	6.47 *	6.01 *	24.08**	...

C) using P_1, P_2, F_1, F_2, BC_1, BC_2, F_3, BCS_1 and BCS_2

m	109.24±2.48**
[d]	3.46±1.73 *
[h]	-18.62±10.7(*)
[i]	-14.41±2.71**
[j]	0.48±6.71ns
[l]	7.80±10.2ns
χ^2	18.56**

Table 105. Estimates of gene effects for AUDPC in cross Ruapuna x Domino

A) using P_1, P_2, F_1, F_2, BC_1 and BC_2

m	109.74±0.95**	107.32±1.73**	119.03±4.49**	107.46±1.73**	104.61±1.95**	119.01±4.49**	108.36±9.54**	104.79±1.95**	108.99±9.55**
[d]	0.51±1.67ns	0.24±1.67ns	-0.01±1.68ns	1.55±1.95ns	-0.01±1.68ns	1.22±1.95ns	-0.02±1.68ns	1.14±1.95ns	1.14±1.95ns
[h]		5.61±3.36(*)	-11.18±6.82ns	5.31±3.37ns	28.07±8.04**	-11.25±6.82(*)	18.76±24.5ns	27.31±8.07**	16.87±24.6ns
[i]			-13.84±4.89**			-13.66±4.90**	-3.75±9.35ns		-4.20±9.35ns
[j]				-10.03±7.59ns		-9.41±7.59ns		-8.66±7.61ns	-8.80±7.61ns
[l]					-26.23±8.53**		-20.66±16.3ns	-25.65±8.55**	-19.40±16.3ns
χ^2	13.74**	10.95**	5.33(*)	9.20 *	1.50ns	1.41ns	1.34ns	0.20ns	...

B) using P_1, P_2, F_2, F_3, BCS_1 and BCS_2

m	111.54±0.51**	108.11±1.40**	113.21±2.18**	107.99±1.40**	105.11±1.94**	113.19±2.18**	114.70±4.99**	104.79±1.95**	114.13±5.01**
[d]	3.63±1.32**	3.37±1.33 *	3.07±1.33 *	1.64±1.94ns	3.19±1.33 *	1.11±1.95ns	3.07±1.33 *	1.14±1.95ns	1.14±1.95ns
[h]		13.39±5.08**	-1.68±7.10ns	13.86±5.09**	39.82±12.9**	-1.48±7.10ns	-10.45±27.3ns	41.97±13.0**	-7.04±27.5ns
[i]			-8.38±2.76**			-8.57±2.76**	-9.60±4.61 *		-9.34±4.61 *
[j]				13.04±10.6ns		14.66±10.6ns		15.38±10.7ns	14.46±10.7ns
[l]					-49.82±22.3 *		12.38±37.3ns	-52.83±22.4**	7.84±37.4ns
χ^2	18.13**	11.16 *	4.99(*)	9.65**	6.18 *	0.04ns	1.84ns	4.10 *	...

C) using P_1, P_2, F_1, F_2, BC_1, BC_2, F_3, BCS_1 and BCS_2

m	110.87±2.47**
[d]	2.71±1.81ns
[h]	10.82±10.8ns
[i]	-6.32±2.68 *
[j]	-2.89±7.14ns
[l]	-14.84±10.1ns
χ^2	5.74ns

Table 106. Estimates of gene effects for AUDPC in cross Briscard x Ruapuna

A) using P_1, P_2, F_1, F_2, BC_1 and BC_2

m	94.15±0.82**	93.05±1.38**	105.38±4.05**	93.02±1.38**	91.63±1.55**	105.35±4.05**	117.60±9.01**	91.61±1.55**	117.85±9.02**
[d]	5.607±1.34**	5.54±1.34**	5.44±1.34**	5.12±1.55**	5.43±1.43**	5.02±1.55**	5.54±1.34**	5.05±1.55**	5.05±1.55**
[h]		2.58±2.62ns	-13.86±5.71 *	2.59±2.62ns	15.25±6.70 *	-13.85±5.71 *	-46.18±22.0 *	15.17±6.70 *	-46.93±22.1 *
[i]			-14.27±4.41**			-14.27±4.41**	-25.67±8.87**		-26.24±8.88**
[j]				3.38±6.19ns		3.38±6.12ns		3.03±6.20ns	3.92±6.21ns
[l]					-14.25±6.94**		21.21±14.0ns	-14.16±6.94 *	21.71±14.0ns
χ^2	14.15**	13.18**	2.58ns	12.88**	8.96**	2.41ns	0.40ns	8.72**	...

B) using P_1, P_2, F_2, F_3, BCS_1 and BCS_2

m	97.75±0.53**	93.46±1.25**	93.92±2.32**	93.52±1.25**	91.56±1.55**	93.91±2.32**	77.97±5.17**	91.61±1.55**	77.63±5.18**
[d]	4.32±1.21**	4.17±1.21**	4.17±1.21**	5.15±1.55**	4.06±1.21**	5.14±1.55**	3.88±1.21**	5.05±1.55**	5.05±1.55**
[h]		17.67±4.67**	16.29±7.51 *	17.57±4.66**	38.58±11.0**	16.38±7.51 *	109.67±28.1**	38.53±11.0**	111.95±28.2**
[i]			-0.64±2.71ns			-0.55±2.71ns	13.58±4.93**		13.98±4.94**
[j]				-10.12±9.94ns		-10.05±9.95ns		-10.21±9.94ns	-12.13±9.96ns
[l]					-44.15±21.1 *		-132.39±38.3**	-44.26±21.1 *	-135.16±38.4**
χ^2	27.81**	13.46**	95.66**	12.42**	9.07**	12.38**	1.48ns	8.02**	...

C) using P_1, P_2, F_1, F_2, BC_1, BC_2, F_3, BCS_1 and BCS_2

m	97.95±2.27**
[d]	4.53±1.47**
[h]	4.86±9.44ns
[i]	-5.23±2.45 *
[j]	0.34±5.89ns
[l]	-10.98±8.42ns
χ^2	24.64**

Table 107. The components of variation in six generations of wheat for the area under the disease progress curve (AUDPC) inoculated with stripe rust pathotype 106E139A-

Cross	D	H	F	E_w	$(H/D)^{1/2}$	$F/(D*H)^{1/2}$
Briscard x Tiritea	102147	33838	-63994	4831	0.58	1.09
Ruapuna x Tiritea	61513	43646	-40904	5365	0.84	0.79
Domino x Tiritea	61683	114560	-69336	27225	1.36	0.83
Otane x Tiritea	87609	179563	-122312	11800	1.43	0.98
Briscard x Otane	6554	22781	-11101	1885	1.86	0.91
Ruapuna x Otane	9173	9173	+69	6473	0.26	0.03
Domino x Otane	8045	13109	+7129	3830	1.28	0.69
Briscard x Domino	7145	3267	-1058	1337	0.68	0.22
Ruapuna x Domino	15716	573	-1609	3110	0.19	0.54
Briscard x Ruapuna	2121	369	+109	963	0.42	0.12

CHAPTER 5

DISCUSSION

A diverse germplasm was used for these experiments to improve the inference base of the results. Also unrelated parents may lead to transgressive segregation for resistance, e.g. for latent period in wheat leaf rust Lee and Shaner (1985b) and Northern corn leaf blight (Hughes and Hooker, 1971). Evidence from these results indicates success in obtaining higher level of resistance in a breeding programme by using diverse germplasm as transgressive segregation was observed. Therefore the present results have a considerable degree of generality.

5.1. Cultivar experiment

In an analysis of variance to compare the resistance of several host cultivars against several races, a significant mean square for cultivars implies that the cultivars differ in general resistance and a significant mean square for the interaction between cultivars and races implies that the cultivars differ in specific resistance (van der Plank, 1968a). This interaction can be caused by variation in the magnitude of the differences between cultivars or by reversals in the ranking of the cultivars with different races. The analysis of 15 cultivars over three pathotypes (Table 2) showed the presence of genetic variation for all components of resistance which agreed with previous results (Cromey, pers. comm.). Significant interactions between cultivars and pathotypes occurred for all characters. These interactions were caused largely by Oroua, Pegasus, Sapphire and Batten. Pathotypes were highly significant for all traits, indicating components of resistance were affected by pathotypes, and that pathotypes were different in aggressiveness on these cultivars. So this is obvious that due to different cultivars and pathotype, the interaction between them should be significant. The significance of the interaction of a set of host and pathotypes indicates the operation of specific resistance (van der Plank, 1968a). It should be mentioned that this is not always true. Knott (1988) presented a hypothetical model with four genes, all having equal and additive effects and four genes for aggressiveness. There was an interaction mean square, but if the effect of each gene was reduced to 10% so that the range was only from 10 to 90% severity, then the interaction mean square was zero.

van der Plank (1978) stated that resistance was horizontal if a set of host genotypes gave constant rankings when tested with different pathogen genotypes. In the case of infection type (Table 3), there was great variation in the ranking of Batten, Pegasus, Sapphire, Oroua and Karamu whereas there was constant ranking for Tiritea, Takahe and Otane. Other cultivars showed nearly a constant ranking. Apart from Batten and Pegasus on which pustules did not form due to action of specific genes, the cultivar rankings for latent period, pustule size and pustule density (Tables 4-6) was similar to that of infection type.

The aggressiveness of the three pathotypes were different on these cultivars. In the case of infection type (Table 7) the most and least aggressiveness pathotype were 232E137A- and 106E139A-, respectively. This was also true for latent period (Table 7) regardless the artificial values for Batten (in response to 111E143A- and 106E139A-) and Pegasus (in response to 106E139A-). For pustule size (Table 7), pathotype 111E143A- produced big pustule whereas pathotype 232E137A- produced small pustules regardless of the artificial values for Batten and Pegasus. For pustule density (Table 7), pathotype 111E143A- produced high pustule numbers on the leaves and pathotype 106E139A- was less aggressive. These results suggest that the components of resistance have some independence in expression. It can also be interpreted that these differences are due to sampling error. However, because these experiments were carried out at three different times (over 2 months period), the environment could probably affect all components and for that reason there were significant differences among means of pathotypes for each trait.

The definition of full and restricted heritabilities is presented in Materials and Methods. For each trait and the difference between full and restricted heritabilities were small. Full heritability can be more important than restricted heritability, because the pathotype was significant and should be regarded in heritability (Table 8).

Genotypic correlation is used by breeders to understand how correlated traits occur, and can be used to indicate the effects of indirect selection and in selection indices. Selection for the character with the higher heritability would be more successful than selection for that with a lower heritability, assuming these two characters are correlated. In breeding work, it would be best to base selection on all components of

resistance.

In the present study, there were highly significant positive genetic correlations amongst infection type, pustule size and pustule density and highly significant negative genetic correlation between latent period with the other components (Table 9). The correlations between traits of each pathotype with each other were almost not significant (Table 10). It is expected that if there are different pathotypes, the correlation between two traits from two pathotypes would be low. If the correlation is high, it might be due to sampling error.

The present study shows that phenotypic correlations were similar to the genetic correlations in direction, but different in magnitude. It was obvious that genetic correlations in general were higher than the corresponding phenotypic correlations. If the two types of correlation are similar to each other, the selection on phenotype will also be a fairly accurate selection of genotype.

It should be noted that there are some limitations in the determination of a genetic correlation. Firstly, estimates of genetic correlations from variance and covariance components are usually subject to rather large sampling error and are therefore seldom very precise. Secondly, genetic correlations are strongly influenced by gene frequencies, so they may differ markedly in different populations (Falconer, 1981). Therefore these results must be regarded as estimates for this population. Thirdly, the genetic correlation will be larger in absolute magnitude than the phenotypic correlation. This results from the very low value of genetic variance component (Baker, 1986). A large value of genetic correlation resulted from this experiment and was in agreement with this discussion.

In support of these experiments, many workers have reported the correlations among components of resistance. There was a negative correlation between infection type and latent period (see Jacobs and Broers 1989; Broers, 1989; Parlevliet and Kuiper, 1977), latent period and pustule size (Kuhn *et al.*, 1980; Lee and Shaner, 1985a; Johnson and Wilcoxson (1978), and latent period and pustule density (Johnson and Wilcoxson, 1978).

In this experiment, high correlations among all components of resistance were observed which is in agreement with other workers mentioned above (also Johnson and Wilcoxson, 1978), and only Denissen (1991) reported that in wheat/leaf rust the correlation between infection frequency and latency period was low. The moderately high genotypic correlation demonstrated that the genes for resistance that were studied may affect one or more of component of resistance such as pustule density, pustule size and length of latent period (see Roelfs, 1988). The selection for one of them would have tended to select the various components of resistance. This was also suggested by Parlevliet and van Ommeren (1975), Ohm and Shaner (1976) and Johnson and Wilcoxson (1978). Selection for long latent period should not be difficult and would tend to select for other components of resistance as well.

In this study, pustule size and pustule density were measured rather than spore production, as the latter is difficult to measure. Spore production is often estimated by the size of the uredium, assuming a close association between spore production and uredium size (Parlevliet, 1985). The variation in latent period, pustule size, and pustule density was clearly but not completely associated. For race-specific cultivars, the variation in pustule size and pustule density was almost completely associated with infection type i.e. the lower the pustule size and pustule density the lower the infection type. It is in agreement with Dehne (1977, quoted by Parlevliet, 1979) who studied these components against wheat stripe rust. But for non-specific cultivars the infection type was not important and cultivars with the same infection type were different in latent period, pustule size and pustule density. These results show that longer latent period, a reduced pustule density and a smaller pustule size tended to go together which is in agreement with Ohm and Shaner (1976) and Parlevliet (1975). Genotypic correlations showed that same gene(s) might partially control these components of resistance. According to correlations between these components of resistance, many workers have reported (in other host and rust) that genes controlling these components of resistance were partially, but not completely, associated (Kuhn *et al.*, 1980; Parlevliet, 1975; Ohm and Shaner, 1976), or linkage between or possible pleiotropic effects of genetic factors controlling these components was suggested (Ohm and Shaner, 1975; Parlevliet, 1986), or they might be controlled by some genes in common (Lee and Shaner, 1985a), but these components might possible be inherit independently (Broers, 1989). Finally, it can be suggested that it is possible to select for these components of resistance in

segregating populations.

