GENETIC ANALYSES OF PERSISTENT ADULT-PLANT RESISTANCES TO WHEAT RUSTS

by

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Statement

This thesis contains no material which has been accepted for the award of any other degree or diploma in any university and, to the best of my knowledge and belief, the thesis contains no material previously published or written by another person, except when due reference is made in the text of the thesis.

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1. INTRODUCTION

Stem rust and leaf rust (incited by *Puccinia graminis* Pers. f.sp. *tritici* Eriks. and E. Henn., and *Puccinia recondita* Rob. ex Desm., respectively) of common wheat (*Triticum aestivum* L. em. Thell.) are important diseases in many areas where wheat is cultivated. Unless controlled by resistant cultivars, rusts may be limiting factors in production in some areas. The wheat industry in northern New South Wales and Queensland has been dependent on stem rust resistant cultivars for almost 30 years. However, during 1973-74 stem rust in southern Australia, a region not as rust prone as the north, caused the loss in wheat production estimated at $100-200 million (Anonymous, 1975).

The most economical method of reducing rust losses is to breed resistant cultivars. This biological control involves none of the problems associated with chemical residues. Initially, breeders utilized resistances conferring high levels of protection and controlled by single genes. Such resistances were readily manipulated during breeding and proved very effective in disease control but after relatively short periods the respective pathogens overcame the resistance genes by simple mutational or recombination changes. Because of repeated failures of these "specific" resistances, many pathologists and breeders have stressed the need to use "non-specific" resistance which, by definition, may offer more permanent protection. However difficulties are involved in distinguishing the two types of resistance particularly when the criteria by which they are defined vary. Generally, it

is accepted that non-specific resistance, is dependent on a multiplicity of host characters which tend to act in unison in permitting host plants to resist a pathogen. However, Hooker (1967) considers that an assurance of the long-term effectiveness of non-specific resistance cannot be given. This was supported by Caldwell (1968) who prefers to use the term "general" resistance and, more recently, by Johnson and Law (1975) who consider "durable" resistance to represent forms of resistance which have persisted despite exposure to pathogens over considerable periods of time.

Because the individual components of non-specific resistance may have small effects on the expression of it, special field and laboratory techniques may be necessary to recognise them. Several field designs were examined in the present work.

Subsequently, an attempt was made to identify and to analyse the genetic nature of the resistance in those cultivars whose resistance would fulfil some, if not all, of the criteria of non-specific resistance. After preliminary epidemiological studies of tetraploid and hexaploid wheats, it became clear that two groups required further investigation, those were the Hope-H44-24 group derivatives of Marquis/ *T. turgidum* L. cultivar Yaroslav emmer and the Marquillo-Double Cross group derivatives of Marquis/*T. turgidum* L. cultivar Iumillo durum. Subsequent genetic experiments were limited to a study of the inheritance of adult-plant stem rust resistance in these and related hexaploid wheats, under field conditions. Where appropriate, studies of seedling populations with *P. graminis tritici* and both adult-plant and seedling assessments with *P. recondita* were made.

In quoting examples of wheat genotypes which have resisted rust for long periods or which might have non-specific resistance, various authors (Anonymous, 1972; Watson, 1968; Caldwell, 1968) have implicated Hope and Marquillo, or their respective derivatives. However, certain reports in the literature in both the United States (Stakman and Christensen, 1953) and Australia (Watson and Luig, 1963; McIntosh *et al.*, 1967) suggested that the 'Hope' and 'Marquillo' derived stem rust resistances had become ineffective. On the other hand, later reassessments of these events suggested that breeders had in some instances over-reacted when certain individual specific gene components had been overcome by the stem rust pathogen.

2. LITERATURE REVIEW

2.1 NOMENCLATURE

A disease response is the result of the interactions of the products of host genes for reaction with those of the pathogen for virulence or avirulence. The genetic system in one organism cannot be investigated without simultaneous study of a corresponding system of the other. From studies of host/pathogen interactions, deductions may be made concerning features of both, or either organisms depending upon the particular circumstance, and various terms have been used to describe the different situations. Disease resistance may be divided into two broad categories: specific and non-specific (Watson and Luig, 1968).

2.1.1 Specific Resistance

When pathogenic isolates vary in ability to produce compatible and incompatible interactions with a particular host genotype, that genotype is said to show specific resistance. That is, the expression of reaction is specifically dependent upon the genotype of the pathogen. The genetics of such variation between isolates of the pathogen have been demonstrated by Flor (1956), Loegering and Powers (1962) and others (reviewed by Person, 1967). Resistance, or low reaction, develops only where the host carries the particular allele for resistance, where the pathogen isolate carries the corresponding allele for avirulence and where the environment is conducive. Any other combination of host and pathogen, or a non-conducive environment results in

susceptibility, or high reaction. Where a particular host possesses more than one gene for resistance and the pathogen isolate possesses the corresponding alleles for avirulence, the infection type is usually similar to, or more incompatible than, that typical of the most incompatible of the individual interactions. The low infection types are usually characteristic of the resistance alleles involved but may be modified to varying degrees by the action of modifying genes in the host (Hooker, 1967; Knott and Green, 1965), in the pathogen (Stakman *et al.*, 1962) and by the environment (Forsyth, 1956). Dominance and epistatic relationships can be determined (Hooker, 1967; Day, 1974) and the individual genes located on particular chromosomes (Sears *et al.*, 1957).

Specific resistance has been designated by an array of terms including, physiologic, seedling, hypersensitive, major gene, non-uniform, differential, mono/oligogenic, racial, race-specific and vertical resistance (Caldwell, 1968; Black, 1970; Van der Plank, 1968). According to Caldwell (1968) each of these terms is either inadequate or obscure in meaning and does not describe the interaction satisfactorily.

2.1.2 Stability of Specific Resistance

Considerable information and experience indicates that resistance controlled by single genes offers only ephemeral protection against pathogens (Watson, 1970a). Mutation is a character of all living organisms and when pathogen variants with virulence occur on a previously resistant host cultivar, that cultivar acts as a medium on which the new variants can increase without competition from the avirulent components of the pathogen population. Australian experience with the wheat

stem rust disease has shown that the pathogen is capable of accumulating virulence genes one at a time, by mutation (Luig and Watson, 1970). These workers therefore proposed that resistances based on gene combinations might be more stable than those based on single genes since multiple mutation in the essentially clonally propagated *P. graminis tritici* would be required in order to overcome resistance. This proposal assumed that all variants of the pathogen had at least two avirulence genes with respect to the host and that the genes constituting the multiple resistance were not deployed individually in other cultivars. While multiple gene resistances are being used in Australia, this latter requirement has not been enforced.

2.1.3 Non-specific Resistance

Non-specific resistance in a particular host genotype is expressed when pathogenic isolates do not vary significantly in their ability to produce incompatible interactions with it.

Terminology in the literature is confusing. As noted by Caldwell (1968), Thurston (1971), Van der Plank (1968) and Watson (1970a) various terms have been used to describe the concept of non-specific resistance e.g. field, partial, horizontal, non-hypersensitive, non-race-specific, generalized, general, tolerance, multigenic, polygenic, minor gene, multiple allele and quantitatively inherited resistance. Most of these terms attempt to describe either the physical nature of the disease, or the mode of inheritance of the resistance, but do not indicate the type of host-pathogen interaction involved. Furthermore, certain listed terms imply that

resistance effective against all strains is polygenically controlled. This may not be true in some instances (Robinson, 1973; Caldwell, 1968).

Robinson (1973) defined vertical resistance as resistance within the genetic capacity of the pathogen to produce virulent variants whereas horizontal was beyond this ability. A more practical term, "durable" resistance, was proposed by workers at the Plant Breeding Institute, Cambridge to describe resistance of lasting effectiveness in the field (Lupton, 1972). By their definition two Australian wheat cultivars Eagle and Kite each with gene *Sr26* have durable resistance. Robinson (1973) lists "durability" as one of six criteria of horizontal resistance. As the definition of non-specific resistance adopted here is essentially that of horizontal resistance (Van der Plank, 1968), durability is considered a criterion of non-specific resistance.

According to Caldwell (1968) the use of the term tolerance to imply general resistance is indefensible by definition. The definition of general resistance given by Caldwell (1968) is equivalent to the definition of non-specific resistance presented here. Caldwell *et al.* (1958) defined a tolerant genotype as one with the relative ability to sustain less loss in yield in the presence of a defined amount of disease. In a tolerant host, a compatible interaction occurs between host and pathogen.

While general resistance is a widely accepted term in the literature (Day, 1974) it does not describe the interaction adequately and may imply host resistance to a number of pathogen species (Hayes, 1973).

2.2 INHERITANCE OF NON-SPECIFIC RESISTANCE

The modes of inheritance of non-specific resistance range from polygenic through oligogenic to monogenic (Caldwell, 1968; Robinson, 1973).

2.2.1 Polygenic Inheritance

Based on theoretical considerations, several workers believe non-specific resistance to be inherited polygenically (Abdulla and Hermsen, 1971; Abdulla, 1971). This implies that no single host gene has an effect large enough to be individually identified and located within a chromosome (Van der Plank, 1968). By their additive nature, an accumulation of such genes in a single genotype may provide resistance approaching the strong interactions exhibited by specific resistances (Black and Gallegly, 1957; Black, 1970; Thurston, 1971; Lewellen *et al.*, 1967). In general, however, non-specific resistance could be conceived as the inhibition of the pathogen by prevention of its rate of increase (Umaerus, 1963; Knott, 1976).

Studies of possible non-specific resistance in wheat where polygenic inheritance patterns were implicated include the *T. aestivum*: *Puccinia striiformis* West system (Henriksen and Pope, 1971; Pope, 1968; Lupton and Johnson, 1970) and the *T. aestivum*: *Urocystis agropyri* (Preuss) Schröet. (flag smut) system (McIntosh, 1968). Pope (1968) entertained the possibility of segregation for as many as twenty "minor" genes in his materials. According to Lewellen *et al.* (1967), minor genes not only modify the expression of major (specific resistance) genes towards lower infection types, but collectively, the minor genes in the absence of major genes,

condition a useful level of resistance. Transgressive segregation for disease reaction was reported in the studies of Pope (1968).

Careful examination of host:pathogen systems is required before classifying such systems as polygenic. When studying the *T. aestivum:Erysiphe graminis* D.C. f.sp. *tritici* Marchal system in a controlled environment, Ellingboe (1975) was able to show that the resistance of Genesee expressed as slow disease development was conditioned by a dominant allele. Earlier studies under field conditions had failed to show the simple nature of inheritance.

2.2.2 Mono/Oligogenic Inheritance

There is evidence in certain instances of resistance with monogenic or oligogenic modes of inheritance which have remained effective over an extended period of time (Caldwell, 1968). Such resistances are controlled by genes having effects large enough to permit individual identication and location (Caldwell, 1968).

2.3 STABILITY OF NON-SPECIFIC RESISTANCE

Non-specific resistance may combine in one host a series of different genetic systems affecting a number of different stages of pathogen development (Watson, 1970a). Such a combination of mechanisms place before the fungus a sequence of mutational barriers which are more difficult to negotiate than the individual mechanisms when present alone (Watson, 1970a). Toxopeus (1956) believed that the genes controlling general resistance in potato to late blight, caused by *Phytophthora infestans* (Mont.) de Bary, are not different from the specific resistance genes except that many more are

involved. The pathogen, he suggests, will slowly accumulate virulence genes in order to overcome each host gene and consequently, the resistance will decline in effectiveness. Neiderhauser (1962) reported some decline in the level of resistance to late blight in Mexico. However, no sudden change in the level of resistance has occurred. In contrast, resistance to flag smut of wheat, incited by *Urocystis agropyri*, has remained effective in Australia since 1922 when it was first exploited (McIntosh, 1968). There has been no experimental evidence to suggest that changes in the flag smut pathogen have occurred.

An assurance of the long-term stability of a resistance considered to be non-specific cannot be given (Hooker, 1967). A monogenic resistance (*Ht1*) in maize, *Zea mays* L., to northern leaf blight disease incited by *Helminthosporium turcicum* Pass. and considered by Caldwell (1968) to be non-specific has recently failed in Hawaii and Australia (Colless, 1975; Hooker, 1976). A resistance which by definition is 'non-specific' at one time will become a specific resistance as soon as virulent variants of the pathogen are identified. However if such a relationship is not observed after years of testing in the field then the evidence in favour of the non-specific classification increases (Caldwell, 1968). These criteria for the qualification of non-specific resistance constitute tests of durability.

2.4 UTILIZATION OF NON-SPECIFIC RESISTANCE

2.4.1 Sources of Non-specific Resistance

Both wild relatives of cultivated crop species and cultivated forms may serve as a source of non-specific resistance genes (Watson, 1970b; Robinson, 1973). Robinson (1973) suggested that horizontal resistance occurs in all plants against all pathogens, but is inadequate in most cultivars. He contends that the accumulation of resistance genes in a single genotype would result in an adequate level of protection. In addition, he suggested, that the wild *Solanum* species have evolved a significant level of resistance in the region of Central and South America where *Phytophthora infestans* is prevalent, and where it is capable of producing a multiplicity of variants by both sexual and asexual processes. Specific resistance has proved completely inadequate in this environment.

The ease with which wild species can be used will depend upon cytotaxonomic relationships with the cultivated forms.

Valuable genes for non-specific resistance may exist in old landrace cultivars (Hooker, 1967; Lupton and Johnson, 1970) which were exposed to the pathogen for long periods of time. A number of cultivated crop species have been studied and shown to possess possible non-specific resistance. The diseases included maize rust incited by *Puccinia sorghi* Schw. (Hooker, 1967), wheat stripe rust (Lupton and Johnson, 1970; Pope, 1968) and oat crown rust caused by *Puccinia coronata* C. da. f. sp. *avenae* (Simons, 1975; Luke *et al.*, 1975).

2.4.2 Detection Techniques

2.4.2.1 Laboratory

Luke *et al.* (1972) measured "slow-rusting" on oat plants (*Avena byzantina* C. Koch) at both the first and third leaf stages in the greenhouse. Adult plants of oats grown in growth cabinets were tested by Luke *et al.* (1972) for crown rust resistance. Lewellen *et al.* (1967) studied the inheritance of stripe rust resistance in controlled environments using seedling plants. The quantities of spores collected from inoculated seedling leaves were used as a measure of resistance to stripe rust by Johnson and Bowyer (1975) and Johnson and Law (1975). Such laboratory tests should be useful in characterizing potential parents for breeding programmes, and distinguishing among relatively small numbers of advanced lines selected as possible cultivars, but as screening methods for large breeding populations, they would involve an impossible amount of work (Guzman, 1964).

2.4.2.2 Field

Field plot designs have ranged from replicated short row experiments (Luke *et al.*, 1975; Henriksen and Pope, 1971; Krull *et al.*, 1965) to split plot designs (Shaner, 1973). Krull *et al.* (1965) included both non-rusted (sprayed) and rusted replicates on which yield comparisons were made. Luke *et al.* (1972) staggered planting dates so that all cultivars would be at different stages of development when the epidemic commenced. Luke *et al.* (1972) suggested an association between "slow-rusting" and maturity. Both artificial epidemics (Luke *et al.*, 1975; Lupton and Johnson, 1970) and natural epidemics (Krull *et al.*, 1965; Henriksen and Pope, 1971; Shaner, 1973) have been employed. The percentage of diseased area of a

particular plant part was assessed at intervals from the commencement of the epidemic (Henriksen and Pope, 1971; Luke *et al.*, 1972, 1975; Shaner, 1973). Henriksen and Pope (1971) multiplied a diseased area rating by a pustule type index to obtain an infection index. Krull *et al.* (1965) classified oat lines into classes with large and small uredia, and statistically analysed grain yields and 200 grain weights on both classes.

2.4.3 Breeding Methodology

Van der Plank (1968), Clifford (1974), Hooker (1967), Lewellen *et al.* (1967) and Watson (1970b) recommend the breeding of cultivars in which genes for specific resistances are combined with those for non-specific resistance. Van der Plank (1968) portrays the role of specific resistance in delaying the start of the epidemic, and that of non-specific resistance in retarding its rate of increase. In addition, Hooker (1967) and Lupton and Johnson (1970) suggest that by appropriate hybridization and selection procedures, it should be possible to combine a high level of non-specific resistance with high yield and quality characteristics. The presence of genes for specific resistance could complicate selection for non-specific resistance, however, the effects of such genes could be removed by selecting only in lines lacking them, (Hooker, 1967) or by utilizing strains that are virulent for them (Watson, 1970b). Derived lines should be tested with the widest possible range of pathogen strains in both the greenhouse and field (Watson, 1970b). Specific resistance genes can be added to established lines by backcross procedures (Watson, 1970b).

Statistical analyses have frequently indicated that relatively few genes were involved in non-specific resistance (Hooker and Saxena, 1971; Luke *et al.*, 1975; Hooker, 1969). Additive genetic variance was of major importance and non-additive components were either absent or of small magnitude (Hooker and Saxena, 1971). Heritability estimates were generally high (Hooker, 1969; Luke *et al.*, 1975). Therefore relatively rapid progress would be expected by selection for resistance using simple plant breeding procedures.

2.5 ADULT PLANT RESISTANCE OF WHEAT CULTIVARS, HOPE AND H44-24

2.5.1 Inheritance of Adult-plant Resistance

The adult-plant stem rust resistance of H44-24 was shown by Goulden *et al.*(1928), Neatby (1931) and Goulden *et al.* (1930) to be genetically independent of its seedling resistances. Goulden *et al.*(1930) indicated that Hope was similar in behaviour to H44-24. Field data indicated that the adult-plant resistance of H44-24 was controlled by a single gene (Goulden *et al.*, 1928). Neatby and Goulden (1930) found that H44-24 in crosses with Marquis, carried a single dominant gene, but Hope possessed two complementary genes. Clark and Humphrey (1933) concluded that Hope carried two independently inherited genes for resistance, whereas H44-24 carried only one. From the cross H44-24/Minhardi, Quisenberry (1931) concluded that genes in addition to those expressed at the seedling stage were necessary to explain adult-plant resistance. A modifier genetic system was suggested by Clark and Humphrey (1933) to account for the variation in rust infection of near immune segregates, however, Clark and Smith (1935) could find no

evidence to establish this system, and attributed variation in infection to micro-environmental fluctuations. Ausemus (1934) indicated that the adult-plant resistance of Hope was independent of that in Marquillo. Pan (1940) concluded that Hope and H44-24 have a gene in common for adult-plant resistance.

The results of early genetic studies are difficult to interpret. Field tests were made in different years under natural, or artificial, epidemics in which the strain composition was not always known. Sears *et al.* (1957) reported that in many instances susceptible parents used in crosses had seedling resistances to at least some strains prevalent during the year of study. In the field, classification of segregating populations into distinct classes may not have always been possible as the heterogeneous data of Goulden *et al.* (1928) suggest.

Since the introduction of suitable cytogenetical techniques for wheat (Sears, 1953) and the publication of the gene-for-gene hypothesis (Flor, 1956), the basis of rust resistance in Hope and H44-24 has been somewhat clarified.

Using a series of chromosome substitution lines Sears *et al.* (1957) suggested that genes on Hope chromosomes 3B and 1D might, by complementary interaction, condition adult-plant resistance. Subsequent results showed that this hypothesis was not correct. Loegering and Powers (1962) indicated that low reaction associated with chromosome 1D (later identified as a single gene and designated *Sr18* (Baker *et al.*, 1970)) was present also in the Marquis parent.

Campbell and McGinnis (1958) studied Redman, an H44-24 derivative, and indicated that it carried three dominant,

complementary genes for adult-plant resistance to race 56, on chromosomes 3B, 4B and 2B.

The results of Sheen *et al.*(1968) suggested a single gene in Conley, a H44-24 derivative, controlling adult-plant resistance to race 15B. Minor genes were implicated in explaining variation within susceptible and resistant classes. Monosomic analysis of rust reaction of Conley implicated chromosomes 5A and 3B, whereas pseudo-black chaff reactions implicated only 3B. The authors concluded that chromosome 3B was responsible for the linked characterspseudo-black chaff and adult-plant resistance.

In backcrosses of Hope and H44-24 to Marquis, Knott (1968) found that both cultivars carry a single dominant gene, *Sr1* (=*Sr9d* (McIntosh, 1973)), for seedling and adult-plant resistance to race 56. However, adult-plant resistance to race 56 was conditioned by two genes, which Knott recognised as *Sr9d* and *Sr2*, following the recommendation of Ausemus *et al.* (1946). Gene *Sr2* enhanced the adult-plant, and seedling, expressions of *Sr9d* (Knott, 1968). In the presence of *Sr9d*, *Sr2* could be detected in the seedling stage. The full seedling resistance of H44-24 and Hope to race 56 was expressed only when both *Sr9d* and *Sr2* were present (Knott, 1968).

Following a study of certain Redman monosomic lines produced by McGinnis, McIntosh (1970) showed that the conclusions drawn by Campbell and McGinnis (1958) were not valid. McIntosh assumed that the seedling reaction of Redman to race 56 was similar to that of Hope, and further, that it was determined by the interaction of genes *Sr9d* on chromosome 2B and *Sr2* on chromosome 3B. From the seedling reactions of F_1 progenies of known somatic chromosome constitutions from

crosses of Redman monosomics 3B, 5B, 2B and 4B with Chinese Spring, it was apparent that the 3B line did not possess *Sr9d*, since hybrids were susceptible, and moreover, the alleged 2B line did not possess *Sr2*, since hybrids gave an intermediate reaction.

Knott (1971) grew the Hope substitution lines in the field and inoculated them with race 56. Line 3B was consistently more resistant than the other lines.

Working with the same substitution set, Brennan (1975) found lines 3B, 6B and 7B to have significantly reduced pustule numbers when inoculated with race 15B in the field. All lines were described as "susceptible" at both seedling and adult stages. Lines 2A, 5A, 7A, 2B, 3B, 6B, 7B, 2D and 3D had smaller reductions in grain weight than did Chinese Spring when rusted and non-rusted comparisons were made. In certain lines, reduced loss of grain weight could be attributed to decreased disease development. Brennan suggested that some lines may have genes conferring a degree of tolerance.

2.5.2 Seedling Resistances of Hope and H44-24

Hope and H44-24 gave differential seedling reactions when tested by Newton *et al.*(1929) and Goulden *et al.*(1930). Goulden *et al.*(1928) and Neatby (1931) showed that H44-24 carried two genes for seedling resistance to race 36. Neatby (1931) noted that only one gene was effective against certain other field strains. Ausemus (1934) established that Hope carried one gene for seedling resistance to race 36. In H44-24, Quisenberry (1931) found one gene for seedling resistance to races 60 and 36.

The only information on virulence is that extractable

from the Stakman differential key (Stakman *et al.*, 1962), but unless the host genes identified in Hope and H44-24 were those responsible for differentiating races, then data from different reports cannot be compared, even when the same race was used. Additionally, it cannot be assumed that all lines of a particular wheat stock were identical.

Sears *et al.*. (1957) tested seedlings of the Hope substitution series with 26 cultures. Lines 4B and 1D each possessed seedling resistance. Loegering and Sears (1966) showed that chromosome 4B possessed *Sr7b*, also present in Marquis, and hence could not account for resistance in Hope. The gene *Sr18*, on chromosome 1D, was also derived from Marquis.

McIntosh *et al.* (1967) located a recessive gene, *Sr17*, on chromosome 7BL of Redman and Hope. Knott (1968) identified a recessive gene effective against 15B-1L (Can) at the seedling stage in crosses involving Hope and H44-24 to Marquis. This gene was subsequently shown to be *Sr17* (Knott, 1971; McIntosh, 1970).

2.6 BREEDING OF HOPE, H44-24 AND THEIR DERIVATIVES IN NORTH AMERICA

Stem rust caused severe and widespread losses in the United States in the years 1904, 1916, 1919, 1920, 1921, 1923 and 1927. Marquis, a leading cultivar suffered severe damage in 1916 and succeeding epidemics. Realizing the seriousness of the problem, McFadden (1930) transferred stem rust resistance from a rust free tetraploid, Yaroslav emmer, to Marquis resulting in the named selections, Hope and H44-24.

Hope, released in 1926, had serious agronomic defects and

was not accepted by growers (McFadden, 1930; Clark and Bayles, 1942). H44-24 was not released as it was inferior to Hope with respect to agronomic characters (McFadden, 1930). Hence, further breeding was necessary. The first series of improved derivatives including, Apex, Rival, Pilot, Regent and Renown were selected from crosses made in 1926, and released in 1939 (Clark and Bayles, 1942).

Newthatch selected from the cross, Thatcher/Hope, was released in 1944 (Stakman and Harrar, 1957; Bayles and Clark, 1954), while Rescue, an Apex derivative, and Redman (Regent/Canus) were released in 1947 (Bayles and Clark, 1954).

The Hope adult-plant resistance has been effective in Nebraska since 1963 (Schmidt - personal communication to McIntosh, 1974). Under heavy spore showers, Scout 66 and Lancer become moderately infected, however rust develops near maturity but does not cause significant damage. Data from the 1974 International Spring Wheat Rust Nursery indicate that Selkirk wheat (McMurachy/Exchange//Redman*3) was resistant at 22 of a total of 24 sites. The average coefficient of infection was 8 on a scale ranging from 0 for the most resistant to 60 for the most susceptible genotypes. However, caution must be exercised when using such data especially where results from one or two sites are clearly different.

Under some conditions, the Hope adult-plant resistance may be inadequate. Abbott (1929) reported that Hope wheat developed considerable stem rust under field conditions in foggy areas of Peru. Selkirk has been heavily rusted in Puerto Rico (Stakman and Rodenhiser, 1958).

Pedigrees of wheats released in the United States and Canada to 1963 reveal that Hope, or H44-24, contributed to the

parentage of at least 40 cultivars (Bayles and Clark, 1954; Briggle and Reitz, 1963). International Maize and Wheat Improvement Centre (CIMMYT) breeders have used North American and Australian Hope and H44-24 derivatives extensively as stem rust resistant parents (Anonymous, 1972). Selections and cultivars from the CIMMYT programme have been distributed on a worldwide basis, and many of these have entered national breeding programs.

2.7 VIRULENCE CHANGES IN RELATION TO HOPE, H44-24 AND THEIR DERIVATIVES IN NORTH AMERICA

Three periods of virulence evolution can be recognised in North America; prior to 1934; 1934 to 1950 when race 56 was predominant; and since 1950 when the race complexes, 15B and 11-32-113, became dominant (Johnson and Green, 1957; Green, 1971).

Because Marquis was widely grown, the prevalent strains were virulent on *Sr7b* carried by Marquis. Newton *et al.* (1929) demonstrated that some field strains were fully virulent on Hope and H44-24 seedlings. However no strains were virulent on Hope and H44-24 at the adult-plant stage (Goulden *et al.*, 1930).

Hope and H44-24 seedlings were resistant to race 56 which became dominant in 1934. Consequently, no field losses were experienced (Peturson, 1958).

Although race 15B was identified in 1939 and was known to be virulent on seedlings of Hope and H44-24 (Knott, 1968), it remained insignificant until 1950 (Stakman and Rodenhiser, 1958). However, superior adaptation to the prevailing weather conditions (Katsuya and Green, 1967) combined with virulence

on *Sr9d* allowed race 15B to take full advantage of the late harvest and produce inoculum during 1950. 15B became well established in southern United States and was responsible for the massive epidemics of 1953, 1954 and 1955 which resulted in significant yield losses.

Not expecting a virulent race to become prevalent over most of North America in a single year, breeders became alarmed. Hope derivatives which had adequate protection to race 56 sustained damage. While not distinguishing the average severity of rust on Thatcher and Redman, Peturson (1958) found from 40 to 60% disease cover during the 1953 and 1954 epidemics in western Canada. Unaccustomed to infection levels of this magnitude, breeders believed that the bread wheats had succumbed completely to 15B. From yield data, a more reliable indicator of damage, from northern United States and Canada, it is evident that the bread wheat cultivars offered some resistance to 15B compared with durum cultivars (Stakman and Rodenhiser, 1958; Peturson, 1958). Maximum yield losses were estimated to be between 30 to 35% during 1953-4 for the bread wheat cultivars comprising Thatcher, Renown, Regent and Redman (Stakman and Rodenhiser, 1958; Peturson, 1958), whereas durum losses over the same period ranged from 75 to 100%. Furthermore, since all the bread wheat cultivars were susceptible to leaf rust, portion of the yield loss must be attributed to this disease as it also, was prevalent (Peturson, 1958). Stakman and Rodenhiser (1958) considered that the durum cultivars Mindum (*Sr9d*), Stewart (*Sr9e*) and Carleton had only specific resistance which 15B overcame, but the Hope derivatives apparently possessed 'general' resistance in addition to specific resistances. By 1955, yield losses

were substantially reduced (Peturson, 1958), following the widespread cultivation of Selkirk whose resistance was essentially that of Redman with *Sr6* added (Green, 1971).

In 1953, a variant of 15B virulent on Selkirk seedlings was identified (Green, 1971). Subsequently, a number of variants within standard races with seedling virulence for Selkirk were identified, but none became a significant component of the *P. graminis tritici* flora in North America (Green, 1971).

Despite the early concern, there are no recent reports detailing strains of stem rust fully virulent on adult-plants of Hope, H44-24 and derivatives.

2.8 BREEDING OF AND VIRULENCE CHANGES IN RELATION TO HOPE, H44-24 AND THEIR DERIVATIVES IN AUSTRALIA

Considerable use has been made of Hope and H44-24 in the breeding of Australian cultivars. Waterhouse made his first cross with Hope in late 1926 (Waterhouse - personal records). A breeding programme was commenced in 1929 at the Waite Agricultural Research Institute, South Australia, having as one of its objectives, the transference of the Hope resistance to Australian cultivars (Phipps *et al.*, 1943). Waterhouse at the University of Sydney released Hofed, while Warigo, Glenwari and Panther were developed in South Australia (Macindoe and Brown, 1968). A series of derivatives then followed - Lawrence (1944), Spica (1952), Hopps and Gala (1960) from Queensland and Mendos (1964) from New South Wales (Macindoe and Brown, 1968).

Until strain 21-Anz-0 appeared in 1954, all Hope derivatives were highly resistant to stem rust as adult-plants, but

suceptible (or moderately susceptible) as seedlings (Watson and Luig, 1963; Phipps *et al.*, 1943). Therefore, the Hope adult-plant resistance protected these cultivars since most were released prior to 1954. During the period 1927 to 1941, selection for Hope resistance was conducted exclusively in the field, as seedling tests were useless (Watson and Luig, 1963).

Strain 21-Anz-0 was avirulent on Hope and derivatives as seedlings (Watson and Luig, 1963). An additional genetic system operated against this strain to confer resistance on seedlings as well as adult plants (Watson and Luig, 1963). Watson and Luig (1963) found that Spica resembled Hope derivatives when seedlings were inoculated with strains 126-Anz-6,7,11 and 21-Anz-0. As the leaf rust and powdery mildew resistances of Spica also paralleled those of Hope, H44-24 and derivatives, the published pedigree was questioned (McIntosh *et al.*, 1967). The recessive gene, conferring seedling resistance, was designated *Sr17* (McIntosh *et al.*, 1967). With the exception of Hopps, all Australian Hope derivatives possessed *Sr17* (McIntosh *et al.*, 1967). When the majority of these were selected, however, during the 1940's, *Sr17* was ineffective. Although *Sr17* is linked with leaf rust and powdery mildew resistances (genes *Lr14a* and *Pm5* respectively: McIntosh *et al.* (1967)), it is unlikely that selection for either (or both) of these resistances could account for the presence of *Sr17* in so many instances. Possibly, *Sr17* enhanced the expression of other genes involved in resistance to such strains.

Strain survey data indicate that Spica, the leading Queensland cultivar from 1954-1956, maintained its resistance

to field strains, predominantly 21-Anz-0 and later 21-Anz-2. During 1957-58, a Spica-attacking strain, 21-Anz-5, was detected. This strain was virulent on seedlings of Hope, H44-24 and derivatives (Watson and Luig, 1963). McIntosh *et al.* (1967) showed that 21-Anz-5 was virulent on plants having *Sr17*. With the widespread cultivation of Spica and Gala, strain 21-Anz-5 became abundant in Queensland, displacing the avirulent strains 21-Anz-0 and 21-Anz-2 (Watson and Luig, 1963). Early maturity and alleged adult-plant resistance (McIntosh - personal communication) have enabled Spica to escape significant damage in recent years. Further south in N.S.W., Glenwari, carrying *Sr17* alone, was withdrawn due to poor baking quality before *Sr17* attacking strains became prevalent. Other Hope derivatives became unpopular or were never grown to any extent.

2.9 PSEUDO-BLACK CHAFF OF HOPE, H44-24 AND DERIVATIVES

Pseudo-black chaff (Broadfoot and Robinson, 1933) of Hope, H44-24 and derivatives has been variously designated black chaff (Hayes *et al.*, 1934), brown necrosis (McFadden, 1939) internodal melanism (Hagborg, 1936) and melanism (Johnson and Hagborg, 1943). As pseudo-black chaff is the term adopted by the Fourth International Wheat Genetics Symposium (McIntosh, 1973), it will be used in this thesis.

The name "black chaff" was used by Smith (1917) to describe a disease caused by *Bacterium translucens* Jones, Johnson and Reddy var. *undulosum* Smith, Jones and Reddy. Later, Smith (1920) changed the name to "bacterial black chaff".

In a list of undesirable characteristics of Hope and H44-24, McFadden (1930) reported the presence of pseudo-black

chaff which he had also noted on the emmer cultivars, Vernal and Yaroslav. Several investigators have reported that Hope, H44-24 and derivatives are very susceptible to attack by the bacterial organism (Hayes *et al.*, 1934), however others expressed uncertainty as to the cause of the condition (Goulden *et al.*, 1929). Upon superficial examination, discoloured stems and glumes resembled the bacterial black chaff disease (Broadfoot and Robinson, 1933), however, a closer inspection revealed an absence of bacteria.

Waldron (1929) reported an "antagonistic relationship" between pseudo-black chaff and stem rust development in hybrids of Hope. On the other hand, Hart and Zaleski (1935) found no antagonistic relationship between stem rust infection and bacterial black chaff.

The aetiology of pseudo-black chaff was investigated by Broadfoot and Robinson (1933), Hagborg (1936), McFadden (1939) and Johnson and Hagborg (1943). Hagborg (1936) and McFadden (1939) were not successful in isolating a causal organism. Various environmental factors have been shown to affect pseudo-black chaff expression (Johnson and Hagborg, 1943), which include high light intensity (McFadden, 1939), high humidity and, more particularly, high temperature (Johnson and Hagborg, 1943) and stem rust inoculum density (McFadden, 1939).

Goulden (1929) and McFadden (1939) intimated very close or complete linkage between stem rust resistance and pseudo-black chaff after repeated failures to break the linkage. Pan (1940) believed this linkage could be broken. Sheen *et al.* (1968) and Kuspira and Unrau (1958) reported that pseudo-black chaff was controlled by a single recessive gene located on chromosome 3B.

2.10 RESISTANCE OF MARQUILLO AND DERIVATIVES

The resistance of Marquillo, Celebration and Thatcher or, the sister line, Double Cross, has been studied by a number of workers. Hayes *et al.* (1925) studied a cross involving a Marquillo sib and a Marquis/Kanred selection, and concluded that the field resistance of the first parent was conditioned by two complementary recessive genes, each inherited independently of the seedling immunity (*Sr5*) of the Marquis/Kanred parent. In the cross Marquis/Iumillo, Thompson (1925) had shown that the resistance of Iumillo was conferred by more than one gene. Neatby *et al.* (1930) reported that field resistance of Marquillo was determined by three or more factors. When Garnet and Reward were used as susceptible parents, many genes appeared to be involved. Neatby (1931, 1933) showed the Marquillo and Double Cross do not possess any genes affecting field resistance additional to those concerned with seedling resistance. Ausemus (1934) interpreted the inheritance of field resistance in Marquillo on the basis of at least three genes. From field studies, Pan (1940) concluded that Double Cross carries two complementary genes for semi-resistance. The field resistance of Thatcher was attributed to the action of at least two recessive genes for resistance by Swenson *et al.* (1947), and to two complementary genes by Koo *et al.* (1951). Marquillo and Thatcher were found to behave similarly to Australian strains and resistance in each case was conferred by a number of genes (Athwal and Watson, 1955). One of the genes was shown to be closely linked with a locus conditioning resistance in Kenya 117A and later identified as *Sr9b*. McIntosh (unpublished) has isolated a gene (*Sr9g*) in chromosome 2B of Thatcher. This gene is also carried by

Marquillo and Celebration and is allelic with *Sr9b* (McIntosh - unpublished). From strain survey results, Luig (unpublished) found that low reaction in the tetraploid international race differentials, Acme and Kubanka, was correlated with that in Celebration. The studies of Sears *et al.* (1957) implicated three chromosomes, 3B, 2B and 6D, in the seedling resistance of Thatcher. Furthermore, they obtained evidence of additional genes that modify rust resistance on chromosomes 6A, 5A, 4D and 1D. Data from the cross Thatcher/Chinese Spring, indicated two recessive genes determining resistance to race 56 (Sears *et al.*, 1957). Chromosome 6D of Thatcher carried *Sr5* (Ausemus *et al.*, 1946).

Sheen and Snyder (1964) produced all twenty-one chromosome substitution lines from a plant which was initially thought to be Marquis but later shown to resemble Thatcher. The Marquis 2B line carried two genes for resistance, one of which was shown to be allelic with *Sr9b*. In the cross, Thatcher 2B/Kenya Farmer 2B, Loegering and Sears (1966) found two independently inherited genes. They suggested, that one of the genes in the Marquis 2B line of Sheen and Snyder might be the same as that in Thatcher 2B. The gene independent of the *Sr9* locus was designated *Sr16*.