In this study differences between latent period in response to different pathotypes can be interpreted genetically rather than being affected by environment. This conclusion agrees with Fuches (1965) who stated that the generation time, i.e. time between date of inoculation and date of sporulation, can be race-dependent (quoted by Stubbs, 1988) who grouped races into slow, normal, and fast, the difference between the first and last group being 4 days.

Resistance has so far been discussed on the basis of attributes taken one at a time. ANOVA only gives a separated analysis of variables, it does not examine the joint relationship between variables. Since all those components act in an epidemic simultaneously and are correlated, it is worthwhile to look at cultivars across all attributes. Also the ranking of cultivars, using all attribute together, cannot be determined by simply measuring the magnitude of the attribute of resistance in a experiment. Such experiments are necessary to measure the attribute themselves, but the effect of attribute overal are difficult to rank cultivars, because these components interact with each other. Multivariate analysis uses variance and covariance matrices to handle multiple characters. In this experiment the results showed significant differences between cultivars (Table 11).

Discriminant analysis (one for each pathotype) was then used to find linear combination of the variables that maximise differences among the genotype as a population (Table 11 & 12). With the use of discriminant analysis following MANOVA, the complex interrelationship among components of resistance can not only be revealed, but also can be taken into account in statistical inference, while ANOVA ignores and fails to exploit these interrelationships. The main purpose of discriminant analysis is to find a linear combination of variables that maximises differences among the hypothesis clarification (genotypes), and to sort the objects into their appropriate groups with minimal error (Bryant and Atchley, 1975). Regarding discriminant analysis for all attributes, most of the variation across characters was explained by the first function of discriminant analysis, and it also could be used in grouping the cultivars. The results obtained in this study demonstrated that the technique of discriminant analysis was useful for grouping and sorting cultivars.

Another method of multivariate analysis such as cluster analysis was used for analysing multiple characters. Pattern analysis comprises mathematical methods of classification and ordination which have been widely applied in agricultural science (Williams, 1976) and which could be used to more advantage in plant pathology (Thompson and Rees, 1979) and plant epidemiology (Kranz, 1974a,b). Numerical taxonomic methods have been useful in differentiating groups of cultivars with very different rust reactions. Analysis of several thousand cultivars, as assessed for disease reactions in some plant breeding programmes, is now feasible by this cluster analysis approach. Cluster analysis have been useful in differentiating groups of cultivars with very different rust reactions. Group 1 was susceptible to all races but group 2 was susceptible only in the seedling test (Fig. 10). Both groups can be used for resistance, because in experiment five and seven the cross of Tiritea x Otane showed transgressive segregation for infection type, latent period and area under the disease progress curve, indicating that such cultivars may possess a non-specific types of resistance. Intercrossing such cultivars and selecting for transgressive resistant segregates would be a good breeding strategy. Group 3 and 5 could be used directly or as parent. According to other experiments and the literature (see Materials and Methods), they showed adult plant resistance and some of them also have known Yr-genes (see Materials and Methods). Other groups have showed the presence of specific resistance gene. They showed specific reaction, so that Oroua (Yr7), Pegasus (Yr1,6), Sapphire (Yr6,7) and Batten (Yr,9) were placed in different groups regarding all attribute whereas with respect to separate components maybe some groups were mixed. It should be mentioned that although Karamu (YrA) was placed in the adult plant resistance group but with some assessment in adult stage and other pathotypes it should probably be placed in other groups. Pattern analysis has been used by few workers in plant pathology area and it was proved to be useful, for instance Syme and Thompson (1986), Rees *et al.* (1979a,b). Thompson and Rees (1979) indicated that the pattern analysis is suited to the analysis of large-scale screening experiments for slow rusting in wheat genotypes and should prove of value with similar epidemiological data for other plant diseases. Pattern analysis provides a valuable complement to other epidemiological methods, by extracting and displaying the main patterns and trends in multivariate data often enables one to obtain new perspectives of the problems under consideration.

To obtain information on the relationships among the several varieties studied,

canonical variate analysis has been employed to separate pathotypes with respect to all components (Table 11 & 12). Canonical variate is a multivariate statistical research tool capable of identifying differences among groups of individuals (or treatments) and improves the understanding of the relationships among several varieties measured within those groups. Canonical variate analysis can be defined as a multivariate statistical technique which determines linear functions of quantitative varieties that maximally separate two or more groups of individuals while keeping each group as compact as possible (Manly, 1986). The canonical discriminant analysis generated a linear model that provided an indication of the differences between the pathotypes on fifteen cultivars of wheat. Canonical discriminant analysis summarized the complex relationships of the data and provided a useful method of reducing the dimensionality of the problem. Canonical discriminant analysis has been successfully employed to find differences between groups, in this experiment pathotypes, on the basis of linear combinations that can depend the understanding of the system under study.

In the present study, following PROC CANDISC the multivariate statistic and F approximation showed that a clear difference between the pathotype mean vectors (all four variables put together come from fifteen cultivars) at P=0.0001 level (Table 11 & 12). The first eigenvalue, 0.63, was much larger than the second one indicating separation between the pathotypes can be accounted for along the first canonical variate. The correlations between the canonical varieties and the original variables indicated that both latent period and pustule size measurements are positively correlated with the first canonical variate, CAN.1, while infection type and pustule density showed a negative association with CAN.1. Since CAN.1 accounts for almost all the separation between the species, it can be concluded that, individually, each of the two variables, latent period and pustule size, contributes to the separation of pathotypes whereas infection type and pustule density tend to bring the pathotype together. The plot of the canonical scores shows the power of the first canonical variate in separating the pathotypes. It is obvious that all pathotypes are quite different from each other. The first canonical discriminant function gave the maximum possible variation between groups with respect to within group variation, and therefore reflects group differences to the greatest degree possible; the second canonical discriminant function captured as much as possible of the group differences not displayed by the first canonical discriminant function, subject to the condition that there is no correlation between two canonical

discriminant functions. The first two canonical varieties were sufficient to account for most of the important pathotype differences with respect to all components. To have a visual display of the groups and to illustrate the discriminating power of each canonical variate in separating the groups and also to identify outlier in the data, it is worthwhile to plot these scores for the first two canonical varieties (Fig. 8).

Several attributes (an extended latent period, small size of pustule and low number of pustule on the leaves) contributed to components of resistance. However, not all cultivars that rusted slowly in this study differed from cultivars that rusted rapidly, as far as individual components of resistance were concerned. Variation within each of the components studied was observed and it should be possible to select components of resistance and develop lines of wheat that possess them in desired combinations. Possibly latent period may be the easiest component to study and to use in a practical way. Although Parlevliet (1988) suggested that infection frequency and spore production may be the most important components of partial resistance to stripe rust. Park and Rees (1989) reported that in response to stripe rust, lower infection type was observed with longer latent periods and/or a lower percentage of leaf area affected on primary leaves of adult plant resistant cultivars. As it will be seen later the components of resistance are quantitative traits (also see Shaner *et al.*, 1978) and within a large group of wheats, these components exhibit continuous variation (Ohm and Shaner, 1975). Because the various quantitative components of resistance act together to affect the course of an epidemic, it is necessary to understand the contribution of each component to the overall effect. As stated earlier, longer latent period, smaller pustule size, and fewer pustules per unit area all play strong roles in retarding disease development.

5.2. Diallel mating design

Knowledge of the mode of inheritance or genetic architecture permits an overall assessment of the probable effects of selection on any generation or population and the probable frequency of specific phenotypes in a population. When specific crosses are being considered, the actual parameter values can be used to predict the response to selection and to estimate the frequencies of recombinant inbred phenotypes. Amongst all mating designs diallel mating designs provide a simple and convenient method for estimating genetic parameters. The major purpose of a diallel cross is the detection and

estimation of additive and dominance variances. It also provides a great deal of genetic information from relatively few parents. As Jinks (1954) stated diallel analysis is a powerful method for obtaining a rapid, overall picture of the genetical structure of a large number of parental lines i.e. overall degree of dominance, of the relative dominance properties of the parents. Different forms of diallel may be found in the literature but among the various forms, the half-diallel technique has certain advantages over the others, giving maximum information about genetic architecture of a trait, parents and allelic frequency (Kearsey, 1965).

The assumptions underlying many genetical systems are: (1) diploid segregation, (2)no difference between reciprocal crosses, and (3) independent action of non-allelic genes (Hayman, 1954b). In the diallel cross, other assumptions are included such as no heterozygosity, no multiple allelism, and no correlation between the gene distribution at each locus. This last condition is not violated if several gene differences exist between the same parents. The analysis treats the corresponding group of genes as if it were a single gene (Hayman, 1957). Almost assumptions were met in these experiments. It has been known that wheat has diploid segregation, and from the literature there were no differences between reciprocal crosses, and all parents were homozygous. Only in some cases there was epistasis which could not meet the assumptions. But it should be noted that although the genetic assumptions may lack validity and despite the deficiencies of the Hayman-Jinks technique, the use of diallels in plant breeding cannot be overlooked. They provide a systematic approach to experimental analysis and also give an overall genetic evaluation which makes identification of crosses with the best selection potential possible in an early generation, especially with the combining ability types.

The models for numerical analysis of a full diallel with or without reciprocal effects are given by Jinks (1954) and Hayman (1954b). It should be noted that the half-diallel in these experiments were analyzed by adopting the least square estimates for a full diallel without reciprocal effects as given by Jinks (1954) and Hayman (1954b). In this case an adjustment for the error components should be made. If a half-diallel is produced and analyzed using the least square estimates as originally derived by Jinks (1954) for a full diallel without reciprocal effects, the dominance components (H_1 and H_2) will be found to be affected severely when the analysis of the data is performed according to a full diallel without reciprocal effects. Consequently, upward and biased

estimates of the dominance components are obtained. This, in turn, will affect the degree of dominance and the proportion of dominant and recessive genes among the parents.

In these diallel experiments, no standard errors of parameters were presented, but it should be noted that diallel analysis does not allow an estimate of the standard errors of the individual statistics (Jinks, 1954).

Biometrical analyses of the diallel for the infection type, latent period, pustule size and pustule density in response to three pathotypes 106E139A-, 111E143A- and 232E137A- and for the area under the disease progress curve in response to pathotype 106E139A- showed that additivity was of major importance in conditioning those traits, because the D components was relatively large for most of the traits.

In the specific diallel, for pathotype 111E143A-, all traits showed additivity whereas for pathotype 232E137A-, additivity occured only for infection type with dominance of major importance for other components (Table 26 & 27). Maybe it is expected because the first pathotype was virulent on all cultivars except Batten and the additive components was the major part, whereas the second pathotype was not virulent on Pegasus, Oroua and Sapphire and dominance should be of major importance.

In the nonspecific diallel, for pathotypes 106E139A- and 111E143A-, additivity was of major importance while for pathotype 232E137A- only pustule density showed additivity and for the other components of resistance dominance was important (Tables 61-63). Because so far there was not any known Yr gene(s) in these cultivars, it is important that in certain cultivars the mode of gene action changed by using different pathotype. This means there was interaction between genes in the cultivars with those in the pathotype. If a single gene controlled the resistance, there would be much interaction between the genotype and the pathotype, but if resistance was controlled by polygenes, the interaction would not be great. Differential interaction of partial resistance was reported with Parlevliet (1977a), but he mentioned that this type of resistance is under polygenic control and has been quite stable.

In the nonspecific field diallel, additivity was ten times greater than dominance for area under the disease progress curve (Table 91). Although the assessment was

different from those in the seedling stage, additivity was so important and selection procedures can be used to select genotypes with higher resistance.

It should be noted that the preponderance of additivity found in these experiments did not necessarily indicate that dominance was lacking. Additivity is the variance of average allele effects and therefore does not mean 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 overdominace, have at least half of their effect estimated as additive (Mather and Jinks, 1982). Even for known Yr gene(s), which showed partially dominance, most of their effect was calculated as additive action.

The narrowsense heritabilities were high or moderately high for all traits, indicating the selection for the resistant genotypes will be effective. This agreed with the importance of additivity. In some cases the narrowsense heritability was greater than their respective broadsense heritability. This is due to negative estimates of H, particularly H_2. Both formulae are identical except for a difference in the numerator coefficient of the H_2 (see Materials and Methods). Hence, when H_2 is negative it causes a greater increase in the numerator of formula estimating H^2_{NS} than for H^2_{BS}, resulting in narrowsense heritability being greater than broadsense heritability.

In the specific diallel, for two pathotypes all narrowsense heritabilities were less than broadsense heritabilities (Table 26 & 27). In the non-specific diallel, the results were the same as for experiment two, except for pustule size in response to pathotype 111E143A- (Tables 61-63). In the nonspecific field diallel, there was a high heritability and broad sense heritability was greater than narrow sense heritability (Table 91).

Negative estimates of H were calculated for some analyses, indicating that dominance was trivial. In the specific diallel and nonspecific field diallel, all H values were positive (Table 26 & 27). In the nonspecific diallel, all H values were positive except for infection type and latent period in response to 106E139A- and for infection type and pustule size in relation to 111E143A- (Tables 61-63). Variance components are conceptually positive, but negative estimate variance components are common in research because of sampling distribution. Possible causes of negative estimates have

been discussed (Searle, 1971; Reeder *et al.*, 1987). These include: inadequate sample size in the experimental design, recording or computation errors, or the invalid assumption of uncorrelated identically distributed random variables. There is also an inherently small probability that the difference between mean squares will be negative, even though the difference of the expected values is positive, because of the sampling distribution of mean squares.

Where positive values of H_1 occurred estimates of the degree of dominance $(H/D)^{1/2}$ were calculated. In the specific diallel, for all components in response to pathotype 111E143A-, partial dominance was present in conditioning resistance (Table 26 & 27). This degree of dominance can be reliable because there was no epistasis. However it is important to realise that in the presence of epistasis, the degree of dominance will be biased upwards (Hayman, 1954b; Jinks, 1954). Correlated gene distribution can also bias the dominance upwards and could also be responsible for inflating the apparent degree of dominance. For pathotype 232E137A- only for infection type, partial dominance was present; for the pustule density dominance and for the latent period and pustule size overdominance were present. Because of epistasis for all attributes, the degree of dominance might be quite precise. In the nonspecific diallel, for almost all components in response to the three pathotypes, partial dominance conditioning of stripe rust resistance and almost all degrees of dominance were reliable (Tables 61-63). In the nonspecific field diallel, partial dominance controlled the area under the disease progress curve (Table 91) and because of the presence of epistasis, maybe the degree of dominance was not valid.

In the specific diallel, in response to pathotype 111E143A-, infection type, latent period, pustule size and pustule density, across all cultivars, were controlled by 1, 1-2, 2-3, and 3-4 genes, respectively (Table 26 & 27); and in the case of pathotype 232E137A-, it was controlled by 1, 2-3, 1-2 and 1 genes for infection type, latent period and pustule size and pustule density, respectively. In the nonspecific diallel, in reaction to pathotype 106E139A- 1, 1-2, 2-3 and 1 genes, in response to pathotype 111E143A- 46, 3-4, 2-3 and 2-3 genes, and in case of pathotype 232E137A- only single genes controlled infection type, latent period, pustule size and pustule density (Tables 61-63). Probably, these differences might be due to differences in virulence genes between pathotypes. In the nonspecific field diallel, stripe rust resistance was controlled by 2-3

genes (Table 91). It can be interpreted that different genes conditioned resistance in response to each pathotype. Even in response to a certain pathotype, different gene(s) controlled components of resistance. It should be noted that in a diallel cross, the number of effective factors is not as clear. Firstly, our estimate must of necessity come from the H statistics which are undoubtedly inferior to those obtained from D statistics. Secondly, any deviation from a random association of gene differences throughout the parental lines leads to a further minimizing of the estimate (Jinks, 1954). The number of effective factors involved in resistance to stripe rust was estimated for these traits which had positive values of H_2. Unfortunately the number of effective factors rarely equals gene number (Mather and Jinks, 1982) and it must be used with caution. However, the estimate of number of genes can give us certain pattem of effective factors.

In the presence of unequal gene frequencies the sign and magnitude of the F value can be used to determine the relative frequencies of dominant to recessive alleles in the parental population and the variation in the dominance level over loci. The value of F will be positive whenever the dominant alleles are more frequent than the recessive alleles, irrespective of whether or not the dominant alleles are increasers or decreases. The dominant alleles were more frequent than the recessive alleles in the specific diallel, nonspecific diallel and nonspecific field diallel for almost all attributes.

In some cases there was a lack of agreement between the analysis of variance (W_r+V_r) and the results of significance tests of departure of the regression slope from unity. For example in the specific diallel, there was lack of agreement for pustule size and pustule density in response to pathotypes 111E143A- and 232E137A-, respectively (Table 26 & 27). In the nonspecific diallel, there was lack of agreement for infection type and pustule density in response to pathotype 106E139A- and for infection type and latent period in reaction to pathotypes 111E143A- and 232E137A- (Tables 61-63). Also in the nonspecific field diallel, there was agreement between the analysis of variance (W_r+V_r) and the result of the significance test of departure of the regression slope from unity (Table 91). It can be concluded that epistasis and/or correlated gene distribution were present, although they were relatively unimportant. Mather and Jinks (1982) noted that the 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

statistic with presumably some bias.

Analyses differed in their sensitivity for detecting epistasis and/or correlated gene distribution. Results of analysis of variance of (W_r-V_r) indicated that there was non-additivity in some cases, whether in the glasshouse diallel or in the field diallel. However, in some cases there was a lack of agreement between these results and those of significance tests of the departure of the regression slope from unity. It was concluded that epistasis and/or correlated gene distributions were present, although they were relatively unimportant.

The W_r+V_r,P correlation coefficients were generally high and significant for most characters. It can be concluded that there was directional dominance in the most of the characters conditioning stripe rust resistance.

The essential points to be gained from the graphical analysis are : 1) the average dominance from the distance between the origin and W_r-intercept of the regression line, 2) the relative proportion of dominant and recessive genes in the parents from the distribution of their respective array points along the regression line of unit slope, and 3) a measure of genetical diversity among the parents from the distance between array points. To test for adequacy of the additive-dominance model, an analysis of variance of W_r-V_r and the regression of W_r on V_r can be carried out. Provided the these analyses show that epistasis or correlated gene distributions are absent, then graphical analysis of W_r/V_r can indicate the distribution of dominant and recessive genes amongst the parents. The above interpretations of the W_r/V_r graph are possible only when a simple additive-dominance model of gene action provides an adequate description of the data. In the presence of non-allelic interactions, the most useful information to be gained is about the existence of such interactions, since the graph itself, being sensitive to interactions, often permits their detection. In almost all cases there was no non-allelic interaction ansiveness are not absolute attributes of a host plant, but are only the expression of its specific interaction with a certain pathotype. The reversal mode of gene action has been reported by other workers (see Lupton and Macer, 1962). In the specific diallel the main aim was to obtain an estimate of the dominance effect of the Yr-genes. In response to pathotype 111E143A- for infection type most cultivars contained Yr-genes have been considered recessive in relation to the Batten contained Yr9. Reversal gene

action was obtained by testing the same genotypes with pathotype 232E137A-, i.e. most cultivars containing Yr-genes have been considered dominant in relation to the Batten. Details are discussed as follows:

In the specific diallel, for all characters, all cultivars showed reversal of dominance in response to two pathotypes (Fig. 11-14). Reversal domiance is expectable because specific gene(s) for resistance was present in all cultivars, except for Tiritea. Although Tiritea is susceptible to two pathotypes, it showed dominance to pathotype 232E137A- and recessiveness to pathotype 111E139A-.

In the nonspecific diallel, in the case of infection type almost all cultivars, except Briscard and Otane, showed reversal of dominance (Fig. 15). Briscard and Otane showed constant recessiveness and dominance to all pathotypes, respectively. In the case of latent period, Otane showed dominance against all pathotypes and the rest of cultivars had reversal of dominance (Fig. 16). In the case of pustule size, only Tiritea and Briscard showed no reversal dominance (Fig. 17) and for pustule density Briscard showed constant recessiveness against all pathotypes (Fig. 18).

In the nonspecific field diallel, there was no reversal dominance because only one pathotype was used. Even by using the F_1 and F_2 the positions of the cultivars did not change on the graph (Fig. 24). All adult plant resistant cultivars showed recessive in the seedling stage d the interpretations of the W_r/V_r graph can be reliable. Also it has been demonstrated that gene dispersion and association cause the W_r/V_r graph to deviate from a straight line of unit slope in characteristic ways, which have a superficial similarity to the effects of complementary and duplicate interactions, respectively (Mather, 1967). Thus, it becomes difficult to discriminant between these two possible phenomena affecting rectilinearity of the W_r/V_r relations. Furthermore, the effects of duplicate interaction and gene association can be quite small causing no detectable departure from the expected linear regression of unit slope. Hence no deviation from the slope of one leads to an inaccurate conclusion regarding the mode of inheritance of a metrical character under investigation.

Genetic diversity among parents was demonstrated by the scatter of the parental array points along the regression line of the W_r/V_r analysis. The analysis of the graphical

statistic in both F_1 and F_2 provided detailed information on the interrelations between the parents in each of the two diallels. The F_1 W_r/V_r regression analysis used in this study has differential sensitivity in the detection of the various types of non-allelic interaction (Jinks, 1954). The F_2 diallel overcomes this difficulty and is therefore desirable for obtaining more reliable information on underlying genetic mechanisms. In all experiments which the F_1 and F_2 were used the results of both diallels were similar. It can be interpreted that the F_1 diallel gave reliable information upon diallel analysis.

Based on analyses of the W_r/V_r graphs for all characters, reversal of mode of gene action was observed by using different pathotypes. Dominance and reces(glasshouse) whereas in the adult stage (field) they showed dominance. Changes in dominance where a gene behaved as recessive in the seedling stage and as a dominant in the older plant was also reported by Favret and Vallega (1953) and Hooker (1967).

There were indications that the W_r/V_r graphs tend to concave upwards for some characters. This was based on the V_r values which were always greater than W_r values. Mather (1967) stated that in diallel crosses, the W_r/V_r graph characteristically concave upwards with complementary, and concave downwards with duplicate interactions. Further, the points tend to cluster at the right or upper end of the line with the complementary, and at the left or lower end of the line with the duplicate relationship.

One possible conclusion is that the reversal of dominance could be due to a higher level of inoculation pressure between experiments or environmental differences. While these experiments were carried out at different times, an attempt was made to have same conditions.

Two kinds of combining ability estimates can be made. The general combining ability describes the average performance of a line in hybrid combinations, which contains mainly additive effects was found to be the major component of variation, although significant specific combining ability, which is a measure of the deviation of crosses from the value expected on the basis of the performance of the parents, is composed of dominance plus interallelic interaction or epistasis variance was present in all cases (Tables 28-29, 64-66 & 92). In all cases the ratio proposed by Baker (1978)

was close to unity, suggesting that additive effects were more important than nonadditive effects for resistance to stripe rust. Judging by the ratios expressing the relative importance of general combining ability and specific combining ability, additive variance was of major importance. This signifies that progeny performance can be effectively estimated on the basis of general combining ability effects, as reaction to the disease appears to be rather uniformly transmitted to all offspring. Line selection in a crop improvement programme should be successful in developing more resistant lines.

The general combining ability, a measure of additive gene action, was highly significant and accounted for a high proportion of the gene action. The specific combining ability, a measure of nonadditive (dominance and epistasis) gene action, was significant. The predominance of general combining ability is reflected in the high estimates of heritability for all traits. However, the mean square for the general combining ability was much greater than that of the specific combining ability. These resistance genes could be manipulated in a breeding programme because of a high level of additive gene effects and high heritability. This experiment was in agreement with other reports. Krupinsky and Sharp (1978) reported the presence of a high value of general combining ability in their diallel for wheat. Kim and Brewbaker (1977) studied the inheritance of slow rusting in corn by means of diallel and generation mean analysis of crosses among 11 inbred corn lines in severe epidemics of *Puccinia sorghi*. They reported that there was significant general combining ability for slow rusting, no heterosis, specific combining ability effects were small, though significant, and broad sense and narrow sense heritabilities were 83% and 47%, respectively. Diallel analysis can estimate general and specific combining abilities that predict which are the best parents and best crosses to use.

The high percentage of additive genetic variance, the low percentage of nonadditive genetic variance, and the high heritability from the second pathotype agree with the same parameters from the first diallel. In this regard Krupinsky and Sharp (1978) reported the presence of additive gene action and high heritability (99% and 90-92% for broad sense and narrow sense, respectively) in minor gene lines. It should be noted that the preponderance of additivity found in this study does not necessarily indicate that dominance was lacking. Additivity is the variance of large 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).

The more useful diallel analysis is probably that involving general and specific combining abilities (Griffing, 1956). 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. Total genotypic variance is equal to $2\sigma^2_{gca} + \sigma^2_{sca}$. Furthermore, assuming no epistasis and a random mating population, the general and specific combining abilities will be functions of additive and dominance variances, respectively. Combining abilities may be related to the genetic components estimated in the present study as follows (Mather and Jinks, 1982) :

$$\sigma^2_G = 2\sigma^2_{gca} + \sigma^2_{sca} = 0.5D - 0.5F + 0.5H_1 - 0.25H_2$$

$$\sigma^2_{sca} = 0.25D - 0.25F + 0.25H_1 - 0.25H_2 - 0.25D_R$$

$$\sigma^2_{gca} = 0.25H_2 - 0.25H_R$$

where D_R and H_R are the random mating (u= v = 0.5) forms of D and H and epistasis is assumed to be absent.

It is pertinent to note several advantages which the combining abilities analysis (Griffing, 1956) has over graphical analysis (Mather and Jinks, 1982). 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 (1982) in which absence of epistasis is an important assumption. However, epistasis occurs widely (Hayman, 1958b) 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, 1982). 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 (Mather and Jinks, 1982).

The Mather and Jinks diallel analysis is dependent upon six assumptions (Hayman,1954b). Gilbert (1958) claimed there were very few cases where these

assumptions imposed by the diallel cross were actually met in a practical breeding situation. Some of these assumptions are critical while others may be overlooked. Independent distribution of genes in the parents was considered the most important assumption for proper interpretation of results (Baker, 1978). The belief that no epistasis exists may often be false. Independent distribution of genes implies that the presence or absence of an allele at a particular locus is statistically independent of the presence or absence of an allele at any other locus. Failure of this assumption will result in an overestimation of the average level of dominance as derived from the genetical analysis (Hayman, 1954b). This may be caused by linkage of genes or from the effect of an insufficient number of parents (Baker, 1978).

It has been concluded that assuming a lack of epistasis cannot be justified if biochemical pathways are considered (Gilbert, 1958). The Hayman-Jinks test for epistasis, based on the W_r/V_r graphical analysis, is only reliable if there is an independent distribution of genes in the parents (Hayman, 1954b). Distortion to the W_r/V_r graph may also be caused by correlation between genes. The Hayman-Jinks method to remove arrays from the diallel table with the occurrence of epistasis until the test of the validity of the additive-dominance hypothesis is satisfied has been criticised. Gilbert (1958) considered that if a set of data contradicts the hypothesis, then it would be better to reject the hypothesis than attempt to correct the data to fit it. The idea of reanalysing the data is only an attempt to find the degree of additivity and dominance underlying the epistasis in the original set.