A dominant allele for resistance on chromosome 3B of Thatcher was assigned the previously deleted designation *Sr12*, which was closely linked with a second recessive allele conditioning a mesothetic reaction to four rust cultures (Sheen and Snyder, 1964). McIntosh (unpublished) has referred to a recessive gene in Celebration, an Australian Double Cross derivative, as *Sr12*, but it appears that the latter has more in common with the unnamed mesothetic factor described above.

Substitution lines in which chromosomes 6A and 2B of Thatcher had been transferred to Chinese Spring, produced significantly less pustules than Chinese Spring (Brennan, 1975).

2.11 BREEDING OF MARQUILLO, THATCHER AND DERIVATIVES IN NORTH AMERICA

Prior to 1917 no completely stem rust resistant cultivars of hexaploid wheat were available (Hayes *et al.*, 1936). Accordingly, crosses were made between common wheats and highly resistant cultivars of *T. turgidum* L., one of which was the Italian cultivar, Iumillo. One Marquis/Iumillo Selection was named Marquillo and another was a parent of Thatcher. In the double cross, Marquis/Iumillo Selection//Kanred/Marquis, selection for adult-plant resistance to many strains was conducted in the field, and resistant lines were tested in the seedling stage for the presence of *Sr5*. Disease assessment trials, involving exposure to all available field strains in artificial epidemics, conducted over seven years showed that Thatcher (average % stem rust infection based on Cobb Scale was 9%) proved more resistant than Ceres (34%) and Marquis (54%) but less resistant than Hope (1%) (Hayes *et al.*, 1936). Thatcher was susceptible to leaf rust (average over 4 years - 59%) while Hope was resistant (4%) (Hayes *et al.*, 1936).

Thatcher proved to be resistant to stem rust in the severe epidemics of 1935 and 1937, and its acreage increased very rapidly, both in the United States and Canada, where it became the leading cultivar (Clark and Bayles, 1942), following severe losses of Marquis and Ceres (Stakman and Harrar, 1957).

Although resistant to stem rust, Marquillo remained a

minor cultivar, owing to a yellow flour defect (Clark and Bayles, 1942).

Believing that Thatcher may become susceptible to certain races under some conditions, breeders crossed it with Hope to broaden the genetic base and to introduce the leaf rust resistance of Hope (Stakman and Harrar, 1957). Newthatch (Hope/3*Thatcher) was released in 1944 (Stakman and Harrar, 1957).

Although Thatcher and derivatives sustained damage during the 1953, 1954 and 1955 epidemics, Green (1971) believed that these derivative cultivars would have been more severely affected had they not possessed "adult-plant resistance inherited from Thatcher".

2.12 VIRULENCE CHANGES IN RELATION TO MARQUILLO, THATCHER AND DERIVATIVES IN NORTH AMERICA

Since the commencement of rust surveys in Canada in 1919, races virulent on Reliance (*Sr5*, *Sr16*) and on Acme and Kubanka (*Sr9g*) have been prevalent (Johnson and Green, 1957; Green, 1971). Thus resistance genes other than *Sr5* and *Sr9g* must have protected Thatcher and its derivatives. No strains that could attack either Thatcher or its derivatives occurred in significant amounts until 1952,when 15B-1 became common (Peturson, 1958).

Race 15B-1 apparently attacked Thatcher and derivatives, but yield data for the epidemic years of 1953, 1954 and 1955 suggested that a degree of resistance remained (Stakman and Rodenhiser, 1958).

Recently in Canada, strains described as "moderately virulent" on the Thatcher derivatives,Manitou and Neepawa,

were isolated (Green, 1971). However, Green considered these strains did not appear to be sufficiently 'aggressive' on the Thatcher derivatives to cause significant damage (Green, 1971), but further increases in the number of genes for virulence with respect to Thatcher types could seriously challenge these cultivars.

2.13 BREEDING OF AND VIRULENCE CHANGES IN RELATION TO DOUBLE CROSS AND DERIVATIVES IN AUSTRALIA

Australian commercial cultivars Celebration, Windebri, and apparently Gatcher, carry the Marquillo adult-plant resistance (Luig and Watson, 1961; Macindoe and Brown, 1968; Luig - personal communication). Celebration was released in 1945, Windebri in 1959 and Gatcher in 1969 (Macindoe and Brown, 1968).

Prior to 1954, prevalent strains were highly avirulent on seedlings of Marquillo and the Double Cross derivatives but virulent for *Sr5*. After 1954 components of standard race 21 were highly avirulent on plants with *Sr5* but considered to be virulent at normal greenhouse temperatures on Marquillo and the Double Cross derivatives lacking *Sr5* e.g. Celebration and Windebri. Iumillo durum gave low reactions to these variants. Subsequently, two components of standard race 34 evolved, both differed from standard race 21 in being virulent on plants with *Sr5*, but one produced lower reactions on seedlings of Celebration and Marquillo than the other (Luig and Watson, 1976). During 1969 and later, variants of standard races 194, 222, 326 and 343 avirulent on Acme, Kubanka and Celebration were isolated. Various collections from Windebri, identified as 21-Anz-9, are distinguishable from most standard race 21 components by high seedling reactions on Iumillo in addition

to Marquillo and Celebration at all temperatures (Luig - personal communication).

3. MATERIALS AND METHODS

3.1 DESIGNATION OF PEDIGREES, ANEUPLOIDS AND CHROMOSOME SUBSTITUTION LINES

The pedigree designation system of Purdy *et al.* (1968) is adopted throughout this thesis. Aneuploid designations and symbolizations follow those proposed by Kimber and Sears (1968). Intervarietal chromosome substitution lines are designated by the recurrent parent, followed by the donor and specific chromosome in parenthesis. For example, Chinese Spring (Hope 3B) or CS(Hope 3B) refers to a Chinese Spring line in which the chromosome 3B pair is replaced by the homologous pair derived from Hope.

3.2 DESIGNATION OF *P. GRAMINIS TRITICI* AND *P. RECONDITA*

Throughout this thesis *P. graminis tritici* and *P. recondita* entities will be designated by the strain formulae used by the Plant Breeding Institute. For both pathogens this is a two-part formula based on the International "standard races" followed by a series of numbers which refer to virulence on supplementary differential genotypes used in the Australasian region. For *P. graminis tritici*, 11 supplementary differentials (Table 1A) are used (Watson and Luig, 1963, 1966; Luig and Watson, 1976). Strain 21-Anz-1,2,5, for example, is standard race 21 on the key of Stakman *et al.* (1962) but, in addition, is virulent on seedlings of McMurachy, Yalta and Renown carrying *Sr6*, *Sr11* and *Sr17*, respectively. By implication, this strain is avirulent with respect to seedlings of the remaining 8 Australasian differential

TABLE 1

Australasian supplementary differentials used in strain classification.

A. *P. graminis tritici*

Differential No.	Cultivar	Accession No.	2n=	Main differentiating gene in Australasia
1	McMurachy	W2086*	42	*Sr6*
2	Yalta	W1373	42	*Sr11*
3	Marquis/Kenya 117A	W2402	42	*Sr9b*
4	C.I. 12632	W1656	42	*SrTt1*
5	Renown	W3125	42	*Sr17*
6	Mentana	W1124	42	*Sr?*
7	Norka	W578	42	*Sr15*
8	Festiguay	W2706	42	*SrW*
9	TAF 2**	W3592	42	*SrAg*^{I}
10	Entrelargo de Montijo	W3560	28	unknown
11	Barleta Benvenuto	W3502	42	unknown

B. *P. recondita*

1	Thew	W203	42	*Lr20*
2	Gaza	W277	28	*Lr23*
3	Spica	W2341	42	*Lr14a*
4	Kenya	W1483	42	*Lr18*
5	Klein Titan	W1633	42	*Lr3KA*
GT	Gatcher	W3201	42	*LrGt*

W numbers refer to the Sydney University Wheat Accession Register.

** *Agr. intermedium* substitution line.

TABLE 2

Pathogen strains, with culture accession numbers, used in seedling and adult-plant studies.

A. *P. graminis tritici*

Strain Designation	Culture No.
21-Anz-0	54129
21-Anz-2	69490
21-Anz-5	65103
21-Anz-9	71178
21-Anz-1,2,5	65020
21-Anz-1,2,3,7,11	70180
21-Anz-1,2,3,7,8,9	731168
21-Anz-2,3,4,5,7	68016
21-Anz-2,3,4,5,6,7	68-L-3
21-Anz-(1)*,2,3,4,5,6,7	66-L-1
34-Anz-1,2,3,5,6 Y (yellow urediospore variant)	66-L-8
34-Anz-1,2,3,6,7	66-L-2
34-Anz-2,4,5,7,11	64231
34-Anz-1,2,3,4,5,6,7	75-L-1
116-Anz-4,5	64726
116-Anz-2,3,7	61352
126-Anz-6,7,11	334
194-Anz-1,2,3,5,6	691042
22-Anz-2,3,7,8	71107
222-Anz-1,2,3,5,6	70-L-2
222-Anz-1,2,3,4,5,6,7	70-L-5
326-Anz-1,2,3,5,6	69822
343-Anz-1,2,3,5,6	73879
TAN (standard race not designated -5,7)	56-E-1
59-E-5,7	59-51A

* Brackets represent incomplete virulence with respect to *Sr6*.

B. *P. recondita*

68-Anz-1,2,3,4,	63312
104-Anz-1,2,3,GT	74606
122-Anz-1,2,3	66-L-1
162-Anz-1,2,3,GT	70201

Except for strains TAN and 59-E-5,7 which originated from the U.S.A., the *P. graminis tritici* and *P. recondita* strains were accessioned from Australian field collections or laboratory mutation experiments of the Plant Breeding Institute.

genotypes. A similar system is used to designate strains of *P. recondita* (Watson and Luig, 1961). In this case, six supplementary differentials are in current use, five listed by these authors and Gatcher (Table 1B). The culture numbers of strains used in the current studies are also listed in Table 2.

3.3 DISEASE ASSESSMENT METHODS

The study of non-specific resistance requires the development of suitable field procedures. It was originally assumed that non-specific resistances would be difficult to detect and would show complex inheritance. Field experiments with the aims of a) developing suitable procedures for the detection and measurement of adult-plant resistance and/or tolerance, and b) the evaluation of potential parent cultivars for future study were conducted at Castle Hill in 1972 and 1973. Small plots of various genotypes were assessed for the rate of disease increase (cumulative disease increase over time) and for 1,000 grain weights of diseased versus non-diseased controls for the same genotype. Infection in these plots originated from inoculated border rows outside the experimental areas. The cultivars examined, listed in Table 3 were grouped according to pedigree or other information.

3.3.1 Hope and H44-24 Derivatives

Published and unpublished information accumulated at this, and other, institutions indicates that Hope, H44-24 and certain derivatives possess adult-plant stem rust resistance that has been, and remains, effective. This resistance was possibly associated with chromosome 3B (Knott, 1971). McIntosh (personal communication) has suggested that Spica must possess

TABLE 3

Cultivars included in adult-plant and seedling studies during 1972, 1973, 1974 and 1975.

Cultivar	Source of Accession where known	Known specific resistance genes		Pedigrees where relevant
		P. gr. tritici	*P. recondita*	
Susceptible Controls				
Chinese Spring	W1806			
Federation	W107		*Lr10*	
Yalta	W1373	*Sr11*		
Line E	W3498			
Svenno	W3623			
Gabo	W1422	*Sr11*	*Lr10*	
Hope and H44-24 derivatives				
Africa Mayo	W3315			Africa/3/Marroqui/2/Hope/Thatcher
Aotea	W2462	*Sr17*	*Lr14a*	Hofed/Cross7//Cross7/Dreadnought
Apex	R.Larson			H44-24/3/Marquis/Iumillo// Marquis/Kanred
Arthur 71				
Cadet	R.Larson			Merit/Thatcher
CS(Hope 3B)	McIntosh			
CS(Hope 7B)	McIntosh	*Sr17*	*Lr14a*	
Gala	W2552	*Sr11 Sr17*	*Lr14a*	Lawrence/Gabo
Glenwari	W1991	*Sr17*	*Lr14a*	Nabawa//Riverina/Hope
Hope	W517	*Sr7bSr9dSr17*	*Lr14a*	Marquis/Yaroslav Emmer
Hofed	W1361	*Sr17*	*Lr14a*	Hope/Federation
H44-24	W1846	*Sr7bSr9dSr17*	*Lr14a*	Marquis/Yaroslav Emmer
Hopps	W2517			Hope/Seafoam/*2/Pusa/Flora3202
Hopps/Federation F4 Selection	L64.211.1.13			
Kenya Page	W3316			
Lancer		*Sr17 Sr9d*		

TABLE 3 (CONT)

Cultivar	Source of Accession where known	Known specific resistance genes		Pedigrees where relevant
		P. gr. tritici	*P. recondita*	
Lawrence	W2048	*Sr7bSr9dSr17*	*Lr14a*	Florence/College
Lerma Rojo 64A	W2977			Yaqui50/N10-B//Lerma52/ Lerma Rojo*2
Redman	W1942	*Sr7bSr9dSr17*	*Lr14a*	H44-24/Reward//Canus
Rescue	R.Larson			Apex/S-615
Renown	W1242		*Lr14a*	H44-24/Reward
Renown	W2346		*Lr14a*	H44-24/Reward
Renown Selection	W3125	*Sr7bSr9dSr17*	*Lr14a*	H44-24/Reward
Samaca				Bonza/Africa Mayo*2
Scout 66		*Sr17Sr9d*		Nebred/2/Hope/Turkey/3/ Cheyenne/Ponca
Selkirk	W2699	*Sr6,Sr7b,Sr9d,Sr17, Sr23*	*Lr10Lr14aLr16*	McMurachy/Exchange//Redman*3
Spica	W2341	*Sr7bSr17*	*Lr14a*	Seafoam/Kamburico//Pusa/ Flora 3202
Warigo	AUS1634	*Sr7bSr17*	*Lr14a*	Nabawa/Hope
Yaqui 50	W2304			Marroqui//Hope/Thatcher
Iumillo derivatives				
Celebration	W1374			Marquis/Iumillo//Marquis/ Kanred/3/Dundee*2
Marquillo	W724			Marquis/Iumillo
Lee	W2084	*Sr11*	*Lr10Lr23*	Hope/Timstein
Thatcher	W1201	*Sr5Sr9gSr16*		Marquis/Iumillo//Marquis/ Kanred
CS(Thatcher 2B)	McIntosh	*Sr9gSr16*		
CS(Thatcher 3B)	McIntosh	*Sr16*		
I*Sr16*Ra	McIntosh	*Sr16*		
Minnesota 12	Luig			
Pato				Tezanos Pinto Precoz/ Sonora64A/Narino59

TABLE 3 (CONT)

Cultivar	Source of Accession where known	Known specific resistance genes		Pedigrees where relevant
		P. gr. tritici	*P. recondita*	
Pato Argentina				
Tezanos Pinto Precoz				
Tobari 66	W3176			Tezanos Pinto Precoz/ Sonora 64A
Others				
Exchange	W1781	*Sr23*	*Lr16*	Warden/Hybrid English
Warden	W217	*Sr23*	*Lr16*	
Acme	W9			
Kubanka	W8			
Australian commercial cultivars				
Gamenya	W2550	*Sr9b*		
Gamut	W3123	*Sr6Sr9bSr11*		
Mengavi	W2551	*SrTt1Sr11*		
Wren	W3002			
Miscellaneous hexaploid cultivars				
Florence	W1844			
Kota	W2024	*Sr7b*		
C273	W3151			
Seafoam	W1221			

TABLE 3 (CONT)

Cultivar	Source of Accession where known	Known specific resistance genes		Pedigrees where relevant
		P. gr. tritici	*P. recondita*	
Tetraploid cultivars				
Amber durum	W1922			
Bansi Strain 168	W990			
Dural	W2738			
Glossy Huguenot	W304			
Kubanka	W575			
Mahrook	W1541			
Marouani	W308			
Marrocos	W2886			
Palestine 2649	W320			
Portugal 24	W905			
Russian 1364/3	W831			
TH559	W1570			
World Collection No.503	W2472			

some adult-plant resistance because it is resistant to strain 126-Anz-6,7,11, which is virulent on plants having *Sr7b* and *Sr17*. The latter virulence should be noted because it is not reflected in the strain formula. Under Australian conditions, Spica and Glenwari are considered susceptible to at least some strains, and were the most commonly grown Hope derivatives when *Sr17* virulent strains were first found in 1959.

3.3.2 Iumillo Derivatives

The hexaploid derivatives of Iumillo are known to have some resistance to many *P. graminis tritici* strains on a world-wide basis. Lee was included in this group rather than the Hope group. Although the parentage of Lee suggests Hope, it possesses none of the known *Sr* genes of Hope. McIntosh (1976 - personal communication) has shown that Lee carries *Sr9g* and *Sr16* in addition to *Sr11*, suggesting involvement of Thatcher, or a related type in its breeding.

3.3.3 Exchange and Warden

Green (personal communication to McIntosh) suggested that Exchange may possess adult-plant resistance to at least some strains. Warden was included as it is one of the parents of Exchange.

3.3.4 Australian Commercial Cultivars

This series of cultivars were included to allow an assessment of adult-plant resistance and/or tolerance in current and recently produced commercial cultivars.

3.3.5 Miscellaneous Hexaploid Cultivars

Luig (personal communication) suggested that Kota may possess some adult-plant resistance. Florence, a Farrer

crossbred, was alleged to have adult-plant resistance (Arnold - personal communication), while Srivastava (1968) indicated that the Indian cultivar, C273, possessed tolerance to rusts.

3.3.6 Susceptible Hexaploid Control Cultivars

Yalta carries *Sr11* but exhibits the "Vertifolia" effect (Van der Plank, 1968) relative to Gabo, which also carries *Sr11*. Clifford (1974) extended the meaning of "Vertifolia" effect to cover comparisons such as this.

3.3.7 Tetraploid Cultivars

Tetraploid wheats frequently exhibit acceptable levels of adult-plant resistance to stem rust (McIntosh - personal communication). The nature of this resistance is not understood. Following the screening of 180 tetraploid cultivars as seedlings, 12 of these which were susceptible to *P. graminis tritici* strains 21-Anz-0, 21-Anz-9, 116-Anz-2,3,7, 126-Anz-6,7,11, 222-Anz-1,2,3,4,5,6,7 and culture 56-E1 were selected for field sowings. Dural was included as a representative of current commercial durum cultivars.

3.4 FIELD DESIGN

3.4.1 1972 Experiment

Thirty-three genotypes selected for study in 1972 were sown in a split plot, latin square in 3 blocks. Three disease treatments were allocated to each block *viz.* i) no disease control, ii) control of leaf rust using the selective fungicide 'RH124' and iii) control of leaf and stem rusts using Dithane M45$^{(R)}$. Cultivars were allocated at random within each treatment. Plots consisted of 3x3' rows, 7" apart.

In order to hinder inter-plot effects, a resistant selection, Tr308, was sown between plots, 4x3' rows across the field and by 3' strips along the field. One row within the 4 rows of Tr308 was left vacant to form a pathway. The experimental blocks were surrounded by inoculated susceptible buffers.

3.4.2 1973 Experiment

In 1973 the design was modified to a split plot of four blocks with 25 cultivars and 2 disease treatments *viz.* i) leaf rust control with 'RH124' and ii) stem and leaf rust control with Dithane M45(R). The 8 treatment blocks were set in the following matrix:

A B A B

B A B A

Cultivars were randomly allocated within treatment blocks. In this experiment, a resistant buffer was considered unnecessary and plots consisted of one 3' row. The experimental area was surrounded by breeding materials in which an epidemic had been induced.

3.5 DISEASE CONTROL

3.5.1 Leaf Rust

The systemic fungicide RH124 (Rohm and Haas formulation number), containing the active agent 4-n-butyl-1,2,4-triazole, was applied by spray to plants and soil at the fourth leaf stage, at the rate of 2 kg active ingredient per hectare. In 1972 specified blocks, and in 1973 the entire experimental area, were sprayed. Following the recommendation of Rowell (personal communication), soil moisture was maintained to allow the continual renewal of a protective dose in the plant.

Protection of fully susceptible cultivars was nearly complete (2% disease cover at flag leaf senescence).

3.5.2 Stem and Leaf Rust

Following artificial inoculation of susceptible checks or breeding materials, specified blocks for full disease control were sprayed every 8 days, or following rain, with Dithane M45(R), the active ingredient of which is mancozeb. The aqueous suspension of 1.5g active agent plus 3 ml. of Triton B(R) (spreader) per litre was applied until all plant surfaces were thoroughly wet. Complete disease control was achieved on susceptible cultivars.

3.5.3 Powdery Mildew

In 1972, the incidence of powdery mildew, caused by *Erysiphe graminis* f. sp. *tritici*, contributed significantly to grain weight variation. Therefore, in 1973, all seed was dusted with the specific systemic fungicide PP149(R) (active ingredient: 5-butyl-2-(ethylamino)-4-hydroxy-6-methylpyrimidine) before sowing. However, this treatment failed to give the effective control which was obtained in Dithane-treated blocks. In order to estimate the effect of powdery mildew, each cultivar at anthesis was given a disease intensity rating ranging from 0 (trace disease incidence) to 5 (maximum mildew intensity for which cultivar Bansi Strain 168 served as a standard for rating 5).

3.6 STRAINS OF RUST PATHOGENS IN THE FIELD

The composition of *P. graminis tritici* and *P. recondita* strains was dictated by the requirements of the breeding programme . *P. graminis tritici* strains present in 1972 and

1973 included 21-Anz-2,4,5; 34-Anz-2,4,5,7,11, 34-Anz-1,2,3,6,7 and 222-Anz-2,3,7,8 and the *P. recondita* strains were 162-Anz-1,2,3,GT and 68-Anz-1,2,3,4.

3.7 EPIDEMIC PROGRESS CURVES

Following the appearance of stem rust in each year percentage disease cover on the uppermost three stem internodes of each of 10 random tillers in each plot was estimated. The weekly measurements were based on a scale given by James (1971). The date of completion of anthesis for each plot was recorded. A disease intensity index for each assessment date was calculated by summing the individual estimates for a specified internode within a plot. The mean disease intensity index and its standard deviation was then calculated from the three replications.

3.8 1,000 - GRAIN WEIGHTS

Under Australian conditions an attack of stem rust usually results in reduced grain weight. Hence 1,000-grain weights were used as estimates of yield reductions sustained by different cultivars in the epidemics. At maturity, all plots were harvested and threshed. Samples of 250 seeds were readily obtained using perspex seed grid (made to specifications of Almgren, 1964). Four 250 seed measurements were made on bulk seed harvested from each plot. As the sampling variances were small, the four weights were summed. Data were analysed by analyses of variance (Kempthorne (1952); Tables 19.1, p.374 and 19.3, p.379 for 1973 and 1972 experiments, respectively).

3.9 GENETIC STUDIES

On the basis of early observations and continued interest in the Hope and Marquillo cultivar groups, various crosses were made for a study of inheritance of the resistances observed.

3.10 SEEDLING TESTS ON HOPE AND DERIVATIVES

Parents were tested as seedlings with eight strains of *P. graminis tritici* in order to confirm their authenticity and to establish their characteristic reactions (Table 4). Both Hopps and CS(Hope 3B) were different from the remainder in the resistant group in that they did not possess known seedling resistance genes.

3.11 GENERATION OF HYBRID POPULATIONS

F_1's were made and grown in the field and greenhouse where various characteristics were examined to ensure that hybrids were genuine. In relation to studies on Hope and derivatives, various resistant parents were crossed with susceptible parents and a number of intercrosses were effected. Resistant cultivars included CS(Hope 3B), Hopps, Hope, Renown Selection, Selkirk and Warigo and susceptible cultivars included Chinese Spring, Federation, Yalta, Svenno, Gabo and W3498.

Celebration and Marquillo were crossed to W3498.

Populations of approximately 300 F_2 plants from each cross were transplanted into the field in 1972, 1973 or 1974. F_2 populations from resistant/susceptible crosses, together with F_1 plants, were protected from stem rust by fortnightly applications of Dithane M45$^{(R)}$.

TABLE 4

Infection types produced on seedlings of resistant and susceptible parents when tested with *P. graminis tritici* strains at <23°C.

Parent	Strains								Known genes for seedling reaction
	21-0	59-5,7	126-6,7, 11	326-1,2, 3,5,6	222-1,2, 3,5,6	194-1,2, 3,5,6	21-1,2,5	21-2	
Resistant									
Hope	;	;1x=	;x2	x2-n	x3n	x3n	x3+n	;	*Sr7bSr9dSr17*
CS(Hope 3B)	x3+n	x3+n	x3+n	x3+n	x3+n	x3+n	x3+n	x3+n	
Hopps	x3+n	;	x3n	x3+n	x3+n	x3+n	x3+n	x3+n	
Renown Sel^{n}	;x=	;	x2	22+	33+	33+	33+	x=	*Sr7bSr9dSr17*
Selkirk	x2=n	0;	;x=	23+cn	33+cn	33+cn	3+cn	0;	*Sr6Sr7bSr9dSr17*
Spica	x=	;x2=	x2	2+	33+	33+	33+	x=	*Sr7bSr17* *Sr23*
Warigo	;x-	;x=	x2	22+n	33+	33+	33+	x=	*Sr7bSr17*
Susceptible									
Chinese Spring	3+	3+	3+	3+	3+	3+	3+	3+	
Federation	3+	2+n	3+	3+	3+	3+	3+	3+	
Yalta	;12=	;12-	;1	3+	3+	3+	3+	3+	*Sr11*
W3498	4	4	4	4	4	4	4	4	
Warden	3-3cn	33+cn	1+cn	33+cn	33+cn	33+cn	33+cn	3cn	*Sr23*
Gabo	;12=	;	;	3+	3+	3+	3+	3+	*Sr11*
Svenno	3+	3+	3+	3+	3+	3+	3+	3+	
CS(Hope 7B)	x+	3+	3+	3+	3+	3+	3+	x+	*Sr17*
I*Sr7b* Ra	3+	2	33+	2	3+	3+	3+	3+	*Sr7b*

Dr. N. H. Luig and Dr. R.A. McIntosh provided 40 F_3 families from Hopps/Federation and 70 F_3 families from CS/CS(Hope 3B), respectively. As difficulties in the classification of adult-plant reactions were anticipated it was decided that initial genetic studies would be based on F_3 families rather than individual F_2 plants.

3.12 FIELD TEST PROCEDURES OF HYBRID POPULATIONS

3.12.1 Direct Field Sowings

3.12.1.1 Seed preparation

Depending on the availability of seed, individual F_3 and F_4 families were replicated two to five times in each year. Certain populations tested in 1973 or 1974 were retested in 1975. Approximately 30 seeds from each family were sown in each replication with the aim of establishing approximately 20 plants in each plot. Within each cross, families were randomized for each replication. A parent plot was included after every tenth family.

3.12.1.2 Field layout

In 1973 and 1974 replicated plots were sown at each of two sites, Plant Breeding Institute, Castle Hill, and Hawkesbury Agricultural College, Richmond. In 1975 only the Castle Hill site was used. Originally, the aim was to study repeatability of results over sites and to ensure against natural calamity. Plots were of two main types. For family classifications, single 3' rows with 14" spacings were used, whereas for single plant classifications 7' rows were sown with a similar quantity of seed. In 1974 and 1975, one replication at Castle Hill was sown in hill plots, or 'clumps',

on a grid of 12"x14". The technique reduced space and still permitted accurate assessment of reactions, but lodging proved a problem in 1975.

3.12.2 Transplantation

Experience gained from the 1973 and 1974 field experiments indicated that single plants could be classified for the 'Hope' adult-plant reaction. In order to allow single plant assessment, F_1, F_2 and some spaced F_3 populations grown in the field were transplanted from pots, a common practice at the Plant Breeding Institute.

3.12.3 Inoculum Increase

All experimental plots were naturally infected from susceptible borders. In order to ensure inoculum, border strips of susceptible genotypes were sown 6-8 weeks prior to normal sowings. Further susceptible strips and borders were included with normal sowings. Strains chosen for field epidemics were applied to the earlier sown border strips by hypodermic injection of an aqueous spore suspension into culms. Each strain was inoculated separately. Some disease, particularly leaf rust occurred naturally.

3.12.4 *P. graminis tritici* Strains

The same strains were used for field epidemics at both sites:

1973 - 21-Anz-2

1974 and 1975 - 21-Anz-1,2,5, 194-Anz-1,2,3,5,6 and 222-Anz-1,2,3,5,6.

The two latter strains are virulent on most of the seedling

resistance genes known to be present in Hope and its derivatives (Table 4), but are not virulent on seedlings of Celebration and Marquillo.

3.12.5 *P. recondita* Strains

A leaf rust epidemic was established in the field at Castle Hill in 1975 using strains 122-Anz-1,2,3, 104-Anz-1,2,3,GT and 162-Anz-1,2,3,GT. Observations were made on leaf rust reaction where parental variability had been noted *viz.* Hopps/W3498.

3.12.6 Grain Weight Measurements

In some instances, 100 grain weights were used to substantiate field rust observations. In the determination of 100 grain weights, two 100 grain samples were taken from threshed population bulks. The data were analysed by analysis of variance (Steel and Torrie, 1960; Table 7.9, p.121).

3.13 ADDITIONAL HOPE DERIVATIVES

Following genetic studies indicating that resistance was more simply inherited than originally assumed, a number of Hope derivatives were assembled for comparative study. Cultivars included, Africa Mayo, Aotea, Arthur 71, Gala, Glenwari, Hofed, Hopps/Federation F_4 Selection, Kenya Page, Lancer, Lawrence, Lerma Rojo 64A, Renown W1242, Renown W2346, Samaca, Scout 66, Redman, Hope, H44-24 and Yaqui 50.

3.14 ADDITIONAL MARQUILLO DERIVATIVES

Adult-plant reactions of Pato, Pato Argentina and Tezanos Pinto Precoz believed to be Marquillo derivatives were observed during 1975.

3.15 SEEDLING TEST PROCEDURE

Seedling studies, employing selected strains, were conducted to determine host cultivar genotypes or to monitor known host genes in hybrid populations. These determinations were necessary to enable field reactions to be correlated with known genotypes with respect to various specific resistance genes. Where parental lines were known to have more than one such gene, strains were chosen to permit classification of each gene separately. In F_3 studies, each population of 20-30 seedlings, sown to a single 10 cm pot, was inoculated with the selected strain at the first leaf stage. For application, urediospores suspended in a light oil, were atomised (10 ml/100 pots) over the seedlings. Inoculated pots were kept overnight in a misting chamber and then spaced on greenhouse benches. When necessary, greenhouse cooling was available. Notes were recorded 12-14 days after inoculation, depending on prevailing conditions, as well as particular pathogen and strain. Seedling infection types were based on the scale proposed by Stakman *et al.* (1962).

The genotype for the *Sr23* locus was inferred from the genotype at the *Lr16* locus. *Lr16* is closely linked in coupling, or allelic, with *Sr23* (McIntosh and Luig, 1973) and the former can be more readily detected in contrast to *Sr23*, since infection types produced by lines possessing *Sr23* can be variable. In general, the effect of *Sr23* on seedling reaction to *P. graminis tritici* strains was minor and did not interfere with classification of genotypes for other loci.

3.16 ADULT-PLANT GREENHOUSE TEST PROCEDURE

Ten adult-plants per family or cultivar (5 plants per 20 cm pot) were inoculated at anthesis using an atomized spray of spores, suspended in a light oil. All plant parts were uniformly covered with inoculum. Inoculated plants were kept overnight in a misting chamber and then placed on benches in greenhouses held at approximately 23°C. When rust pustules on the susceptible controls were fully developed all plants were scored.

3.17 CYTOGENETIC STUDIES

3.17.1 Chromosome Arm Location of *Sr12*

Sears *et al.* (1957) and Sheen and Snyder (1964) located certain seedling resistance gene(s) of Thatcher on chromosome 3B. McIntosh (personal communication) has referred to a recessive gene in chromosome 3B, which is characterised by a mesothetic infection type, as *Sr12*. To investigate this resistance, F_3 lines from the cross CS ditelosomic 3BL/CS(Thatcher 3B) were studied in the seedling stage. In addition, certain euploid and aneuploid materials derived from Apex, Rescue and Cadet, provided by Dr. R. Larson, Agriculture Canada, Lethbridge, Alberta, Canada, were examined.

3.17.2 Somatic Chromosome Counts

Mitotic studies were performed on root tips pre-treated for 24 hours in ice-water, or for 4 hours in a saturated aqueous solution of α-bromonaphthalene, fixed in Farmer's fixative, or glacial acetic acid, respectively, hydrolysed in IN HCl at 60°C for 10 minutes and stained in leuco-basin fuchsin.

3.17.3 Chromosome Arm Location of Hope Adult-plant Resistance

Sheen *et al.* (1968), Knott (1971) and McIntosh (personal communication) have suggested that the Hope 3B chromosome carries the factor(s) controlling the adult-plant reaction. In order to investigate this resistance, all Hope chromosome substitution lines (originally produced by Dr. W.Q. Loegering, Uni. of Missouri, U.S.A.) and the respective parents were tested in 1975. Additionally, since CS(Hope 3B), Hopps and Warigo were known to have adult-plant resistance, certain crosses with Chinese Spring aneuploids were studied. Thirdly, a set of Redman monosomic lines (provided by Dr. A.B. Campbell, Agriculture Canada, Winnipeg, Manitoba, Canada) and certain materials derived from Apex, Cadet and Rescue wheats, obtained from Canada, were examined.

EXPERIMENTAL RESULTS

4. DISEASE ASSESSMENT TECHNIQUES

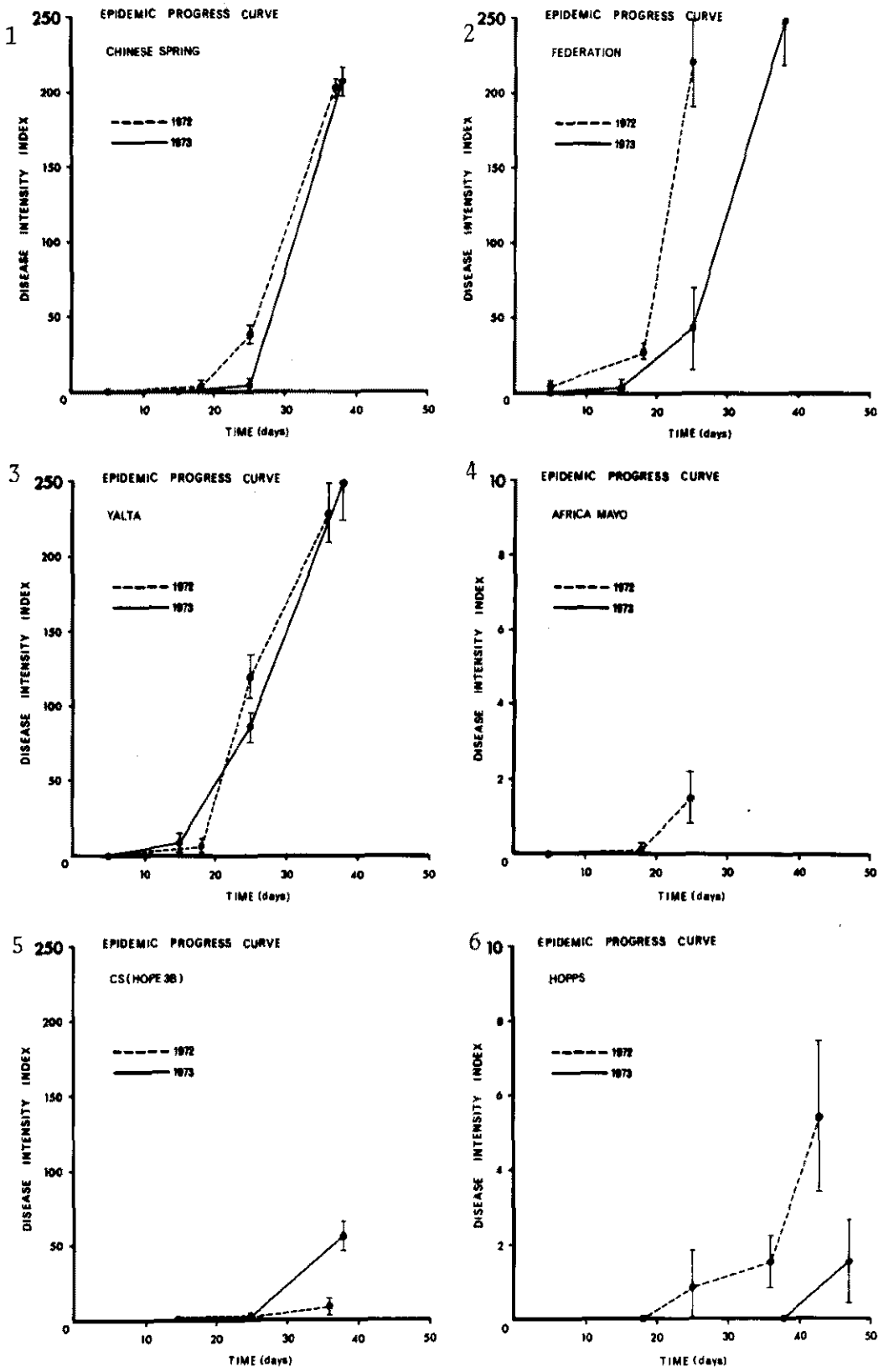
4.1 EPIDEMIC PROGRESS CURVES

Epidemic progress curves, constructed by plotting disease intensities against time, are given in Fugures 1-40. Disease intensities for the second uppermost internode were plotted, as the stem and sheath in this region remained viable and infective over the longest period of the epidemic, thus offering the greatest opportunity for disease development. Three disease intensity scales *viz.* 250, 50 and 10 have been used in the figures 1-40 to indicate the low level of disease development on certain cultivars. Disease development was largely dependent on maturity. Late maturing cultivars were exposed to higher inoculum levels at higher temperatures and longer day lengths than early maturing cultivars, hence epidemic progress curves for cultivars of differing maturities can be compared only with difficulty. The number of days to the completion of anthesis (Table 6) was used as a guide to maturity. Cultivars grown over both years generally behaved similarly. The disease assessment studies indicated a need to record disease data at optimum times for each cultivar.

4.2 YIELD PARAMETER - 1,000 GRAIN WEIGHT

Analyses of variance conducted on 1,000-grain weights from 1972 and 1973 trials are presented in Table 5. Mean 1,000-grain weights for various disease controlled and non-controlled plots for each year are listed in Table 6. Disease control treatments and cultivar effects were highly significant ($P<0.01$), while the disease control treatment x

FIGURE



Disease progress curves produced by plotting cumulative pustule numbers against time for 40 genotypes tested over one or two years.

FIGURE

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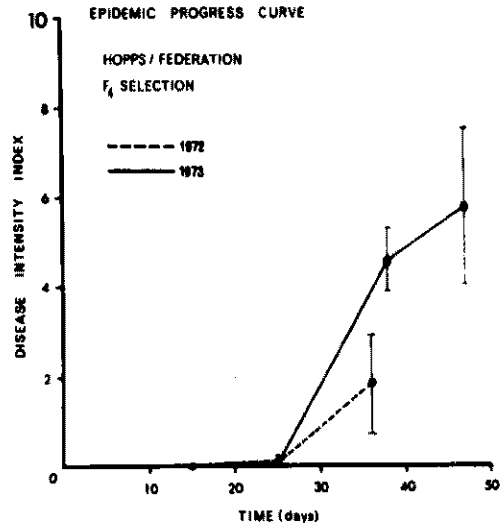

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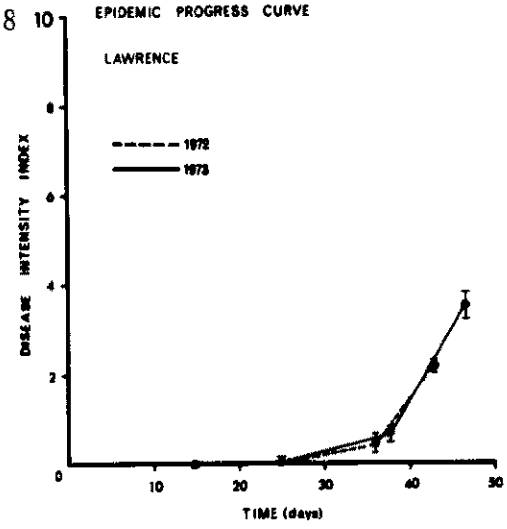

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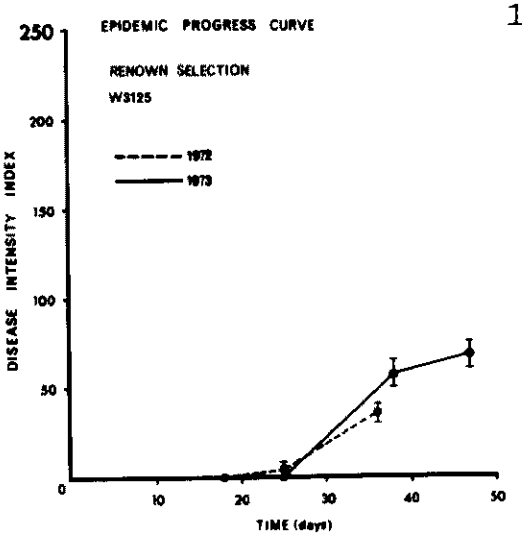

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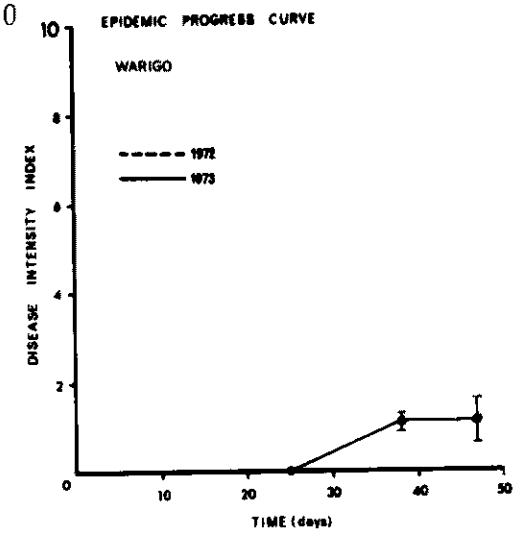

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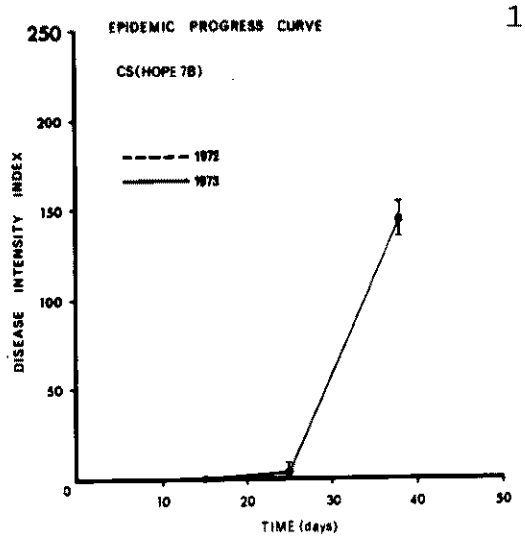

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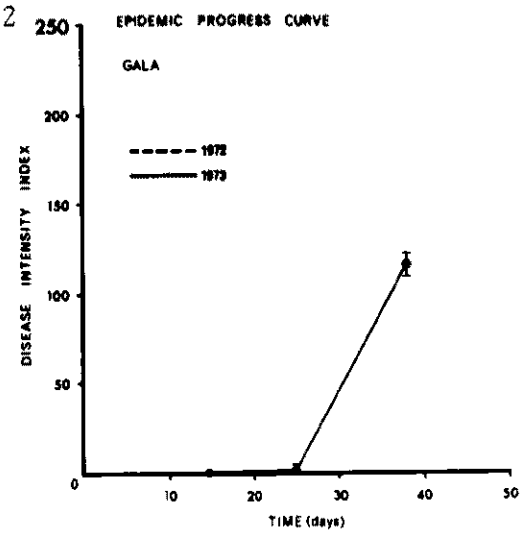

FIGURE

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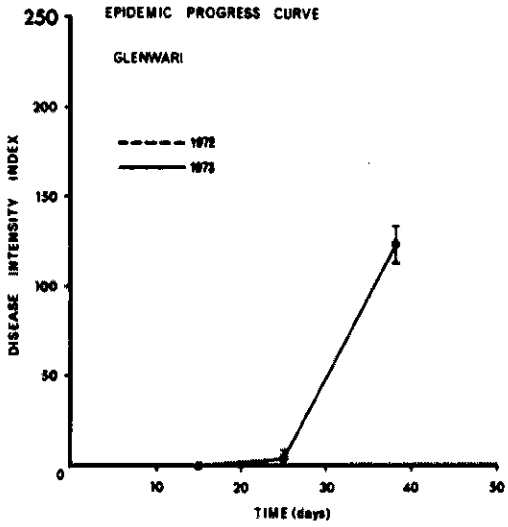

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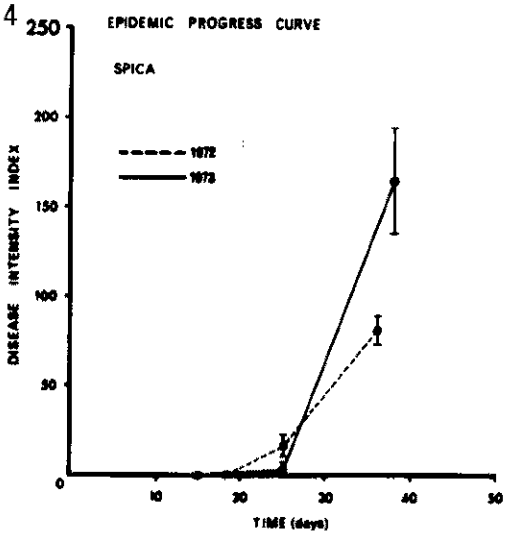

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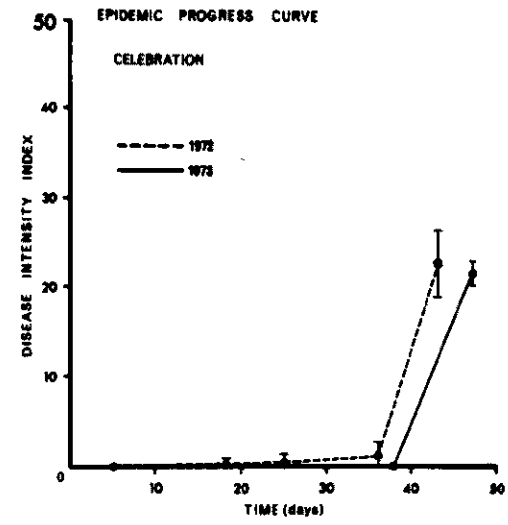

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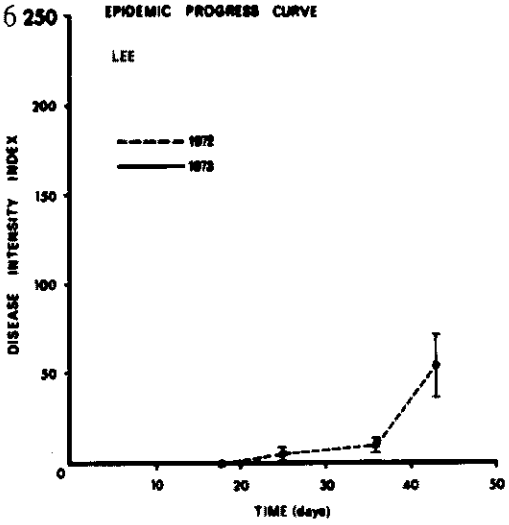

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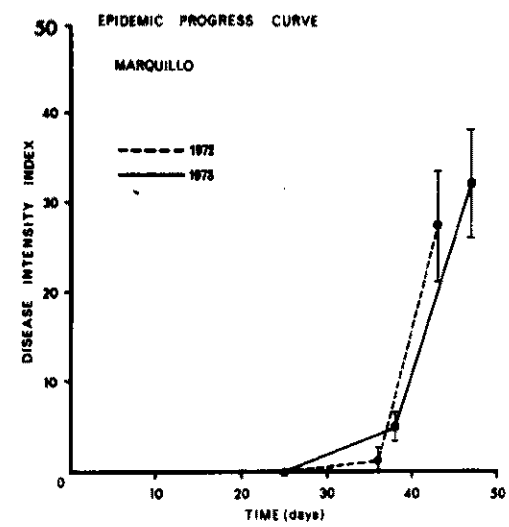

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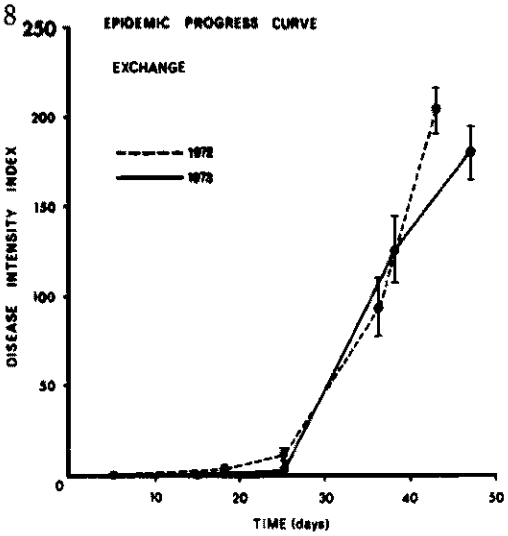

FIGURE

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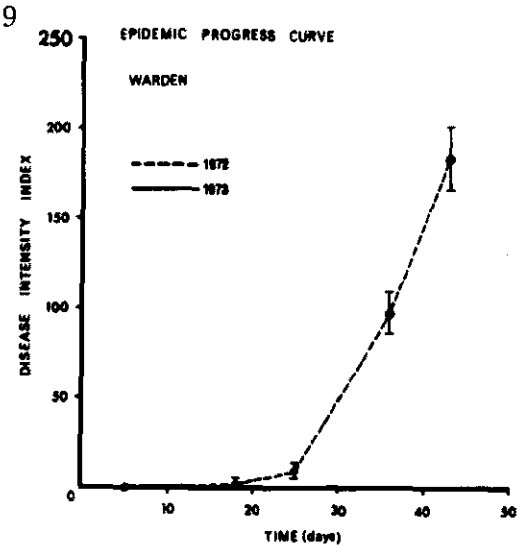

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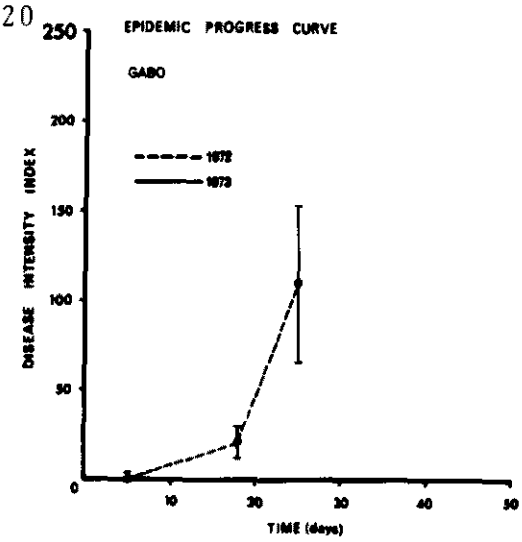

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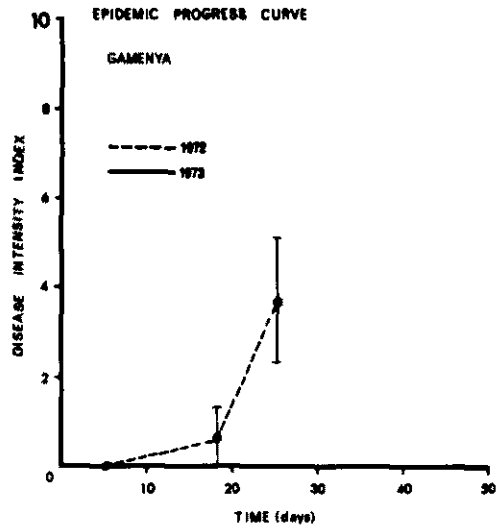

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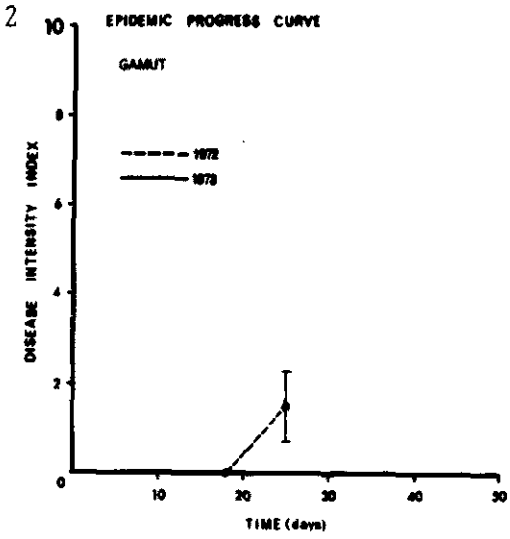

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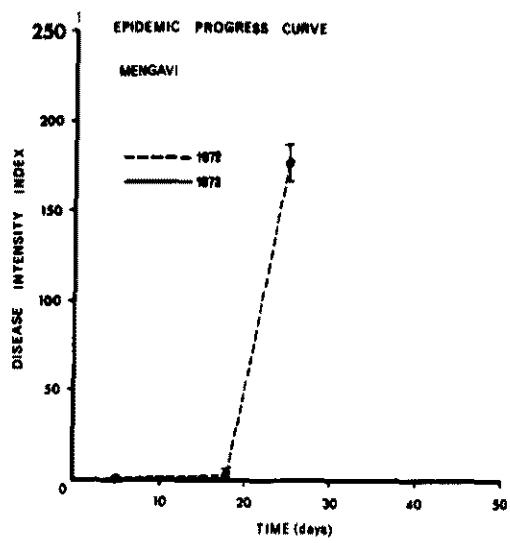

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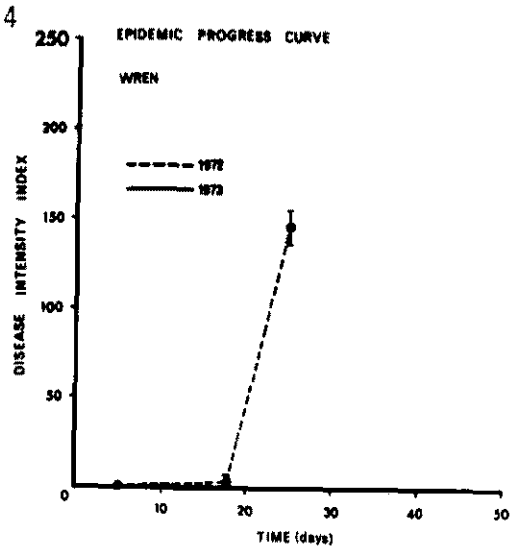

FIGURE

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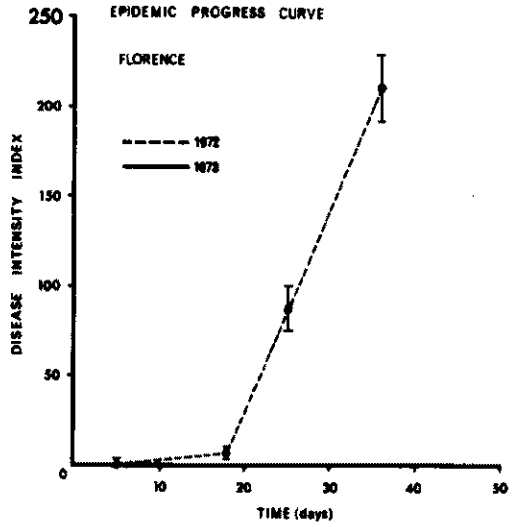

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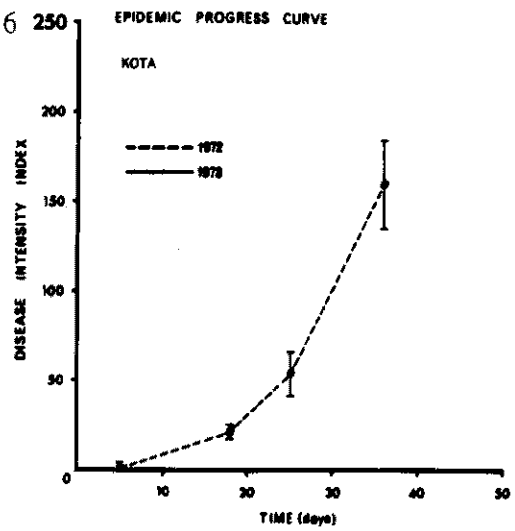

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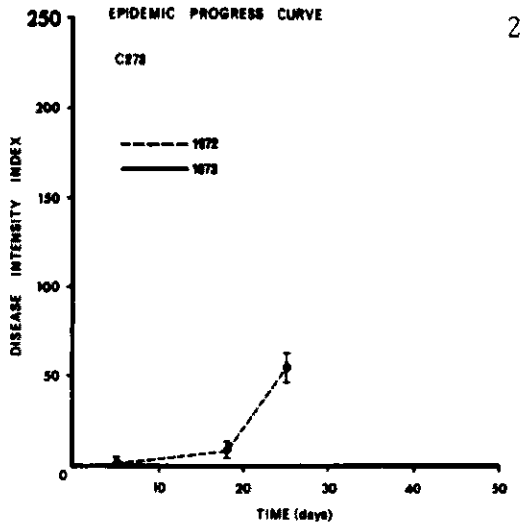

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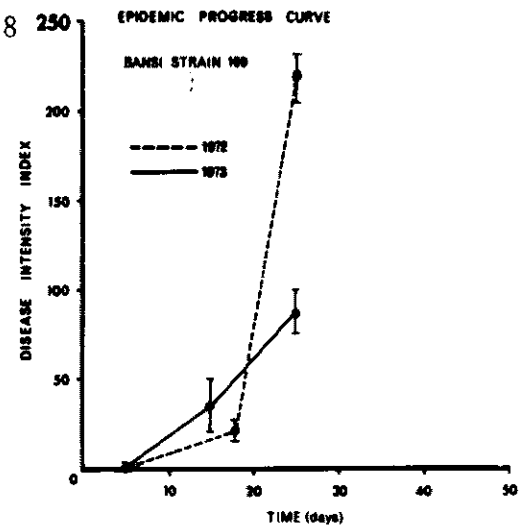

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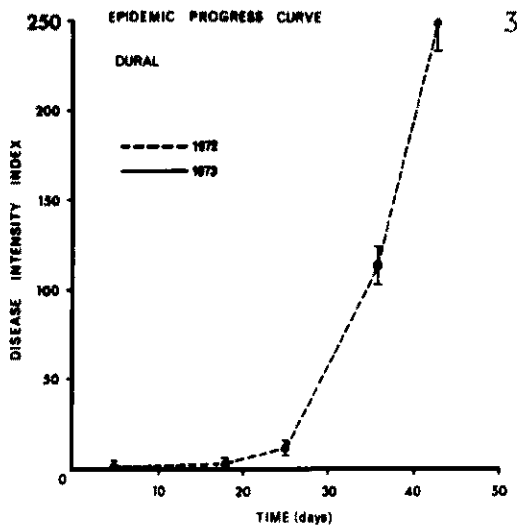

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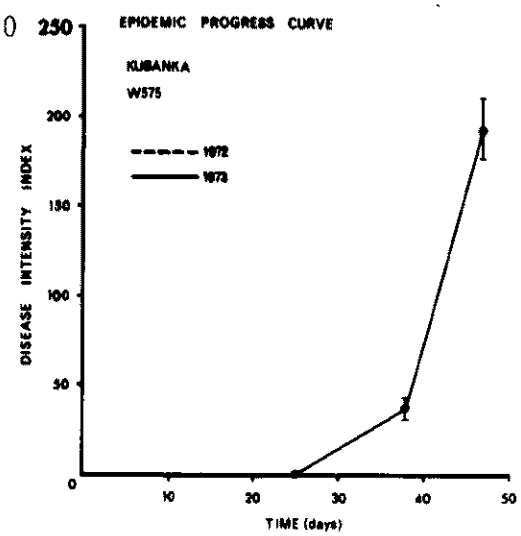

FIGURE

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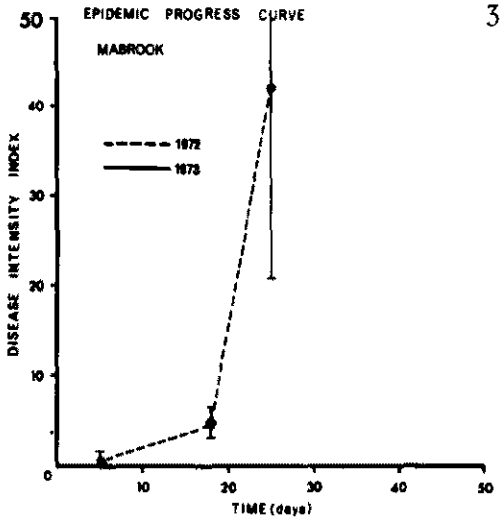

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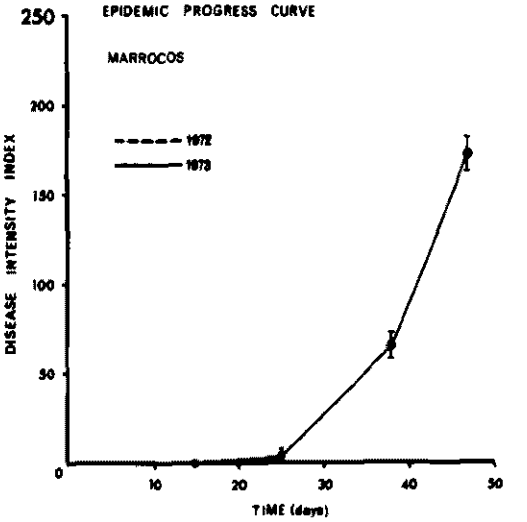

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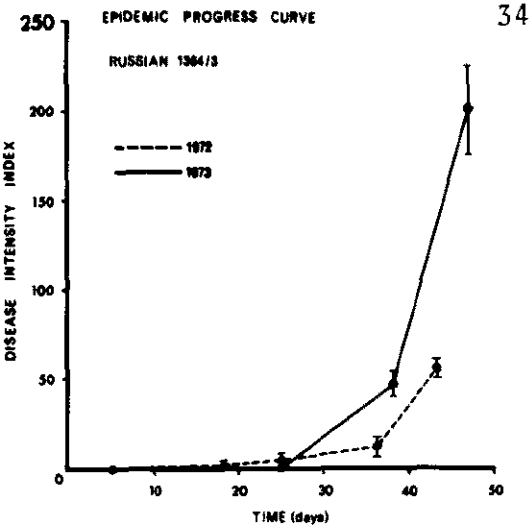

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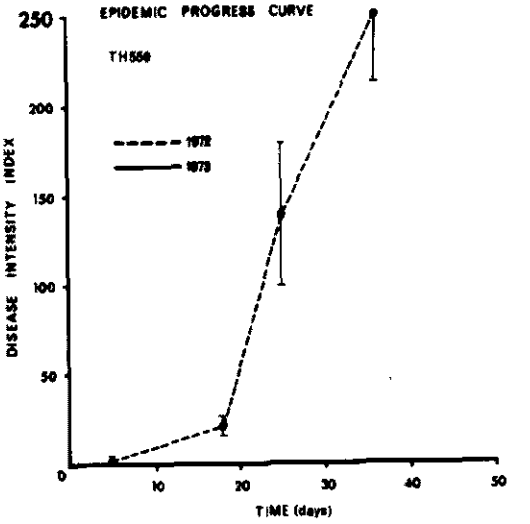

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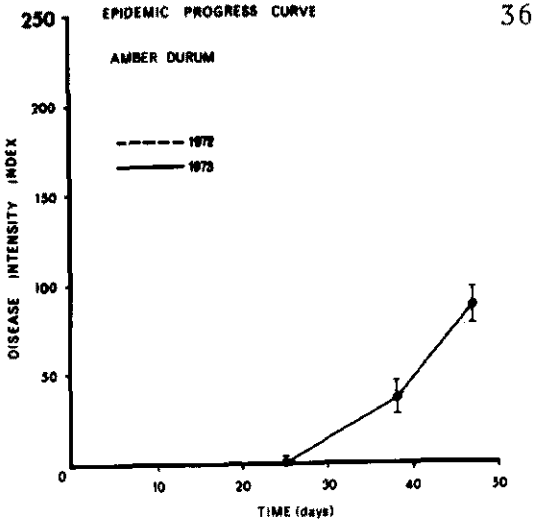

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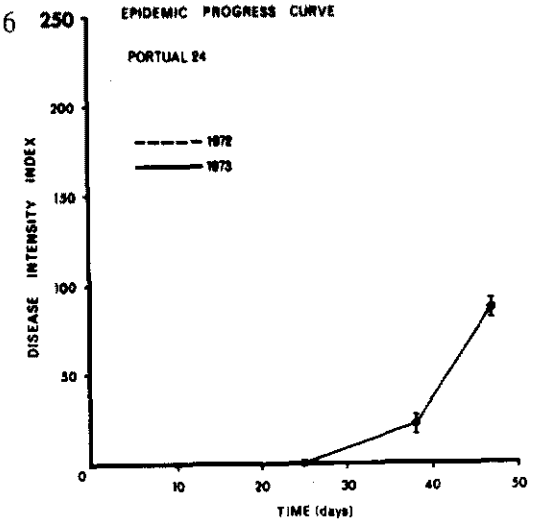


FIGURE

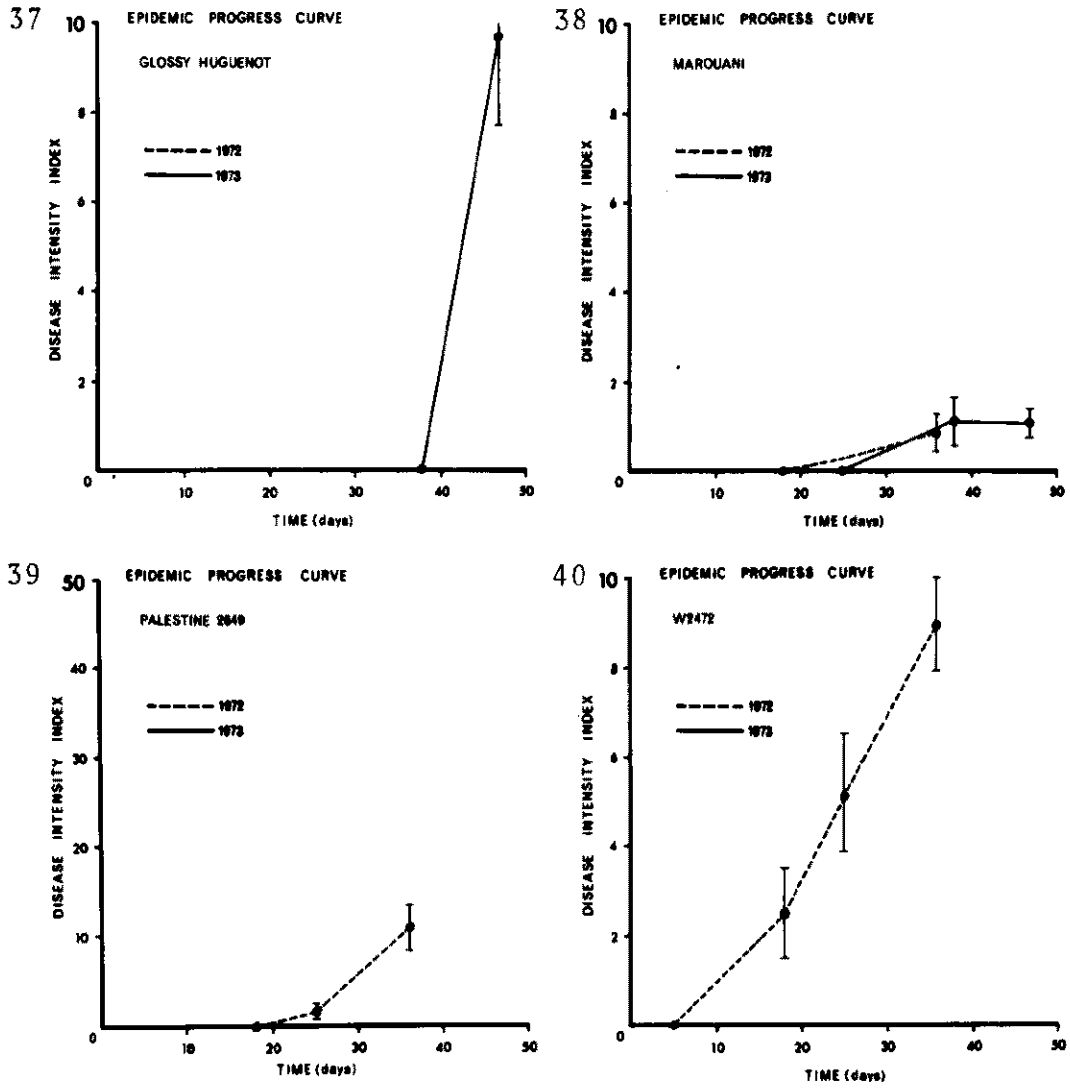

37
EPIDEMIC PROGRESS CURVE
GLOSSY HUGUENOT
1972
1973
DISEASE INTENSITY INDEX
TIME (days)
38
EPIDEMIC PROGRESS CURVE
MAROUANI
1972
1973
DISEASE INTENSITY INDEX
TIME (days)
39
EPIDEMIC PROGRESS CURVE
PALESTINE 2649
1972
1973
DISEASE INTENSITY INDEX
TIME (days)
40
EPIDEMIC PROGRESS CURVE
W2472
1972
1973
DISEASE INTENSITY INDEX
TIME (days)

TABLE 5

Analysis of variance for the effects of stem rust on 1,000 grain weights during 1972 and 1973.

Source of variation	df	MS	F
1972*			
Rows	2	18.53	
Columns	2	38.42	
Disease control treatments	2	798.05	39.76**
Cultivar	32	433.95	21.62**
Disease control x Cultivar interaction	64	34.22	1.70**
Error	191	20.07	
1973			
Disease control treatments	1	1811.66	108.86**
Replications	3	9.93	
Error (a)	3	16.64	
Cultivars	22	400.12	65.31**
Disease control treatments x cultivar interaction	22	48.79	7.96**
Error (b)	132	6.13	

* three missing plots

** $P<0.01$

TABLE 6

Mean 1,000-grain weights (g) for disease controlled and non-controlled plots of various cultivars in 1972 and 1973.

Cultivar	1972					1973				1972 and 1973	
	1,000-grain weights g.			A/B%	Maximum stem rust intensity rating	1,000-grain wts. g.		C/D%	Maximum Stem Rust intensity rating	Powdery* Mildew intensity rating	Approx No. of days to completion of anthesis
	No disease control (A)	Leaf Rust control	Full disease control (B)			Leaf Rust control (C)	Stem & Leaf Rust control (D)				
Chinese Spring	21.6	21.1	24.9	87	203	26.9	32.2	81	209	1	136
Federation	17.9	17.2	34.6	52	222	23.7	35.8	66	250	4	136
Yalta	25.7	26.3	38.2	67	227	29.8	42.2	71	250	4	126
Selkirk	30.1	29.2	33.4	93	0	32.1	36.9	87	0	4	146
Africa Mayo	33.9	35.2	33.6	101	1.5					2	120
CS(Hope 3B)	21.3	21.5	21.2	101	9	27.9	26.6	105	58	2	130
Hopps	34.2	32.7	37.7	91	5	39.5	39.9	99	1.5	3	141
Hopps/Federation Selection	23.4	28.2	34.6	68	2	34.7	37.8	92	6	4	141
Lawrence	31.0	37.4	36.8	84	10	40.6	47.1	86	18	4	141
Renown Selection	23.4	30.0	30.8	76	36	25.7	31.7	81	67	4	146
Warigo						26.7	29.4	91	1	2	146
CS(Hope 7B)						26.9	30.5	88	145	2	136
Gala						36.3	44.9	79	118	3	126
Glenwari						38.5	49.9	77	125	3	136
Spica	54.0	52.4	51.3	105	82	43.8	50.6	86	165	2	116
Celebration	36.1	37.8	38.7	93	23	44.2	46.4	95	21	3	141
Lee	30.6	31.2	32.7	94	55					1	136
Marquillo	33.2	35.3	34.5	96	27	36.1	39.5	91	32	2	146
Exchange	23.8	26.4	38.6	62	206	24.7	43.1	57	180	2	146
Warden	26.2	25.4	38.2	69	185					3	146
Gabo	34.3	37.0	39.3	87	111					3	120
Gamenya	34.1	39.6	37.8	90	4					1	120
Gamut	41.7	42.7	38.0	107	1.5					2	120
Mangavi	36.8	39.9	41.8	86	177					2	120
Wren	22.0	22.1	29.0	76	146					4	120

TABLE 6 (CONT)

Cultivar	1972					1973				1972 and 1973	
	No disease control (A)	Leaf Rust control	Full disease control (B)	$\frac{A}{B}$%	Maximum stem rust intensity rating	Leaf Rust control (C)	Stem & Leaf Rust control (D)	$\frac{C}{D}$%	Maximum Stem Rust intensity rating	Powdery* Mildew intensity rating	Approx No. of days to completion of anthesis
Florence	39.4	37.4	39.6	99	215					3	130
Kota	34.1	31.0	39.9	85	162					3	130
C273	42.9	42.5	40.4	106	56					2	116
Bansi Strain 168	22.1	24.1	32.0	69	215	25.3	36.9	69	87	5	116
Dural	21.6	22.8	36.3	60	253					4	146
Kubanka									194	4	160
Mabrook	31.7	34.9	41.1	77	42					4	116
Marrocos	27.1	29.5	41.2	66	37	31.5	39.6	80	174	3	153
Russian 1364/3	21.8	23.9	29.1	75	55				200	1	160
TH559	24.0	27.6	31.2	77	283					3	126
Amber durum						31.1	40.6	77	87	3	148
Portugal 24						42.5	53.1	80	86	2	153
Glossy Huguenot						39.6	42.1	94	10	1	153
Marouani	36.5	36.0	35.8	102	1	51.3	52.7	97	1	2	153
Palestine 2649	28.2	34.2	33.8	83	10					3	136
W2472	15.0	19.7	20.1	75	9					3	126
Mean	29.6	31.3	35.2			33.7	39.9				

1972 l.s.d. for cultivar means (P=0.05) = 5.8g.

1973 l.s.d. for cultivar means (P=0.05) = 3.5g.