Results directly relevant to practical breeding can be determined from estimating general and specific combining abilities and their effects. This information is useful for evaluating the performance of hybrids or the potential of a hybrid breeding programme. There the combining ability diallel has the advantage that it is not subject to the restriction of the assumptions of the Hayman-Jinks diallel. The use of the Hayman-Jinks diallel analysis means that additional information can be obtained from the progeny as well as the parents. This includes : 1) dominance-recessive relations, 2) genic interactions, 3) probable linkage associations, and 4) number of effective factors. Other important information, including heritabilities can be obtained from both the Hayman-Jinks and combining abilities diallels. Even though Hayman-Jinks' analyses do provide extra information about the genetical systems of the plant materials studied, the Griffing

analysis provides sufficient information for the practical breeder. This is specially so in cases where epistasis and/or correlated gene distribution are present. It has been shown that the presence of correlated gene distribution has no effect on the general and specific combining ability estimates (Nassar, 1965). The presence of epistasis was included in the model as well (Matzinger and Kempthome, 1956; Griffing, 1956).

A good diallel comparison was presented by Arunachalam (1976) who compared and contrasted the combining abilities analysis and that of Mather and Jinks and concluded that the former provided all the information that a breeder will need from a diallel cross.

Both forms of diallel in this study agreed that additive gene action was predominant and that dominance, and epistasis involving dominance was minor. However, the F_1 diallel analysis has the important advantages of relatively short commitments of time and materials. An F_1 diallel analysis as used in this study is not as genetically informative as a generation means analysis (Mather and Jinks, 1982). However, it does highlight the relative importance of additive and dominance genetic variance and provide the estimates of narrowsense and broadsense heritability. The latter are of fundamental importance in estimating genetic advances in plant breeding programmes. The results were also supported by combining ability analysis where the predominant role of gene effects was established through the higher values of the predictability ratio. Thus, resistance in wheat may be improved through concentrating desirable genes through selection such as by pedigree or modified mass pedigree selection.

The parents in the diallel appeared to meet the necessary assumptions of diallel analysis such as a broad range of variability represented by the parental cultivars. Other assumptions of the diallel such as normal diploid segregation or bivalent behaviour during meiosis, and the absence of reciprocal or maternal effects are not suspected in wheat or from these results. Bias associated with linkage disequilibrium was expected to be minimal in this study, because data were generated from the homozygous parents and the F_1 generation. For these reasons parents and F_1's have been preferred to segregating generations to study certain aspects of quantitative genetics.

However it is important to note here that even though additivity predominates in most of the characters studied, this will often be true even when much dominance of the classical type exists. This is because the heritable portion of the continuous variation in quantitative genetic studies depends on genes which are transmitted in Mendelian fashion (classical type). These classical genetic genes are acting in polygenic systems where their effects complement one another. These effects sometimes act in simple additive fashion (additivity), but sometimes interact in such a way that the net effect is not the sum of the effects of individual genes.

The problem of epistasis has not been overcome. The type of epistasis can be more accurately determined by a generation means analysis (Hayman, 1958a, 1960b) but an accurate measurement of additive and dominance variance cannot be determined in the presence of epistasis. This has not been solved with the removal of arrays from the diallel table in the Hayman-Jinks analysis, so the results of generation means are presented thereafter.

5.3 Factorial mating designs

One purpose of this study was to determine whether cultivars with the seedling resistance can affect or modify the expression of resistance of cultivars with the adult plant resistance and *vice versa*. There are some reports about it; for example Lewellen *et al*. (1967) reported that minor genes may modify the expression of major genes so that a lower infection type results. Other types of modifying expression of resistance gene(s) have been reported for instance Lupton and Macer (1962) showed that the genetic background could give an increased level of resistance to race 2B in the heterozygous state. Dyck and Samborski (1968) reported that leaf rust resistance in the wheat cultivar behaved as a dominant or partially dominant trait in one cross and behaved as a recessive trait in another. It was suggested that a modifying factor (gene) within a susceptible cultivars could reverse the dominance to recessiveness for resistance. Also Johnson and Wilcoxson (1979) reported that very slow-rusting lines were obtained from F_5 families of certain fast-rusting x fast-rusting crosses of barley infected with *Puccinia hordei*. Their report suggested that even barley cultivars very susceptible to *Puccinia hordei* may posses modifying genes that gave rise to higher levels of resistance in crosses with other susceptible barley cultivars. Lee and Shaner,

1985b reported that the longer latent periods in the F_1 and transgressive segregation in four F_2 populations of crosses between slow-rusting cultivars may be due to modifiers, epistasis, or complementation in the heterozygous state. Leisle and Martens (1988) reported that the expression of Sr13 resistance varies with the genetic background. To summarize some facts arising from the literature as follows: 1) Major genes are modified by minor genes and *vice versa*; 2) Major genes together exhibit additive effects and so do minor genes; 3) Major genes and minor genes together express modifying and additive effects; 4) Major genes mask minor genes and *vice versa*; 5) Major gene resistance is enhanced by minor genes and *vice versa*; 6) One dose of a major gene may confer susceptibility, whereas two, three or four doses may confer increasing levels of resistance; 7) Several genes collectively conditioning horizontal resistance individually condition vertical resistance and single genes controlling vertical resistance collectively condition horizontal resistance; and 8) Genes may be major in one background and minor in another (Nelso (1978).

It was common in these experiments that minor genes were masked by seedling gene(s). The crosses containing Batten (Yr9) had an almost constant reaction to pathotype 111E143A- whereas the crosses of Oroua (Yr7), Pegasus (Yr1,6), Sapphire (Yr6,7) and Karamu (YrA) had different reactions to pathotype 232E137A-, depending on the cross (Table 40 & 41). Estimates of variance components showed that the major part of variation in seedling resistant cultivars was additive (Table 42). It can be concluded that transgressive segregation will be observed even in crosses of seedling resistant cultivars. This is in agreement with the comment of Johnson (1988) who stated that transgressive segregation for resistance could arise from the interactions or additive effects of race-specific genes.

Utilization of combinations of both major and minor host genes for disease protection is logical (Allan and Purdy, 1967; Lewellen *et al.*, 1967). In actual plant breeding practice, incorporating both forms of resistance may be difficult, particularly if the major host genes are epistatic to the minor genes. Dyck and Kerber (1985) stated that there is an increasing consensus that all types of resistance must be utilized in the development of a breeding strategy to produce cultivars with stable rust resistance. They also reported that major specific genes controlling resistance to stripe rust in wheat have been easily overcome by the pathogen.

5.4. Generation means analysis

By initiating a breeding programme to produce cultivars resistant to stripe rust, it is essential to obtain some information on the mode of inheritance of the resistant genes. The present information available for additive, dominance and digenic epistatic gene effects and their relative importance in the inheritance of stripe rust resistance is inadequate. To analyze gene action, a genetic model was developed by Hayman (1958a, 1960b), which estimated the additive and dominance gene effects, and partitioned the digenic epistatic effects into different components from the means of six generations of a cross.

In the present study, the crosses were made without reciprocals for experiments two, four, five, six and seven. Because there was no evidence for cytoplasmic effect in the inheritance of stripe rust resstance, a resistant parent could contribute resistance genes equally as either the male or the female parent without the possibility of losing some of the resistance (Milus and Line, 1986a; Gerechter-Amitai and Grama, 1974). A review of the literature on the inheritance of stripe rust resistance showed that there is no report of cytoplasmic inheritance (Röbbelen and Sharp 1978). No cytoplasmic effects were also reported in leaf rust studies (Bjarko and Line, 1988b). Only Krupinsky and Sharp (1978) reported the presence of cytoplasmic effects in the their cultivars that they studied for stripe resistance.

Generation mean analysis aims to do two things ; first to detect the effects of specific types of gene action, second to estimate the contribution of a particular components to the overal variation. Analysis of generation variances as well as of generation means can be carried out, and this produces complementary information for interpreting the genetic architecture. Analysis of the generation means and variances was carried out separately to provide complementary information. Analysis of generation means was used to detect additive and dominance effects and the presence of epistasis, using scaling tests (Mather and Jinks, 1982). A joint scaling test is more powerful than any of the other tests in detecting epistasis and also estimates the relative contributions of additive and dominance effects to the variation. If epistasis is present this can be partitioned into effects due to digenic interactions - homozygous x homozygous [i],

homozygous x heterozygous [j] and heterozygous x heterozygous [l]- by fitting a model containing these parameters to the observed means. Failure to obtain a fit with a model of additive, dominance and digenic epistasis parameters implies the presence of higher order interactions or interactions between linked loci. To distinguish between these requires a specific range of backcross generations (Hill, 1966) which were not available in this experiments. The more generations available, the more parameters that can be fitted to the data. If experiments are replicated over environments, further parameters specifying the interaction of genetic effects with the environment can be defined.

A problem in interpreting the analysis of generation means is that the parameters specifying the effects are the balance effects of all segregating loci. This means that additive and additive-related interaction parameters are a function of the degree of dispersion of increasing genes between the parents, and dominance effects are the net product of the direction of dominance at each locus. Consequently, estimates of additive effects could be small because there is a high degree of dispersion rather than because there is little variation. Similarly dominance could be small because of ambidirectional contributions. If the additive effect is small and nonsignificant while the dominance estimate is large and highly significant, this indicates a high degree of dispersion of increasing genes between the parents combined with strong directional dominance; a classical explanation of F_1 heterosis (Snape, 1987). Genetic variances, on the other hand, are not influenced by balance, because they are the sum of squared effects of each locus and hence express the total variation of additive and dominance effects. If epistasis is demonstrated by the analysis of generation means, caution has to be used in interpreting what are biased estimates of additive and dominance variances (Snape, 1987).

The use of different generation means to estimate magnitudes of gene action and the conformity of the genetic system goveming the expression of a character to an additive-dominance model was proposed by Cavalli (1952) and has been illustrated by Hayman (1958a, 1960b) and Mather and Jinks (1982). The computational presentation by Mather and Jinks (1982) does not specifically indicate the general statistical nature of the methodology nor the potential for generalization to more complex genetic models. Rowe and Alexander (1980) tried to clarify computational aspects of the methodology and indicate the generalization to more complex genetic models. They gave the general

formulation of weighted least squares procedure using matrix notation. They also stated that the standard errors of the parameter estimates given by Mather and Jinks (1982) were underestimated. No reasons were given to explain such a statement. The weighted method of Mather and Jinks, which is more realistic according to genetic theory, is based on the assumption that the populations have nonhomogeneous variance. Rowe and Alexander's method is based on the assumption that the populations have homogeneous variances. In the latter case, there is actually no need to do a weighted least squares analysis. So, it was logical to use weighted least squares analysis in these experiments (five and seven).

All the genetic models used in the study of quantitative inheritance have involved certain assumptions in order to simplify statistical procedures. Some of the assumptions are more important than others in causing bias in the estimates of gene effects. Anderson and Kempthome (1954) used the following assumptions in the development of their genetic model: (1) multiple alleles absent; (2) linkage absent; (3) lethal genes absent; (4) constant viability for all genotypes; (5) environmental effects additive with genotypic value. There would be no serious bias expected in the estimates of the parameters from assumptions 1, 2 and 3. Since the only segregating populations used in this study are the F_2, F_3, first backcross and backcross selfed generations of a cross between two homozygous lines, multiple alleles would be present only if the parental lines were not homozygous or if mutation occurred. Lethal genes are not likely to be present in crosses since the parental inbred lines used in the study have been maintained by selfing for many generations. Viability was constant. According to Mather and Jinks (1982) the assumptions of generation mean analysis are: (1) parents are homozygous, (2) no genotype x environment interactions, (3) resistance genes are associated in one parent, (4) linkage equilibrium for the epistatic models. These assumptions are almost same as the assumptions of Anderson and Kempthome (1954), but in the cross of two resistant parents, the assumption of association of resistance gene in one parent might cause bias in the estimated parameters. In the cross of two resistant parents, there are different genes for resistance in both parents, so it is not convenient to use generation mean analysis for that cross, because it is assumed that the resistant genes by which the parents differ are associated in one parent (Mather and Jinks, 1982). This point applies to the crosses Ruapuna x Domino (for infection type), Domino x Otane and Briscard x Ruapuna (for latent period); Ruapuna x Domino and

Briscard x Ruapuna (for area under the disease progress curve). But using generation mean analysis for the cross with two resistant parents gives genetic information about certain pattems and there is the possibility that they are opposite for the genes by which they differ.

A failure of the additive-dominance model to fit the data must imply that one (or more) of the assumptions on which the model is based is in fact invalid (Mather and Jinks, 1977). One of the assumptions is that the genes show simple autosomal inheritance and sex-linked, matemal elements and other things cause the departure of inheritance from simple autosomal, the model would not then be appropriate and would be found to fail in its fit with an adequate body of observational results (Mather and Jinks, 1977). Since wheat is an hermaphroditic plant, sex-linkage cannot be involved. In the additive-dominance model, it is assumed that the genes involved are independent of each other in producing their effects. If the genes act in a multiplicative fashion or the genes are additive in their effects on the linear dimensions of an organ but the character is effectively an area which is reflected the square of the sum, by transforming and changing the scale of data, the genes make their own independent contributions to the phenotype (Mather and Jinks, 1982). It is possible to transform the data to a more appropriate scale and to carry out analysis successfully using the simple additive-dominance model on the transformed data. The only justification for any transformation that may be used is that it works (Mather and Jinks, 1977). For infection type,latent period, and area under the disease progress curve (before generation means analysis the data were transformed using natural logarithms, see Materials and Methods), neither log nor square root transformations of the data decrease chi-square values for the models. Even though these models were significant, not all components of the models may be significant.