* Powdery mildew intensity rating

0 - trace disease incidence to 5 maximum mildew intensity.

cultivar interactions were significant at P=0.05, in both years. In 1972, the overall treatment means for no disease control, leaf rust control and full disease control were 29.7g, 31.3g and 35.2g, respectively. The l.s.d. (P=0.05) value for comparing treatment means was 1.69g, which indicated that leaf rust did not significantly effect grain weight, with the exception of Lawrence, Renown Selection and Palestine 2649. On the other hand, certain differences in grain weight were due to the effect of powdery mildew rather than stem rust. Powdery mildew ratings for each cultivar appear in Table 6.

4.3 CULTIVARS

4.3.1 Hope and H44-24 Derivatives

Cultivars within this group, except CS(Hope 3B), Spica and African Mayo, completed anthesis within a 6 day period. The exceptions were 10, 26 and 20 days earlier, respectively. Selkirk was immune in both years as no strains present had combined virulence for *Sr6* and *Sr17*. However, there was sufficient inoculum of strains virulent for either *Sr6* or *Sr17*, that there could be no delayed effects on epidemic progress. Cultivars Africa Mayo, CS(Hope 3B), Hopps, Hopps/Federation Selection, Lawrence, Renown Selection and Warigo were clearly resistant. Africa Mayo, Hopps, Hopps/Federation Selection and Warigo gave mean disease intensity indices (over 2 years) ranging from 1 to 6 at maturity, whereas the comparable values for CS(Hope 3B), Lawrence and Renown Selection ranged from 9 to 67. Africa Mayo and CS(Hope 3B) were not

strictly comparable with the other cultivars due to early maturity. The adult-plant resistance in these various Hope derivatives was generally characterised by some pustules occurring for several centimetres above nodes whereas, below the nodes, stems were relatively free of rust. These eight cultivars showed symptoms of pseudo-black chaff (Broadfoot and Robinson, 1933). Despite the adult-plant resistance, Lawrence, Renown Selection, Selkirk and Hopps/Federation Selection suffered grain weight losses in each year. This was attributed to powdery mildew infection rather than to rust.

CS(Hope 7B), Gala, Glenwari and Spica were relatively susceptible, free of pseudo-black chaff and carried pustules on all stem sections. Spica was not significantly diseased until 10 days prior to maturity, possibly due to early maturity. Grain weights suggest that Spica escaped damage in 1972 but sustained some loss in yield in 1973, a result consistent with the respective epidemic progress curves.

4.3.2 Marquillo, Celebration and Lee

The epidemic progress curves and grain weights for Celebration and Marquillo signified that both possessed adult-plant resistance. The resistance of Marquillo was not as effective as that expressed by Hopps and Warigo, but was similar to that shown by CS(Hope 3B). Both Celebration and Marquillo remained relatively free of rust until 7 days prior to maturity, when small pustules appeared. At maturity, the mean disease intensity indices of Celebration and Marquillo were 23 and 32, respectively. Lee was relatively more susceptible than Marquillo, giving an index of 55 and maturing 10 days earlier. McIntosh (1976 - personal communication)

showed that Lee does not carry *Sr12*, hence its relative susceptibility. Grain weight data suggested that Lee may possess some adult-plant resistance or tolerance. Hayden (1956) considered Lee tolerant to stem rust.

4.3.3 Exchange and Warden

Although alleged to possess adult-plant resistance, Exchange and Warden were clearly susceptible as indicated by disease progress curves (Figs. 18,19) and significant grain weight losses. They had similar maturities and reached a specified level of disease 14 days later than Yalta and 7 days later than Chinese Spring, but this reflected the maturities of these cultivars relative to Yalta and Chinese Spring.

4.3.4 Australian Commercial Cultivars

Gabo, Gamenya, Gamut, Mengavi and Wren were of similar maturities. Gabo, Mengavi and Wren were susceptible reaching disease intensity indices of 111, 177 and 146, respectively. Since strains 34-Anz-1,2,3,6,7 and 222-Anz-2,3,7,8 were only minor constituents of the initial field rust flora in 1972, Gamut and Gamenya matured before these strains had increased, hence both cultivars appeared relatively resistant. Grain weight losses reflected the epidemic progress curves of the five cultivars.

4.3.5 Florence, Kota and C273

Despite alleged adult-plant resistance, both Florence and Kota were susceptible in terms of disease progress curves. The similar grain weights from sprayed and unsprayed plots of Florence suggest that this cultivar could be rust tolerant. Due to very early maturity, C273 probably escaped disease damage. Hence, reported tolerance of C273 may be due to disease

escape through early maturity.

4.3.6 Susceptible Hexaploid Cultivars

As expected Chinese Spring, Federation and Yalta were susceptible with mean disease intensity indices of 210, 235 and 235, respectively. All sustained statistically significant grain weight losses.

4.3.7 Tetraploid Cultivars

All tetraploid cultivars had similar maturities, except Bansi Strain 168 and Mahrook which matured 25 days earlier. The tetraploid cultivars fell into three disease intensity categories, susceptible, intermediate and resistant. Bansi Strain 168, Dural, Marrocos, Mahrook, Kubanka, Russian 1364/3 and TH559 were susceptible. Despite early maturity Bansi Strain 168, was extremely susceptible to rusts, and powdery mildew. All suffered significant grain weight losses.

Amber durum and Portugal 24 were intermediate in response, giving maximum disease intensity indices of 87 and 86, respectively, and sustaining significant grain weight losses.

Glossy Huguenot, Marouani, Palestine 2649 and W2472 possessed adult-plant resistance, as suggested by maximum disease intensity indices of 10, 1, 10 and 9, respectively, and non-significant grain weight differences when diseased and sprayed treatments were compared.

5. INHERITANCE OF ADULT-PLANT RESISTANCE TO STEM RUST IN HOPE WHEAT AND DERIVATIVES

Notes were recorded three times during the period of epidemic development which extended from the late ear initiation stage. Differences in adult-plant reaction were more readily seen during an optimal period for each population, depending upon maturity. This optimal period covered the milk to early-dough stages.

5.1 PARENTAL ADULT-PLANT REACTIONS

The reactions of various parents are illustrated in Plate 1. As observed in the disease progress experiments, only CS(Hope 3B) among the selected resistant parents showed significant rust development, but in this case disease developed only with the approach of senescence.

5.2 GENETIC ANALYSIS OF ADULT-PLANT REACTIONS IN CROSSES OF RESISTANT AND SUSCEPTIBLE PARENTS

5.2.1 F_1

In 1975, five F_1 plants from each of ten crosses involving resistant and susceptible parents were sown in the rust nursery together with the respective parents. As indicated in Table 7, all hybrids were susceptible, showing that resistance was recessive to susceptibility. The reactions of F_1 plants are illustrated in Plate 2.

5.2.2 F_2

The F_2 segregations for various crosses are presented on Table 8. Only two phenotypic classes, resistant and susceptible, were distinguished.

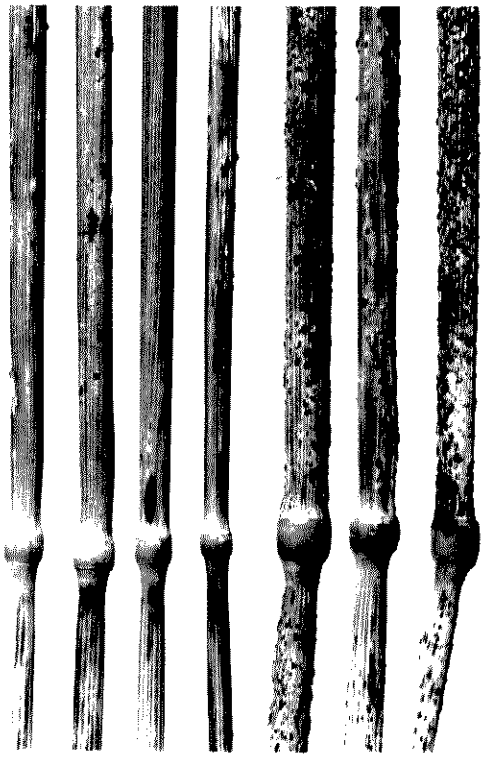
PLATE 1 Adult-plant reactions of F_3 lines and respective parents.

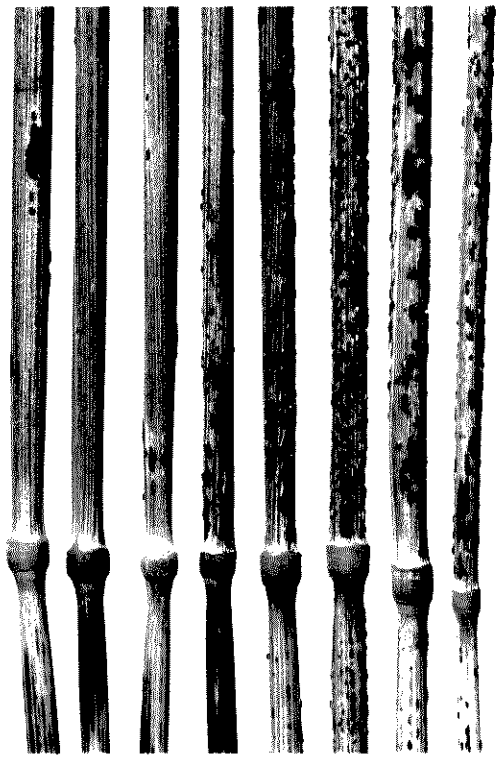
a) Gabo/Selkirk: 1 Selkirk; 2-4 Resistant segregates; 5-6 Susceptible segregates; 7 Gabo.

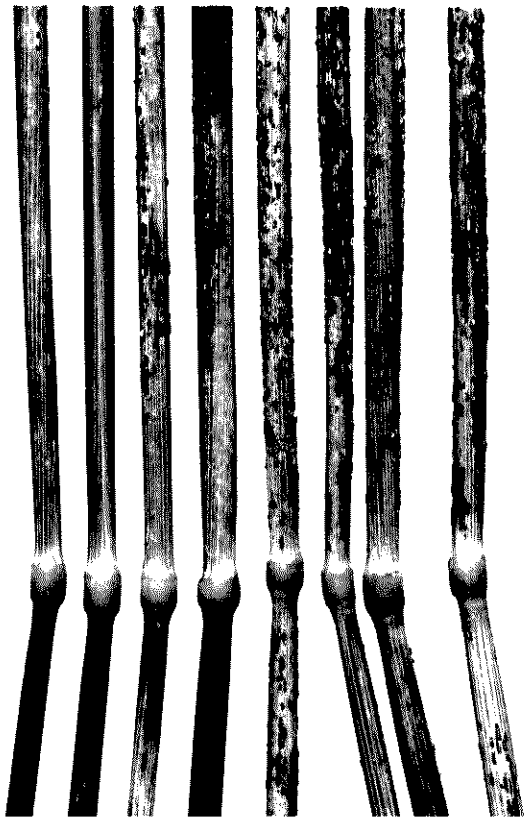
b) Warigo/W3498: 1 Warigo; 2-4 Resistant segregates; 5-6 Susceptible segregates; 7 W3498.

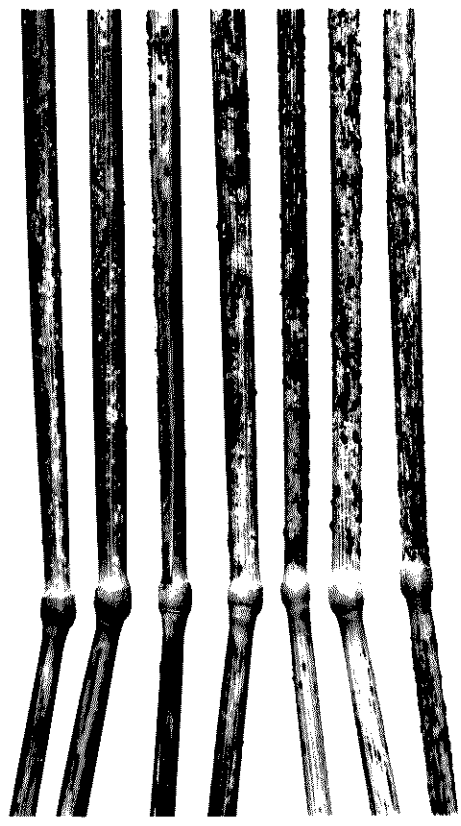
c) Hopps/Federation: 1 Hopps; 2-4 Resistant segregates; 5-7 Susceptible segregates; 8 Federation.

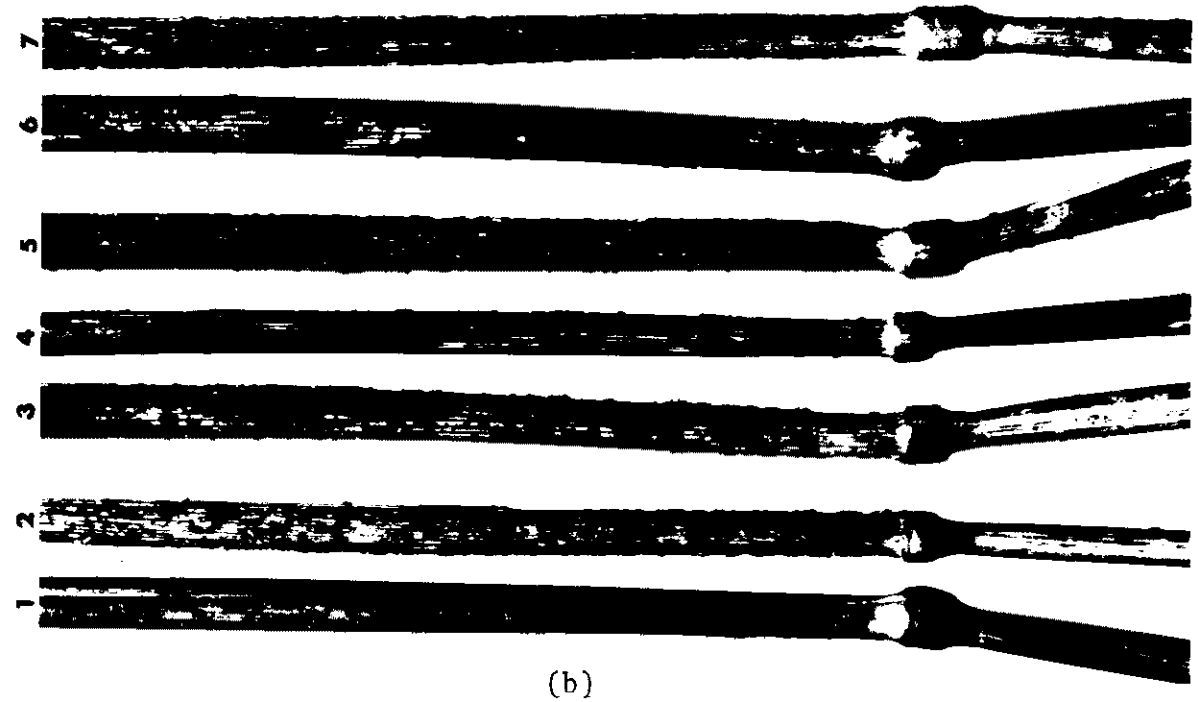
d) Renown Selection/W3498: 1 Renown Selection; 2-4 Resistant segregates; 5-6 Susceptible segregates; 7 W3498.



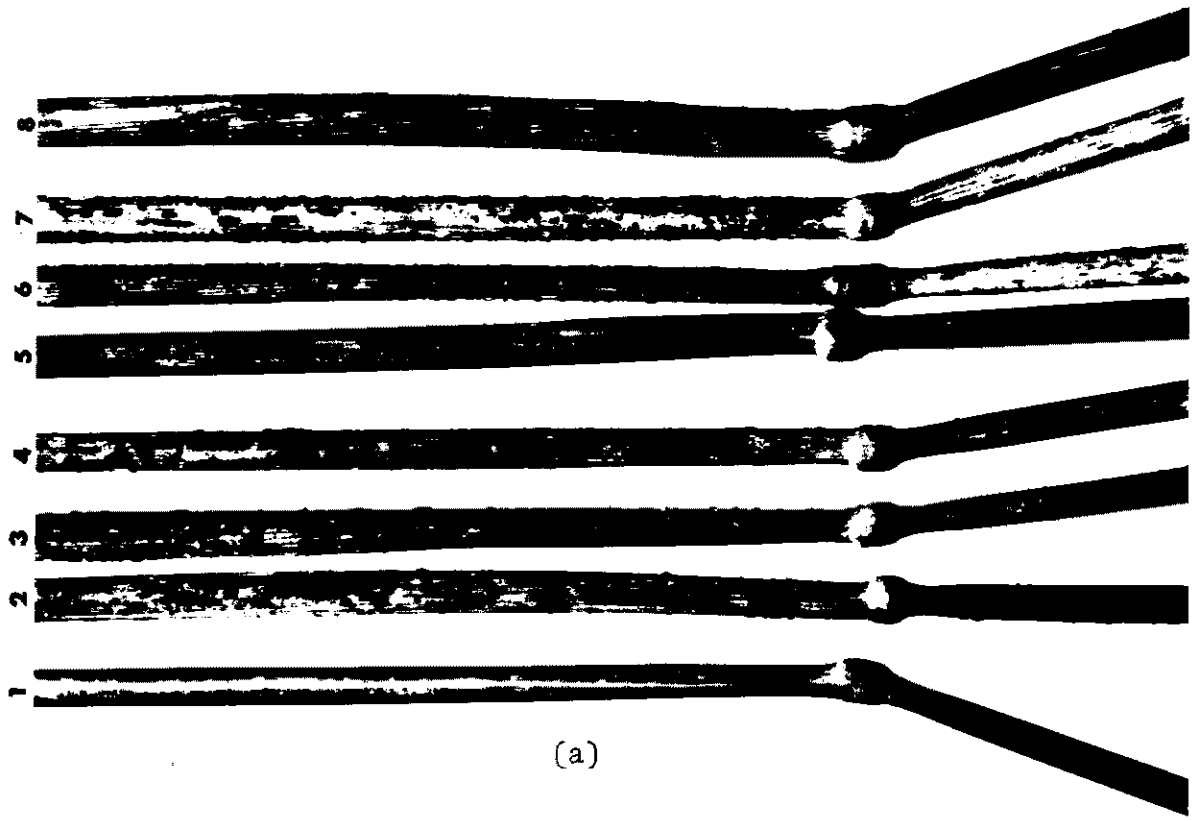








(b)



(a)

PLATE 2 Adult-plant reactions of F_1 hybrids and parents.

a) 1 Hopps; 2 Hopps/Yalta; 3 Hopps/W3498; 4 Hopps/CS; 5 Hopps/CS(Hope 3B); 6 W3498; 7 Yalta; 8 CS(Hope 3B).

b) 1 Warigo; 2 Warigo/Yalta; 3 Warigo/CS; 4 Warigo/CS(Hope 3B); 5 Chinese Spring; 6 CS(Hope 3B); 7 Yalta.

TABLE

Adult-plant reactions of F_1 plants from crosses of resistant and susceptible parents.

		Susceptible parent					
		Chinese Spring	Gabo	Spica	Yalta	W3498	Warden
Resistant parent	CS(Hope 3B)	S*	N/A**	N/A	N/A	N/A	N/A
	Hopps	S	N/A	S	S	S	S
	Selkirk	N/A	S	N/A	N/A	S	N/A
	Warigo	S	N/A	N/A	S	N/A	N/A

* susceptible

** not available

TABLE 8

Segregation for adult-plant reaction in F_2 populations derived from crosses of resistant and susceptible parents.

Cross	Designation	Resistant	Susceptible	$\chi^{2*}_{1:3}$
Hopps/CS	HX73.38.1	27	62	1.35
Spica/Hopps	HX72.151.1	17	44	0.27
Hopps/W3498	HX72.76.2	10	28	0.04
W3498/Hopps	HX72.176.2	15	39	0.22
Hopps/Yalta	HX72.73.3	19	46	0.62
Total segregation for Hopps		88	219	2.19
Gabo/Selkirk	HX72.48.1	29	77	0.32
Warigo/CS	HX73.71.1	24	55	1.22
Warigo/Yalta	HX73.79.1	18	47	0.25
Total segregation for Warigo		42	102	1.33
Heterogeneity χ^2				0.86
Total segregation for all crosses		159	398	3.43

* Value for significance at P=0.05, with 1 df is 3.84, with 7 df is 14.07.

The segregations for each cross, for crosses involving each resistant parent and for the overall total, fitted a hypothesised ratio of 1 resistant: 3 susceptible. This suggests that adult-plant resistance in each of the resistant parents is controlled by recessive alleles at a single locus.

5.2.3 F_3

F_3 family classifications for various crosses involving CS(Hope 3B), Hopps, Renown Selection, Selkirk and Warigo are summarised in Table 9 and illustrated in Plate 1. Three phenotypic classes, homozygous resistant, segregating and homozygous susceptible were distinguished. The various crosses were grown and classified during 1973, 1974 or 1975, and in a number of instances, the same lines from certain crosses were studied in more than one year. In those crosses where the same lines were studied in two different years, only the data set with the larger total number of lines was used to obtain the various parent total values in Table 9. The observed family classifications fitted a ratio of 1 homozygous resistant: 2 segregating: 1 homozygous susceptible confirming that reaction in each instance was determined by segregation of alleles at a single locus.

Where families were tested in different years the classifications in the CS/CS(Hope 3B) cross corresponded in every instance, reflecting the reliability with which families could be classified, and also indicating that strain composition did not affect the results. Of 136 F_3 families in Hopps/Yalta, 122 had been examined in 1974. One hundred and seventeen were similarly classified in both years. Three families classified

TABLE 9

Segregation class frequencies of F_3 families in various crosses involving resistant and susceptible parents when tested as adult-plants.

(HR = homozygous resistant; Seg = segregating; HS = homozygous susceptible)

Cross	Designation	Year tested	HR	Seg	HS	$\chi^{2*}_{1:2:1}$
CS/CS(Hope 3B)	HX72.180.1**	1973	12	37	21	2.54
"	" **	1975	10	32	17	2.23
Hopps/Federation	HX67.211.1	1975	11	23	12	0.05
Spica/Hopps	HX72.151.1	1975	16	34	12	1.10
Hopps/Warden	HX72.71.1	1975	11	41	13	4.57
Hopps/W3498	HX72.76.1	1974	20	37	13	1.63
"	HX72.76.2	1975	15	41	21	1.22
Hopps/Yalta	HX72.73.1**	1974	31	68	26	1.37
"	HX72.73.1**	1975	34	72	30	0.71
Heterogeneity χ^2						3.97
Total segregation for Hopps			107	248	101	3.68
Renown/W3498	HX72.116.1	1975	17	34	9	3.20
Selkirk/W3498	HX72.130.1	1975	36	60	24	2.40
Gabo/Selkirk	HX72.48.1**	1974	40	63	38	1.65
"	" **	1975	54	86	55	2.72
Total segregation for Selkirk			90	146	79	2.44
Warigo/Svenno	HX72.181.1	1974	43	108	46	1.92
W3498/Warigo	HX72.182.1**	1974	25	42	27	1.15
"	" **	1975	42	87	51	1.10
Total segregation for Warigo			85	195	97	1.21
Heterogeneity χ^2						19.71
Total segregation for all crosses			309	655	303	1.51

* Value for significance at P=0.05 with 2 df is 5.99, 10 df is 18.31 and 22 df is 33.92.

** Certain F_3 families were retested in 1975.

homozygous susceptible in 1974, segregated in 1975, and two families classified as homozygous resistant in 1974, segregated in 1975. These classification differences were probably due to chance or to misclassification in one, or other, year. As the resistant phenotype was recessive, no resistant plants might have been sampled in the 1974 samples of the first three discrepant lines. It is more difficult to account for the disparity between homozygous resistant and segregating classifications, but error during subsampling, randomisation and sowing could not be discounted completely. One hundred and thirty-five F_3 families from the cross Gabo/Selkirk tested during 1975 had been tested in 1974. Three families classified as homozygous susceptible in 1974, were classified as segregating in 1975. From 180 F_3 families in W3498/Warigo examined during 1975, 92 had been studied during 1974. All 92 families were similarly classified in each year.

Between 15 and 30 plants within each of 34 F_3 segregating families from the cross Hopps/Yalta, 36 F_3 families from Gabo/Selkirk and 21 F_3 families from W3498/Warigo were individually classified for adult-plant reaction during 1975. Two phenotypic classes, resistant and susceptible were recognised. Summarised results appear in Table 10 and individual family results in Appendix 1. Although segregations for a single F_3 family from each of the crosses Hopps/Yalta and Gabo/Selkirk deviated significantly from expectation for a ratio of 1 resistant: 3 susceptible (Appendix 1), two such instances in a total of 91 tests is near expectation. Data were homogeneous as indicated by χ^2 tests of heterogeneity. Again, the total segregation ratios for each cross, as well as the overall total for the three crosses, fitted the hypothesised segregation at a single locus.

TABLE 10

Pooled frequencies of resistant and susceptible F_4 plants derived from segregating F_3 families tested in 1975.

Cross	Designation	No. of F_3 families	Resistant	Susceptible	$\chi^2_{1:3}$	P value
Hopps/Yalta	HX72.73.1	34	168	544	0.75	>0.3
Gabo/Selkirk	HX72.48.1	36	232	632	1.58	>0.2
W3498/Warigo	HX72.182.1	21	108	285	1.29	>0.2
Total segregation for all crosses		91	508	1461	0.67	>0.4
Total χ^2 for all crosses 3 df					3.62	>0.3
Heterogeneity χ^2 2 df					2.95	>0.2

5.2.4 $\underline{F_4}$

In 1973, a minimum of two and maximum of five single random spikes were harvested from 20 F_3 rows of the cross CS/CS(Hope 3B). Similarly, in Hopps/Federation, from three to six single plants were taken from 39 F_3 space-planted lines. Both groups were tested for adult-plant reaction in 1975. For the purpose of analysis, to make the contribution from each F_3 family unbiased, results from the first two F_4 lines tested in each F_3 family in the cross CS/CS(Hope 3B) and first three F_4 lines in each F_3 family in Hopps/Federation were considered. The analysis for each cross was then repeated using the last two and last three F_4 results, respectively. Because of increased homozygosity (75%) in F_4, the results, summarised in Table 11, were tested for conformity to a hypothesised ratio of 3 homozygous resistant: 2 segregating: 3 homozygous susceptible.

The data were consistent with the hypothesis that the adult-plant resistances in CS(Hope 3B) and Hopps were determined by alleles at single loci. The behaviour of the F_4 lines reflected the classified behaviour of the respective F_3 families. Of 38 F_4 lines derived from segregating F_3 families in CS/CS(Hope 3B), 12 were homozygous resistant, 18 were segregating and 8 were homozygous susceptible ($\chi^2_{1:2:1}$ = 0.95; P 2 df >0.5). Similarly, the F_4's derived from segregating F_3 families in Hopps/Federation were classified, 30 homozygous resistant, 46 segregating and 24 homozygous susceptible ($\chi^2_{1:2:1}$ = 1.36; P 2 df >0.5).

Hence, all tests on F_2, F_3 and F_4 results showed that the observed results could be explained on the basis of segregation at single loci, with resistance being recessive in each instance.

TABLE 11

Frequencies of F_4 families in segregation classes for two crosses when tested in 1975. (HR = homozygous resistant; Seg = segregating; HS = homozygous susceptible)

Cross	Designation	F_4 lines/ F_3 family	HR	Seg	HS	$\chi^{2*}_{3:2:3}$
CS/CS(Hope 3B)	HX72.180.1	first two	19	11	10	2.83
		last two	20	9	11	2.83
Hopps/Federation	HX67.211.1	first three	50	23	44	2.19
		last three	45	29	43	0.05

* Value for significance at P=0.05 is 5.99.

5.3 GENETIC ANALYSIS OF INTERCROSSES OF RESISTANT PARENTS

5.3.1 F_1

Adult-plant reactions of five F_1 plants from various intercrosses involving CS(Hope 3B), Hope, Hopps, Renown Selection, Selkirk and Warigo are presented in Table 12.

TABLE 12

Adult-plant reactions of F_1 plants derived from intercrosses of resistant parents when tested in 1974.

Female parent	Male parent				
	CS(Hope 3B)	Hope	Hopps	Renown Selection	Selkirk
Warigo	R*	N/A**	R	R	R
Selkirk	N/A	R	R	N/A	
Renown Selection	N/A	R	R		
Hopps	R	R			

* resistant
** not available

Because resistance was shown to be recessive in resistant/susceptible crosses, the simplest hypothesis to explain the resistance of the various hybrids is that the same allele is involved in each instance. If the alleles determining resistance were different the hybrids should have been susceptible.

Two 1,000 grain weight measurements were made on the 5 F_1's and respective parents in each of the crosses, CS/CS(Hope 3B), Hopps/CS(Hope 3B), Hopps/CS, Warigo/CS(Hope 3B) and Warigo/CS grown in 1975. Cross and parent means with the analysis of variance are presented in Table 13.

TABLE 13

Analysis of variance, cross and parent means for 1,000 grain weight measurements for five crosses.

Source of variation		df	M.S.	F
Among plants		44	0.892	
Treatment (genotypes)	A	8	4.654	$\frac{A}{B}$ 82.54**
Among plants within genotypes (experimental error)	B	36	0.061	$\frac{B}{C}$ 3.37*
Among samples within plants (sampling error)	C	45	0.018	
Total		89		

* P<0.05;
** P<0.01.

Cross and parent means (1,000 grain wt. in g.)

CS/CS(Hope 3B)	10.96
Hopps/CS(Hope 3B)	27.92
Hopps/CS	20.05
Warigo/CS(Hope 3B)	25.92
Warigo/CS	18.49
Chinese Spring	10.29
CS(Hope 3B)	18.91
Hopps	24.29
Warigo	28.95
l.s.d. (P=0.05)	1.15

The resistant/resistant F_1's and CS(Hope 3B) had significantly higher 1,000 grain weights than their respective resistant/susceptible F_1's and Chinese Spring, reflecting the recessiveness of the resistance. This assumes that the level of heterosis for grain weight under rust free conditions in hybrids involving

CS and CS(Hope 3B), when each was crossed to a third parent were similar. The grain weights of Chinese Spring and CS(Hope 3B), grown in rust free plots during 1972 and 1973, were not significantly different (Refer section 4.2), indicating a probable similarity in the genetic factors determining grain weight.

5.3.2 $\underline{F_2}$

Adult-plant reactions were determined on single F_2 plants from various intercrosses in 1974. All plants were resistant. Table 14 lists the numbers of plants tested in each cross.

TABLE 14

Numbers of resistant F_2 plants in intercrosses of resistant parents when tested in the field in 1974.

Cross	Designation	No. of progenies	Number of plants
Hope/Hopps	HX72.9.1,2 & 3	3	267
Hope/Renown Selection	HX73.6.2	1	18
Hope/Selkirk	HX73.1.1 and 2	2	290
Selkirk/Warigo	HX73.2.1	1	794
Warigo/Hopps	HX73.5.1	1	746
Warigo/Hopps	HX73.3.1 and 2	2	985
Total			3200

5.3.3 $\underline{F_3}$

Adult-plant reactions of F_3 populations from various intercrosses involving CS(Hope 3B), Hope, Hopps, Renown Selection, Selkirk and Warigo were studied in 1974 or 1975. All families, the numbers of which are listed in Table 15, were resistant in each cross.

TABLE 15

Numbers of resistant F_3 families in various crosses between resistant parents tested in two years.

Cross	Designation	Year tested	No. of families
Hopps/CS(Hope 3B)	HX73.17.1	1975	57
Hopps/Hope	HX73.32.1	1975	30
Renown Selection/Hopps	HX72.102.1	1974	73
Hopps/Selkirk	HX72.67.1	1974	71
Hopps/Warigo	HX73.75.1	1975	25
Warigo/CS(Hope 3B)	HX73.72.1	1975	90
Warigo/Renown Selection	HX73.5.1	1975	98
Selkirk/Warigo	HX73.2.1	1975	100
Hope/Selkirk	HX73.1.1	1975	30
Total			574

In Hopps/Selkirk, no susceptible F_3 families were observed, but within five different families, single susceptible plants were recorded. These plants were considered to be outcrosses in F_2, or contaminants introduced during machine threshing of F_2 populations. In each instance, one or other of these explanations was supported by the non-typical spike morphology shown by the plants. Again, in the cross Renown Selection/Hopps, two families contained single susceptible plants. Cultivar Hopps is renowned for its instability which is attributed to outcrossing. Possibly, this tendency also occurs in its hybrids. The F_2 and F_3 data suggest that the gene(s) determining adult-plant resistance in Hope and various Hope or H44-24 derivatives are allelic, or very closely linked. Since the F_1 plants were resistant, the hypothesis of allelism is preferred.

5.4 GENETIC ANALYSIS OF SEEDLING REACTION OF CS(HOPE 3B)

CS(Hope 3B) consistently expressed infection type "x33+n" when tested with a range of *P. graminis tritici* strains including 59-E-5,7, 126-Anz-6,7,11, 34-Anz-2,4,5,7,11, 116-Anz-4,5, 326-Anz-1,2,3,5,6, 343-Anz-1,2,3,5,6, 21-Anz-1,2,5 and 222-Anz-1,2,3,5,6. The infection type was not strictly mesothetic, as "3+" pustules associated with necrotic regions predominated, while small pustules of "1" type occurred to a lesser extent. Pustules of "2" type did not occur (Plate 3). Chinese Spring produced infection type "3+", when tested with the above strains under similar conditions. Following a preliminary experiment, involving tests of ten F_3 families and parents of the cross CS/CS(Hope 3B) with strains 59-E-5,7, 126-Anz-6,7 and 34-Anz-2,4,5, strain 59-E-5,7 was selected for a more comprehensive genetic study of F_3 families from the cross CS/CS(Hope 3B). This culture appeared to produce the largest contrast between parents. Furthermore, differences between the respective parents were increased by obtaining the maximum levels of infection, in warm (20-25°C), well lit (~2500 ft candles) conditions. Cool conditions and/or reduced light intensity depressed development of necrosis.

5.4.1 F_1

Five F_1 seedlings produced infection type "3+" when inoculated with strain 59-E-5,7, indicating the recessiveness of the CS(Hope 3B) reaction.

5.4.2 F_3

Of the 47 F_3 families, tested as seedlings with 59-E-5,7, 10 were homozygous low in reaction (infection type "x33+n"), 18 were homozygous high (infection type "3+") and 19 segregated.

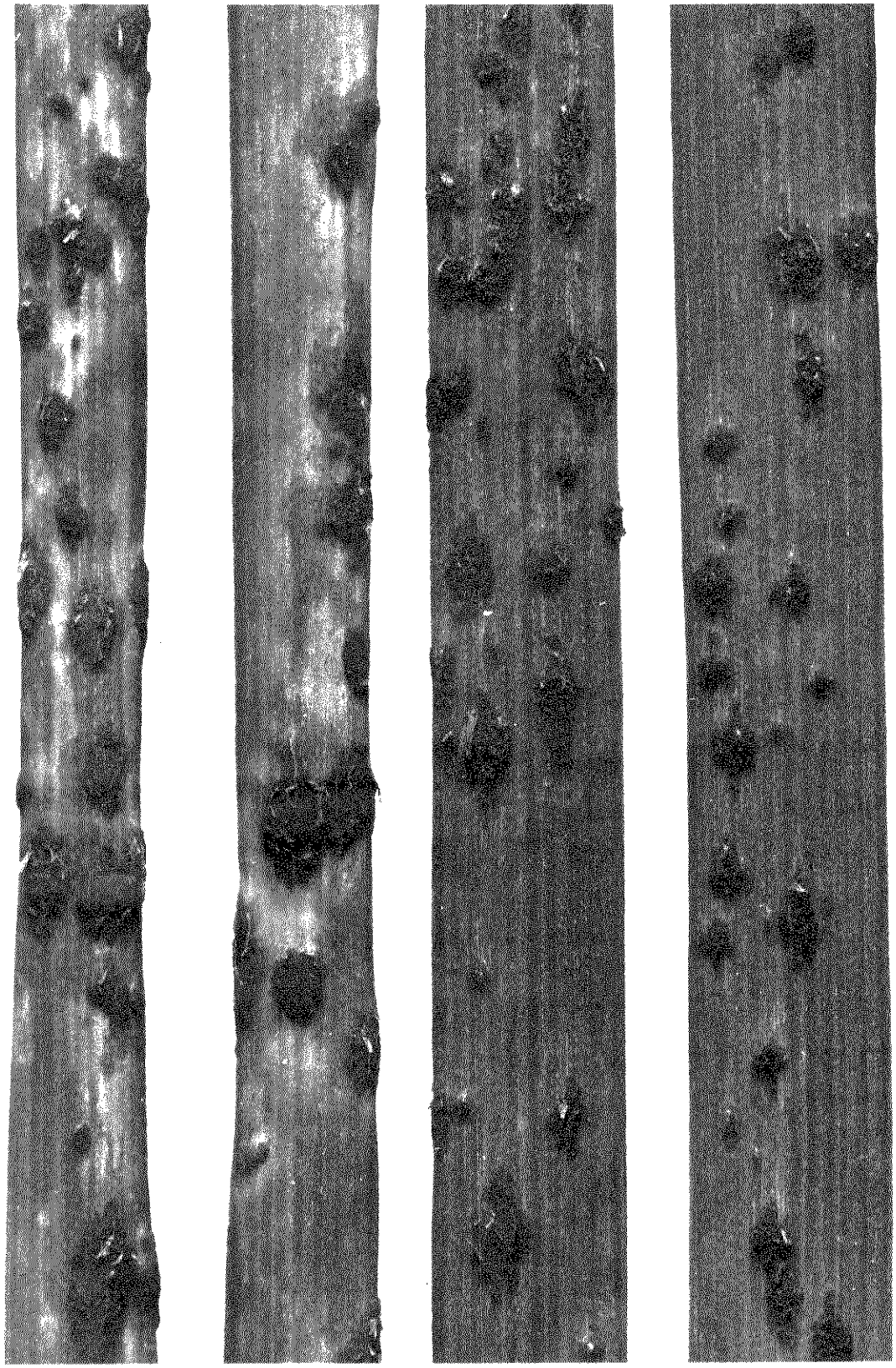


PLATE 3 Left to right: Infection types produced on adaxial primary leaf surfaces when the following lines were inoculated with *P. graminis tritici* strain 59-E-5,7 - 1 CS(Hope 3B); 2 and 3 contrasting F_3 segregates; 4 Chinese Spring.

This result tentatively suggested segregation at a single locus ($\chi^2_{1:2:1}$ = 4.45; P value 2 df >0.1).

Seedlings within the 19 segregating F_3 families were individually classified for low reaction (it "x33+n") or high reaction (it "3+") (Table 16).

With one exception, all families segregated in accordance with the hypothesised ratio of 1 low reaction: 3 high reaction. However, the χ^2 value for the total segregation frequency was highly significant (P<0.01). This was a consequence of cumulative excessive numbers of individuals with high reaction. This deviation may have been due to a consistent difficulty in classification. The F_3 segregation data indicated that the seedling reaction of CS(Hope 3B) to strain 59-E-5,7 may be controlled by a single recessive gene. However, individual family segregations fitted the ratio 1 low reaction: 4.05 high reaction, calculated from the total segregation, which may indicate a more complex inheritance behaviour.

5.4.3 Correlation of Seedling Reaction to 59-E-5,7 with Adult-plant Reaction

Of the 47 F_3 families classified at the seedling stage, the results of 43 corresponded with the adult-plant reactions. Four families classified homozygous high reaction as seedlings, segregated in the field. These results indicate that linked genes may be involved. However, the occurrence of recombinants in one group suggests that misclassification may be involved, possibly due to either failure to detect the small numbers of segregates with low reaction, or alternately the gene may be impenetrant in some lines. Such a result is also consistent with the deficiency of segregates with low reaction in the

TABLE 16

F_3 seedling segregation within segregating F_3 lines of the cross CS/CS(Hope 3B) when tested with strain 59-E-5,7.

Line designation	Low reaction (it "x33+n")	High reaction (it "3+")	$\chi^2_{1:3}$	$\chi^2_{1:4.05}$
3052	5	35	3.33	1.34
3053	6	27	0.82	0.05
3054	11	35	0.03	0.49
3056	8	33	0.66	0.01
3057	4	25	1.94	0.66
3058	7	24	0.09	0.15
3062	4	35	4.52*	2.23
3071	6	25	0.53	0.01
3072	10	25	0.24	1.69
3074	6	18	0	0.41
3079	6	31	1.52	0.30
3081	7	20	0.01	0.64
3091	4	16	0.27	0.01
3096	3	26	3.32	1.63
3098	7	17	0.22	1.33
3101	3	26	3.32	1.63
3105	6	23	0.29	0.01
3108	8	14	1.52	3.82
3112	5	15	0	0.34
Total segregation for all families	116	470	8.47**	0
Total χ^2 df 19			22.64	
Heterogeneity χ^2 df 18			14.17	

Values for significance, 1 df at P=0.05 is 3.84, at P=0.01 is 6.64, 18 df at P=0.05 is 28.87.

overall seedling studies.

It is not clear from this result if alleles determining the low seedling reaction of CS(Hope 3B) are identical with those determining adult-plant resistance to field strains.

Hopps and Warigo consistently exhibited an infection type "x33+n" when tested with strains virulent for the various designated specific resistance genes which these cultivars possess. In contrast, the adult-plant susceptible cultivars Aotea, Gala and Glenwari gave an infection type "3+". Classification of F_3 families from Hopps/W3498, Hopps/Yalta, Hopps/Federation and Warigo/W3498 proved difficult, and close correlations between seedling determinations and adult-plant reactions were not obtained. F_3 families that were homozygous resistant as adult-plants did not always express the infection type "x33+n". However, 10 families from Hopps/Federation which were clearly homozygous it"x33+n" as seedlings were adult-plant resistant in each instance.

Because seedling and adult-plant reactions were not completely correlated, either because of linkage or because of variable penetrance of a single factor at the seedling stage, it was apparent that breeding programmes would not be aided by seedling selection for infection type "x33+n".

5.4.4 Effect of Hope Adult-plant Resistance on 1,000 Grain Weights in the Presence of Stem Rust

Thousand grain weights were determined on 9 homozygous resistant and 9 homozygous susceptible F_3 families from CS/CS(Hope 3B) grown in 1973. Family and treatment means, together with an analysis of variance with equal replications and subsamples, are presented in Table 17. Kernel samples are illustrated in Plate 4.

TABLE 17

Analysis of variance of 1,000-grain weights and family and treatment means for F_3 families of CS/CS(Hope 3B).

Source of variation		df	MS	F
Among families		17	0.4501	
Treatment (resistant/susceptible)	A	1	5.6330	$\frac{A}{B}$ 44.54*
Among families within treatments (experimental error)	B	16	0.1265	$\frac{B}{C}$ 84.98*
Among samples within families	C	18	0.0015	
Total		35		

* $P<0.01$.

Family and treatment means (g)

Homozygous resistant	Homozygous susceptible
24.74	13.76
24.10	12.15
19.09	12.77
14.62**	14.69
19.98	13.56
19.54	12.83
22.78	11.61
25.03	15.03
22.91	14.68
21.79 ± 3.40	13.46 ± 1.20

l.s.d. (P=0.05) for treatment means is **1.60**

** This line was retested in 1975 and shown to be resistant but of poor vigour.

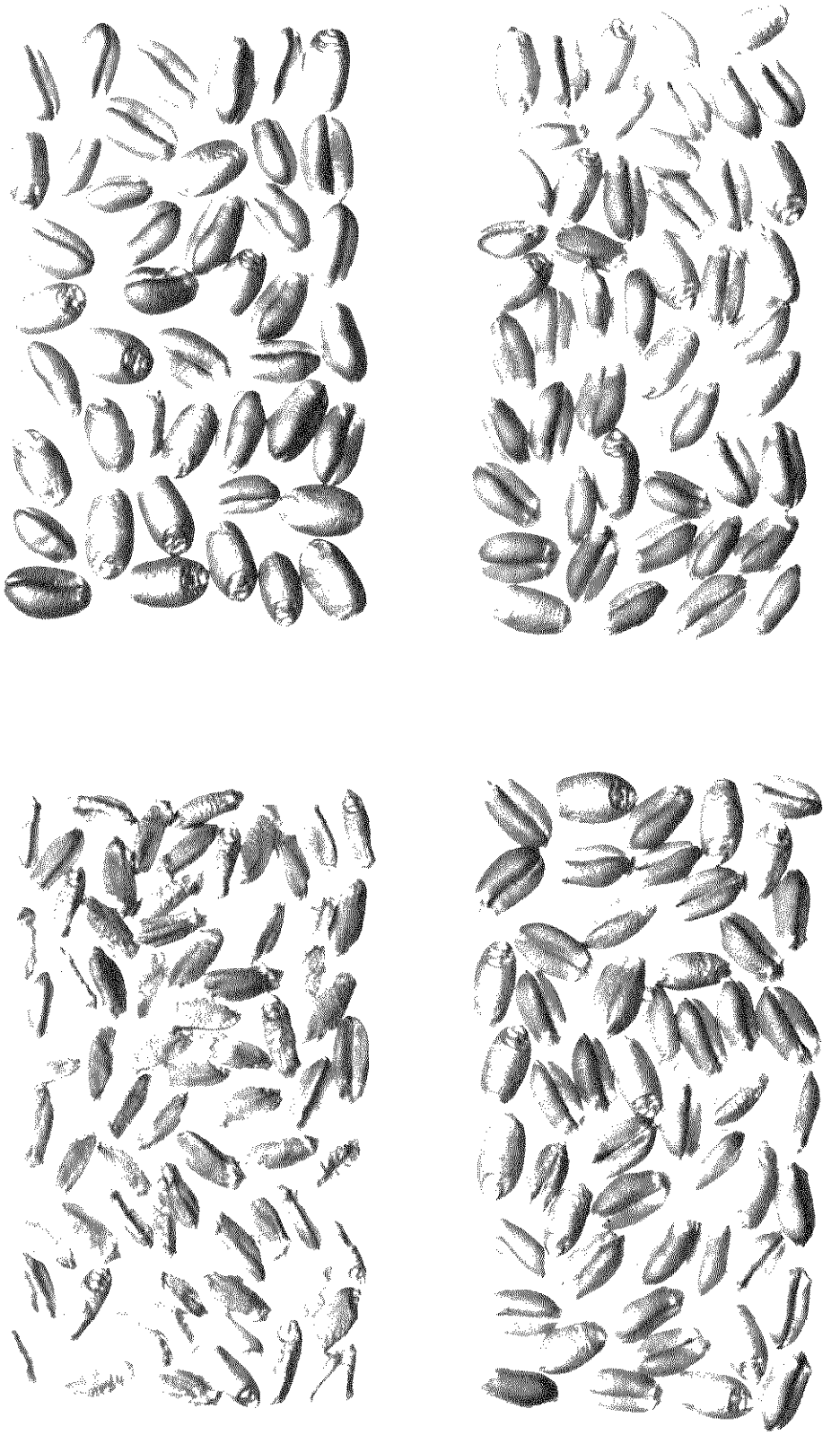


PLATE 4 Effect of stem rust on grain weights of CS(Hope 3B) (top) and Chinese Spring (bottom). Left: rusted, Right: non-rusted.

The significant variation associated with disease reaction and the clear divergence of the treatment means showed that the gene in CS(Hope 3B) offered considerable protection against grain weight loss in the presence of rust. The significant variation associated with families within treatments indicated significant environmental and/or genotypic variation for 1,000 grain weight between the families.

5.5 GENETIC ANALYSIS OF VARIATION IN EXPRESSION OF ADULT-PLANT RESISTANCE

Some variation in the amount of rust occurring on resistant plants, in both the resistant/susceptible and resistant/resistant crosses, was consistently apparent. The inheritance of the variation in expression of the adult-plant resistance was studied in F_3 families from Warigo/CS(Hope 3B) and F_4 lines derived from homozygous resistant F_3 families in Gabo/Selkirk. Results from field experiments in 1972 and 1973 indicated that the second uppermost internode offered the greatest opportunity for disease development. A disease intensity rating was allocated for each plant on the basis of the percentage disease cover for each tiller. The analysis of each cross is discussed separately.

5.5.1 Warigo/CS(Hope 3B)

Warigo/CS(Hope 3B) F_3 populations were selected for study, because variation in disease intensity in this cross ranging from 0 to 30% was greater than other crosses such as Warigo/Renown Selection and Selkirk/Warigo (0-10%) (Plate 5). Note, in that plate the disease intensity ratings apply to greater areas than those visible. Further rust developed only with the approach of senescence. The degree of variation in disease

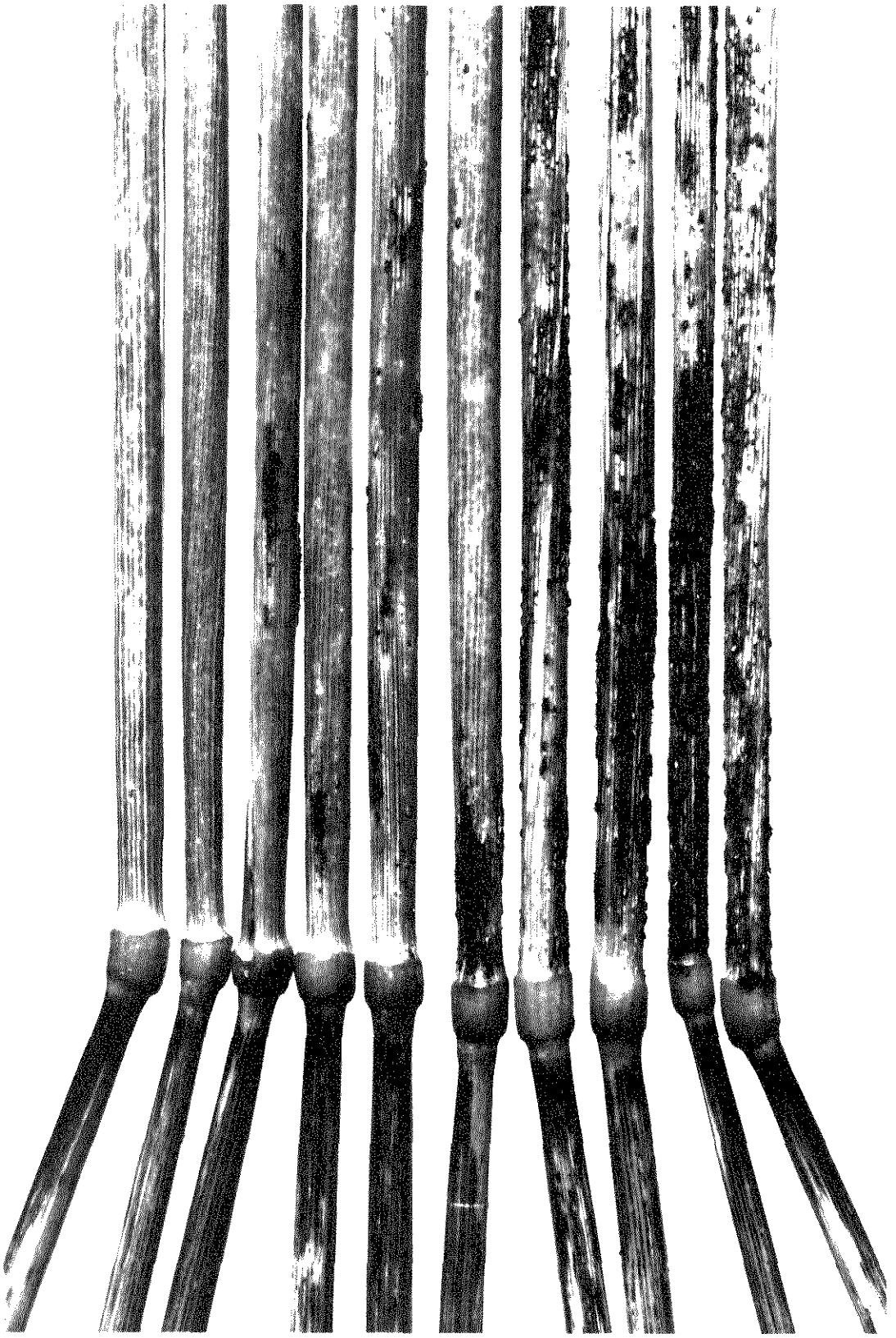


PLATE 5 Variation in the expression of resistance in F_3 lines of Warigo/CS(Hope 3B).
Left to right: Disease intensity ratings.

1-2.	0-2%
3-4.	2-5%
5-6.	5-10%
7-8.	10-20%
9-10.	>20%

intensity within each cross appeared to be related to the performance of the parents. As they neared maturity Hope, Hopps, Selkirk, Renown Selection and Warigo were diseased to a similar degree, 0 to 5%, whereas on CS(Hope 3B), the comparable disease intensity ranged from 10 to 30%. Two replications of approximately 20 individual plants in 91 F_3 families were assessed for disease intensity at maturity. Three categories of family grouping were made a) homozygous low in which F_3 family members had disease intensities ranging from 0 to 5%, b) homozygous high in which F_3 family members had disease intensities ranging from 10 to 30% and c) segregating in which F_3 intensities ranged from 0 to 30% or covered a major section of this range. Frequency distributions of disease intensity for individual F_3 families are presented in Appendix 2. Independent results from each replicate of the 91 lines agreed in 86 (96%) instances. In the five lines where classifications were not in agreement, a final decision was based upon the summed data for each line. Plots adjacent to the susceptible buffer rows tended to have higher disease intensities, presumably reflecting exposure to larger amounts of inoculum. Of the 91 F_3 families classified, 28 were classified homozygous low, 18 were homozygous high and 45 segregated. As the observed distribution of families conformed with a ratio of 1 homozygous low: 2 segregating: 1 homozygous high ($\chi^2_{1:2:1} = 2.203$; $P>0.3$), it was suggested that variation in the level of adult-plant resistance in this cross was associated with segregation at a single locus. Since Warigo, Selkirk and Renown Selection were grouped in the lower category of resistance and since F_3 populations from Warigo/Renown and Selkirk/Warigo did not segregate beyond the

range 0-10% disease, it is suggested that these cultivars may each carry the same modifying allele. Since Hope reacts similarly to Renown Selection and Warigo and presumably carried the modifier, it therefore appears likely that the modifying locus is not linked with the adult-plant reaction locus, presumably located in chromosome 3B.

5.5.2 Gabo/Selkirk

Measurable variation in disease intensity was observed on homozygous resistant F_3 lines tested during 1974. The variation was measured in the field in 1975 using 4 or 5 F_4 lines derived from each of 15 random homozygous resistant F_3 families. Frequency distributions of disease intensity for each F_4 line were grouped into three disease reaction categories as in the Warigo/CS(Hope 3B) cross. An analysis of frequencies of disease categories is presented in Table 18. For analysis, the first four F_4 results for each F_3 family were considered and the analysis was repeated using the last four F_4 results.

TABLE 18

Frequencies of disease categories of F_4 lines derived from homozygous resistant lines in Gabo/Selkirk.

	Homozygous low	Segregating	Homozygous high	$\chi^{2*}_{3:2:3}$
First four F_4 results/F_3 family	21	20	19	2.31
Last four F_4 results/F_3 family	22	19	19	1.62

* Value for significance at P=0.05 is 5.99.

The results were consistent with an hypothesis that adult-plant resistance was modified by segregation at a single locus. From the F_4 data, the parent F_3 families were classified, 3 homozygous low, 10 segregating and 2 homozygous high. Although the sample of 15 families is very small, the distribution is consistent with a ratio of 1:2:1 ($\chi^2_{1:2:1} = 1.80$; $P>0.3$). F_4 lines derived from segregating F_3 families were classified 9 homozygous low, 20 segregating and 11 homozygous high ($\chi^2_{1:2:1} = 0.2$; $P>0.6$).

5.6 THE EFFECT OF ALLELES FOR SPECIFIC RESISTANCE IN SEEDLINGS ON THE EXPRESSION OF ADULT-PLANT RESISTANCE

5.6.1 Gabo/Selkirk

Correlation analysis of the F_4 disease category data with the segregation behaviour at the *Sr6*, *Sr7b*, *Sr17* and *Lr16* loci are presented on Table 19. To provide equal contributions to each F_3 family, 3 F_4 lines were included in each case.

As indicated by the non-significant heterogeneity χ^2 values, there was no evidence indicating that genotypes at the four loci influenced the expression of adult-plant resistance. The heterogeneity χ^2 value for *Sr17* and adult-plant reaction approached a P value of 0.1, but this deviation was considered insufficient to implicate an interaction between *Sr17* and the adult-plant reaction. F_4 segregation at the *Sr6* locus did not fit the expected 3:2:3 ratio. Although segregation at the *Sr6* locus of the parent F_3 families fits the expected 1:2:1 ratio ($\chi^2_{1:2:1} = 3.14$; $P>0.2$), an excess over the expected number of *Sr6 sr6* families was obtained (1 *Sr6Sr6* : 10 *Sr6sr6* : 3 *sr6sr6*). This appears to be a chance deviation due to a small sample size. The major component of

TABLE 19

Association of genotype at the *Sr6*, *Sr7b*, *Sr17* and *Lr16/Sr23* loci with the expression of adult-plant resistance in F_4 families from homozygous resistant F_3 lines in Gabo/Selkirk.

		Modification of resistance in F_4 families			
		Homozygous low	Segregating	Homozygous high	Total
Sr6 Sr6		4	1	1	6
Sr6 sr6		9	5	7	21
sr6 sr6		4	5	6	15
	Total	17	11	14	42
Sr7b Sr7b		7	6	4	17
Sr7b sr7b		3	1	2	6
sr7b sr7b		6	7	9	22
	Total	16	14	15	45
Sr17 Sr17		3	1	2	6
Sr17 sr17		0	6	5	11
sr17 sr17		11	9	8	28
	Total	14	16	15	45
Lr16 Lr16		3	7	7	17
Lr16 lr16		4	2	3	9
lr16 lr16		8	7	4	19
	Total	15	16	14	45

TABLE 19 (CONT)

Chi-square analysis

	Association of the expression of adult-plant resistance with:							
	Sr6		*Sr7b*		*Lr17*		*Lr16*	
	χ^2	P	χ^2	P	χ^2	P	χ^2	P
$\chi^2_{5:2:3}$ specific resistance gene	16.57	<0.01	4.01	>0.1	14.35	<0.01	0.72	>0.3
$\chi^2_{5:2:5}$ low vs high adult-plant resistance expression	0.32	>0.8	0.93	>0.5	2.70	>0.1	2.70	>0.1
$\chi^2_{9:6:9:6:4:6:9:6:9}$ Total segregation	18.82	<0.05	7.35	>0.3	24.45	<0.01	7.38	>0.3
χ^2 heterogeneity 4 df	1.93	>0.7	2.41	>0.5	7.40	>0.1	3.96	>0.3

deviation in the total χ^2 is attributable to deviation in the segregation at the *Sr6* locus.

Similarly, segregation at the *Sr17* locus was not in accordance with the hypothesised ratio of 3:2:3. Of 15 parent F_3 families, the classifications were 6 *Sr17 Sr17*, 7 *Sr17 sr17* and 2 *sr17 sr17* ($\chi^2_{1:2:1} = 2.20$; $P>0.3$). This excess of F_3 lines of genotype *sr17 sr17* was presumably a chance effect.

5.6.2 Warigo/CS(Hope 3B)

F_3 adult-plant resistance categories and genotypes for the *Sr7b* and *Sr17* loci are jointly presented in Table 20. The chi-square analysis of heterogeneity gave no evidence that particular alleles at these loci influenced the expression of adult-plant resistance.

TABLE 20

Association of genotypes at the *Sr7b* and *Sr17* loci with the expression of adult-plant resistance in F_3 families from Warigo/CS(Hope 3B).

	Expression of resistance in F3 families			
	Homozygous low	Segregating	Homozygous high	Total
Sr7b Sr7b	5	3	1	9
Sr7b sr7b	9	16	7	32
sr7b sr7b	4	9	4	17
Total	18	28	12	58
Sr17 Sr17	4	8	2	14
Sr17 sr17	11	20	7	38
sr17 sr17	5	5	4	14
Total	20	33	13	66

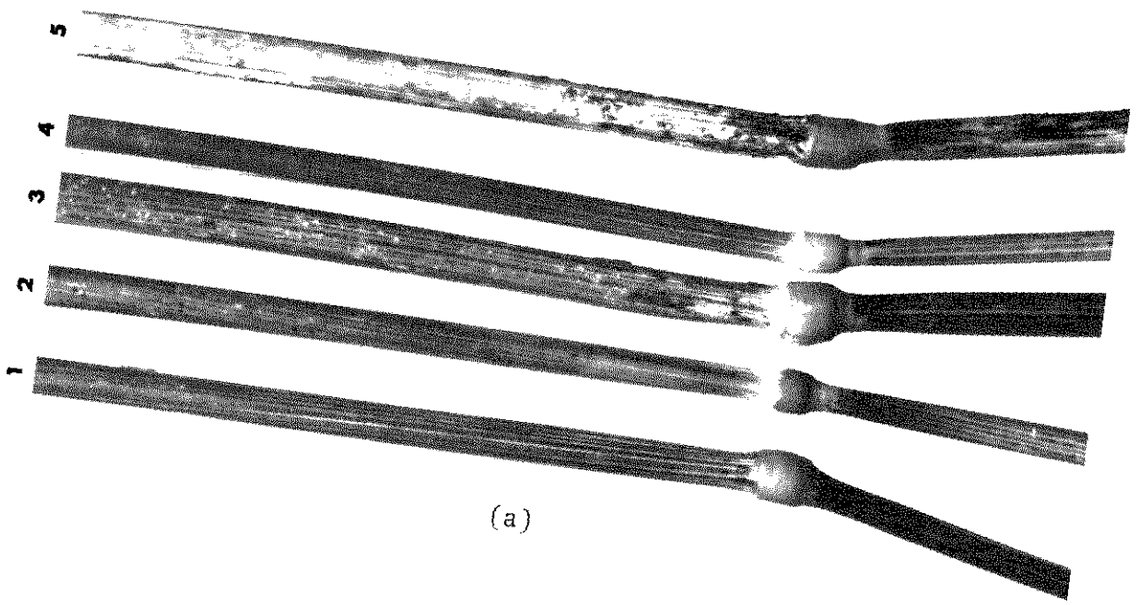
TABLE 20 (CONT)

Chi-square analysis

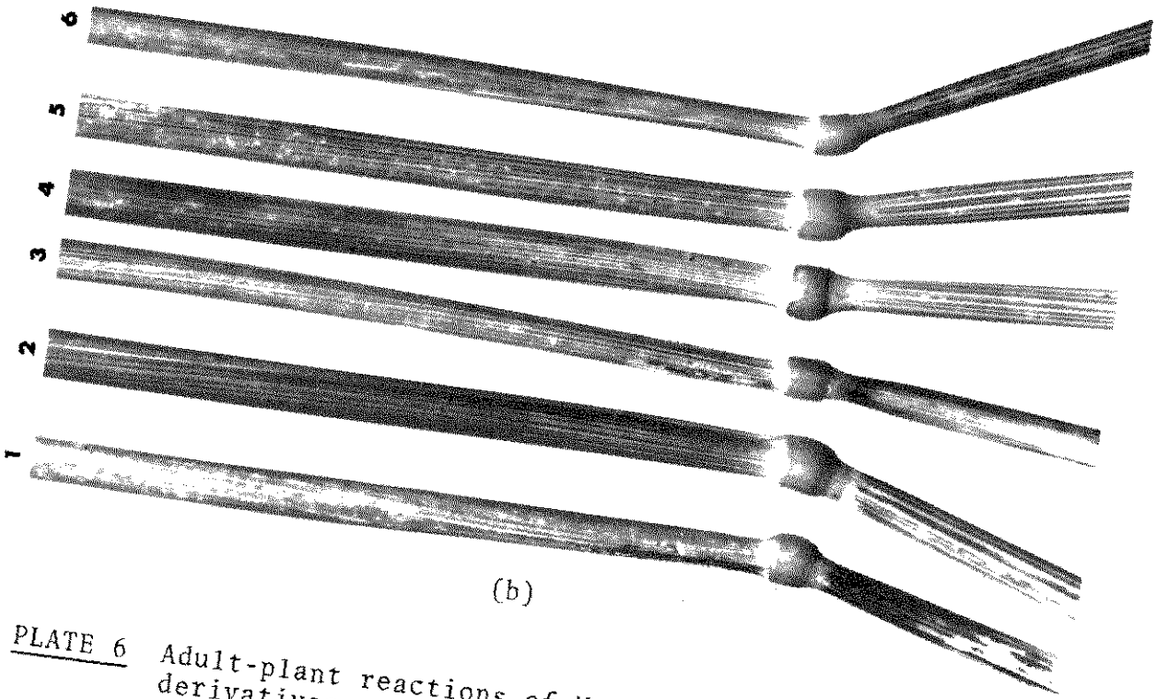
	Association of the expression of adult-plant resistance with:			
	Sr7b		*Sr17*	
	χ^2	P	χ^2	P
$\chi^2_{1:2:1}$ specific resistance gene	2.87	>0.2	1.52	>0.3
$\chi^2_{1:2:1}$ low vs high adult-plant resistance expression	1.31	>0.5	1.48	>0.3
$\chi^2_{1:2:1:2:4:2:1:2:1}$ Total segregation	6.02	>0.5	4.42	>0.7
Heterogeneity χ^2 4 df	1.84	>0.7	1.42	>0.8

5.7 ADULT-PLANT REACTION OF ADDITIONAL HOPE AND H44-24 DERIVATIVES

Adult-plant reactions of H44-24 and Hope derivatives, Africa Mayo, Aotea, Arther 71, CS(Hope 7B), Gala, Glenwari, Hofed, Hopps/Federation F_4 Selection, Kenya Page, Lancer, Lawrence, Lerma Rojo 64A, Renown W1242, Renown W2346, Samaca, Scout 66, and Yaqui 50 were assessed in 1974 and/or 1975 when strains present were virulent for *Sr17*. Aotea, CS(Hope 7B), Gala, and Glenwari were susceptible. All remaining cultivars were resistant and expressed pseudo-black chaff, suggesting that these cultivars probably possess the 'Hope' type adult-plant resistance (Plate 6).



(a)



(b)

PLATE 6 Adult-plant reactions of Hope and Marquillo derivatives.
Left to right:
a) 1 Scout 66; 2 Arthur 71; 3 Samaca; 4 Tobari 66; 5 Lerma Rojo 64A.
b) 1 Yaqui 50; 2 Kenya Page; 3 Africa Mayo; 4 Tezanos Pinto Precoz; 5 Pato; 6 Chris (note absence of black sub-nodal regions in 4 and 5).

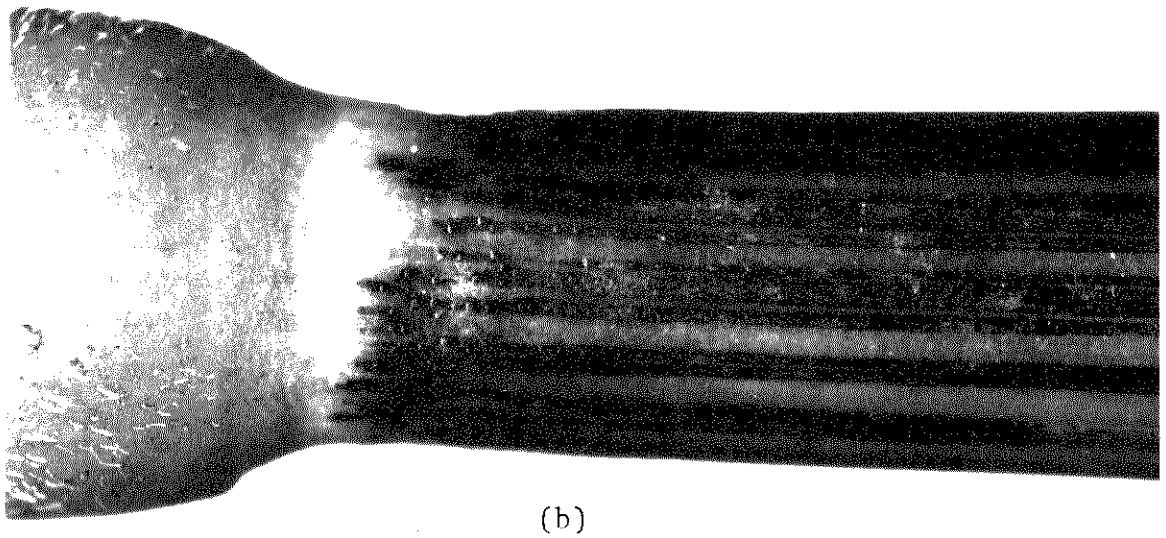
5.8 GREENHOUSE TESTS OF ADULT-PLANT REACTIONS OF HOPE DERIVATIVES

Adult plants of the Hope derivatives, CS(Hope 3B), Hopps, Renown Selection, Selkirk, Spica and Warigo and susceptible controls Chinese Spring and Yalta were tested in the greenhouse using strains 21-Anz-1,2,5 and 194-Anz-1,2,3,5,6. It was not possible to differentiate between resistant and susceptible cultivars, thus it was necessary to conduct genetic studies in the field. Presumably, environmental conditions within the glasshouse, including temperature, humidity, inoculation conditions and inoculum density allowed significant disease development on the resistant cultivars.

5.9 PSEUDO-BLACK CHAFF

Adult-plant resistance to stem rust in Hope, H44-24 and derivatives is associated with a physiological condition known as pseudo-black chaff (Broadfoot and Robinson, 1933; Johnson and Hagborg, 1943). In affected tissue pigment develops in chlorenchyma cells located in stems below the nodes, as well as in the glumes, and necks as illustrated in Plate 7. Microscopic examination showed that the cells in pigmented areas were disrupted and were presumably dead (Plate 8). Strong pseudo-black chaff development is unsightly and undoubtedly inhibits photosynthesis, probably sufficiently in some instances to affect yield. Observations on some materials supplied by Australian wheat breeders for testing in the Australian National Rust Program in 1975 suggested that excessive expression of pseudo-black chaff caused stem weakening and fracture near the top node. Furthermore, in northern Australia, there has been confusion between blackening

(a)



(b)

PLATE 7 Development of Pseudo-black chaff.

a) glumes and paleas.

b) below uppermost stem node. (x10)

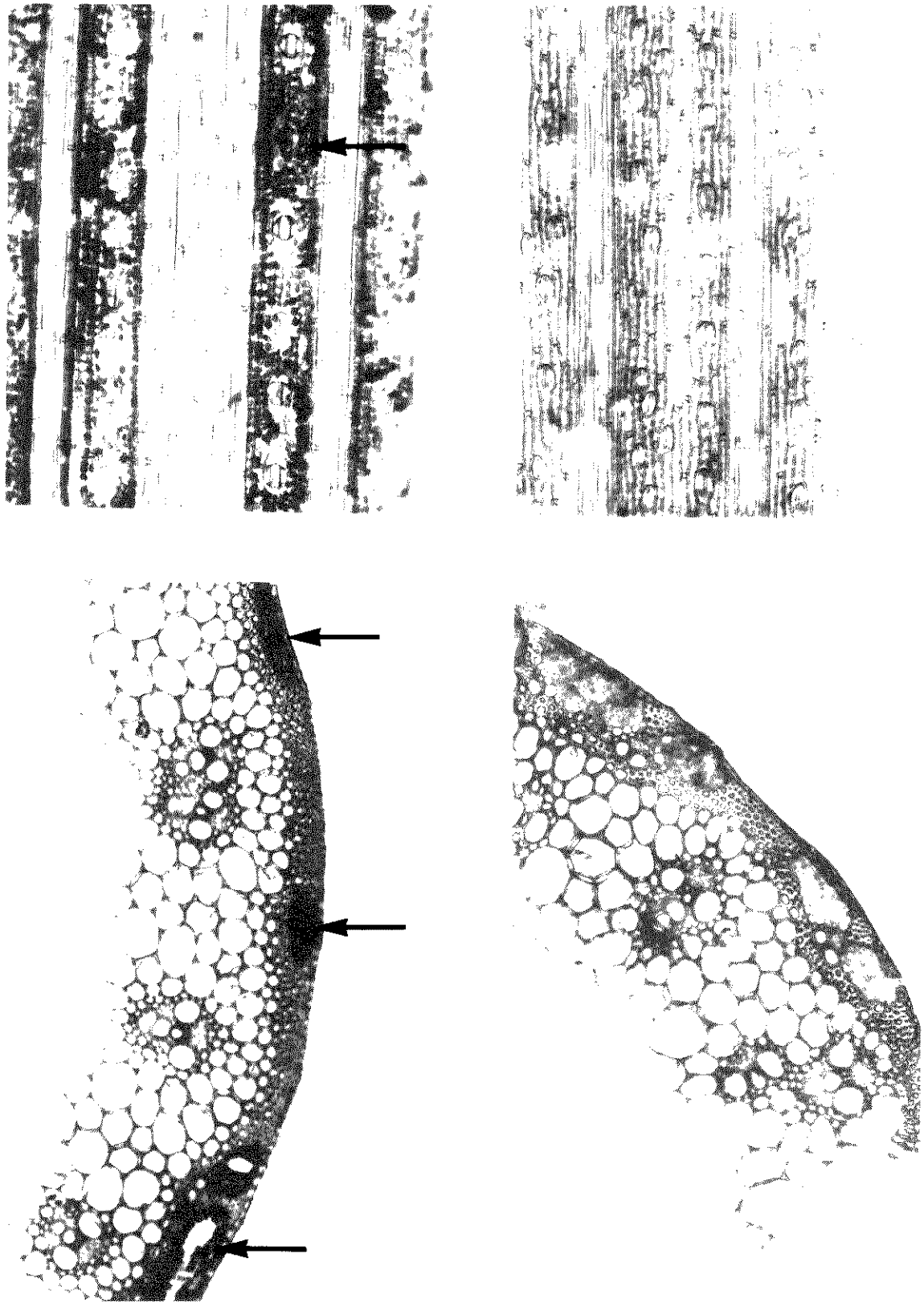


PLATE 8 Pseudo-black chaff development in sub-nodal chlorenchyma cells.
Top - longitudinal stem sections
Bottom - transverse stem sections
Left - Hopps : pigmented
Right - Yalta : non-pigmented
(note pigmented tissues arrowed) (all x70)

in cultivar Gatcher which may carry adult-plant stem rust resistance derived from Hope and H44-24, and susceptibility to *Septoria nodorum* Berk. The possible predisposition of cultivars with pseudo-black chaff to this and other pathogens has not been studied.

5.9.1 Inheritance of Pseudo-black Chaff

5.9.1.1 Parental reactions

Of the stem rust resistant genotypes studied, CS(Hope 3B), Hopps and Warigo were approximately similar in expression of pseudo-black chaff, whereas Selkirk and Renown Selection showed less pigmentation at Castle Hill in 1974 and 1975. The latter, North American cultivars, are not strictly comparable with CS(Hope 3B) and the Australian cultivars, because they are later flowering, and hence are subject to higher temperatures during the pigment development. The susceptible parents, Chinese Spring, Gabo, Spica, Yalta and W3498 did not develop pseudo-black chaff (Plate 1).