The analysis of generation means proved to be a simple and useful procedure for investigating the gene action involved in the inheritance of stripe rust resistance in these crosses. The estimates of gene effects together with the scaling test and chi-square for all crosses have been calculated. Adequacy of the three-parameter model led to derivation of estimates free from linkage, if present, for additive and dominance effects for the experiments. Assuming that the results were not biased by genotype environment interactions, the lack of fit of the three-parameter model provide evidence of gene

interactions in the crosses. Information as to kind and magnitude of epistatic effects for those characters was provided by the six-parameter model. As noted before, the test of the adequacy of the three-parameter model provides information on the absence or presence of gene interactions. The test of adequacy of scale (chi-square) is important because in most cases additive and dominance components of variances are estimated assuming the absence of gene interaction (Mather and Jinks, 1982). Adequacy of scale must satisfy two conditions: namely, additivity of gene effects and independence of heritable components from non-heritable ones. Mather and Jinks (1982) proposed that if the three-parameter model is not adequate, the six-parameter model must be tested and nonsignificant component(s) should be removed and then the model should be tested with the rest of components. Despite the method of Mather and Jinks (1982), in these experiments all models, two-, three-, four-, five- and six-parameter, were used for a complete understanding of the genetic systems for the character studied (Tables 82, 87 & 97-106). According to Mather and Jinks (1982), if a simple model provides a good fit to the data, there is no basis for assuming a more complex situation. For either infection type and latent period six crosses followed the method of Mather and Jinks (1982) i.e. by omitting nonsignificant parameter(s), the precision with which the remainder are estimated should be increased (increase in mean and decrease in its standard error). In other crosses, after removing the nonsignificant parameter(s) in the six-parameter model, the chi-square was still significant, indicating that the model was not appropriate (i.e. did not follow the method of Mather and Jinks) and then one concluded the presence of trigenic interaction, linkage or both. In fact we could not fit an appropriate model to it, however, by using all possible models, the best fit (all components were significant with a low standard error and chi-square was not significant) was obtained. This suggests that to obtain an appropriate model and also not to be confused with trigeneic interaction or linkage, using all models is necessary to understand the best genetic model.

For area under the disease progress curve, generation mean analysis was tested with three sets of generations (Tables 97-106), the first set involved P_1, P_2, F_1, F_2, BC_1 and BC_2, the second one involved P_1, P_2, F_2, F_3, BCS_1 and BCS_2, and the third one involved P_1, P_2, F_1, F_2, BC_1, BC_2, F_3, BCS_1 and BCS_2. The two-, three-, four-, five-, and six-parameter models were tested for the first two sets of generations. Usually, the first set is used as the basic generations in those experiments which do not need too much

seed for the F_1 and backcrosses to estimate gene effects in the field trials. At least six generations are necessary for the estimation of the six parameters m, [d], [h], [i], [j] and [l]. The most convenient experiment involves all generations is the first set (Hayman, 1958). In some plants such as wheat, producing sufficient seed for the F_1 and backcrosses for the field trial is usually more difficult, time consuming and extra labour is required. In such a case, using the second set of generations P_1, P_2, F_2, F_3, BCS_1 and BCS_2 is more convenient, but the precision of this method is less than the first one. By using the nine generations nothing obtains beyond digenic interaction. To obtain trigenic interactions at least ten generations are needed (Mather and Jinks, 1982). The results of these three methods were not similar in any cross. In general, more interactions in method two were significant than in method one. In methods one and two the goodness of fit was obtained for all crosses, indicating nothing beyond the digenic interactions. The method three had less significant interaction than the other methods and also the goodness of fit for all crosses was not obtained, except for crosses of Briscard x Otane and Ruapuna x Domino. This indicated that indicating the model was not appropriate (or probably the presence of trigenic interaction, or linkage, or interaction with environment, or combinations of them). The lack of fit to digenic models may be due to more complex genetic control of resistance such as trigenic interactions, linkage of interacting loci, or the large environmental variance associated with the phenotypes in the crosses studied (Mather and Jinks 1982). Bhullar *et al.* (1978) reported that when more statistics of F_2-derived generations were included in addition to the within family variances of the basic six generations to estimate additive-dominance model parameters, the fit of the model was not good.

In some cases only the additive part, [d] or the dominance part, [h], was significant and epistasis was not significant. It can be expressed since the value for epistatic gene action (even if it could be large) is an average for all resistance genes and epistasis may be obscured by the background genotype. Epistasis may not be detected if only one or a few gene pairs exhibited epistasis and other gene pairs acted additively, or if there were several epistatic interactions with opposite effects (positive and negative components) that cancelled one another. The nonsignificance of nonallelic interaction in this study, particularly the [j] effects, may be due to the cancelling of positive and negative effects from different loci as previously mentioned. In the nonspecific glasshouse generation means (Tables 82 & 87), genetic models assuming epistatic

interaction adequately described the gene action in all crosses, except the cross Ruapuna x Tiritea for infection type and cross Briscard x Domino for latent period, only the additive, [d], part was significant and neither dominance nor epistasis was significant. In this experiment the additive x dominance [j] was significant in crosses Briscard x Tiritea, Domino x Tiritea, Otane x Tiritea, Briscard x Otane for infection type, whereas [j] was significant in crosses Domino x Tiritea and Ruapuna x Domino for latent period. The major role of the additive x dominance effect suggests that the development of pure line resistant wheat lines may not be feasible since this type of epistasis is not fixed by selection under self-fertilization. Selection for stripe rust resistance in early segregating generations would be highly inefficient. Rather, the development of resistant hybrid cultivars possessing reduced disease damage would appear to be favoured. But in most of crosses, [j] was not significant either for infection type and or for latent period indicated that selection is useful in selecting resistant lines with lower infection type and longer latent period. In this experiment, the cross Ruapuna x Domino for infection type and cross Briscard x Ruapuna for latent period had not any significant additive, [d], part in the model, indicating selection is not effective at least in early generations.

Usually evidence for polygenic resistance existed because the additive-dominance model was not a fitted model for all crosses in experiments five and seven. For example neither for infection type nor for latent period, the additive-dominance model was observed (Tables 82 & 87). In the nonspecific field generation means, the additive-dominance model was observed in crosses Briscard x Tiritea and Otane x Tiritea, only by using the first method (Tables 97-106). Also all the models, whether fitted to the means of the basic six or all the nine generations, gave a consistent values of additive effects, [d], indicating the possibility of deriving highly resistant lines in further generations.

For each cross, all the components of the appropriate model were highly significant. Overall epistatic effects were of importance. If there is no epistasis and additive dominance is adequate to the polygenic system, the F_2 mean is expected to be half-way between the F_1 mean and the mid-parent value i.e. $F_2=1/4(P_1+P_2)+1/2F_1$ (Mather and Jinks, 1977). When the observed value of the F_2 tends to be higher than the expected one, this might indicate interaction effects operating, although very slight and insignificant when compared with the dominance and additive effects ;

In the nonspecific glasshouse generation means, whether for infection type or latent period (Tables 82 & 87), only in the cross Otane x Tiritea for infection type were all components fitted (m, d, h, i, j and l). In the other crosses, although all three epistatic components were not significant in the fitted model, at least one epistatic effect was significant. With regard to the individual epistatic gene effects, [i], [j] and [l] effects appeared to contribute to the performance of infection type and latent period in the crosses studied. The magnitude of the estimates of [i], [j] and [l] over all ten crosses revealed that significant epistatic gene effects were present and important in the inheritance of stripe rust resistance. In the case of infection type over all ten crosses, additive x additive, additive x dominance and dominance x dominance gene effects made equal and considerable contributions (Table 82). But in the case of latent period dominance x dominance gene effects made considerable contribution and additive x additive effects were also of large magnitude (Table 87). Additive x dominance gene effects were of less importance. Since more epistasis appeared, it is logical to assume that more gene or factor control of these traits as Cockerham (1959) noted that epistatic gene action is not uncommon in the inheritance of qualitative traits, and that there is no sound biological reason why this type of gene action should be less common in the inheritance of quantitative traits. As the number of genetic factors conditioning a trait increases, it seems reasonable to suppose that the number of interactions among factors will also increase. These results agree with the results of Milus and Line (1986b) who attempted to quantify epistasis for stripe rust resistance. These results indicated that additive x dominance and dominance x dominance are almost as important as additive x additive, but based on the performance of inbred lines of corn, Stuber (1970) suggested that genetic models that estimate additive, dominance, and additive x additive interactions are realistic for analysis of quantitative variation in most self-pollinated species. One consequence of the different gene effects on the choice of a breeding strategy is that line selection following repeated self-fertilization would be expected to raise levels of resistance due to the predominant additive gene effects. The additive x additive epistatic components increasing resistance are likewise fixable in pure inbred lines. Dominance may be exploited but only if hybrid wheat is the objective of the breeding programme. If the sign of [j] and [l] effects are positive, they can contribute to increase disease levels (for example for infection type). Because neither the simple nor the epistatic dominance gene effects can be fixed in homozygous lines and operate in opposing directions, it may be necessary for selection pressure to be mild in early selfed

generations and be intensified when homozygousity is approached.

In the field experiment, whether by method one or method two (Tables 97-106), in almost all crosses at least one of the interactions was significant. With regard to the individual epistatic gene effects, [i], [j] and [l] effects appear to contribute to the performance of area under the disease progress curve in the crosses studied. The magnitude of the estimates of [i], [j] and [l] over all ten crosses revealed that significant epistatic gene effects were present and important in the inheritance of stripe rust resistance. In both methods over all ten crosses, dominance x dominance gene effects made a considerable contribution and additive x additive and additive x dominance gene effects made an equal contributions but of very small magnitude in comparison with dominance x dominance. A significant dominance x dominance interaction component indicates an interaction between the heterozygous states of the loci involved. This would help explain the lower than expected mean values of the F_1 populations of the resistant x resistant crosses. In the case of the third method over ten crosses, the additive x additive effect was more important than the other interactions, additive x dominance was of least importance and dominance x dominance was of intermediate importance.

The opposite signs of [h] and [l] on the basis of the digenic epistasis model indicate duplicate or opposing types of gene interactions. In order to analyze the nature of classical epistasis, the sign of the [h] and [l] components were screened in those crosses where both components were significant, after the omission of certain less important parameters. Four crosses out of ten for infection type (Table 82) and six cross out of ten for latent period (Table 87) possessed opposite signs for the [h] and [l] components, thereby suggesting the predominance of a duplicate type of epistasis. In all crosses duplicate interaction does not create any difficulty for selecting plants with lower infection type and longer latent period, but in experiment five in cross Otane x Tiritea, dominance in the direction of low infection type along with a positive [l] effect would indicate duplicate epistasis which is undesirable in selection. Dominance towards longer latent period and significant [l] effects indicated a duplicate type of epistasis for latent period, suggesting that difficulty would be encountered in selecting for longer latent period. For the field experiment (Tables 97-106), in general, duplicate interactions did not play an important role as well. In fact, Mather (1967) stated that complementary interaction increased the variance of segregating families and populations, but duplicate

interactions generally decreased the variance. Thus selection towards a single optimum phenotype can favour duplicate but not complementary interactions. Complementary interactions could perhaps sometimes be favoured by disruptive selection towards two or more optima.

It should be noted that the sign of parameters [d] and [j] depends upon the parents being considered as P_1 or P_2. The sign of [j] would change correspondingly in most cases but the sign of the other parameters would be unaffected. The negative signs of [h] indicates partial dominance in the direction of reduction of the character. The opposite sign of additive, [d], and additive x additive, [i] genetic components shows the oppositional nature of the interactions. Two, four and five crosses out of ten showed oppositional nature of interaction for infection type (Table 82), latent period (Table 87) and area under the disease progress curve (Tables 97-106), method two, respectively.

Gene dispersion was also verified by comparing the magnitude of [h] and [l] and the higher estimates of the [h] component over [d]. If parents used in a cross are in the association phase (gene with increasing effects in one parent and gene with decreasing effect in the other parent), the [h] component is always smaller when compared to [d]. However, if they are in the dispersion phase (gene with increasing and decreasing effects are randomly distributed among parents), the estimates of the [h] component are always higher than [d] due to the accumulation of dominant parental genes in the hybrid. Most of the crosses, whether in the glasshouse (Tables 82 & 87) or field (Tables 97-106), showed that they were in association phase.

The estimates of epistasis as well as dominance and additive gene action may have been influenced by genotype-environment interactions in both the three-parameter and six-parameter models. The possible importance of genotype-environment interactions could be determined by conducting tests in several environments. However, all of the models that had a goodness of fit, in both the glasshouse experiments (infection type and latent period) and the field experiment (method one and two), indicated no presence of genotype-environment interactions or trigenic interactions, linkage, or some combination of these.

The present of linkage among genes may cause an important bias in the

estimates derived from the data on early generations of the crosses. Early generations of a cross were considered in experiments five and seven, and an equilibrium of linkage relations is improbable (Comstock and Robinson, 1952; Mather and Jinks, 1982). Therefore, if there is epistasis, bias due to linkage relations would be present in the estimates of the gene effects (Kempthorne, 1957). In order to minimize the undesirable linkage effects, the use of populations derived by intermating the F_2 individuals of a cross, as recommended by Hanson (1959) in self-pollinated crops, should be preferred. Presumably, recombination would occur at a high rate and the resulting populations may provide a wider spectrum of variability. The most serious bias would be expected to occur in the estimates of the [i] and the [j] effects. However, apparent linkage bias might be due to trigenic or higher epistasis. Where interallelic interactions are not involved, the estimates of gene effects are not biased by linkage relationships.

In no case, whether in the glasshouse or field experiments, was the F_1 significantly better than the more desirable parent. This indicates that nonadaptive gene action for infection type, latent period and area under the disease progress curve was of minor importance. In the case of area under the disease progress curve the degree of dominance for Ruapuna x Domino was 1.21, indicating overdominance for resistance, the F_1 mean being similar to that of the resistant parent. It is therefore possible that the gene action may be one of complete dominance, rather than overdominance for resistance.