5.9.1.2 F_1

Five F_1 plants from each of 10 crosses involving carrier and non-carrier parents were studied in 1975. All hybrids (Table 21 and Plate 2) were free of pseudo-black chaff symptoms, hence the condition appears to be recessive.

TABLE 21

Pseudo-black chaff reactions of F_1 plants derived from crosses between carrier and non-carrier parents.

(N = no pseudo-black chaff symptoms; N/A = not available)

	Non-carrier parent				
Carrier Parent	CS	Gabo	Spica	Yalta	W3498
CS(Hope 3B)	N	N/A	N/A	N/A	N/A
Hopps	N	N/A	N	N	N
Selkirk	N/A	N	N/A	N/A	N
Warigo	N	N/A	N/A	N	N/A

5.9.1.3 F_2

The F_2 segregations obtained in 1975 for various crosses involving Hopps and Warigo are presented in Table 22. Only two phenotypic classes, pseudo-black chaff and non-pseudo-black chaff were distinguished.

5.9.1.4 F_3

F_3 family classifications for various crosses involving CS(Hope 3B), Hopps, Selkirk, Renown Selection and Warigo are summarised in Table 23. Three phenotypic classes, homozygous pseudo-black chaff, segregating and homozygous non-pseudo-black chaff were distinguished in tests covering three years.

For the F_2 and F_3 data, the segregations for each cross, for crosses involving each carrier parent, and for the overall total, fitted hypothesised ratios of 1 pseudo-black chaff: 3 non-pseudo-black chaff and 1 homozygous pseudo-black chaff: 2 segregating: 1 homozygous non-pseudo-black chaff, respectively. Therefore, pseudo-black chaff in each of the

TABLE 22

Segregation for pseudo-black chaff reaction in F_2 populations derived from crosses of carrier and non-carrier parents.

Cross	Designation	Pseudo-black chaff	Non-pseudo-black chaff	$\chi^{2*}_{1:3}$
Hopps/CS	HX73.38.1	27	62	1.35
Spica/Hopps	HX72.152.1	17	44	0.27
Hopps/W3498	HX72.76.2	10	28	0.04
W3498/Hopps	HX72.176.2	15	39	0.22
Total segregation (Hopps)		69	173	1.60
Warigo/CS	HX73.71.1	24	55	1.22
Warigo/Yalta	HX73.79.1	18	47	0.25
Total segregation (Warigo)		42	102	1.33
Heterogeneity χ^2				0.44
Total segregation (all crosses)		111	275	2.91

* Value for significance at P 1 df = 0.05 is 3.84,

5 df is 11.07.

TABLE 23

Segregation class frequencies of F_3 families in various crosses involving carrier and non-carrier parents.

(HBC = homozygous pseudo-black chaff; Seg = segregating
HNBC = homozygous non-pseudo-black chaff)

Cross	Designation	Year	HBC	Seg	HNBC	$\chi^{2*}_{1:2:1}$
CS/CS(Hope 3B)	HX72.180.1	1973	12	37	21	2.54
Hopps/W3498	HX72.76.1	1974	20	37	13	1.63
Hopps/Yalta	HX72.73.1	1974	31	68	26	1.37
Total segregation (Hopps)			51	105	39	2.65
Renown/W3498	HX72.116.1	1975	17	34	9	3.19
Gabo/Selkirk	HX72.48.1	1974	40	63	38	1.65
Selkirk/W3498	HX72.130.1	1975	36	60	24	2.40
Total segregation (Selkirk)			76	123	62	2.28
W3498/Warigo	HX72.182.1	1974	25	42	27	1.15
Heterogeneity χ^2						12.37
Total segregation (all crosses)			181	341	158	1.56

* Value for significance at P 2 df = 0.05 is 5.99, 12 df is 21.03.

carrier parents is controlled by recessive alleles at a single locus.

5.9.2 Intercrosses of Carrier Parents

5.9.2.1 F_1

Pseudo-black chaff reactions based on 5 F_1 plants from various intercrosses involving CS(Hope 3B), Hope, Hopps, Renown Selection, Selkirk and Warigo are presented in Table 24.

TABLE 24

Pseudo-black chaff reactions of F_1 plants derived from intercrosses of carrier parents when tested in 1974.

(BC = pseudo-black chaff; N/A = not available)

	CS(Hope 3B)	Hope	Hopps	Renown	Selkirk
Warigo	BC	N/A	BC	BC	BC
Selkirk	N/A	BC	BC	N/A	
Renown	N/A	BC	BC		
Hopps	BC	BC	N/A		

Since previous results showed that pseudo-black chaff was controlled by recessive alleles at a single locus in each genotype, the simplest hypothesis to explain the F_1 reaction is that the same allelic pair is involved in each parent. If different loci were involved the simplest model predicts that F_1's would not exhibit blackening.

5.9.2.2 F_3

Pseudo-black chaff reactions of F_3 populations from various intercrosses involving CS(Hope 3B), Hope, Hopps, Renown Selection, Selkirk and Warigo were studied in 1975. All families, the numbers of which are listed in Table 25, failed to segregate.

TABLE 25

Numbers of F_3 families studied for pseudo-black chaff reaction in various crosses.

Cross	Designation	No. of families
Hopps/CS(Hope 3B)	HX73.17.1	57
Hopps/Hope	HX73.32.1	30
Warigo/Renown Selection	HX73.5.1	98
Warigo/CS(Hope 3B)	HX73.72.1	90
Selkirk/Warigo	HX73.2.1	100
Hope/Selkirk	HX73.1.1	30
Total		315

The F_3 data confirmed the conclusion from F_1 observations that the same recessive alleles are carried by the various parents.

5.9.3 Association of the Pseudo-black Chaff Phenotype with Adult-plant Resistance

The pseudo-black chaff phenotype was completely associated with adult-plant resistance to stem rust in each F_2 and F_3 population listed in Tables 22 and 23. This complete correlation between pseudo-black chaff reaction and adult-plant reactions suggests that pigment formation and resistance may be determined by the same recessive alleles. Alternatively, recessive alleles at closely linked loci may control the two characters. As the behaviour of all 680 F_3 lines, listed in Table 23, to pseudo-black chaff and rust reaction were completely associated, the maximum applicable recombination frequency (r) at P=0.05 was calculated from the expression (Hanson, 1959):

$$(2r - \frac{3}{2} r^2) = 1 - (0.05)^{\frac{1}{680}}$$

$$[1 - (2r - \frac{3}{2} r^2)]^{680} = 0.05$$

$$r = 0.2\%$$

5.9.4 Genetic Modification of Pseudo-black Chaff

In early genetic studies on Selkirk it was noted that certain segregates showed levels of pseudo-black chaff development far in excess of that shown by Selkirk, suggesting that the degree of pigmentation could be modified. Variation in the intensity of the pseudo-black chaff phenotype was observed in 1975 in F_3 populations of Selkirk/Warigo and Warigo/Renown Selection, and in homozygous stem rust resistant F_3 families derived from Selkirk/W3498. Certain F_3 families and single plants were relatively free of pigment while remaining resistant to stem rust. This variation is illustrated in Plate 9.

Measurable variation in pigmentation was observed on homozygous resistant F_3 lines in Gabo/Selkirk in 1974. Based on a visual assessment at maturity of the mean pigmentation for each F_3 family, 3 families were selected as having low pigment and 8 families were selected for high pigment. The pigmented area below the top stem internodes for 10 plants per family were summed and averaged to give a mean family pigmentation. These families were similarly ranked at maturity in 1975. Five F_4 lines derived from each of the 11 F_3 families were assessed in 1975. Following a visual assessment at maturity of the average pigmentation on 10 F_4 plants per family, the mean F_4 family pigmentation was calculated and the families ranked. All F_4 lines derived from the 3 F_3 families with low pigment showed less

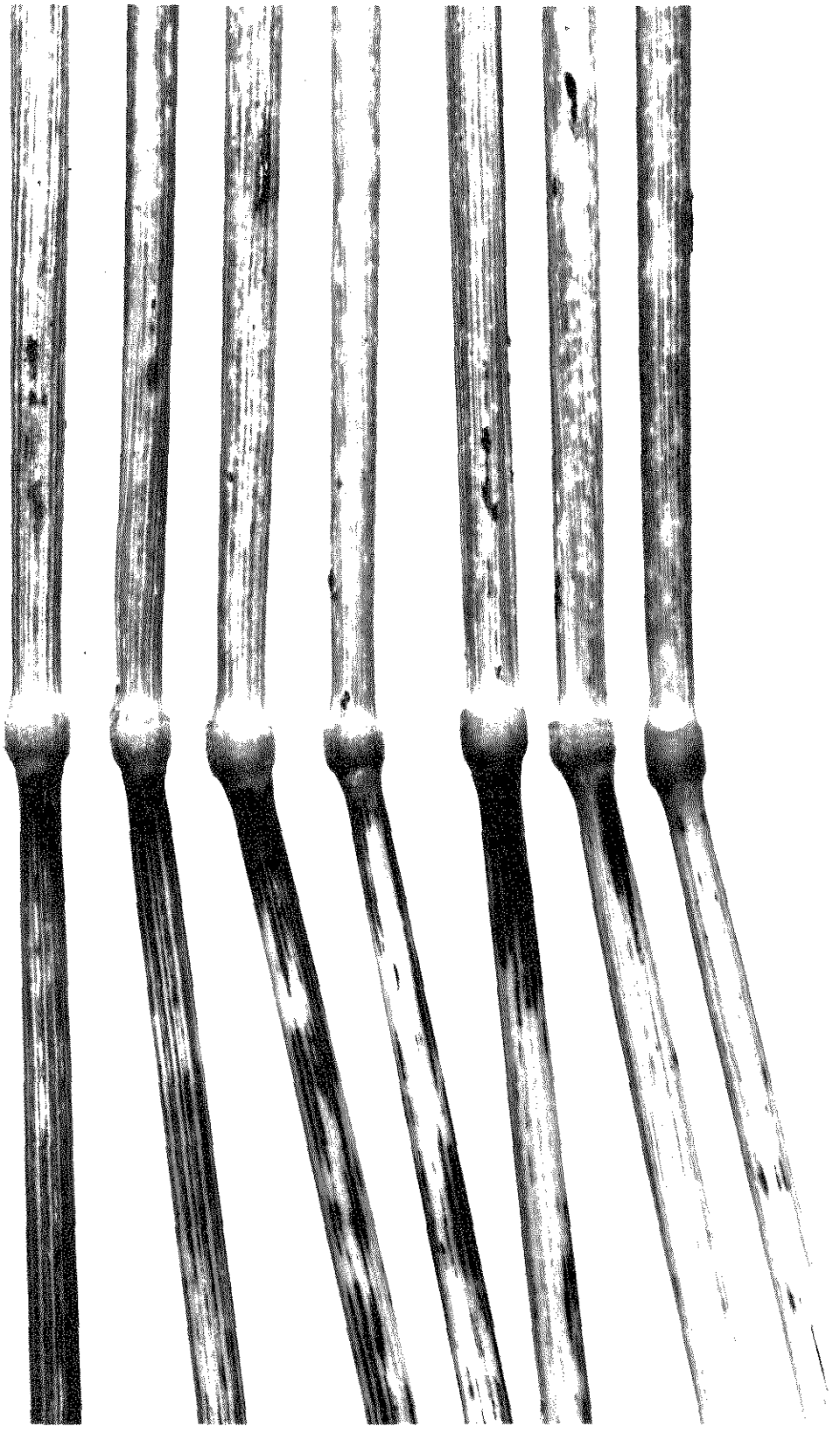


PLATE 9 Variation in sub-nodal pigment development in F_3 lines of Warigo/Renown.

pigmentation than F_4 lines derived from F_3's with high pigment. However, certain plants within these low pigment lines could not be distinguished from plants in high pigment lines. There was no correlation between disease intensity and degree of pigment development.

These results suggest that the expression of pseudo-black chaff can be significantly modified. Apparently, segregates with very low levels of pigment can be selected while retaining rust resistance.

5.10 ADULT-PLANT RESISTANCE OF SPICA TO STEM RUST

F_3 families from Spica/W3498 together with Spica, W3498 and Seafoam were inoculated in the greenhouse with a range of *P. graminis tritici* strains at the seedling and adult-plant stages.

5.10.1 Inheritance of Adult-plant Reaction

Separate groups of ten plants in 45 F_3 families were inoculated at anthesis with strains 126-Anz-6,7,11 and 34-Anz-1,2,3,4,5,6,7. These strains were chosen as both had produced low disease intensities on adult-plants of Spica in an earlier greenhouse study. Both strains are virulent on seedlings with *Sr7b* and *Sr17*, present in Spica. Two reaction classes, the first represented by few small pustules per unit area of stem and similar to Spica (low reaction), and the second by many large pustules per unit area and similar to W3498 (high reaction), were distinguished with each strain. Families were classified as homozygous low, segregating and homozygous high reaction. As shown in Table 24, adult-plant classifications for both strains were so similar that the exceptions were considered misclassifications, particularly with 34-Anz-1,2,3,4,5,6,7 where

greater difficulty in classification was experienced. Resistance appeared to be due to a single gene. Seedlings of the 45 F_3 families were inoculated with strains 21-Anz-1,2,3,7,11 and 326-Anz-1,2,3,5,6 in order to classify segregation of the *Sr17* and *Sr7b* loci, respectively. As shown in Tables 26 and 27, genotypes with respect to these loci were independent of the locus controlling adult-plant reaction to 126-Anz-6,7,11 and 34-Anz-1,2,3,4,5,6,7.

In order to compare the adult-plant reactions of Spica and its Seafoam parent, replicated pot sowings of these and W3498 were inoculated at anthesis with eight *P. graminis tritici* strains (Table 28). Reactions were visually assessed on a quantitative disease intensity scale, ranging from 0-5 (0 = disease free, 0.5 = trace, 5 = fully susceptible as represented by 21-Anz-5 on W3498) by 4 independent observers. The relatively small standard deviations of the mean disease intensities indicated similar observer assessments for each line. The data confirmed previous results indicating that Spica possesses adult-plant resistance to strains 126-Anz-6,7,11 and 34-Anz-1,2,3,4,5,6,7. Furthermore, Spica displayed a significant level of resistance to all cultures except 21-Anz-5 despite the fact that all were virulent for *Sr17* and *Sr7b*. By contrast, Seafoam displayed a significant level of resistance only to 126-Anz-6,7,11.

In the same experiment, 5 F_3 families homozygous low, and 4 families homozygous high, in reaction to 126-Anz-6,7,11 were selected for similar tests with 34-Anz-1,2,3,4,5,6,7, 21-Anz-2,3,4,5,7 and 126-Anz-6,7,11 (Table 29). Again, those lines previously classified as homozygous low displayed less disease than those classified as homozygous high in tests with

TABLE 26

Frequencies of F_2 genotypes determined from F_3 progeny tests in cross Spica/W3498.

(HL = homozygous low: Seg = segregating: HH = homozygous high)

		Adult-plant reaction to 126-Anz-6,7,11			
		HL	Seg	HH	Total
Adult-plant	HL	13	0	0	13
reaction to	Seg	2	19	0	21
34-Anz-1,2,3,4,5,6,7	HH	0	2	9	11
	Total	15	21	9	45
Sr7b Sr7b	HL	3	5	3	11
Sr7b sr7b	Seg	7	9	3	19
sr7b sr7b	HH	5	7	3	15
	Total	15	21	9	45
*Sr17 Sr17**	HL	2	6	3	11
Sr17 sr17	Seg	8	11	4	23
sr17 sr17	HH	5	4	2	11
	Total	15	21	9	45

* *Sr17* designates the recessive allele for low reaction (McIntosh, 1973).

Chi-square Analysis

	Association of adult-plant reaction with:			
	Sr7b		*Sr17*	
	χ^2	P	χ^2	P
$\chi^2_{1:2:1}$ adult-plant reaction	1.80	>0.3	1.80	>0.3
$\chi^2_{1:2:1}$ seedling reaction	1.79	>0.3	0.02	>0.98
$\chi^2_{1:2:1:2:4:2:1:2:1}$ Total segregation	4.29	>0.8	4.16	>0.8
χ^2 heterogeneity 4 df	0.70	>0.95	2.33	>0.5

TABLE 27

Frequencies of F_2 genotypes determined from F_3 progeny tests in cross Spica/W3498.

(HL = homozygous low: Seg = segregating: HH = homozygous high)

		Adult-plant reaction to 34-Anz-1,2,3,4,5,6,7			
		HL	Seg	HH	Total
Sr7b Sr7b	HL	3	5	3	11
Sr7b sr7b	Seg	5	9	5	19
sr7b sr7b	HH	5	7	3	15
	Total	13	21	11	45
Sr17 Sr17	HL	3	5	3	11
Sr17 sr17	Seg	7	11	5	23
sr17 sr17	HH	3	5	3	11
	Total	13	21	11	45

Chi-square Analysis

	Association of Adult-plant reaction with:			
	Sr7b		*Sr17*	
	χ^2	P	χ^2	P
$\chi^2_{1:2:1}$ adult-plant reaction	0.38	>0.8	0.38	>0.8
$\chi^2_{1:2:1}$ seedling reaction	1.79	>0.3	0.02	>0.98
$\chi^2_{1:2:1:2:4:2:1:2:1}$ Total segregation	2.73	>0.95	0.60	>0.99
χ^2 heterogeneity 4 df	0.56	>0.95	0.20	>0.99

TABLE 28

Mean disease intensities produced on Spica, Seafoam and W3498 when tested as adult-plants with eight *P. graminis tritici* strains and scored by four observers.

Mean and standard deviation

Strain	Spica	Seafoam	W3498
21-Anz-5	4.4±0.2	4.9±0.1	5.0**
21-Anz-2,3,4,5,7*a	2.2±0.3	4.6±0.2	4.9±0.1
21-Anz-2,3,4,5,6,7*b	1.3±0.1	3.9±0.1	4.1±0.1
21-Anz-(1),2,3,4,5,6,7*c	3.0±0.3	4.8±0.1	4.9±0.1
34-Anz-2,4,5,6	2.3±0.3	4.1±0.2	4.8±0.1
34-Anz-1,2,3,4,5,6,7*e	1.0	3.6±0.2	5.0
126-Anz-6,7,11	0.4±0.1	0.5±0.1	4.3±0.2
222-Anz-1,2,3,4,5,6,7*d	1.3±0.1	4.4±0.2	5.0
Mean over strains	2.0	3.9	4.7

* a-e represents sequential culture selections from recurrent EMS treatments.

** standard for susceptibility on 0-5 scale.

TABLE 29

Mean disease intensities produced on nine selected homozygous lines from Spica/W3498 when classified by 4 independent observers.

(H = homozygous high reaction: L = homozygous low reaction)

F_3 line designation	Previous classification to 126-Anz-6,7,11	Mean and standard deviation			
		21-Anz-2,3,4,5,7	34-Anz-1,2,3,4,5,6,7	126-Anz-6,7,11	Mean over strains
6497	H	4.8±0.1	4.0	1.5	3.4
6502	H	3.8±0.1	4.4±0.2	1.5±0.2	3.2
6503	H	4.0±0.2	4.0±0.2	1.9±0.1	3.3
6510	H	4.1±0.1	3.9±0.1	1.6±0.1	3.2
6531	H	4.0±0.2	4.4±0.1	1.5±0.2	3.3
Mean		4.1	4.1	1.6	3.3
6505	L	2.6±0.4	3.0	0.5	2.0
6513	L	2.9±0.2	3.5±0.2	0.4±0.1	2.3
6515	L	2.4±0.2	2.3±0.1	0.4±0.1	1.7
6517	L	3.3±0.2	2.3±0.1	0.4±0.1	1.9
Mean		2.8	2.8	0.4	2.0
Mean over lines		3.5	3.5	1.1	
Spica) from Table 27		2.2±0.3	1.0	0.4±0.1	
W3498)		4.9±0.1	5.0	4.3±0.2	

all three strains. However, as can be seen from the mean values in Table 29, the amount of disease on the homozygous low lines with 21-Anz-2,3,4,5,7 and 34-Anz-1,2,3,4,5,6,7 exceeded the amount of disease produced by 126-Anz-6,7,11 on the homozygous high lines. With 126-Anz-6,7,11 none of the homozygous high lines approached the amount of disease produced by the W3498 parent (i.e. 4.3±0.2 in Table 28). On the other hand, the overall mean responses to 21-Anz-2,3,4,5,7, the wild progenitor and 34-Anz-1,2,3,4,5,6,7, the derived strain, by the homozygous low and homozygous high lines were identical indicating that the avirulence in 34-Anz-1,2,3,4,5,6,7 observed in Table 27 was a characteristic of 21-Anz-2,3,4,5,7 and also of the unrelated strain 34-Anz-2,4,5,6.

5.10.2 Seedling Studies

At relatively low temperatures (<23°C), Spica seedlings produced infection type "3-3" when inoculated with 126-Anz-6,7,11 and 34-Anz-1,2,3,4,5,6,7. Strain 21-Anz-5 produced infection type "3+". However, repeated attempts to classify segregation of parental differences in cross Spica/W3498 were not successful.

5.11 ASSOCIATION OF ADULT-PLANT STEM RUST AND LEAF RUST RESISTANCES IN HOPPS

5.11.1 Adult-plant Leaf Rust Resistance in Hopps

Standard cultures of *Puccinia recondita* Rob. ex Desm. strains, 68-Anz-1,2,3,4, 122-Anz-1,2,3 and 162-Anz-1,2,3,GT were virulent on Hopps seedlings, but adult-plants in the field were resistant (max. disease cover 0.5%) to a mixture of these strains, whereas W3498 was susceptible.

5.11.2 Inheritance of Adult-plant Reaction

5.11.2.1 F_1

F_1 plants from Hopps/W3498 tested in 1975 were resistant indicating dominance of resistance.

5.11.2.2 F_2

F_2 plants from Hopps/W3498 were classified in two phenotypic classes, resistant and susceptible. Of 190 plants, 133 were resistant and 57 were susceptible. This segregation conformed with a ratio of 3 resistant: 1 susceptible ($\chi^2_{3:1}$ = 2.53; P>0.1) indicating a single dominant gene conditioning low reaction.

5.11.2.3 F_3

F_3 families derived from 78 F_2 plants of known leaf rust reaction, 44 resistant and 34 susceptible, were tested in 1975. Three phenotypic classes, homozygous resistant, segregating and homozygous susceptible were distinguished. F_3 families derived from the resistant group were 11 homozygous resistant and 33 segregating ($\chi^2_{1:2}$ = 1.38; P>0.2). All 34 F_3 families derived from susceptible F_2 plants were homozygous susceptible.

Seventy-seven random F_3 families from the same cross were assessed for leaf and stem rust reactions in 1975. A joint analysis of leaf and stem rust reactions appears in Table 30. The non-significant heterogeneity χ^2 value provided no evidence to suggest linkage between the single loci involved in adult-plant reactions to stem rust and leaf rust.

TABLE 30

Joint classifications of leaf rust and stem rust reactions in Hopps/W3498.

(HR = homozygous resistant: Seg = segregating: HS = homozygous susceptible).

		Stem rust reaction			
		HR	Seg	HS	Total
Leaf rust reaction	HR	7	7	3	17
	Seg	6	24	11	41
	HS	2	10	7	19
Total		15	41	21	77

Chi-square Analysis	χ^2	P
$\chi^2_{1:2:1}$ leaf rust reactions	0.43	>0.8
$\chi^2_{1:2:1}$ stem rust reactions	1.22	>0.5
$\chi^2_{1:2:1:2:4:2:1:2:1}$ total segregation	7.78	>0.3
χ^2 4 df (heterogeneity) linkage	6.13	>0.15

5.12 INHERITANCE OF SEEDLING RESISTANCE TO LEAF RUST IN CS(HOPE 3B)

Warigo, Chinese Spring, Hope and H44-24 produced infection type "3+", whereas CS(Hope 3B) produced infection type ";" when inoculated with *P. recondita* strain 122-Anz-1,2,3. Of 58 F_3 seedling families from Warigo/CS(Hope 3B), 15 were homozygous for low reaction, 29 segregated and 14 were homozygous for high reaction, indicating segregation for allelic differences at a single locus ($\chi^2_{1:2:1} = 0.03$; $P>0.8$). Pooled frequencies of low and high reactions within the 29 segregating families were 473 and 154, respectively, further supporting the single locus hypothesis and indicating dominance of the allele for low reaction ($\chi^2_{3:1} = 0.06$; $P>0.95$). Individual family results are presented in Appendix 3. Twenty F_3 families, 6 homozygous low, 6 homozygous high and 8 segregating were tested as seedlings by Dr. P.L. Dyck, Agriculture Canada, using "race 15" (culture 64-156) which on Selkirk, Warigo and CS(Hope 3B) produced infection types "4", "33+" and ";1-", respectively. The six families homozygous low with 122-Anz-1,2,3 were homozygous low with race 15 (its ";1-", ";1" or ";1+"). The six families homozygous high with strain 122-Anz-1,2,3 segregated for a second factor (it "2,2+") with race 15. Of the families segregating with 122-Anz-1,2,3, 5 segregated infection types ";1" and "2+", whereas 3 segregated infection types ";1", "2+", "33+" to race 15. The Canadian results suggest that CS(Hope 3B) carries at least two genes determining low reaction to race 15, one of which appears identical to that operating against 122-Anz-1,2,3.

6. RESISTANCE OF CELEBRATION, MARQUILLO AND THATCHER TO STEM RUST

6.1 INHERITANCE OF THE ADULT-PLANT REACTIONS IN CELEBRATION, MARQUILLO AND THATCHER

Epidemic progress curves obtained during 1972 and 1973 demonstrated adult-plant resistance in Celebration and Marquillo (Figs. 15,17). Preliminary observations on field sown F_3 families from Celebration/W3498 in 1974 indicated the need to score adult-plant reactions at least three times during the epidemic. Differences in adult-plant reactions were more readily discerned at an optimal period for each population depending upon the maturity. The optimal period extended over the milk to late-dough stages of development. At this later stage, the reactions of Celebration and Marquillo were readily distinguished from W3498. Although Celebration matured 5 days earlier than Marquillo, the former had more disease than the latter at a corresponding maturity stage. Similar differences were apparent in 1975 when detailed genetic studies were undertaken.

6.1.1 Crosses of Resistant and Susceptible Parents

6.1.1.1 F_1

F_1 hybrids of Celebration/W3498 and Marquillo/W3498 were susceptible indicating that resistance was recessive (Plate 10b).

6.1.1.2 F_3

F_3 family classifications for the two crosses Celebration/W3498 and Marquillo/W3498 are summarised in Table 31. Three phenotypic classes, homozygous resistant, segregating and homozygous suceptible were distinguished.

TABLE 31

Segregation class frequencies of F_3 families in crosses of Celebration and Marquillo with W3498 when classified as adult-plants.

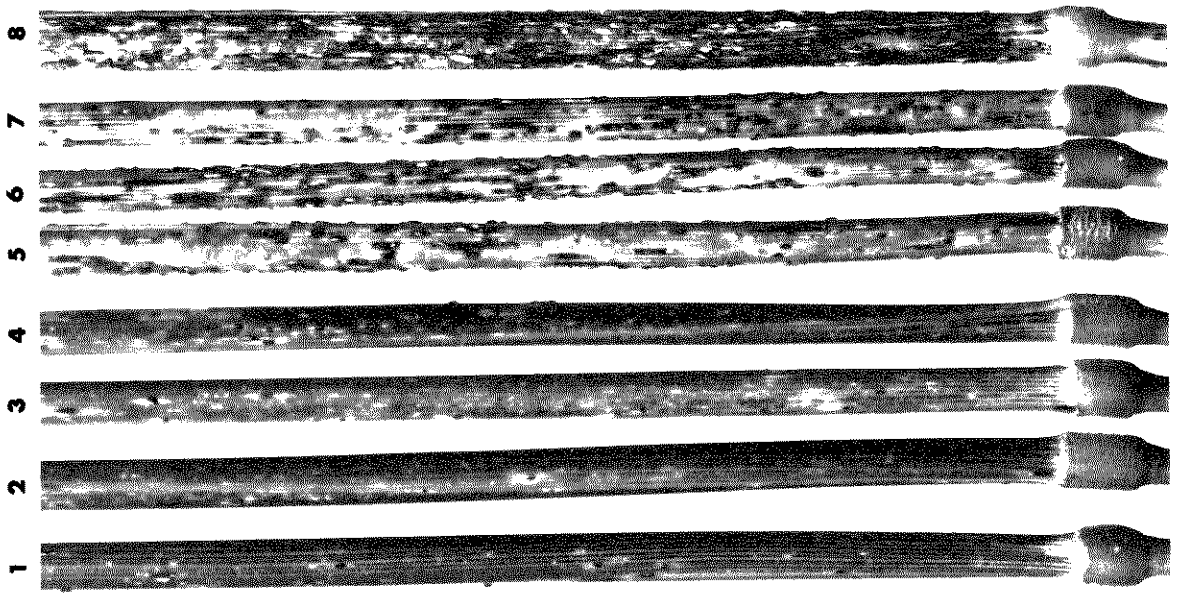
(HR = homozygous resistant; Seg = segregating; HS = homozygous susceptible).

Cross	Designation	HR	Seg	HS	$\chi^{2*}_{1:2:1}$
Celebration/ W3498	HX72.15.1	25	52	22	0.43
Marquillo/ W3498	HX72.96.1	25	54	21	0.96
Total segregation		50	106	43	1.33

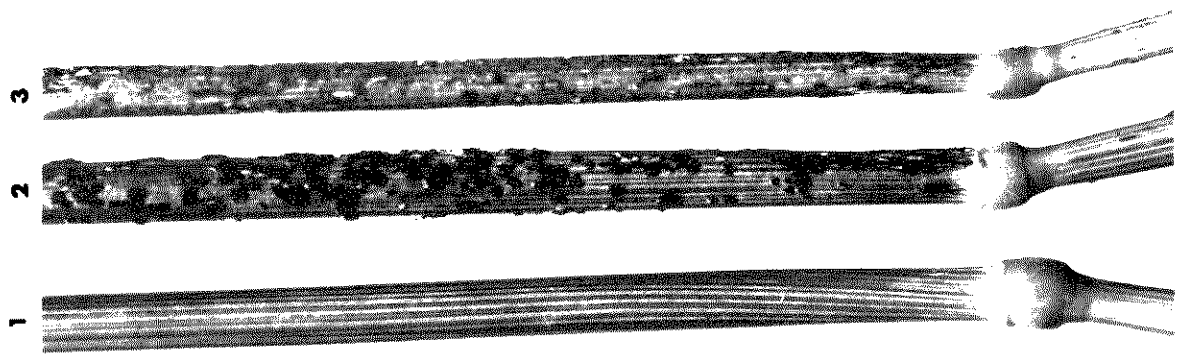
* Value for significance at P=0.05 is 5.99.

In each cross, the observed family distributions fitted a ratio of 1 homozygous resistant : 2 segregating : 1 homozygous susceptible, indicating that reaction in each case was determined by segregation of alleles at a single locus.

Nearer maturity, it became apparent that homozygous resistant families from Marquillo/W3498 could be subdivided when three further classes, "homozygous low", "segregating" and "homozygous high" were distinguished. Plants in the low reaction class carried 2 to 5% disease cover on the penultimate internode and were similar to Marquillo, whereas those in the high reaction class carried 15 to 20% disease cover (Plate 10). Using these criteria, the 25 homozygous resistant families, appearing in Table 31, were sub-classified, 6 homozygous low, 15 segregating and 4 homozygous high, suggesting independent segregation of alleles at a second locus ($\chi^2_{1:2:1}$ = 1.32;



(a)



(b)

PLATE 10 Adult-plant reactions.

a) F_3 lines from Marquillo/W3498.
1 Marquillo; 2-4 Resistant segregates - genotype *Sr12 Sr9g*; 5-7 Intermediate - *Sr12 sr9g*; 8 Susceptible - *sr12 sr9g*.

b) 1 Celebration; 2 Celebration/W3498 F_1; 3 W3498.

P 2df > 0.5). There was no apparent variation in disease intensity among homozygous susceptible families. This suggested that the second locus only enhanced the resistance and that it had no effect when alone.

Subdivision of homozygous resistant families in a similar manner in Celebration/W3498 was not possible. Some variation was present, the most resistant families displayed 15-20% disease cover while the least resistant showed 20-30%.

One-thousand grain weights for 13 homozygous resistant and 13 homozygous susceptible F_3 families in Celebration/W3498 are listed in Table 32. Mean 1,000 grain weights of 13.85g for homozygous susceptible families and 25.52g for homozygous resistant families and the highly significant F ratio for disease groups, clearly indicated the effect of rust resistance in these materials.

6.1.1.3 Thatcher chromosome substitution lines

A series of lines in which single chromosomes of Thatcher were individually substituted into Chinese Spring (produced by Dr.E.Sears, Uni. Missouri, U.S.A.), together with Thatcher and Chinese Spring were tested in 1975. Except for CS(Thatcher 2B) and CS(Thatcher 3B), all substitution lines and Chinese Spring were susceptible. Neither CS(Thatcher 2B) nor CS(Thatcher 3B) was as resistant as Thatcher. An analysis of variance (completely random design) of 1,000 grain weights (Table 33) showed that CS(Thatcher 2B) and CS(Thatcher 3B) were significantly heavier than Chinese Spring and the other substitution lines. The grain weights of CS(Thatcher 4D) and CS(Thatcher 6B) exceeded that of Chinese Spring at the P=0.05 level, but compared with the 2B and 3B lines these were small effects and

TABLE 32

Mean 1,000 grain weights and analysis of variance for homozygous resistant and homozygous susceptible F_3 families from Celebration/W3498

Line designation	Homozygous resistant (g)	Line designation	Homozygous susceptible (g)
6711	24.26	6703	11.63
6724	25.49	6704	15.29
6741	22.63	6721	18.29
6763	28.46	6726	10.07
6765	23.68	6734	7.33
6768	31.77	6744	20.63
6771	27.35	6757	12.77
6774	21.33	6775	9.45
6789	23.41	6781	12.17
6791	31.16	6782	12.16
6793	24.37	6787	16.54
6811	28.42	6792	15.84
6813	19.45	6805	17.86
Mean	25.52		13.85
$S_{\bar{x}}$	3.56		3.75

l.s.d. (P=0.05) = 4.57

Analysis of variance

Source of variation	df	MS	F
Between rust reactions (A)	1	17.4630	
Between lines within rust reactions (experimental error) (B)	24	0.2944	$\frac{A}{B}$ 59.31**
Between samples within lines (sampling error) (C)	26	0.0077	$\frac{B}{C}$ 37.83**
Total	51		

**P<0.01

may have been due to chance.

TABLE 33

Mean 1,000 grain weights and analysis of variance for Thatcher chromosome substitution lines and Chinese Spring.

Substitution Line	Mean 1,000 grain weight (g)	Line designation	Mean 1,000 grain weight (g)
CS(Thatcher1A)	8.85	5B	10.01
2A	10.36	6B	12.12*
3A	9.46	7B	9.58
4A	8.78		
5A	7.64	1D	10.48
6A	9.49	2D	8.88
7A	8.93	3D	9.98
		4D	11.67*
1B	11.33	5D	9.82
2B	19.38**	6D	10.84
3B	20.58**	7D	8.27
4B	7.50		
		CS	8.67

* 1.s.d. (P=0.05) for means = 2.95

** 1.s.d. (P=0.01) for means = 4.01

Analysis of variance

	df	MS	F
Between lines	21	509.49	23.99**
Within lines	22	21.24	
Total	43		

** P<0.01.

6.2 GENETIC ANALYSES OF SEEDLING REACTIONS OF CELEBRATION, MARQUILLO AND THATCHER

6.2.1 Parental Reactions

Many strains are avirulent on seedlings of Celebration, Marquillo and Thatcher. The infection types produced by seedlings of these cultivars, the Thatcher chromosome substitution series, Chinese Spring and various differential genotypes when inoculated with cultures of 126-Anz-6,7,11 34-Anz-2,4,5,7,11, 326-Anz-1,2,3,5,6, 34-Anz-1,2,3,4,5,6,7, 59-E-5,7 and culture 56-E-1 are presented in Table 34. Following infection the temperature was kept below 20°C for 5 days.

In instances where strains were virulent on plants with *Sr5*, Thatcher and Celebration reacted similarly. Marquillo produced lower infection types, possibly reflecting its greater resistance in the field. Of the Thatcher substitution lines, 6D and 4D reacted in the same way as seedlings possessing *Sr5*. This result was expected for substitution line 6D as the 6D chromosome carries it. Line 4D presumably carries *Sr5* as a result of chance transference during backcrossing. Both substitution lines 2B and 3B produced low infection types with 5 of the 6 cultures, and 3B was only slightly lower than Chinese Spring with strain 34-Anz-1,2,3,4,5,6,7 while 2B gave a high reaction. However, neither line produced infection types as low as Thatcher. Loegering and Sears (1973) showed that both Thatcher 2B and 3B possessed *Sr16*. However, I*Sr16*-Ra which behaved as expected for their test culture, 59-E-5,7, and similarly with 56-E-1, produced infection types higher than the respective substitution lines, indicating that both lines possessed additional gene(s). The infection types

TABLE 34

Infection types produced on seedlings of Celebration, Marquillo, Thatcher, Thatcher chromosome substitution lines and certain differentials when inoculated with strains of *P. graminis tritici*.

Line	*P. graminis tritici* culture and strains					
Behaviour for *Sr5*	56-E-1 avirulent	126-6,7,11 virulent	34-2,4,5,7,11 virulent	326-1,2,3,5,6 avirulent	34-1,2,3,4,5,6,7 virulent	59-E-5,7 avirulent
Celebration	0;=	;x-	;x-	;	x	0;
Marquillo	0;=	0;	0;	0;	;x-	0;-
Thatcher	0;=*	;x-	;x-	0*	x	0*
CS(Thatcher 2B)	;12=	3-	2+3-	2-	3+	12=
CS(Thatcher 3B)	0;=	x2-	x-	x3	x+3+	x2=
ISr16-Ra	2=	3-3	3-3	3-3	33+	2=
Acme	0;	;x+	2+3-	2	3+	
Kubanka	3+	;	33+	2-	33+	
CS	3+	3+	3+	3+	3+	3+
W3498	3+	4	4	4	4	4
CS(Thatcher 1A)		3+	3+	3+		
2A		3+	3+	3+		
3A		3+	3+	3+		
4A		3+	3+	3+		
5A		3+	3+	2+3-		
6A		3+	3+	3+		
7A		3+	3+	3+		
1B		3+	3+	3+		
4B		3+	3+	3+		
5B		3+	3+	3+		
6B		3+	3+	3+		
7B		3+	3+	3+		

TABLE 34 (CONT)

	56-E-1 avirulent	126-6,7,11 virulent	34-2,4,5,7,11 virulent	326-1,2,3,5,6 avirulent	34-1,2,3,4,5,6,7 virulent	59-E-5,7 avirulent
CS(Thatcher 1D)		3+	3+	3+		
2D		3+	3+	3+		
3D		3+	3+	3+		
4D		3+	3+	0;*		
5D		3+	3+	3+		
6D		3+	3+	;x=*		
7D		3+	3+	3+		

* *Sr5*

further suggested that additional genes must be different. In the case of CS(Thatcher 2B), McIntosh (personal communication) has shown that an additional gene in chromosome 2BL is allelic with *Sr9* (hereafter designated *Sr9g* (McIntosh, 1976 - personal communication)). Since the infection type on CS(Thatcher 5A) is similar to that produced on CS(Thatcher 2B), it is assumed that CS(Thatcher 5A) carries *Sr9g* through a chance segregation. Detailed evidence showing the occurrence of a second gene (*Sr12*) located in chromosome 3B of CS(Thatcher 3B) is presented in section 7.1.1 of this thesis.

The infection types produced on various cultivars were dependent on post-incubation temperatures (Table 35). Behaviour attributable to *Sr12* was dependent upon temperature, as well as the particular strain, as indicated by infection types on CS(Thatcher 3B) (*Sr12*, *Sr16*). By contrast, the expressions of *Sr9g* and *Sr16* were relatively stable as indicated by the infection types produced by CS(Thatcher 2B) (*Sr9g*, *Sr16*) and I*Sr16*-Ra.

By choosing cultures of appropriate strains, e.g. 326-Anz-1,2,3,5,6 or related types to best identify *Sr9g*, 126-Anz-6,7,11 or 34-Anz-2,4,5,7,11 to identify *Sr12* and 59-E-5,7 or 56-E-I to identify *Sr16*, and by manipulating the temperature, it was possible to study the seedling reactions of hybrid lines derived from Celebration and Marquillo with respect to each of these genes, and to correlate the genotypes determined using specific cultures and environments with field reactions to a mixture of strains.

TABLE 35

Effect of temperature on infection types produced by various cultivars to strains of *P. graminis tritici*.

Cultivar	Temperature	Infection types			
		126-Anz-6,7,11	34-Anz-2,4,5,7,11	326-Anz-1,2,3,5,6	59-E-5,7
Marquillo	low*	0;=	0;=	0;	0;=
	high**	;1-	;1-	;	;
Celebration	low	;x-	;x-	;x-	0;
	high	x-	xx-	x12=	x-
Thatcher	low	;x-	;x-	0***	0***
	high	x-	xx-	0***	0***
CS(Thatcher 3B)	low	x2-	x-	x3	x2=
	high	x3	x33+	x33+	x2+
CS(Thatcher 2B)	low	2+3-	2+3-	2-	12=
	high	3	3-3	22+	22+
I*Sr16*-Ra	low	3-3c	3-3c	3-3c	2=
	high	3c	33+	33+	2+
CS	low	3+	3+	3+	3+
	high	4	4	4	4

* <20°C for 5 days following inoculation.

** >20°C for 5 days following inoculation.

*** *Sr5*.

6.2.2 Reactions of F_3 Lines in Celebration Crosses

6.2.2.1 Strains 126-Anz-6,7,11 and 34-Anz-2,4,5,7,11

F_3 families from Celebration/W3498 produced similar reaction groupings when tested with 126-Anz-6,7,11 and 34-Anz-2,4,5,7,11. Two infection type groups were distinguished with each strain, ";x" and "33+,3+" with 126-Anz-6,7,11, and "x+3" and "33+,3+" with 34-Anz-2,4,5,7,11. Three phenotypic classes, homozygous low reaction, segregating and homozygous high reaction were obtained with each strain. Of 83 families classified with both strains, 20 were homozygous low, 47 segregated and 16 were homozygous high in reaction. This conforms with a predicted ratio of 1:2:1 ($\chi^2_{1:2:1} = 1.84$; $P>0.3$) suggesting segregation of alleles at a single locus. In order to investigate dominance or recessiveness of the allele for low reaction, segregation ratios (Appendix 4) for 27 F_3 lines were pooled (Table 36). These results, for each strain, show that the allele for low reaction presumed to be *Sr12* was recessive.

6.2.2.2 326-Anz-1,2,3,5,6

When F_3 families in Celebration/W3498 were tested with strain 326-Anz-1,2,3,5,6, four infection type groups ";x2-,;x2": "2-2,2": "x3,x+3" and "33+,3+" were distinguished. Fifty-two F_3 families were classified into 9 phenotypic classes, as indicated in the matrix (Table 37) assembled on the assumption that two genes were involved, one concerned in the production of

TABLE 36

Pooled frequencies of F_3 plants with low and high reactions in Celebration/W3498 when tested with 126-Anz-6,7,11 and 34-Anz-2,4,5,7,11.

Strain	No. of families pooled	Low reaction	High reaction	$\chi^2_{1:3}$	P
126-Anz-6,7,11	18	110	327	0.01	>0.9
34-Anz-2,4,5,7,11	9	50	156	0.92	>0.3
Total segregation for both strains	27	160	483	0.01	>0.9
Total χ^2 for both strains 2 df				0.92	>0.3
Heterogeneity χ^2 1 df				0.92	>0.3

TABLE 37

Matrix of segregation class frequencies for F_3 families in Celebration/W3498 when tested with 326-Ana-1,2,3,5,6.

(it = infection type)

Celebration it ";x2-": W3498 it "3+"

			Total
it ";x2-,;x2" 5	it";x2-,;x2,2-2,2" 8	it"2-2,2" 6	19
it";x2-,;x2,x3,x+3" 3	it";x2-,;x2,2-2,2,x+3,x3,33+,3+" 13	it"2-2,2,33+,3+" 6	22
it"x3,x+3" 3	it'x+3,x3,33+,3+" 6	it"33+,3+" 2	11
Total 11	27	14	52

	χ^2	P
$\chi^2_{1:2:1}$ Columns	0.43	>0.8
$\chi^2_{1:2:1}$ Rows	3.70	>0.1
Total segregation $\chi^2_{1:2:1:2:4:2:1:2:1}$	10.23	>0.2
Joint segregation χ^2 6 df	6.1	>0.3

TABLE 38

Pooled frequencies of F_3 seedlings in infection type classes in Celebration/W3498 when tested with 326-Anz-1,2,3,5,6.

	No. of families pooled	F_2 genotype	";x2-,;x2"	"2-2,2"	"x3,x+3"	"33+,3+"	$\chi^2_{3:9:1:3}$	P
	6	*Sr12sr12 Sr9gsr9g*	23	82	10	25	0.78	>0.8
Total χ^2 18 df							10.49	>0.9
Heterogeneity χ^2 17 df							9.71	>0.9
							$\chi^2_{3:1}$	
	6	*Sr12Sr12 Sr9gsr9g*	92		31		0.002	>0.95
Total χ^2 6 df							7.25	>0.2
Heterogeneity χ^2 5 df							7.25	>0.2
							$\chi^2_{3:1}$	
	3	*sr12sr12 Sr9gsr9g*		41		15	0.09	>0.7
Total χ^2 3 df							6.75	>0.05
Heterogeneity χ^2 2 df							6.66	<0.02

infection type "X" (columns) and the other with infection type "2" (rows).

A chi-square analysis of the data showed that it conformed with an hypothesis of two independently segregating genes. Both genes were necessary for the expression of low reactions approaching those of the Celebration parent. No line gave reactions identical with Celebration, suggesting that further genetic modification was involved. When results of Table 37 were compared with the data already available for strains 126-Anz-6,7,11 and 34-Anz-2,4,5,7,11, the column groupings corresponded with the homozygous resistant, segregating and homozygous susceptible classifications previously obtained, implying involvement of *Sr12*. The allele involved in row classifications was assumed to be *Sr9g*. In order to determine dominance of *Sr9g*, segregation ratios within different groups of selected F_3 lines (Appendix 5) were pooled (Table 38). These results showed that *Sr9g* was dominant irrespective of the genotype at the *Sr12* locus and that *Sr12* was recessive in the presence of *Sr9g*.

6.2.2.3 Culture 56-E-1

In F_3 families of Celebration/W3498 inoculated with culture 56-E-1 at low temperature (<20°C) four infection type groups, ";x-,;x", "2-,2", "x3" and "3+" were recognised. On the basis of these groups, 50 F_3 families were arranged in a matrix presented in Table 39. There appeared to be two genes segregating, an allele for low reaction which gave infection type "x3" (Rows), the other producing infection type "2-" or "2" (Columns).

TABLE 39

Segregation class frequencies of F_3 families from Celebration/W3498 when tested with culture 56-E-1.

(it = infection type)

Celebration it ";x=": W3498 it "3+"

			Row Totals
it ";x-,;x" 5	it ";x-,;x,x3" 7	it "x3" 2	14
it ";x-,;x,2-,2" 5	it ";x-,;x,x3,2-2,3+" 13	it "x3,3+" 5	23
it "2-,2" 5	it "2-,2,3+" 4	it "3+" 4	13
Column Totals 15	24	11	50

	χ^2	P
$\chi^2_{1:2:1}$ columns	0.73	>0.5
$\chi^2_{1:2:1}$ rows	0.68	>0.7
Total segregation $\chi^2_{1:2:1:2:4:2:1:2:1}$	3.92	>0.8
Joint segregation χ^2 6 df	2.51	>0.8

The chi-square analysis also indicated that Celebration possessed two independent alleles for low reaction to 56-E-1. Alone the respective alleles determined infection types "2-" or "2" and "x3" but when combined a complementary interaction (Category IV interaction of Loegering and Powers

1962) was exhibited. Of the 50 F_3 families tested with 56-E-1, the mesothetic reactions (row classifications) were identical with classifications for *Sr12* vs *sr12* when the lines were tested with 34-Anz-2,4,5,7,11, however, 56-E-1 detected an additional factor, although 56-E-1 is virulent on seedlings with *Sr9g* (see Kubanka, Table 34). This additional factor gave an infection type similar to that produced by I*Sr16*-Ra. Thus the factor is possibly *Sr16*. A test of linkage between the *Sr9g* and *Sr16* was made, as Sears and Loegering (1968) showed that these loci were carried in the long arm of chromosome 2B. Table 40 summarises the joint classification frequencies, presumably for *Sr9g* and *Sr16*, among 32 F_3 families in Celebration/W3498 when tested with strain 326-Anz-1,2,3,5,6 and culture 56-E-1. The chi-square analysis suggested that the two loci were inherited independently which supports the findings of Loegering and Sears (1968). To investigate the dominance of *Sr16*, segregation frequencies within groups of F_3 lines (for individual line ratios see Appendix 6) were pooled (Table 41). The results showed that *Sr16* was dominant irrespective of the genotype at the *Sr12* locus and that the *Sr12* allele was recessive in the presence and absence of *Sr16*.

One F_3 family from Celebration/W3498 gave an infection type "33+" when tested with 34-Anz-2,4,5,7,11, 126-Anz-6,7,11 and 326-Anz-1,2,3,5,6 and infection types "2" and "2-" to 59-E-5,7 and 56-E-1, respectively, which supported the proposal that Celebration probably carries *Sr16*. *Sr12* and *Sr9g* were not present in this line.

TABLE 40

Joint segregation frequencies in infection type groupings from Celebration/W3498 when tested with strain 326-Anz-1,2,3,5,6 and culture 56-E-1.

326-Anz-1,2,3,5,6:- Celebration it ;x2-: W3498 it 3+

56-E-1 :- " it ;x- : " it 3+

Culture 56-E-1

Strain 326-Anz-1,2,3,5,6	it";x-,;x" ";x-,;x,2-,2" "2-,2"	it";x-,;x,x3" ";x-,;x,x3,2-2,3+" "2-,2,3+"	it"x3" "x3,3+" "3+"	Row Totals
it";x2-,;x2" ";x2-,;x2,2-2,2" "2-2,2"	2	2	1	5
it";x2-,;x2,x3,x+3" ";x2-,;x2,2-2,2,x+3, x3,33+,3+" "2-2,2,33+,3+"	7	9	2	18
it"x3,x+3" "x3,x+3,33+,3+" "33+,3+"	3	4	2	9
Column Totals	12	15	5	32

	χ^2	P
$\chi^2_{1:2:1}$ columns	3.19	>0.2
$\chi^2_{1:2:1}$ rows	1.50	>0.4
Total segregation $\chi^2_{1:2:1:2:4:2:1:2:1}$	5.38	>0.7
Heterogeneity χ^2 6 df	0.69	>0.99

TABLE 41

Pooled frequencies of F_3 seedlings in infection type classes in Celebration/W3498 when tested with culture 56-E-1.

	No. of families pooled	F_2 genotype	";x-,;x"	"2-,2"	"x3"	"3+"	$\chi^2_{3:9:1:3}$	P
	13	*Sr12sr12 Sr16sr16*	76	204	32	62	4.81	>0.1
Total χ^2 39 df							27.94	>0.9
Heterogeneity χ^2 38 df							23.13	>0.95
							$\chi^2_{1:3}$	
	5	*Sr12sr12 Sr16Sr16*	28	88			0.01	>0.9
Total χ^2 5 df							3.44	>0.5
Heterogeneity χ^2 4 df							3.43	>0.4
							$\chi_{3:1}$	
	7	*Sr12Sr12 Sr16sr16*	112		35		0.11	>0.9
Total χ^2 7 df							1.35	>0.98
Heterogeneity χ^2 6 df							1.24	>0.98

TABLE 41 (CONT)

	No. of families pooled	F_2 genotype	";x-,;x"	"2-,2"	"x3"	"3+"	$\chi^2_{3:9:1:3}$	P
							$\chi^2_{3:1}$	
	4	*sr12sr12 Sr16sr16*		82		23	0.14	>0.7
Total χ^2 4 df							1.21	>0.8
Heterogeneity χ^2 3 df							1.07	>0.7
							$\chi^2_{1:3}$	
	5	*Sr12sr12 sr16sr16*			28	83	0.01	>0.9
Total χ^2 5 df							0.4	>0.9
Heterogeneity χ^2 4 df							0.39	>0.98

6.2.3 Reactions of F_3 Lines of Marquillo Crosses

6.2.3.1 Strains 126-Anz-6,7,11 and 34-Anz-2,4,5,7,11

When 92 F_3 families from Marquillo/W3498 were tested with 34-Anz-2,4,5,7,11 at low temperature (<20°C), two infection type groups, ";x=,;x-;x" and "33+,3+" were distinguished. Three phenotypic classes in the frequencies 30 homozygous low reaction, 45 segregating and 17 homozygous high reaction were obtained. This distribution was attributed to segregation at a single locus ($\chi^2_{1:2:1} = 3.72$; $P>0.1$). The results for 40 families tested with 126-Anz-6,7,11 at similar temperatures were identical with those obtained with 34-Anz-2,4,5,7,11. However, segregates with the lowest infection types, ";0", were more incompatible with 126-Anz-6,7,11 than with 34-Anz-2,4,5,7,11 (it ;x=). Assuming the 40 family samples chosen on the basis of sufficient seed remaining after other tests, was random,the observed segregation classes, 13 homozygous low reaction, 23 segregating and 5 homozygous high reaction fitted a single gene segregation ratio of 1:2:1 ($\chi^2_{1:2:1} = 3.73$; $P>0.1$).

Between 15 and 40 seedlings in each of 34 F_3 families had been tested with 34-Anz-2,4,5,7,11 and in each of 17 F_3 families with 126-Anz-6,7,11. The pooled individual classifications (Appendix 7) appearing in Table 42 indicate that the allele for low reaction was recessive.

6.2.3.2 Strain 326-Anz-1,2,3,5,6

In F_3 families of Marquillo/W3498 inoculated with 326-Anz-1,2,3,5,6 at low temperature (<20°C), four infection type groups, "0;,;,;x=,;x-": "2-2,2,22+": "x,x+x,x+3" and "33+,3+" were recognised. Assuming the likelihood of

TABLE 42

Pooled frequencies of seedlings with low and high reactions in segregating F_3 families from Marquillo/W3498 when tested with 34-Anz-2,4,5,7,11 and 126-Anz-6,7,11.

Strain	No. of Pooled F_3 lines	Low reaction*	High reaction**	$\chi^2_{1:3}$	P
34-Anz-2,4,5,7,11	34	181	479	2.07	>0.1
126-Anz-6,7,11	17	120	337	0.39	>0.5
Total segregation for both strains	51	301	816	2.25	>0.1
Total χ^2 2 df				2.46	>0.1
Heterogeneity χ^2 1 df				0.21	>0.6

* infection types ";x=,;x-,;x" with 34-Anz-2,4,5,7,11 and ";0,;x=" with 126-Anz-6,7,11.

** infection types "33+, 3+" with both strains.

segregation for two genes, the 92 F_3 families were arranged into the matrix on Table 43.

TABLE 43

Segregation class frequencies of F_3 families from Marquillo/W3498 when tested with 326-Anz-1,2,3,5,6.

(it = infection type)

Marquillo it "0;": W3498 it "3+"

			Row Totals
it "0;,;,;x=,;x-"	it"0;,;,;x=,;x-,2-2,2,22+"	it "2-2,2,22+"	
8	12	4	24
it "0;,;,;x=,;x-,x,x+3,x+x"	it"0;,;,;x=,;x-,2-2,2,22+,x,x+3,x+x,33+,3+"	it "2-2,2,22+,33+,3+"	
16	27	7	50
it "x,x+x,x+3"	it"x,x+x,x+3,33+,3+"	it "33+,3+"	
6	6	6	18
Column Totals 30	45	17	92

	χ^2	P
$\chi^2_{1:2:1}$ columns	3.72	>0.1
$\chi^2_{1:2:1}$ rows	1.48	>0.3
Total segregation $\chi^2_{1:2:1:2:4:2:1:2:1}$	8.30	>0.3
Joint segregation χ^2 6 df	3.10	>0.7

The data suggested that Marquillo possessed two independent alleles for low reaction to strain 326-Anz-1,2,3,5,6. Alone, the respective alleles determined infection types "2-2,2,22+" and "x+x,x+3,x" but when combined a complementary interaction was exhibited. Some variation within infection type

groups was impossible to resolve.

Mesothetic reactions in families tested with 326-Anz-1,2,3,5,6 also occurred when the same families were tested with 34-Anz-2,4,5,7,11, indicating that *Sr12* was involved. The second factor for low reaction appeared to be *Sr9g*.

Between 15 and 35 seedlings from each of 7 F_3 families segregating "2-2,2,22+" and "33+,3+" were individually classified after inoculation with 326-Anz-1,2,3,5,6 (Table 44).

TABLE 44

Individual plant frequencies in F_3 families in Marquillo/W3498 segregating for infection types "2-2,2,22+" and "33+,3+" when tested with 326-Anz-1,2,3,5,6.

Line designation	Low reaction (it"2-2,2,22+")	High reaction (it"33+,3+")	$\chi^2_{3:1}$	P
11385	24	12	1.33	>0.2
11401	27	15	2.57	>0.1
11425	15	5	-	
11486	13	5	0.08	>0.7
11509	19	6	0.02	>0.8
11512	16	4	0.26	>0.6
11519	13	6	0.44	>0.5
Total segregation for all families	127	53	1.89	>0.1
Total χ^2 7 df			4.70	>0.5
Heterogeneity χ^2 6 df			2.81	>0.8

6.2.3.3 Culture 56-E-1

When 53 F_3 families of Marquillo/W3498 were tested with culture 56-E-1 at low temperature (<20°C) two infection type groups, low "0;=,0;-,0;" and high "33+,3+" were recognised. Twenty families were homozygous low, 23 segregated and 10 were

homozygous high conforming only poorly to an hypothesised ratio of 1:2:1 ($\chi^2_{1:2:1} = 4.70$; $P>0.05$). Between 16 and 30 seedlings in each of 21 segregating F_3 families were tested with 56-E-1 and individually classified (Table 45). Of 411 plants classified, 309 were low in reaction and 102 gave high reactions; the frequencies conforming very closely to a ratio of 3:1 ($\chi^2_{3:1} = 0.01$; $P>0.9$) and indicating segregation at a single locus with low reaction dominant.

Family classifications with 56-E-1 were identical with those obtained using 34-Anz-2,4,5,7,11. However, whereas low reaction was dominant using 56-E-1 it was recessive using 34-Anz-2,4,5,7,11 and 126-Anz-6,7,11. In the Celebration cross, *Sr12* was recessive using culture 56-E-1. Hence, Marquillo may possess a second factor completely linked in coupling with *Sr12*, but which interacts only with culture 56-E-1. Because of its unspecified genes for avirulence 59-E-5,7 was not considered suitable for use with these materials. Assuming that the *Sr12*-like behaviour in Marquillo to culture 56-E-1 was determined by two genes linked in coupling, the maximum recombination frequency (r) at P=0.05 was calculated from the expression given by Hanson (1959):

$$(2r - \tfrac{3}{2}r^2) = 1 - (0.05)^{53}$$

$$r = 6.5\%$$

None of the F_3 families gave plants which produced an infection type "2-" similar to that produced on I*Sr16*-Ra when inoculated with 56-E-1. This result showed that *Sr16* was not present in Marquillo and, furthermore, confirmed that 56-E-1 is virulent on plants having *Sr9g* which was detected using strain

TABLE 45

Classification of F_3 plants from segregating families in Marquillo/W3498 when tested with 56-E-1.

Line designation	Low reaction (it"0;=0;-,0;")	High reaction (it"33+,3+")	$\chi^2_{3:1}$	P value
Marquillo	0;=			
W3498		3+		
11480	14	2	1.33	>0.2
11483	16	4	0.27	>0.6
11484	12	6	0.67	>0.4
11487	11	10	4.91	>0.02
11488	15	4	0.16	>0.6
11490	19*	1	4.27	>0.02
11495	16	7	0.36	>0.5
11498	12	7	1.40	>0.2
11500	11	5	0.34	>0.5
11504	15	7	1.77	>0.1
11509	18	4	1.86	>0.1
11510	10	6	1.33	>0.2
11514	19	5	1.73	>0.1
11516	19*	2	2.90	>0.05
11517	14	3	0.49	>0.4
11518	16	2	1.77	>0.1
11521	14	5	0.03	>0.8
11522	17	4	0.37	>0.5
11525	11	5	0.34	>0.5
11530	17	4	0.54	>0.4
11532	13	4	0.03	>0.8
Total segregation for all families	309	102	0.01	>0.9
Total χ^2 21 df			26.87	>0.1
Heterogeneity χ^2 20 df			26.86	>0.1

* more recent tests confirmed segregation.

326-Anz-1,2,3,5,6.

6.3 CORRELATION OF SEEDLING AND ADULT-PLANT REACTIONS

6.3.1 Strains 34-Anz-2,4,5,7,11 and 126-Anz-6,7,11

6.3.1.1 Celebration/W3498

The classification of 67 F_3 families as seedlings using strains 34-Anz-2,4,5,7,11 and 126-Anz-6,7,11 corresponded with adult-plant reactions (Table 46), determined in the field when

strains 21-Anz-1,2,5, 194-Anz-1,2,3,5,6 and 222-Anz-1,2,3,5,6 were present.

TABLE 46

Comparison of F_3 family adult-plant field classifications in Celebration/W3498 with seedling classifications using 34-Anz-2,4,5,7,11 or 126-Anz-6,7,11.

Adult-plant reaction

Reaction to		Homozygous resistant	Segregating	Homozygous susceptible
34-Anz-2,4,5,7,11	HR	20	0	0
or				
126-Anz-6,7,11	Seg	0	34	0
	HS	0	0	13

6.3.1.2 Marquillo/W3498

Adult-plant reactions of 42 F_3 families corresponded with seedling reactions obtained using 34-Anz-2,4,5,7,11 and 126-Anz-6,7,11 (Table 47).

TABLE 47

Comparison of F_3 family adult-plant field classifications in Marquillo/W3498 with seedling classifications using 34-Anz-2,4,5,7,11 and 126-Anz-6,7,11.

Adult plant reaction

Reaction to		Homozygous resistant	Segregating	Homozygous susceptible
34-Anz-2,4,5,7,11	HR	13	0	0
and				
126-Anz-6,7,11	Seg	0	24	0
	HS	0	0	5

The complete correlation between the reactions of F_3 families from both Celebration/W3498 and Marquillo/W3498 indicated that adult-plant resistance was determined by *Sr12*.

6.3.2 Strain 326-Anz-1,2,3,5,6

As indicated in section 6.1.1.2, the homozygous resistant F_3 families in Marquillo/W3498 were classified into three classes, homozygous low, segregating and homozygous high reaction. Of 17 F_3 families classified on this basis, all were homozygous low reaction with respect to the factor producing an infection type "x3" (*Sr12*), when inoculated with 326-Anz-1,2,3,5,6. This indicated that the modifying gene was *Sr9g*. Furthermore, the seedling reactions of 8 selected single head progenies taken from each of the 17 families corresponded with adult-plant classifications as above (Table 48).

Ninety-one F_3 plants of known seedling reaction to 326-Anz-1,2,3,5,6 were grown in the field during 1975. Forty-one plants which had produced infection type ";x-" were resistant, while 12 plants with an infection type "x3" were rusted at maturity but had developed rust at a relatively late stage. Of the field susceptible plants, 11 had given infection type "2-2" and 27 infection type "3+". This indicated that *Sr9g* gave no protection in 1975 when present alone.

The adult-plant classifications were substantiated by 1,000 grain weights determined on 8 plants from each of the seedling infection classes (Table 49). Clearly, those seedlings which gave infection type ";x-" with 326-Anz-1,2,3,5,6 in the seedling stage were much less affected by rust than either the "x3" and "2-2" groups. It is suggested that the ";x-" class involved genotypes combining both *Sr9g* and *Sr12*, while "x3" represented *Sr12* alone.

TABLE 48

Segregation class frequencies of single head progenies when tested with 326-Anz-1,2,3,5,6 in Marquillo/W3498 selected from plants in F_3 families of known adult-plant reaction.

(L = "homozygous low", Seg = "segregating", H = "homozygous high")

Line designation	F_2 genotype	Adult-plant reaction of F_3 line	No. of single head progenies in infection type classes		
			it";x-,;x"	it";x-,;x,x3"	it"x3"
1612	*Sr9g sr9g Sr12 Sr12*	Seg	2	3	3
1622	*Sr9g Sr9g Sr12 Sr12*	L	8	0	0
1636	*sr9g sr9g Sr12 Sr12*	H	0	0	6
1637	*Sr9g sr9g Sr12 Sr12*	Seg	5	0	3
1638	*Sr9g Sr9g Sr12 Sr12*	L	8	0	0
1640	*Sr9g Sr9g Sr12 Sr12*	L	8	0	0
1641	*Sr9g sr9g Sr12 Sr12*	Seg	2	2	0
1647	*Sr9g sr9g Sr12 Sr12*	Seg	3	1	2
1648	*sr9g sr9g Sr12 Sr12*	H	0	0	8
1656	*Sr9g sr9g Sr12 Sr12*	Seg	4	4	0
1665	*Sr9g sr9g Sr12 Sr12*	Seg	0	3	1
1674	*Sr9g sr9g Sr12 Sr12*	Seg	6	1	1
1679	*Sr9g Sr9g Sr12 Sr12*	L	8	0	0
1682	*Sr9g sr9g Sr12 Sr12*	Seg	3	3	2
1686	*Sr9g Sr9g Sr12 Sr12*	L	6	0	0
1687	*Sr9g sr9g Sr12 Sr12*	Seg	3	3	0
1704	*Sr9g sr9g Sr12 Sr12*	Seg	5	1	2
Marquillo	*Sr9g Sr9g Sr12 Sr12*	L	";x-"		
W3498	*sr9g sr9g sr12 sr12*	H	"3"		

TABLE 49

Mean 1,000 grain weights from F_3 plants in Marquillo/W3498 of known infection type to 326-Anz-1,2,3,5,6 and grown in a field rust nursery.

F_3 plant designation	Seedling Infection Type	Mean 1,000 grain weight (g)	Field Reaction
13176	";x-"	33.59	Resistant
13183	"	33.49	"
13184	"	39.20	"
13198	"	26.65	"
13199	"	30.31	"
13235	"	27.24	"
13239	"	27.72	"
13240	"	32.41	"
Mean	"	31.33	"
$S\bar{x}$		**4.24**	
13202	"x3"	20.34	late rusting
13203	"	17.58	" "
13204	"	24.04	" "
13205	"	15.04	" "
13245	"	21.88	" "
13246	"	15.75	" "
13247	"	21.20	" "
Mean	"	19.34	" "
$S\bar{x}$		**3.35**	
13256	"2-"	16.57	susceptible
13257	"	21.04	"
13258	"	20.62	"
13259	"	13.17	"
13268	"	10.31	"
13270	"	9.72	"
13271	"	11.08	"
13273	"	10.47	"
Mean	"	14.12	"
$S\bar{x}$		**4.68**	
13229	"3+"	11.60	susceptible
13232	"	13.43	"
13252	"	15.30	"
13253	"	16.68	"
13254	"	17.24	"
13260	"	12.69	"
13263	"	17.71	"
13267	"	13.27	"
Mean	"	14.74	"
$S\bar{x}$		**2.30**	

l.s.d. for means (P=0.05) = **2.59**

TABLE 49 (CONT)

Analysis of variance

Source of variation	d.f.	MS	F
Among plants	31	1.23	
Between Treatments (A)	3	10.18	$\frac{A}{B}$ 37.70**
Among plants within (B) treatments (expt. error)	28	0.27	
Among samples within (C) plants (sampling error)	32	0.01	$\frac{B}{C}$ 23.71**
Total	63		

** $P<0.01$.

6.4 ADULT-PLANT REACTION OF ADDITIONAL MARQUILLO DERIVATIVES

Pato, Pato Argentina (these lines probably similar), Tobari 66 and Tezanos Pinto Precoz were resistant at the adult-plant stage in 1975 (Plate 6). Pseudo-black chaff did not develop on these cultivars. When compared to Celebration, Marquillo and Thatcher, the adult-plant reaction of these cultivars was similar suggesting that they are 'Marquillo' derivatives. Recently, McIntosh (1976, personal communication) has found that Tezanos Pinto Precoz carries *Sr13*.

7. CYTOGENETIC STUDIES

7.1 CHROMOSOME ARM LOCATION OF *Sr12*

7.1.1 Cytogenetic Confirmation of the Location of *Sr12* in Chromosome 3B

Seedling samples from F_3 lines in the cross CS ditelosomic 3B/CS(Thatcher 3B) were tested with strains 59-E-5,7 and 126-Anz-6,7,11 and scored somatically for chromosome constitution. With 126-Anz-6,7,11 only *Sr12* was segregating,but with strain 59-E-5,7 (at relatively low temperatures) segregation for two genes was apparent. One of these was presumed to be *Sr16*, whose presence in CS(Thatcher 3B) had been demonstrated by Loegering and Sears (1973) whereas the second, producing an infection type "x12=", apparently was the same as that operating against 126-Anz-6,7,11. The frequencies of reaction classes for various F_2 chromosome constitutions are summarised in Table 50.

Classification of *Sr12* versus *sr12* (or its deficiency) using 126-Anz-6,7,11 indicated that genotype was completely correlated with chromosome constitution, as all disomic F_2 plants were homozygous low, monotelodisomic plants segregated and ditelosomic F_2 plants produced high homozygous reactions. These results indicated that *Sr12* is located on the short arm of chromosome 3B, or possibly in the long arm, but so near to the centromere that no recombinants were recovered in the sampled population of 78 zygotes, or 156 gametes. As expected segregation with respect to *Sr16* and *sr16* (in chromosome 2B) was independent of chromosome 3B constitution.

Results obtained by Dr. P.L. Dyck, Agriculture Canada,

TABLE 50

F_2 chromosome constitutions and genotypes as determined by F_3 progeny tests in the cross CS ditelosomic 3BL/CS(Thatcher 3B).

F_2 chromosome constitution	F_2 genotype			
	Sr12Sr12	*Sr12sr12* or *Sr12* -	*sr12sr12* or - -	Total
42	24	0	0	24
$42t^{L**}$	0	46	0	46
$42tt^{L**}$	0	0	8	8
Total	24	46	8	78

	Sr16Sr16	*Sr16sr16*	*sr16sr16*	Total	$\chi^{2*}_{1:2:1}$
42	9	12	3	24	3.00
$42t^{L**}$	12	25	9	46	0.76
$42tt^{L**}$	2	6	0	8	3.00
Total	23	43	12	78	3.92

* value for significance at P=0.05 is 5.99.

** Includes 1 telo- and 2 telo-chromosomes, respectively.

for 69 of the lines using "race 56" (C17) were similar to the classifications obtained using strain 126-Anz-6,7,11, indicating segregation of the same gene.

7.1.2 Apex, Cadet and Rescue

Infection types produced when seedlings of Apex, Cadet and Rescue, and various aneuploid and substitution line derivatives were inoculated with 4 strains of *P. graminis tritici* are

presented in Table 51. From the response with 326-Anz-1,2,3,5,6 it was apparent that Apex and Rescue genotypes possess *Sr5* in common with Thatcher, whereas Cadet does not. With 126-Anz-6,7,11, Apex and Cadet produced similar infection types to Thatcher, Celebration and Marquillo, where *Sr12* was mainly responsible. Rescue produced infection type "x3-" which was similar to many of the *Sr12* segregates in Celebration and Marquillo crosses, and for which *Sr12* was probably responsible. Rescue ditelo 3BL produced infection type "33+", the expected result if the gene involved was *Sr12*, and was located in chromosome 3BS. Furthermore, the parental Rescue monosomic 3B line segregated "x3-" and "3+" as expected for disomic and monosomic segregates, respectively. The ";12=" and ";1" infection types produced by Rescue when infected with 222-Anz-1,2,3,5,6 and 343-Anz-1,2,3,5,6 both of which are virulent for *Sr5* suggested that Rescue possesses *Sr9g*. This suggestion was further supported by the "2-" infection types produced in Rescue monosomic 3B and Rescue monotelosomic progeny seedlings not possessing *Sr12*. Hypothesised and established genotypes for the various lines are listed in the Table 51. The low infection type produced by S-615(Apex 3B) further indicated the presence of *Sr12* in chromosome 3B. The results showed that reciprocal substitution of the 3B chromosomes in Rescue and Cadet had no phenotypic effects upon the respective recurrent parents indicating that the 3B chromosomes are probably identical in genotype. Furthermore, the infection type of Rescue was not changed by 3B of Cadet and Cadet was not altered by 3B of Rescue, indicating that the infection type ";" to strain 126-Anz-6,7,11 in Cadet resulted from modifying gene(s). These differences are similar to those

TABLE 51

Seedling reactions of parent lines and various aneuploids derived from Apex, Cadet, S-615 and Rescue to certain strains of *P. graminis tritici*.

Line	Infection types				Hypothesised genotype
	126-6,7,11	326-1,2,3,5,6	222-1,2,3,5,6	343-1,2,3,5,6	
Apex	;	0;	;1=	;	*Sr5* *Sr12* *Sr9g*
S615	3+	3+	33+	33+	- - -
S615(Apex 3B)	x	x+	x+2	x+2	- *Sr12* -
Rescue	x3-	0;	;12=	;1-	*Sr5* *Sr12* *Sr9g*
Rescue(Cadet 3B)	x3-	0;	;12=	;1-	*Sr5* *Sr12* *Sr9g*
Rescue monosomic 3B	x3-,3	;	;12=,2-	;1-,2=	*Sr5* *Sr12*/-&*sr12sr12* *Sr9g*
Rescue ditelosomic $3B^L$	33+	;	-	-	*Sr5* -
Rescue monotelosomic $3B^L$	3	0;	2	2-	*Sr5* - *Sr9g*
Cadet	;1	x3+	2	2-2	- *Sr12* *Sr9g*
Cadet(Rescue 3B)	;	x3+	2	x2	- *Sr12* *Sr9g*
Chinese Spring	4	3^{++}	3+	3+	- - -
Thatcher	;	0	;1=	;1=	*Sr5* *Sr12* *Sr9g* *Sr16***
Marquillo	0;	;x-	;	;	- *Sr12* *Sr9g*
Celebration	;	;x	;1=	;1=	- *Sr12* *Sr9g* *Sr16*
CS(Thatcher 2B)	33-	2-	2-	2-	- - *Sr9g* *Sr16*
CS(Thatcher 3B)	x2-	x3+	x3+	x3	- *Sr12* - *Sr16*
I*Sr16*Ra	3-3	3-3	3-3	3-3	- - - *Sr16*
Mi12*	;x	x33+	x3+	x33+	- *Sr12* - -

* *Sr12* line obtained from Cereal Rust Laboratory, Minnesota, U.S.A.