5.4.1. Generation variance

Partitioning the mean variances of the basic six generations into additive, (D), dominance, (H), covariance of additive-dominance, (F), genetic and additive environmental, (E), can enable us to estimate the magnitude and sign of dominance, and heritability (Mather and Jinks, 1982). Because the F [F=(dh)] is a linear function of the h's and so, like h, can take sign: it is in fact a weighted sum of the h's, the weights being the corresponding d's. Where the F is positive the genes from the larger parent (e.g. high infection type as susceptible, longer latent period as resistant, and high rust severity as susceptible) show a preponderance of dominance over their alleles from the other parent, and where the F is negative the genes from the smaller parent show the dominance (Mather and Jinks, 1977). It should be noted that these statistic can be

estimated in the diallel cross, but here these statistics belongs to two parents (one cross) not a set of parents (diallel cross). So it is informative to estimate these statistics from the generation means experiments. In almost all experiments, whether in the glasshouse (Tables 83 & 88) or the field (table 107), the sign of the F value was in agreement with the sign of the degree of dominance resulting from the deviation of the F_1 from the mid-parent; and it showed that, in each cross, the genes from the resistant parent (low infection type and severity) were preponderantly dominance over their alleles from the susceptible parent whereas in the case of latent period it was reversed.

If the h and d are constant in magnitude for all the k gene pairs segregating in a certain cross, $(H/D)^{1/2}$ provides an estimate of the degree of dominance. If h and d are not constant in magnitude $(H/D)^{1/2}$ provides an estimate of the average dominance of the genes (Mather and Jinks, 1982).
If the $(D*H)^{1/2}$ is equal to F, it means the measure of dominance is constant in both magnitude and sign for all the gene-pairs involved (Mather and Jinks, 1982). In almost all of crosses, whether in the glasshouse (Tables 83 & 88) or the field (table 107), d and h were not constant in magnitude and $(H/D)^{1/2}$ is the average degree of dominance rather than the degree of dominance.

5.4.2. Heritability

Heritability estimates provide information on the transmission of characters from the parents to the offspring and thus facilitate evaluation of genetic and environmental effects in phenotypic variation and aid in selection. Also the speed of progress under selection following a cross of two lines will depends on heritability (Mather an Jinks, 1982). Heritability estimates with genetic advance enable breeders to predict the real genetic gain under selection so that they can anticipate improvement from different types and intensities of selection. Broad-sense heritability is based on total genetic variance which consists of fixable and nonfixable components. Hence, narrow-sense heritability estimates based on additive genetic variance (fixable component) are important in predicting the effectiveness of selection in a genetically heterogeneous population. Matzinger *et al.* (1960) noted that failure to include epistatic in heritability estimates may bias estimates of additive genetic variation and predicted gain from selection. It has been observed that heritability estimates increase with greater similarity between the parents

and are otherwise dependent on experimental design (Simmonds, 1979). In this study, five cultivars were intercrossed, whether in the glasshouse experiments or in the field experiment, in a diallel fashion and tested in replicated trials. Therefore, it can be believed that a reasonable order of magnitude for the heritability of reaction to infection type, latent period, and area under the disease progress curve by stripe rust for the material studied has been established. Warner (1952) stated that in estimating heritability from the variance of the backcrosses and the F_2, the assumptions appropriate to the use of the analysis of Mather and Jinks (1982) should be fulfilled. These are: first, independence of genotype and environmental variance, and second, additivity of genic effects over the various loci. High heritability was obtain in the glasshouse and field experiments; for example for infection type (Table 80), the average broadsense and narrowsense heritabilities over ten crosses were 0.76 and 0.61 respectively; and for latent period (Table 85) were 0.60 and 0.44 for broadsense and narrowsense heritabilities. And narrowsense heritability estimates, whether for infection type or latent period, were high for the crosses involving Ruapuna. For the area under the disease progress curve (Table 95), the average of broadsense and narrowsense heritabilities over ten crosses were 0.73 and 0.51, respectively.

If heritability values for latent period were high, significant progress in cultivar improvement programmes should be possible. The heritability values were greatest in the crosses of shorter with longer latent period parents, as might be expected. Nevertheless, progress in selecting for longer latent period would have been possible in all crosses. The effectiveness of selection agreed with Johnson and Wilcoxson (1979) who reported that the heritability of barley infected by leaf rust was low or moderately low and even with low heritability selection for longer latent period was effective.

In general, for area under the disease progress curve, the heritability of resistant by susceptible crosses was higher than for other crosses, which agreed with Johnson and Wilcoxson (1979), and narrowsense heritability estimates were the highest for the crosses involving Ruapuna and Briscard. Ruapuna and Briscard are resistant to stripe rust and their resistance appears to be readily identifiable in the segregating populations. Heritability estimates indicate that it may be possible to select for genotypes containing higher numbers of resistance genes and thus obtain enhanced resistance to stripe rust in their crosses. These results agreed with the results of Bjarko and Line (1988a) who

reported that the heritability of the area under the disease progress curve for leaf rust resistance was from 0.92 to 0.21, depending on the cross. Gavinlertvatana and Wilcoxson (1978) reported that the heritability of slow rusting in spring wheat infected with *Puccinia recondita* f.sp. *tritici* in the field, ranged from 55 to 87%. High heritability was reported by other workers (Johnson and Wilcoxson 1979; Luke *et al*. 1975). Estimated heritability for the Ruapuna and Briscard crosses were reasonably high, suggesting that genetic advance could be made for slow rusting by selection criteria based on the AUDPC in crosses between cultivars.

Genetic data are also of practical interest in plant improvement. For example for infection type, estimated rates of genetic gain in this study averaged, over ten crosses, 43% of the F_2 mean (Table 80). Also for latent period, estimated rates of genetic advance, over ten crosses were 11% of the F_2 mean (Table 85). For area under the disease progress curve, estimated rates of genetic advance averaged over ten crosses were 28% of the F_2 mean (Table 95). High heritability of infection type coupled with the importance of additive type of gene action indicate that rapid progress in stripe resistance improvement can be achieved.

The usefulness of estimates of heritability as a practical tool of the plant breeder depends on several factors. In the first place estimates of heritability provide information on the relative practicability of the selection: high heritability in the F_2 indicates effective selection on an individual plant basis is possible. A plant breeder, faced with a problem in an unfamiliar crop or on a character about which little is known, might find some heritability studies useful in order to attack the problem more intelligently. The usefulness of heritability studies would also depends on the effort and expense required to gain such information.

It should be noted that sometimes the heritability estimate is greater than the theoretical limit, i.e., 1.00, which may be ascribed to several causes; sampling errors, differential responses of the F_2 vs. the backcrosses to the environment, and nonallelic interactions can result in an upward bias of heritability estimates, as measured by Warner's method (1952). Heritability and genetic advance generally agreed in showing those characters for which selection in the F_2 would lead to substantial improvement. It should be noted, however, that a moderate heritability estimate for the all traits studied,

in some crosses, was associated with the higher genetic advance. This may be due to a large environmental variation for this trait in the F_2 population. Reporting genetic advance and heritability should therefore be more informative in genetic and breeding studies than showing either of them alone. The ultimate usefulness of these estimates should be evaluated by conducting selection programmes and comparing predicted with realized gains.

5.4.3. Number of genes

In many experiments it is important to distinguish whether a character is controlled by a few genes of major effect, or by many genes of minor effect, since this can have important consequences for selection strategy (see Literature Review) as well as being intrinsically interesting. The most powerful way of identifying the number of genetic factors segregating in wheat is to use aneuploid techniques. These allow the variation to be partitioned into the effects of individual chromosomes and then into the effects of individual arms or regions of those chromosomes. This method estimates the numbers and locations of factors and also the relative size of effects. However, these techniques require the use of aneuploid stocks and a certain level of expertise in cytogenetic manipulations, which are not always readily available. The alternative is to use methods of quantitative genetics which, although they do not establish the identity of individual factors, do give an estimate of how many are segregating. In using these techniques, however, it is important to realize that they estimate the number of units that are segregating, which is not necessarily the same as the number of different gene loci, and are thus termed the number of effective factors rather than genes. To highlight this distinction see Mather and Jinks, 1982.

In the quantitative method, there are two approaches to the estimation of the number of gene, or more correctly effective factors, controlling continuous variation. One is the method of moments and the other is the genotype assay. (1) The method of moments is based on a comparison of the square of the genotypic range with the estimated genetic variance. The ratio of these two statistics is expected to increase as the number of genes controlling a trait increases. By assuming that the trait is controlled by independent genes with equal additive effects and that the parents of the cross represent the limits of genotypic expression, it is possible to estimate the number of

genes. It is generally agreed that estimates from this method are represent the minimum gene number. Modifications have been suggested for use when dominance is believed to be present (Multize and Baker, 1985a, b). (2) Jinks and Towey (1976) developed experimental designs, based on genotype assay, that could detect segregation within polygenes. The estimated number of genes controlling three characters in tobacco increased as the generations advanced from the F_2 to the F_8, and this increase was attributed to segregation within effective factors. Their studies implied that populations had variation that was hidden in balanced polygenic systems composed of a large number of genes. In inbreeding species, variation will remain hidden once genes become homozygous as a result of selfing. This variation can be exposed by making repeated crosses among selected lines to break old linkage groups and form new, more favourable linkage groups. Once favourable combinations of genes are selected, they should remain linked and segregate together most of the time. Therefore progeny with enhanced levels of resistance should be sources of durable resistance for breeding programmes.

The underlying assumptions of the equations which were used to estimate the number of genes are: (1) no systematic relation between mean and variance, (2) no linkage of genes, (3) no epistasis, (4) the relevant genes are of equal effect, (5) one parent supplies only plus alleles of those genes in which the two parents differ, whereas the other parent supplies only minus alleles, and (6) an equal degree of dominance for all plus alleles. It is unlikely that all of these assumptions will be met in practice, particularly that all factors are of equal effect, and estimates are generally likely to be underestimates of the true number of effective factors segregating in a cross (Mather and Jinks, 1982). Assumption number five could not be ascertained for any of the crosses. Mulitze and Baker (1985a) discussed the assumptions required in estimating gene number by the method of moments. The most important assumption, that the parents represent extreme genotypes, i.e., that all resistance genes occur in one parent, is probably correct for the four resistant x susceptible crosses (Briscard, Ruapuna, Domino and Otane with Tiritea) studied in the glasshouse and field experiments. Lack of dominance is another assumption met to a close approximation. Other assumptions, that epistasis and linkage are absent and that effects of all loci are equal, are problematical but probably less crucial to the validity of the method. Multize and Baker (1985a) suggested that problems caused by unequal effects at different loci may not be overly

important in method of moments estimates of numbers of genes. They also stated that the most important assumption for applying the method of moments procedure is that the parents represent the extreme genotypes. The F_3 was used to estimate the number of genes in the field experiment which can be more reliable than those estimated using the F_2, because Multize and Baker (1985a) stated that gene number estimates from the method of moments procedure are expected to be more reliable if carried out in generations beyond the F_2. This is due partly to the reduction in bias caused by dominance.

The genotypic assay was designed to be less dependent on assumptions than some of the other biometrical procedures. Use of the method does require certain assumptions concerning linkage and epistasis but does not require assumptions about levels of dominance and equality of effects over loci. Hill and Avery (1978) showed how the number of effective factors changes with generation in genotype assay and how this depends on initial coupling/repulsion relationships.

However, these gene number estimates must be used with extreme caution. Falconer (1981) stated that since the estimation of the number of loci is necessarily so imprecise it does not seem worthwhile to discuss in detail its limitations or the errors that may have been introduced by the assumptions that were made. The results of the various methods to calculate the number of genes should be looked at with caution as several prerequisites are not met and the estimates of the various methods do not match. Despite these pitfalls an estimate was made of the number of genes in each parent. In the glasshouse and field experiments the analyses estimate the minimum number of genes. However, the resistance genes could be linked and could segregate as a group or effective factor. So the formula would estimate the number of effective factors and the number of individual genes would be greater. Milus and Line (1986a) explained the differences between gene numbers and effective factors. The effective factor hypothesis can also explain why transgressive segregation for enhanced resistance was frequently observed among progeny from crosses between susceptible parents (Pope, 1968; Sharp *et al.*, 1976; Krupinsk and Sharp, 1979; Wallwork and Johnson, 1984). Susceptible cultivars may have genes for resistance that are in balanced polygenic combinations (Johnson, 1984) i.e. genes for resistance are cancelled by linked genes for susceptibility. When two susceptible cultivars with different genes are

crossed, recombination within effective factors can replace some of the genes for susceptibility with genes for resistance and produce genotypes with more genes for resistance than for susceptibility. Pope (1968) hypothesized that genes controlled functions in a sequence of events leading to resistance. In this model, each gene alone has no effect, but high levels of resistance can be achieved when the necessary combination of gene is produced by crossing.

For infection type (Table 81) the number of genes for Briscard, Ruapuna and Domino in cross with Tiritea (susceptible) were estimated as 1-3, 2-4 and 1-3 genes, respectively. For Briscard in a cross with Otane, the number of genes was estimated as 1-3 genes, as was estimated with Tiritea. Polygenic inheritance was reported by other researchers, for example Johnson (1978) hypothesized that some of the genes controlling durable stripe rust resistance were linked and inherited as polygenes (effective factors), since a large part of the resistance was readily transferred in breeding programmes. Law *et al.* (1978) reported the presence of at least three genes on chromosome 5B'-7B' that controlled adult-plant resistance to stripe rust in several wheat cultivars. The number of segregating genes in other crosses was estimated as less than unity implying that both cultivars carry the same number of genes for resistant, or this may be due to ambidirectional distribution of the genes between the parents, or due to unequal gene effects or both. For latent period (Table 86) the number of genes for Briscard, Ruapuna and Domino (longer latent period parents) in crosses with Tiritea (susceptible) were estimated as 1-3, 1-4 and 1 genes, respectively. For Briscard in a cross with Otane, the number of genes was estimated 1-2 genes, less than estimated with Tiritea. These results of latent period are supported by other researchers. Kuhn *et al.* (1980) estimated that two genes controlled the inheritance of longer latent period in Suwon 85 wheat to leaf rust, also Lee and Shaner (1985a,b) estimated that one to three genes controlled the inheritance of longer latent period in wheat to leaf rust, depending on the cross. In the field experiment (Table 96), based on the different formulae of Wright (1968), Milus and Line (1986a) and Bjarko and Line (1988a) the number of genes for the resistant x susceptible crosses were estimated as 2-9, 3-12, 3-8, 1-5 for Briscard, Ruapuna, Domino and Otane in crosses with Tiritea for area under the disease progress curve. This polygenic resistance was corroborated by other researchers. For example Milus and Line (1986a) reported that the area under the disease progress curve in some cultivars with durable resistance to stripe rust was controlled by 2 to 3 genes but the

range of number of genes was from less than unity for resistant x resistant crosses to 70 for resistant x susceptible crosses (Milus and Line, 1986b). Luke *et al.* (1975) estimated 2 to 13 genes for crown rust resistance in Red Rustproof oats. Skovmand *et al.* (1978b) estimated that 2 to 12 genes were segregating for slow stem rusting in wheat, depending on the cross. Gavinlertvatana and Wilcoxson (1978) estimated 3 to 31 genes for slow leaf-rusting in spring wheat, depending on the cross. Bjarko and Line (1988a) reported that two to three genes, or possibly more, controlled slow leaf-rusting in each parent but the range of numbers of genes was 0-9, depending on the cross.

In general, the large number of resistance genes in the Ruapuna x Tiritea cross estimated by some formulae is probably an overestimate of the real number of genes. Other formulae gave a most reasonable estimates of number of genes. Briscard, Ruapuna and Domino have different resistant genes from each other, because low estimate of the number of genes in Briscard x Ruapuna, Briscard x Domino and Ruapuna x Domino and transgressive segregation for resistance and susceptibility in their crosses means that each parent contributed different resistance genes. Thus, if there is no transgressive segregation in resistant x susceptible cultivars, it means the number of genes from the resistant parent can be estimated truly. Since an effective factors consist of linked genes, the estimated number of genes must be expected to increase as generations advance, because linkage groups will continue to be broken in later generations. However, it is clear that stripe resistance is governed by several minor genes.

Again it should be noted that the presence of linkage, dominance, or unequal effects at different loci will cause an underestimation of the actual number of segregating genes present, while the presence of epistasis may cause either an overestimation or an underestimation of the actual number of segregating genes. Inheritance of resistance to stripe rust frequently proved more complex in the field than in the glasshouse. Since all of these crosses were tested in the field for area under the disease progress curve, high environmental variance might have increased the estimated number of genes.

5.4.4. Continuous distribution and transgressive segregation

The continuous variation, whether in the glasshouse (Fig. 21 & 22) or the field

(Fig. 35-44), was observed in the F_2 frequency distributions of all crosses. All traits showing a continuous curve can be said to show quantitative inheritance as opposed to qualitative inheritance, in which the traits show distinct classes, because quantitative genetic traits are characterized by continuous distributions which include gene effects and effects due to the interaction of genotype and environment. The individual gene effects can rarely be measured. The genes must be considered together and statistical procedures used to obtain basic genetic information. (Allard, 1960) stated that the continuous distribution in segregating populations of crosses may be due to the segregation of several genetic factors, low heritabilities, or both. On the other hand, the continuous variation does not necessarily imply polygenic inheritance (see Thompson, 1975). Continuous variation may even be conditioned monogenically provided that nonheritable (environmental) effects are large. Additive resistance is more affected by environmental factors than dominant resistance. A normal distribution of a phenotypic trait can be seen from the segregation of only one gene in the F_2 populations when heritability is low (Hoff and McDonald, 1980). There is some evidence to support both sides of this controversy. For example Johnson and Wilcoxson (1979) stated that continuous variation is evidence of quantitative inheritance for barley-barley leaf rust, and Kuhn *et al.* (1980) reported that assuming quantitative inheritance solely on the basis of the presence of continuous variation in segregating population is not valid. But in these experiments in the glasshouse and field, the heritability was high and it can be concluded that continuous variation is as evidence of quantitative inheritance. It should also be noted that if the F_2 frequency distribution is multi-modal, it suggests the involvement of major genes in the expression of stripe rust resistance which were not observed in all adult plant resistant cultivars.

In most crosses, the F_2 distribution was continuous and skewed towards susceptibility (higher infection type and shorter latent period) and for the field experiment it was skewed towards resistance (low severity). The lack of normal distribution may be due to the presence of dominance, epistasis, or linkage between the resistance genes. Skewness in frequency distributions in a particular direction suggests dominance toward that direction. Parlevliet (1978) mentioned three possible causes for skewness in case of latent period: (1) dominance effects; genes for a longer latent period would be inherited in a recessive manner, (2) the latent period of the most susceptible genotype may represent a physiological barrier and gene action cannot be fully expressed at such

low latent period values, and (3) geometric cumulative gene action which means that the more genes that are present the larger is their effect. In this experiment, the first cause was of importance. Jacobs and Broers (1989) concluded that the first reason (mentioned above) most likely was the most important one and they excluded the second one. Geometric cumulative gene action was not excluded. It causes the F_2 mean to be closer to the F_1 than to the midparent value.

In the glasshouse (Fig. 21 & 22) and field (Fig. 35-44) experiments, transgressive segregation was observed in for resistance and susceptibility. The transgressive segregation whether for resistance or susceptibility clearly originated from a combination of the genetic components from both parents of each cross. Transgressive segregation toward resistance between two susceptible parents indicates that some of the resistance genes in both parents must be different. This can be supported with other workers such as Johnson and Wilcoxson (1979) and Lee and Shaner (1985a, b). This is not unexpected because the genes of the two parents differ in their effect on the disease and originated from different ancestors. If transgressive segregation for susceptibility is observed in crosses between resistant cultivars, this indicates that each of the resistant cultivars has different genes for stripe rust resistance. Johnson (1988) stated that transgressive segregation for resistance could arise from interactions or additive effects of race-specific, or from the transfer of race-specific genes from a suppressive to an expressive background. It could also arise from the accumulation of resistance genes of the type associated with durable resistance. It would not be possible in advance to predict which of these possibilities had been achieved, because, so far, there were not any known Yr genes in these adult plant resistant cultivars and for all crosses the F_2 frequency distribution was uni-modal, transgression resulted from the accumulation of resistance genes of the type associated with durable resistance. Also it should be noted that the absence of resistance segregants in the F_2 or F_3 indicates that these resistance genes did not have major effects; and also the larger population sizes and later generations and more severe disease pressure, might have facilitate to obtain individual(s) with more resistance than either parents. Transgressive segregation was reported by other workers (Pope 1968); Krupinsky and Sharp 1978 and 1979; Wallwork and Johnson 1984; and Grama *et al.* 1984). It should be noted that transgressive segregation does not necessarily imply additive gene action.

To sum up, infection type and latent period in wheat infected with *Puccinia striiformis* behaved genetically as a quantitatively inherited trait in that the progenies were continuously distributed (from high to low infection type or from shorter to longer latent period) and transgressive segregation occurred almost in all crosses. For both resistant x resistant or susceptible x susceptible crosses, the F_2 distributions were continuous but almost symmetrical and not skewed toward either parent. Distributions of individual plant values indicated that these segregating populations followed a normal distribution. This together with high heritability suggests that infection type and latent period were quantitatively inherited and not controlled by a single gene. In the resistant x resistant crosses, the highest levels of resistance obtained through transgressive segregation came from those cultivars which themselves possessed either the most resistance or the most susceptibility. This agreed with the results obtained by Allan and Purdy (1970). Infection type and longer latent period in Briscard, Ruapuna and Domino were partially recessive. Only Otane was dominant which had a shorter latent period in comparison with those cultivars. Partial recessiveness was reported by other researcher (Lewellen *et al.*, 1967; Pope, 1968; Krupinsky and Sharp, 1978; Röbbelen and Sharp, 1978; Krupinsky and Sharp, 1979; Wallwork and Johnson, 1984; Milus and Line, 1986a). These results indicate that resistance to stripe rust is recessive to susceptibility and support the conclusions of Milus and Line (1986b) who concluded that durable stripe rust resistance seems to be recessive rather than dominant. In other rusts similar results were reported. Lee and Shaner (1985a, b) reported that the inheritance of latent period in six slow-leaf-rusting wheat cultivars was controlled by recessive or partially recessive genes. Knott (1988) reported that for both stem and stripe rust of wheat, there is increasing evidence that polygenic resistance is recessive and it may take several genes to produce appreciable resistance. Recessive or partially recessive control of slow rusting has been reported for several rust-cereal interactions as well (Jacobs and Broers, 1989; Kuhn *et al.*, 1980; Luke *et al.*, 1975; Parlevliet, 1976 & 1978; Sharp *et al.*, 1976). If resistance is recessive, a hybrid breeding programme with those material would not produce good resistant genotypes. Nevertheless, hybridization between them followed by selection for higher resistance may be promising.

For the field experiments, the area under the disease progress curve distributions of F_2 and F_3 populations of all crosses, either susceptible x resistant or resistant x resistant, were continuous and only for crosses Briscard, Ruapuna and Domino with

Tiritea were thus skewed toward the resistant parent. In the crosses between susceptible cultivar, Tiritea, and resistant cultivars, transgressive segregation toward low area under the disease progress curve was observed, indicating that some gene(s) from Tiritea contribute to resistance. In crosses between two resistant cultivars, a high proportion of susceptible progeny was produced. This is consistent with the results obtained by Allan and Purdy (1970). Transgressive segregation toward resistance indicates that different genes for slow rusting resistance from different sources show additive effects. Thus, we should be able to develop greater levels of slow rusting by intercrossing diverse slow rusting cultivars to provide durability of resistance. If the additive gene action is due to interaction between alleles at different loci, as implied by the joint scaling tests, then transgressive segregation is expected. Transgressive segregation in this study supported the finding of Pope (1965) and Krupinsky and Sharp (1979). Wallwork and Johnson (1984) who reported transgressive segregation for adult plant resistance to stripe rust. These results are supported by other work (Milus and Line, 1986a; Allan *et al.*, 1963; 1966; Hendriksen and Pope, 1971; Lewellen and Sharp, 1968; Sharp *et al.*, 1976; Bjarko and Line, 1988a). These result also agreed with other evidence that slow rusting is inherited as a quantitative character (Gavinlertvatana and Wilcoxson, 1978; Johnson and Wilcoxson, 1979). However it is not yet clear to what extent the genes operating in one cultivar are identical with those operating in another, and there is no direct information concerning the corresponding genes which must be present in the rust (Manner, 1988). However, progress could be made in breeding work by selecting moderately slow rusting lines in the field using the area under the disease progress curve and making the desired crosses with these lines. This cycle could then be repeated until the desired lines had been developed.

5.5. Component of resistance

There is little information on inheritance of components of resistance in wheat in response to stripe rust. In an epidemic wherein cycles overlap and susceptibility often corresponds with the duration of the crop, two sequences greatly condition the rate of epidemic progression, namely the latent period and the sporulation. The latent period is the time, generally expressed in days, which separates germination and penetration from the appearance of new sporulating sori. This period depends on the climate, host, and the parasite itself. Both (latent period and the sporulation) sequences are generally

considered as factors of aggressiveness and not of virulence (Rapilly, 1979). Slow rusting wheat cultivars exhibit longer latent periods, smaller and fewer uredinia, and less spore production than susceptible cultivars. In the field, the effects of these slow- rusting components are cumulative over several infection cycles, resulting in slow rust development (Shaner and Hess, 1978). The latent period is highly correlated with disease development in the field and is the easiest component of slow rusting to measure in the greenhouse (Shaner, 1980; Shaner and Finney, 1980). Latent period is also the most important component of partial resistance (Zadoks, 1971; Shaner and Finney, 1980; Lee and Shaner, 1985a,b). Latent period has been reported as the component measured with least error (Kuhn *et al.*, 1978; Shaner *et al.*, 1978; Shaner and Finney, 1980). Therefore, it can be a good selection criterion in large scale greenhouse screening.

There is no published information on the inheritance of latent period in response to stripe rust, but there is some information about differences in latent period (Park and Rees, 1989; Cromey, 1992a). The present study provides information on the inheritance of latent period from crosses between one susceptible and four adult plant resistant cultivars. For the susceptible cultivar Tiritea, pustules began to appear about 10 days after inoculation whereas in adult plant resistant cultivars it was longer and in the F_2 plants of some crosses it reached to 19 days. Longer latent period also was reported by Park et al (1988). The effects of the slow-rusting character on the components of resistance may be expressed by longer latent period, restricted pustule size and fewer pustules per unit area. Since these experiments were carried out by different pathotypes at different time (over two months period), an attempt to maintain steady environmental factors was made, especially temperature (15±1) and light-intensity. One can conclude that differences observed between latent period were affected by environment rather than by host or pathotype, but it should be noted that the latent period of stripe rust depends mainly on temperature (Rapilly, 1979). The duration of the latent period does also depend on the number of infection sites, but can be affected by the physiological state of the host (Rapilly, 1979). However, it is presently difficult to take advantage of these differences to increase the longer latent period of varieties to obtain slow rusting epidemics, especially because as Fuchs (1972) reported that the behaviour of a race on a host is not always consistent; sometimes it is slow, sometimes it is fast in latency. Parlevliet (1975) measured latent period at different developmental stages and growing

conditions and stated that the relative latent period of the different cultivars was not affected by temperature. Other environmental factors, like day-length and light-intensity did not seem to have any effect either but the developmental stage of the plant and the genotype on the other hand influenced the latent period greatly and results can be reliable.

Latent period is an important part of the components of resistance (Johnson and Wilcoxson, 1978; Parlevliet, 1978; Shaner *et al.*, 1978). Latent period, the time between infection and production of secondary inoculum from that infection, is one of the main elements that controls the rate of development of wheat stripe rust. The variation in latent period on a leaf may be considerable, and it seems to be an inherent feature of the disease and not due to experimental error because it does not diminish under rigorously controlled conditions. One point should be noted that, in all experiments, the first day after inoculation on which any lesion produce secondary inoculum was characterized as the latent period and this approach did not use all the available data because it is clear that variance as well as mean values for latent period differ among host genotypes (see Shaner, 1980). To solve this problem, Shaner (1980) proposed the use of probit analysis, but in these genetic studies, large numbers of plants (more than 4000) had to be examined every day, and daily examination of all plants was difficult and time-consuming. It should be noted that in these experiments latent period was measured on the first leaves of adult plant resistant cultivars. To have a sound criterion for selection, latent period should be measured at the adult stage as well because Cromey (1992a) reported that the differences among cultivars in latent period were small at the seedling stage compared to those on flag leaves (also Parlevliet, 1975). In these experiments, the differences in latent period among the cultivars were quite significant and also the number of plants was a limiting factor to the measurement of latent period in the glasshouse.

5.6. Adult plant resistance

Various workers have warned of the genetic vulnerability of the established sources of seedling resistance to stripe rust (McIntosh, 1988). Adult plant resistance to stripe rust in wheat had been widely used (Stubbs, 1985) and in some cases it was eroded (Priestley, 1978; Stubbs, 1985; Caldwell, 1968) while in some cases it was

durable (Milus and Line, 1986a,b). In the interaction of wheat with *Puccinia striiformis*, race-specificity of resistance can also be found in adult plant resistance and is most readily detected in the adult stage (Priestley, 1978; Cromey, 1992a; Ezzahiri and Roelfs, 1989). So these cultivars should be tested with other pathotypes to assure their durability.

In the present study, the infection type and latent period were controlled by 1-4 recessive genes in Briscard, Ruapuna and Domino, whereas the area under the disease progress curve was controlled by 2-13 dominant genes. It can be interpreted that different genes are responsible for resistance in the seedling and adult stages or if they are the same, probably some modifying genes change the mode of gene action and this increases the number of genes. This finding agrees with Bennett (1981) who reported that adult plant resistance in wheat to powdery mildew does not correspond with resistance in seedling leaves, suggesting that different factors may sometimes be associated, but did not agree with Park and Rees (1989) who suggested that resistance in seedlings of cultivars with adult plant resistance may be governed by the same factor(s) that control adult plant resistance in them, or by some additional resistance factor(s) (also see Qayoum and Line, 1985). However, Cromey (1992a) stated that in adult plant resistant cultivars the differences in resistance between cultivars could be discerned at both seedling and adult growth stages. The differences were slight on the seedlings, where all susceptible and adult plant resistant cultivars had susceptible or moderately susceptible infection types, but differences in uredinium density were great enough to allow for some separation of cultivars. Dyck *et al.* (1966) concluded that studies on the inheritance of adult plant resistance are difficult for several reasons. First, the presence of genes for seedling resistance in most varieties masks the expression of adult plant resistance. Second, modifying genes seem to be very important in adult plant resistance and third, both the genes and their modifiers are very sensitive to environmental change.

5.7. Durability

In breeding for disease resistance, one objective may be to produce cultivars with durable resistance. Breeders are interested in durable resistance because it appears to be race non-specific or at least to be much more stable to changes in pathogen

virulence. Plant breeders generally agree that breeding for resistance should not depend solely on race-specific genes. Resistance that is race non-specific and controlled by a number of genes may be long-lasting because directional selection pressure on the pathogen will be minimal. Durable resistance is that resistance which has been adequate against the disease for a number of years over a range of environments and pathogen cultures (Johnson, 1978, 1981a, 1984). Johnson's definition does not necessarily imply future durability. It makes judgement of durability retrospective, and possible only when it is too late for practical breeding purposes. Johnson (1988) proposed that the best way to enhance the probability of achieving durable resistance in new cultivars is to transfer resistance from sources already identified as durable. As yet there is no simple way to identify the precise genetic components that are associated with the durable resistance to stripe rust of wheat, so the method is designed to retain as much of the resistance as possible from durably resistant sources. It should be noted that breeders are interested in all good (i.e. agronomic or physiologic) characters. For example transferring resistant gene(s) by backcrossing to Australian wheats, established sources of durable resistance, but most sources are poorly adapted to Australian wheat-growing conditions, and are red-seeded and stem rust susceptible (McIntosh and Wellings, 1986). Also, the occurrence of a pathotype of *Puccinia striiformis* in New Zealand with increased pathogenicity on some wheat cultivars with adult plant resistance such as Brock (Cromey, 1992b) emphasises the difficulty of recognising durable resistance to stripe rust amongst adult plant resistant cultivars. However, the use of resistance that has been effective over a range of environments, cultures, and years is certainly more likely to lead to a cultivar with durable than untested resistance or resistances that are known to have failed elsewhere.

In these experiments, it was attempted to study inheritance of resistance in locally adapted cultivars by measuring components of resistance. Durability of the genes involved in polygenic systems such as partial resistance (Parlevliet, 1988) or slow rusting (Wilcoxson, 1981) can be a highly desired property. Although partial resistance or slow rusting has been a stable trait over a relatively long period of time (Pope, 1965; Sharp, 1972), race-specificity was seen in them (Johnson and Taylor, 1972; Parlevliet, 1977a). However, according to Parlevliet (1976 and 1977a), the partial resistance is under polygenic control and has been quite stable. It is obvious that the long-lasting of a resistance, either monogenic or polygenic, is dependent upon the genetics of host-

pathogen interactions and not upon the genetics of resistance. The reasons for stability of polygenic general resistance is based in part upon genetic probabilities. Knott (1988) stated that resistance can be durable for two reasons, first, the pathogen cannot develop a highly virulent or aggressive race or, if one is produced, it is not competitive and second, a highly virulent or aggressive race, for whatever reason, does not come into contact with the resistant host. This is different from pyramiding of genes which is not durable (Johnson, 1988; van der Plank, 1968b). It should be noted that slow development of disease does not imply durability. It was pointed out by Johnson (1988) that slow development of disease (slow rusting) can be due to one of at least three possible causes: first, race-specific, adult plant or incomplete resistance, second, a low frequency of pathogenicity for a race-specific gene in a mixed population of races, so that cultivars possessing the gene receive a low frequency of matching infection, and third, slow rusting of a durable, apparently race-nonspecific type (also see Nelson, 1978). In these experiments, it can be concluded that the third reason most likely was the most important one. Slow development of disease, which appears to be race nonspecific and durable, has been found in wheat and efforts to find cultivars with this resistance have continued for the last several years (Milus and Line, 1986a,b; Luke *et al.*, 1972, 1975; Hughes and Hooker, 1971; Kontt and Padidam, 1988; Kim and Brewbaker, 1977; Gavinlertvatana and Wilcoxson, 1978; Parlevliet, 1978).

Numerous authors have reported that a higher level resistance than is present in the parents can be obtained from transgressive segregation in segregating generations, for instance in stripe rust (Krupinsky and Sharp, 1979; Wallwork and Johnson, 1984). Johnson (1988) pointed out that selecting genotype(s) with transgressive segregation can be obtained from different wheat crosses. It could therefore be a useful way of increasing resistance by crosses among locally adapted wheats, rather than trying to transfer resistance from unadapted sources thought to possess durable resistance. He mentioned that although this resistance will be race-nonspecific but it is not warranted. There is no proof that a race matching all potentially race-specific genes can be found to screen the segregating progeny.

Knowledge of the type of gene action involved in the expression of a character is helpful in deciding on the breeding procedures to be used for improvement of the character. Whereas dominance and some forms of epistasis would tend to favour the

production of hybrids, additive gene action signifies that standard selection procedures would be effective in bringing about advantageous changes in the character. These result demonstrated that the additive component was of major important and selection for higher resistance should be effective. also that this was supported by high heritability and genetic advance. Sources of durable resistance can be selected from crosses of moderately resistant and susceptible commercial cultivars. Since transgressive segregation for higher resistance occurred, some progeny from these crosses should have higher levels of resistance than the parents and would be a useful source of resistance in a breeding programme. This interpretation agreed with Krupinsky and Sharp, (1979) who stated that selection for significant resistance from crosses of susceptible commercial cultivars or cultivars with an intermediate level of resistance is a valid method for accumulating sources of resistance. Once the resistance is accumulated, it can be manipulated, as demonstrated with the additive, minor-gene lines. Elite progeny are likely to be frequent if all parental genotypes have satisfactory agronomic and quality performance. Thus, acceptable segregants could probably be selected from space-planted, advanced bulk populations of genotypes carrying resistant genes.

The study showed that selection among F_2 plants of almost all crosses should be highly effective for low infection type, longer latent period and low area under the disease progress curve. Estimates of heritability suggest the degree to which modification is possible through selection of resistant genotypes and a high estimate for expected genetic advance. Based on the result of gene action analysis in these crosses of wheat, it is suggested that simple recurrent selection or pedigree schemes for higher resistance may be effectively employed to isolate resistant plants from the population. It should be noted that selection for partial (polygenic slow rusting) resistance tends to select cultivars improved for several or even all the components (see Parlevliet and van Ommeren, 1975). There is no doubt that polygenic resistance is difficult to use in a routine breeding programme because it is impossible to select for if genes for specific resistance to the races being used are present. Second, the frequency of resistant plants in crosses is low and selection must be carried out over several generations and finally on a family basis. If breeders want to use polygenic resistance, they must be prepared to put considerable effort into it (Knott, 1988). The limited presence of dominance, [h], and duplicated epistasis would tend to retard the pace of progress through selection in early

generations. Thus, selection for stripe rust resistance would be more effective if the dominance and epistatic effects were reduced after a few generations of selfing.

The discussion on the inheritance of infection type ,latent period and area under the disease progress curve in response to stripe rust is in agreement with the literature (see Simons, (1975); Luke *et al.*, (1975); Lewellen *et al.*, 1967; Hendriksen and Pope, 1971; Sharp and Volin, 1970; Pope, 1968; Lupton and Johnson, 1970). Finally a breeding programme must be based on reliable selection criteria. Reliability is partly determined by the repeatability of result, so this needs to be more experiment on these cultivars carried out.

CONCLUSIONS

1. ANOVA showed that large genetic variation existed in cultivars for all components of resistance, even there were differences among cultivars with the adult plant resistance with respect to all components of resistance.

2. Multivariate methods were useful tools to ordinate cultivars across all attributes simultaneously and to ascertain patterns of response across pathotypes.

3. Adult plant resistant cultivars were ranked constantly for all attributes when tested with three pathotypes whereas those cultivars with known Yr gene(s) differed in ranking.

4. Pathotypes were different in aggressiveness on cultivars with respect to all components of resistance. In general, pathotype 232E137A- was more aggressive than other pathotypes.

5. Eight clusters were formed by cluster analysis. All seedling resistance, adult plant resistance and susceptible cultivars were in separate clusters.

6. Significant genotypic correlations were observed between components of resistance, suggesting that linkage between (or possible pleiotropic effects on) genetic factors controlling these components.

7. Components of resistance had higher correlations in response to pathotype with less aggressiveness than pathotype with more aggressiveness.

8. Heritabilities for infection type and latent period were higher than for pustule size and pustule density.

9. In general, whether for seedling resistant or adult plant resistant cultivars, additive component of variation was the major genetic component of resistance and this was substantiated by high narrowsense heritability values. Sometimes partial dominance was important and additive x additive epistatic played a minor role.

10. The changing of pathotype, whether for seedling resistant or adult plant resistant cultivars, led to changes in apparent gene action for some cultivars (especially those with known Yr genes), but for some other cultivars, the type of gene action was almost constant.

11. In crosses between cultivars with seedling resistance and those with adult plant resistance, the additive components of variation resulted mostly from seedling resistant cultivars.

12. In general, seedling resistance factors could mask contributions from adult plant resistant cultivars.

13. In response to one pathotype, the number of genes for each attribute was different from each other. Also in response to different pathotypes for any one attribute the gene number was different. This suggested that different factors were conditioning the components of resistance for each pathotype.

14. In all crosses, susceptible x susceptible, susceptible x resistant and resistant x resistant, transgressive segregation was observed in the F_2 and F_3 distribution for infection type, latent period and rust severity and in both directions, suggesting that genes in resistant cultivars were different from one another. Also it suggests the possibility of obtaining higher resistant genotypes from these crosses.

15. Generation means analysis indicated that the modes of gene action for infection type and latent period were different, suggesting different resistance factors controlled them and it agreed with the different number of genes for these two attributes.

16. The additive genetic component was major for infection type, latent period and rust severity. It was supported with high heritability for these attributes. In general, over ten crosses, heritability for infection type was higher than that for latent period and rust severity. High heritability for these attributes suggested that genotype-environment interaction was not important, under the conditions of this experiment.

17. Epistasis, especially additive x additive, was present for infection type, latent

period and rust severity, suggesting that resistance (low infection type, long latent period and low rust severity) was controlled by polygenes rather than monogenes.

18. In adult plant resistant cultivars, the number of genes at the adult stages for rust severity was far more than for the seedling stages, whether for infection type or latent period. It can be suggested that different effective factors controlled resistance in the seedling stage (low infection type and longer latent period) and the adult stage (low rust severity).

19. The mode of gene action of adult plant resistant cultivars in the seedling stage, whether for infection type or latent period, was recessive but in the adult stage, rust severity, was dominant.

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