** Thatcher assumed to carry *Sr16* as CS(Thatcher 3B) and CS(Thatcher 2B) both possess *Sr16*.

involving Marquillo and Celebration in section 6.2.1.

7.2 CHROMOSOME ARM LOCATION OF HOPE ADULT-PLANT RESISTANCE

7.2.1 Chromosome Substitution Lines of Hope

In 1975, adult plants of Chinese Spring were susceptible and Hope resistant. Of the substitution lines only CS(Hope 3B) was resistant. This result differed from that of McIntosh *et al.* (1967) because the strains present in 1975 were virulent for *Sr17*, hence lines CS(Hope 6B) and CS(Hope 7B) were susceptible. These workers failed to recognise the phenotypically lesser effect of CS(Hope 3B) compared with the lines carrying *Sr17*.

An analysis of variance of 1,000 grain weights and line means taken from rusted plots are presented in Table 52. The mean 1,000 grain weight of CS(Hope 3B) was clearly different from all other lines and Chinese Spring. Although not detected by disease ratings, variation between the remaining lines could have been due to differences in disease intensity not recognised by the field assessment method, to inherent grain weights, or to chance deviations. These data suggest that chromosome 3B contributed very largely to adult-plant reaction.

7.2.2 Cytogenetic Analysis

7.2.2.1 CS(Hope 3B)

Three F_2 populations from CS monotelosomic 3B/CS(Hope 3B), each derived from monosomic F_1 plants, were examined for rust reaction in the field. Owing to the poor fertility of bagged heads on F_1 plants grown during the summer in the glasshouse, only small F_2 populations were obtained. Variation in plant

TABLE 52

Analysis of variance of 1,000 grain weight measurements on Hope chromosome substitution lines.

Source of variation	df	MS	F
Between lines	21	0.17	7.8**
Among lines (Error)	22	0.02	
Total	43	0.09	

** P<0.01.

Line Means (g)

CS(Hope 1A)	12.30*	1B	11.55	1D	12.54*
2A	12.70*	2B	6.96	2D	11.12
3A	11.09	3B	21.43**	3D	13.59*
4A	9.87	4B	9.69	4D	10.10
5A	9.89	5B	11.46	5D	8.22
6A	9.71	6B	11.08	6D	7.57
7A	11.10	7B	12.82*	7D	10.27
Chinese Spring	8.67				

*1.s.d. (P=0.05) = 3.02.

**1.s.d. (P=0.01) = 4.10.

height at maturity was also noted. The somatic chromosome constitution of F_2 plants was deducted from mitotic counts on 5 grain samples (F_3) taken from each plant. A decision to designate a plant as disomic was made if 3 consecutive counts were 2n=42. Alternatively, a decision to designate a plant as monosomic was reached when two plants with 2n=41 had been scored. Of 23 F_2 plants, 5 were disomic, 17 were monosomic and 1 was apparently monotelosomic for the long arm. Both disomic and monosomic plants were resistant compared with CS, but monosomic plants were more rusted than the disomics. The monotelosomic plant was susceptible. Additionally the three plant types differed in height - disomics were tall (mean 115 ± 15.4 cm), monosomics were intermediate (95 ± 14.6 cm) and the monotelosomic was relatively short (64 cm). Two short susceptible plants which had failed to set seed were assumed to have been nullisomic. It was concluded that the allele for resistance is in chromosome 3B and that it is hemizygous-effective. Since the derivative monotelosomic for the long arm was susceptible, the resistance allele must be situated in the short arm.

7.2.2.2 Hopps and Warigo

F_2 populations derived from monosomic F_1 plants in crosses CS monotelosomic 3B/Hopps and CS monotelosomic 3B/Warigo and euploid F_1s in from Hopps/CS and Warigo/CS were examined for rust reaction (Table 53).

Because earlier results for Hopps and Warigo have shown that adult-plant resistance was controlled by a recessive allele, the segregation ratios were tested for conformity with a 1:3 ratio. Segregation in the euploid populations conformed

TABLE 53

Segregations for reaction in monosomic and euploid F_2 populations.

Cross	F_1 Chromosome No.	Resistant	Susceptible	$\chi^{2*}_{1:3}$
CSMT3B/Hopps	41	13	4**	24.02
Hopps/CS	42	27	62	1.35
CSMT3B/Warigo	41	20	2**	50.97
Warigo/CS	42	24	55	1.22

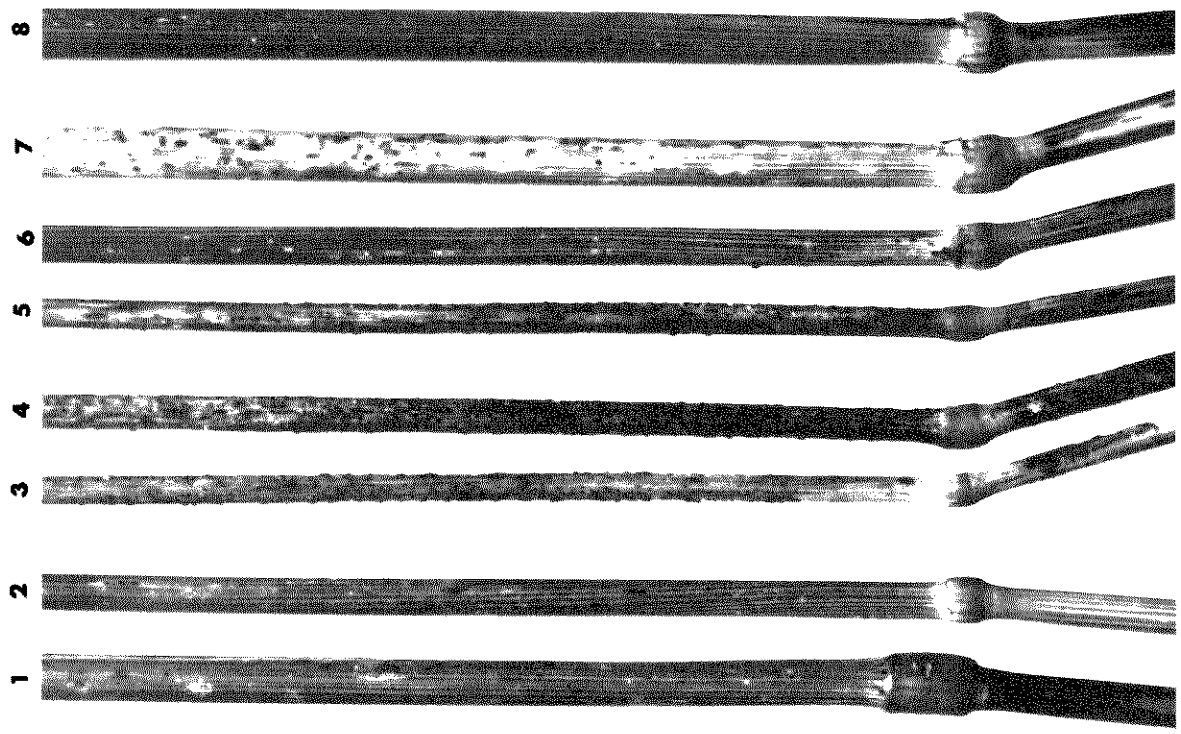
* value for significance at P=0.05 is 3.84.
** short stature as well as susceptible.

with this hypothesis, but that in monosomic populations clearly deviated. The relatively few susceptible plants in these populations were much shorter than the resistant plants and similar in height to the susceptible plant in the CS monotelosomic 3B/CS(Hope 3B) cross, indicating that they were nullisomic or monotelosomic. Mitotic examination of progenies from one susceptible plant in CS monotelosomic 3B/Warigo indicated that this plant was monotelosomic for the long arm of chromsome 3B. The remaining susceptible plants were sterile. These results confirmed the hemizygous-effective behaviour of a resistance allele in chromosome 3BS.

7.2.2.3 Redman Monosomics

Four monosomic plants and a variable number of plants of unknown chromosome number in each Redman monosomic line were grown under rust epidemic conditions. In anticipation of the importance of chromosome 3B, 6 nullisomic, 19 monosomic, 20

disomic and 1 plant monotelocentric for the long arm of 3B had been somatically identified before transplantation to the field. Redman was resistant and Chinese Spring control was susceptible. Redman monosomic lines 6A and 1D were uniformly susceptible. Apart from line 3B, the other monosomic lines were uniformly resistant. In line 3B, nullisomic plants died as tufts prior to elongation and could not be assessed for disease reaction, the monotelosomic elongated weakly and was susceptible to rust. Disomic plants were more resistant than monosomic 3B plants (Plate 11). Observation on pseudo-black chaff reaction indicated that this character was lacking in lines 6A and 1D. Presumably, at completion of the back-crossing programme the genotypes for adult-plant reaction and the closely associated pseudo-black chaff character remained heterozygous, and the monosomic plants subsequently selected for line maintenance were homozygous susceptible. Lack of genotype reconstitution was noted in several lines. For example, both monosomic and disomic plants in 1A and 7A flowered and matured much earlier than Redman. These lines apparently lacked the long day requirement of Redman and hence flowered early under Australian short-day conditions. In studies on seedling reaction, McIntosh (1970) indicated that lines 3B and 2B did not represent the reconstituted Redman genotype with respect to seedling reaction to North American *P. graminis tritici* "race 56". According to a genetic model he proposed, Redman seedlings produced infection type ";" to "race 56" as a result of Category IV interaction involving *Sr9d* and a gene (probably the adult-plant resistance allele) in chromosome 3B. Redman monosomic 3B gave infection type "3+" due to lack of *Sr9d* in its chromosome 2B, whereas line 2B produced infection type



1 Rescue cultivar; 2 Cadet (Rescue 3B); 3 Cadet monotelosomic 3BL; 4 Cadet ditelosomic 3BL (3 and 4 derived from a monotelosomic 3BL line); 5 Cadet monotelosomic 3BL; 6 Cadet monosomic 3B; 7 Cadet disomic 3B (5, 6 and 7 derived from a monosomic 3B line); 8 Cadet cultivar.

1 Chinese Spring; 2 Redman disomic 3B; 3 Redman monosomic 3B; 4 Redman monotelosomic 3BL (2, 3 and 4 derived from a monosomic 3B line); 5 Redman cultivar.

PLATE 11 Adult-plant reactions

"2+" due to *Sr9d* and a lack of the gene in chromosome 3B. Since line 2B was resistant in the present study, it would appear that for McIntosh's model to be correct, the modifier of *Sr9d* must be different from the recessive gene for adult-plant resistance. Additionally, McIntosh (1970) showed that Redman lines 2B and 4B, although having different seedling phenotypes to "race 56", were both monosomic for chromosome 2B. These results explained the uncertainty of gene locations claimed for Redman by Campbell and McGinnis (1958). Apparently, the Redman monosomic series was fully reconstituted with respect to *Sr17* (McIntosh *et al.*, 1967). The present study with the Redman monosomics supported the earlier conclusions that chromosome 3B was involved, that the resistance was hemizygous-effective and that the short arm of 3B was involved.

7.2.2.4 Apex, Cadet and Rescue

The pedigree of Apex is given as H44-24/Double Cross and Rescue as Apex/S-615. Both cultivars were resistant and expressed pseudo-black chaff in 1975. The pedigree of Cadet is Merit/Thatcher. The parentage of Merit was not available, but adult-plants of Cadet expressed pseudo-black chaff in 1975, suggesting that H44-24 or Hope might be involved.

Seedlings of known chromosome numbers from various chromosome 3B aneuploids and chromosome substitution lines, involving Apex, Cadet, S615 and Rescue were transplanted in the field in 1975, and assessed for rust reaction (Table 54 and Plate 11).

The substitution of chromosome 3B of S615 by its homologue from Apex resulted in adult-plant resistance, again suggesting, but not proving involvement of chromosome 3B.

TABLE 54

Reactions of parent lines and various aneuploids derived from Apex, Cadet, S615 and Rescue.

(Rust reactions: R = resistant; S = susceptible.
Pseudo-black chaff: + = present; - = absent).

Line	Chromosome No.	Stem Rust Reaction	Pseudo-black chaff
Apex	42	R	+
S615	42	S	-
S615(Apex 3B)	42	R	+
Cadet(Rescue 3B)	42	R	+
Rescue(Cadet 3B)	42	R	+
Cadet	42	R	+
Cadet monosomic 3B	-42 (3 plants)	R	+
	-41 (4 plants)	semi-R	+
	$-41t^{L*}$ (1 plant)	S	-
Cadet monotelosomic 3B	$-42tt^{L**}$ (1 plant)	S	-
	$-41t^{L*}$ (3 plants)	S	-
Rescue	42	R	+
Rescue monosomic 3B	-41 (8 plants)	semi-R	+
Rescue monotelosomic 3B	$-42tt^{L**}$ (2 plants)	S	-
	$-41t^{L*}$ (1 plant)	S	-

** including 2 telosomes and

* including 1 telesome.

Monotelosomic and ditelosomic 3BL plants for both Cadet and Rescue were susceptible. Additionally,one newly isolated monotelosomic 3BL plant in the Cadet monosomic 3B line was also susceptible. The reciprocal 3B substitution lines of Cadet and Rescue behaved similarly as adult plants, indicating that these cultivars may carry similar genes in their respective chromosomes 3B. Although not stated by Larson,it is assumed that the monotelosomic lines had been isolated earlier from the respective monosomics,and hence apart from absence of 3BS, were similar in genotype. Again,chromosome 3BS was implicated in rust reaction, resistance was hemizygous-effective but disomy of chromosome 3BS produced a higher level of resistance than monosomy.

8. DISCUSSION AND CONCLUSIONS

Construction of epidemic progress curves clearly demonstrated the effectiveness of adult-plant resistances in Hope and Marquillo and certain of their respective derivatives to Australian stem rust strains, and indicated a need for genetic analyses of these resistances. By calculating areas under epidemic progress curves, Wilcoxson *et al.* (1974) showed that adult-plants of Hope, Redman and Thatcher were relatively resistant, Lee intermediate and Selkirk immune when tested with stem rust race 15B, to which Hope and Selkirk gave low reactions as seedlings. The adult-plant responses of all cultivars were similar to those obtained in the present studies, although some rust developed on Selkirk in 1974 and 1975, when strains virulent on seedlings were present in the field.

Apart from Florence which appeared to possess rust tolerance on the basis of grain weight determinations, the remaining hexaploid cultivars did not exhibit detectable adult-plant resistances or tolerances. Wilcoxson *et al.* (1974) classified Exchange as relatively resistant, whereas in the present studies it was susceptible. Although Webster gave a high reaction to race 15B at the seedling stage, adult-plants were classified relatively resistant by Wilcoxson *et al.*, however in Australia resistance derived from Webster is now ineffective (Luig and Watson, 1970).

Tetraploid cultivars selected for susceptibility as seedlings fell into three rust intensity categories as adult-plants *viz.* susceptible, intermediate and resistant. Cultivars Glossy Huguenot, Marouani, Palestine 2649 and W2472 exhibited

sufficient resistance for disease to have no measurable effects on grain weights. Tetraploid wheats have proved valuable sources of rust resistance in the past as exemplified by Hope and Marquillo. The present small survey illustrated the availability of further resistances which could be expected in tetraploid wheats, or which possibly could be transferred to hexaploid wheats.

After two years of study of disease assessment techniques, this approach was discontinued. The study of epidemic progress and preparation of progress curves proved time consuming and was therefore expensive. Other factors such as maturity differences and variable reactions to additional diseases made comparisons between progress curves and between grain weight measurements relatively difficult to interpret. On the other hand, equally useful data could have been obtained from two or three observations on small plots between the stages of heading and dough ripeness. As far as the genotypes studied in the current work were concerned, significant differences in disease reactions could be determined by visual analysis of small plots, and a preliminary study of F_3 lines in the cross CS/CS(Hope 3B) in 1973 confirmed that visual classification could be extended to segregating populations.

Although a satisfactory leaf rust epidemic developed in the field during 1972, this disease did not cause statistically significant effects on grain weight. Phipps (1938), Peturson *et al.* (1948) and Keed and White (1971) reported grain weight losses attributable to the effects of leaf rust.

Watson and Luig (1968) suggested that the cultivar Hopps may possess a genetically complex resistance to stem rust, since it appeared to fit their criteria for non-specific resistance.

Genetic analysis of F_1, F_2, F_3 and F_4 populations from crosses involving the Hope derivatives CS(Hope 3B), Hopps, Renown Selection, Selkirk and Warigo indicated that adult-plant resistance was determined by recessive alleles at a single locus common to all lines. Goulden *et al.* (1928), Knott (1968) and Sheen *et al.* (1968) found that the adult-plant resistances of Hope, H44-24 and Conley, respectively, were controlled in each instance by a single gene. Goulden *et al.* (1930), Neatby and Goulden (1930), Clark and Humphrey (1933) and Pan (1940) concluded that Hope and H44-24 have a gene in common for adult-plant resistance. Presumably, Knott (1968) detected the same gene and presumably, this gene was transferred to Conley. Knott (1968) designated this gene as *Sr2* following the recommendation of Ausemus *et al.* (1946). It is presumed further, that the same gene was identified in the current studies.

The results of four different experiments indicated that *Sr2* was located in the short arm of chromosome 3B. *Sr2* was incompletely hemizygous-effective, an unusual condition for a recessive gene. Disomy of chromosome 3BS produced a greater level of resistance than monosomy. Sheen *et al.* (1968) associated chromosome 3B with *Sr2*, but failed to note the hemizygous-effective nature of the resistance as indicated by their data. The observed F_2 segregation ratio from CS monosomic 3B/Conley obtained by Sheen *et al.* was 43 resistant: 26 susceptible compared with ratios ranging from 4:4 to 3:12 for other non-critical monosomic populations. When compared with the present results of 22:1 from CS monotelosomic 3BL/CS(Hope 3B), 13:4 from CS monotelosomic 3BL/Hopps and 20:2 from CS monotelosomic 3BL/Warigo, 37% susceptible F_2 plants from the CS monosomic 3B/Conley cross appears to be an

excessively high nullisomic frequency. Two alternative explanations may be suggested. As the resistance is hemizygous-effective, only nullisomic 3B or monotelosomic 3BL plants can be fully susceptible. However, chromosome 3B could have undergone a relatively high rate of misdivision producing monotelosomic plants at a higher than usual frequency. Somatic chromosome counts of F_2 progenies from CS monotelosomic 3BL/CS(Hope 3B) indicated that chromosome 3B did misdivide to give monotelosomic individuals, but the observed frequency was relatively low at approximately 4%. Secondly, it is possible that monosomic plants from the Conley cross were more susceptible than disomic plants, owing to the dosage effect observed in the CS(Hope 3B) cross, thus some of the monosomic plants may have been classified as susceptible. Both explanations may partly account for the high proportion of susceptible plants observed by Sheen *et al.*

One thousand grain weights determined on homozygous resistant and homozygous susceptible F_3 families from CS/CS(Hope 3B) demonstrated that *Sr2* in CS(Hope 3B) offered considerable protection against grain weight loss in the presence of rust. Since CS(Hope 3B) becomes considerably rusted near to harvest ripeness, even greater protection could be expected with the other Hope derivatives which do not rust to the same extent.

Classifications of adult-plant reactions were consistent over two years of field testing. Furthermore, a small summer sowing of lines tested during autumn showed that single plants could be readily classified for *Sr2* during both spring and autumn. In contrast, resistant and susceptible cultivars were not differentiated in one greenhouse test. Evidently the

greenhouse conditions were conducive to rust development, since resistant cultivars produced disease intensities similar to those produced by susceptible controls. Hart and Zaleski (1935) reported that the Hope resistance may be ineffective under artificial conditions in the greenhouse, owing to reduced light intensity and high humidity.

It was found necessary to record adult-plant reactions over an optimal period which extended from the milk to early dough stages of development. At maturity, certain genotypes showed rust intensities approaching those of susceptible genotypes, but the rust developed only with the approach of senescence. McIntosh *et al.* (1967) failed to detect *Sr2* possessed by Renown Selection when using strains virulent on plants with *Sr17*, but did indicate their belief that Renown W2346 possessed additional resistance. They assessed the final adult-plant reactions at maturity when differences are less clear.

It was evident that the amount of stem rust development on resistant plants in both resistant/susceptible and resistant/resistant crosses was variable. Hope, Hopps, Selkirk, Renown Selection and Warigo were diseased to a similar degree near maturity, whereas CS(Hope 3B) carried significantly more disease and furthermore, the degree of variation in disease intensity within resistant/resistant crosses appeared to be related to the reaction of the parents. Results from an inheritance study involving Warigo/CS(Hope 3B) and homozygous resistant lines of Gabo/Selkirk indicated that *Sr2* was modified by alleles at a single locus in each cross. In the case of Hope, any modifying genes are not associated with chromosome 3B, since substitution of the entire chromosome

produces a greater degree of susceptibility in CS(Hope 3B) than is shown by Hope. Sheen *et al.* (1968) implicated a system of minor genes to explain variation within resistant and susceptible classes in a Conley cross.

In the genetically complex system of inheritance suggested by Watson and Luig (1968), genes presumably have approximately equal effects. However, in the model proposed here, the presence of *Sr2* is essential before any resistance is expressed, and further modification is effected by one or few genes.

Watson and McIntosh (personal communication) suggested that alleles for specific resistance may interact with, and enhance, the expression of adult-plant resistance even though the particular strains present may carry the corresponding genes for virulence. Clifford (1974), Rees (personal communication) and Caldwell (personal communication) have suggested that specific resistance genes may offer a broad spectrum residual resistance to strains with corresponding genes for virulence. Indeed, Ellingboe (1975) described a specific instance where this was apparent in the wheat powdery mildew system. In Warigo/CS(Hope 3B) and Gabo/Selkirk crosses there was no evidence that the expression of *Sr2* was affected by the specific resistance alleles *Sr6*, *Sr7b*, *Sr17* and *Sr23*, or by their contrasting susceptibility alleles. This conclusion was valid for those instances where strains carried the corresponding genes for virulence or where the actual gene had no effect on adult-plant reaction. In this latter respect, McIntosh and Luig (1973) claimed that *Sr23* could be detected at the seedling stage with all Australian strains. *Sr23* provides no protection to adult-plants and had no effect on

Sr2 expression in Selkirk. Segregation in the Selkirk and Warigo hybrid populations demonstrated the possibility of combining specific resistance alleles with the non-specific resistance, *Sr2*. *Sr2* can be classified 'non-specific' as pathogen variants with proven virulence have not been isolated in 50 years of commercial use. In order to provide effective broad spectrum resistance, Van der Plank (1968), Hooker (1967), Lewellen *et al.* (1967) and Watson (1970b) have recommended the breeding of cultivars in which genes for specific and non-specific resistances are combined.

Genetic analysis of pseudo-black chaff showed that pigment development was governed by recessive alleles, confirming the findings of Kuspira and Unrau (1958) and Sheen *et al.* (1968). The locus involved was completely or very closely linked with *Sr2*. Goulden (1929) and McFadden (1939) intimated very close or complete linkage following repeated attempts to isolate recombinants, whereas Pan (1940) reported linkage but believed the linkage could be broken. Sheen *et al.* (1968) reported recombination values ranging from 21 to 24% in crosses involving Conley, but these values appear to be too high on the basis of present data.

The degree of melanin pigmentation appears to be modified by relatively few genes. Although Selkirk and Renown Selection with long day requirements for flowering are later under Australian conditions, they were relatively less pigmented than CS(Hope 3B), Hopps and Warigo despite the fact that higher temperatures experienced late in the season were more conducive to pigment development (Johnson and Hagborg, 1943). It appears therefore that Selkirk and Renown Selection possess a genetic system(s) which reduces the degree of

pigmentation. Hybrid populations, homozygous for the pseudo-black chaff factor and derived from crosses of Selkirk, Renown Selection and Warigo showed variation in the degree of pigment development. Certain lines selected for low and high levels of pigmentation gave progenies which behaved similarly to the respective parent lines. Hence, it is suggested that alleged recombinants for *Sr2* and pseudo-black chaff, reported by Sheen *et al.* (1968) from Conley crosses, were individuals which carried recessive alleles at the *Sr2* and the pseudo-black chaff loci but in which one character was significantly modified relative to the other.

Variation in the degree of pigment development was not associated with disease intensity as variation in rust reaction was observed while the pseudo-black chaff phenotype remained relatively constant and *visa versa*. All plants with *Sr2* developed pigment but certain individuals and/or lines were comparatively free of pigment at maturity. From a breeding standpoint, this finding is important, since *Sr2* can be utilized without the deleterious characters associated with pseudo-black chaff development. Furthermore, it should be possible to select for *Sr2* indirectly by selecting for pseudo-black chaff. This could obviate the need to conduct field rust tests. However, any approach of this kind would need to be very carefully applied, as the marker being selected must be suppressed to the maximum extent. McFadden (1939) proposed a test to identify carriers of *Sr2* prior to flowering by inducing pigment formation around the site of a hypodermic inoculation using *P. graminis tritici* urediospores. Preliminary tests of this procedure confirmed McFadden's method, however, plants which produced pigment about the site of inoculation

became strongly pigmented at the late dough stage. Moreover, the procedure was unreliable in detecting carriers which were relatively free of pigment at the later maturity stage.

As rust reaction and pigmentation are modified independently, it could be argued that a) each character is controlled by a different locus and each modified by a separate genetic system,or b) the pleiotropic characters are separately modified.

A group of 13 Hope derivatives reported (Anonymous 1972; Watson, 1970b; Caldwell, 1968) to be sources of non-specific resistance, or to have resisted rust over many years of exposure were field resistant and expressed pseudo-black chaff,suggesting that they probably possess *Sr2*. Presumably *Sr2* has played a significant role in the long-term resistance of these cultivars.

Hope derivatives which carry *Sr2* tend to give seedling reactions with some necrotic flecks (;) in an otherwise susceptible (3+) reaction,but classification for this reaction type in segregating lines was not always possible. Cultivars Aotea, Gala and Glenwari which lack *Sr2* do not show the flecking. Knott (1968) and Loegering (personal communication to McIntosh) found that *Sr2* which conditioned adult plant resistance to race 56 could be detected in seedlings by interaction with *Sr9d*,but this method is not possible using Australian cultures,since all are virulent for *Sr9d*.

It has been suggested by McIntosh (personal communication) that Spica possesses adult-plant resistance to stem rust. However,the situation is confusing because Spica may escape disease damage due to early maturity. Epidemic progress curves and grain weight analysis in 1972 and 1973 suggested that

both theories may apply. While Spica does not carry *Sr2* and was regarded as a susceptible parent in crosses studied in the field, greenhouse tests showed that Spica varied in adult-plant reaction to strains virulent on *Sr7b* and *Sr17*. Spica showed a varying but significant level of resistance compared with W3498, to strains 21-Anz-2,3,4,5,7, 21-Anz-2,3,4,5,6,7, 21-Anz-(1),2,3,4,5,6,7, 34-Anz-2,4,5,6, 34-Anz-1,2,3,4,5,6,7, 126-Anz-6,7,11 and 222-Anz-1,2,3,4,5,6,7 but not to 21-Anz-5. Apparently 21-Anz-5, isolated on commercial crops of Spica, was highly adapted in its ability to infect and produce disease on Spica. Similarly, it is suggested that certain strain(s) of *P. striiformis* became adapted on the adult-plant resistant Joss Cambier in Britain in 1971 (Clifford, 1974).

Although, 15 lines from Spica/W3498 were homozygous resistant compared with W3498 to both 126-Anz-6,7,11 and 34-Anz-1,2,3,4,5,6,7, all produced more disease than Spica suggesting that the resistance is enhanced by the genetic background of Spica. However, *Sr7b* and *Sr17* in Spica did not influence the expression of the adult-plant reaction. The behaviour of Seafoam, a parent of Spica, was generally similar but intermediate to that of Spica and W3498 and was clearly more resistant to 126-Anz-6,7,11 than to other strains.

Under field rust conditions there was no evidence of segregation in reaction among F_3 lines derived from Spica. No significant variability could be identified in F_3 lines of CS/Spica nor among F_3 lines not possessing *Sr2* in the Spica/Hopps hybrid populations. On the other hand, certain F_3 families with *Sr2*, both early and late maturing, from Spica/Hopps were free of stem rust in the field

in 1974,whereas none of the homozygous resistant F_3 families from Hopps/Yalta were rust-free. This suggests that the resistance carried by Spica may have enhanced the expression of *Sr2*. An inheritance study conducted in the greenhouse suggested that the adult-plant resistance in Spica was possibly determined by alleles at a single locus.

Unpublished data of Green (personal communication to McIntosh) in Canada, Rajaram (personal communication) in Mexico and at this Institute indicate that adult-plants of Hopps are resistant to all strains of *P. recondita* with which they have been tested. In this study,adult-plant resistance was governed by a single dominant allele which segregated independently of *Sr2*. Whether this allele is different from those adult-plant leaf rust resistance genes already designated is not known. Of 18 clearly distinguishable loci for resistance,only three *Lr12*, *Lr13* and *Lr22* are of the adult-plant type. Since *Lr22* was derived from *T. tauschii* (Coss.) Schmal. subsequent to the breeding of Hopps,it is unlikely that the gene identified in this study is *Lr22*. Whether the Hopps allele is similar to *Lr12* or *Lr13* is unknown. Rajaram (personal communication) indicates that Hopps possesses resistance to *Puccinia striiformis* in Mexico. Having resistance to all three rusts, Hopps is a very useful parent in breeding.

CS(Hope 3B) was found to possess seedling resistance to all cultures of *P. recondita*, whereas Hope was susceptible to strains virulent on *Lr14a* and Chinese Spring seedlings were susceptible to all strains. The origin of seedling resistance in CS(Hope 3B) is not understood. Possibly, it was carried by the Hope donor used by Sears but resistance of this type in Hope has not been reported in other studies. In the current

studies, a single dominant allele appeared to determine this resistance; however, studies on the same F_3 lines by Dr. P.L. Dyck using Canadian cultures, suggested segregation of two genes for low reaction, one of which was identical to that detected with the Australian culture. No attempt was made to study the inheritance of adult-plant leaf rust resistance in CS(Hope 3B), because Chinese Spring is resistant at the adult-plant stage. Hence, the seedling resistance would need to be transferred to a genotype susceptible at the adult-plant stage before it could be properly assessed for breeding purposes. A further possible explanation of the seedling resistance of CS(Hope 3B) is that the gene(s) controlling adult-plant resistance of Chinese Spring are expressed in the seedling stage when combined with *Sr2*. This model predicts that segregation in the crosses such as CS(Hope 3B)/Warigo and CS(Hope 3B)/CS should be similar and relatively simple. However, in a cross such as CS(Hope 3B)/Federation segregation indicative of gene interaction is expected, as both *Sr2* and the Chinese Spring genes would segregate.

Adult-plant resistance in Marquillo and Celebration was primarily determined by recessive alleles at a single locus. Correlation studies with seedling reactions suggested that a gene, designated *Sr12* and characterised by mesothetic seedling infection types, governed adult-plant resistance. Cytogenetic studies on seedlings of CS(Thatcher 3B), Apex, Rescue and Cadet indicated that *Sr12* was located in the short arm of chromosome 3B and was hemizygous-ineffective. This location is consistent with that of Sheen and Snyder (1964).

As resistant Marquillo derivatives neared maturity,

alleles at an independent locus enhanced the expression of adult-plant resistance attributable to *Sr12*, but these alleles gave no adult-plant resistance when alone. This modification of resistance was attributed to an allele at the *Sr9* locus, *Sr9g* (McIntosh, personal communication). Although resistant lines from the Celebration cross displayed variation in adult-plant disease intensity, resistant lines could not be further classified as with the Marquillo cross.

In resistant lines, the rate of disease increase was slow by comparison with susceptible lines, although at maturity lines carrying *Sr12* alone were significantly rusted. This 'slow-rusting' characteristic again underlines the necessity to record field notes during the pre-harvest ripeness periods of development.

Where strains were virulent for *Sr5*, Celebration and Thatcher reacted similarly as seedlings, whereas Marquillo produced lower infection types, possibly reflecting its greater field resistance. Furthermore, the infection types attributable to *Sr12* were consistently lower in the Marquillo than the Celebration cross. However, the general character of the infection type, as well as pedigrees, suggested that the same resistance allele, *Sr12*, was involved in both genotypes. Known genes did not account for this difference.

Lines 2B and 3B of the Chinese Spring (Thatcher) chromosome substitution series, showed resistance at both the seedling and adult-plant stages, but neither were as resistant as Thatcher. There was no evidence that line 6A considered to be important in Canada (Brennan, 1975) was different from Chinese Spring. Although Loegering and Sears (1973) showed that both CS(Thatcher 2B) and CS(Thatcher 3B) carried *Sr16*, a

comparison of the seedling infection types produced on Chinese Spring, *Sr16* isogenic line, and their substitution lines indicated that the lines possessed additional genes. In the case of CS(Thatcher 2B), McIntosh (personal communication) identified *Sr9g*, and in the present studies *Sr12* was located on chromosome 3B of CS(Thatcher 3B). *Sr9g* is present in many Thatcher derivatives as well as the durum cultivars Acme, Kubanka and Iumillo (McIntosh and Luig, unpublished). From the adult-plant susceptibilities of segregates possessing *Sr9g* alone and I *Sr16*-Ra, CS(Thatcher 2B) (*Sr9g Sr16*) was predicted to be susceptible, however the resistance observed may have resulted from interactions involving *Sr9g* and *Sr16*.

Grain weight analyses conducted on the Marquillo and Celebration crosses, together with the Thatcher substitution line series, substantiated the adult-plant reaction data and demonstrated the effectiveness of *Sr12 Sr9g* combinations in comparison with lines carrying either gene alone.

Infection types produced on CS(Thatcher 3B) indicated strain variability as well as sensitivity of *Sr12* (**Table 35**) to temperature. For seedling studies the use of certain strains such as 126-Anz-6,7,11 and 34-Anz-2,4,5,7,11 permitted easier classification than the use of other strains. Infection at low temperatures increased the level of incompatibility. These sensitivites could explain the failure of Loegering and Sears (1973) and Green and Dyck (1975) to detect *Sr12*. In the present studies, *Sr12* was classified using the 59-E-5,9 strain employed by Loegering and Sears, but the infection types produced were higher than those produced using 126-Anz-6,7,11. On the other hand, the low infection types produced by *Sr9g* and *Sr16*, in seedlings, were relatively temperature stable.

Using certain North American cultures it was demonstrated that Celebration probably carries *Sr16* whereas Marquillo does not.

Culture 56-E1, avirulent on *Sr12* and *Sr16*, produced infection types on Marquillo and Celebration identical with those obtained using strains 34-Anz-2,4,5,7,11 and 126-Anz-6,7,11. However, in the Marquillo cross, lines known to be segregating with recessiveness of *Sr12*, using strains 126-Anz-6,7,11 and 34-Anz-2,4,5,7,11, segregated in a dominant pattern with 56-E1. On the other hand, similar lines in the Celebration cross were tested with 56-E1 and these strains showed recessive gene segregations. It appears therefore that Marquillo carries a second allele closely linked in coupling with *Sr12*. Presumably 56-E1 is avirulent for this allele as well as *Sr12*, whereas all other strains employed were virulent. Since Celebration and Marquillo were derived from the same source, Iumillo, the presence of the *Sr12* allele in both must be implied. Sheen and Snyder (1964) obtained similar results when studying a line considered to be CS(Thatcher 3B). They assumed two closely linked loci, one dominant, the other recessive, determining low reaction and the recessive allele was characterised by a mesothetic infection type. Recombinants were not recovered. Indeed, they suggested the use of symbol *Sr12* for the dominant allele, whereas assuming two genes are involved, its current use more likely applies to the recessive allele.

Many cultivars including Apex, Cadet, Newthatch, Rescue and Yaqui 50 apparently combine *Sr12* and *Sr2* in coupling and since both are located in chromosome 3B they may show genetic linkage. This linkage could be studied in such lines as

S615(Apex 3B),where from present predictions both genes should occur and other genes in Apex (*Sr5*, *Sr9g*) should be absent.

While *Sr2* can be classified 'non-specific', *Sr12* cannot be so classified with confidence. Recently, Green and Dyck (1975) reported strains virulent on adult-plants of Thatcher, but they suggest that Thatcher resistance should still be used in breeding programmes as the resistance remains effective against prevalent strains in Canada. Furthermore, in Australia, strain 21-Anz-9, occasionally isolated from commercial crops of Windebri (Celebration derivative), is almost fully virulent on seedlings of Marquillo, Celebration and Iumillo and may be adapted to these cultivars at the adult-plant stage (Luig, personal communication). Although 21-Anz-9 is avirulent on most specific resistance genes carried by current commercial cultivars, variants combining the virulence of 21-Anz-9 with virulence on plants with the other genes may arise,and such variants could cause damage to cultivars carrying *Sr12*.

Under certain environmental conditions, such as high humidity, temperature and abundant inoculum, *Sr2* may not provide sufficient protection to prevent some yield losses (Stakman and Rodenhiser, 1958), especially in cultivars where *Sr2* is the only effective resistance gene present. In North America, epidemics in 1953, 1954 and 1955 involving race 15B, caused losses,as the race was apparently well adapted to cultivars possessing *Sr2*,and was virulent on all other specific resistance genes carried by these cultivars (*Sr7b*, *Sr9d*, *Sr17*). In all three years, abundant moisture and inoculum favoured rust development (Peturson, 1958).

The persistent effectiveness of *Sr2* and *Sr12* could be due to a) an extremely low mutation rate in the direction of

virulence for the corresponding gene in the pathogen, b) stabilizing selection (Van der Plank, 1968) or c) the genes may not follow the gene-for-gene relationship (Ellinghoe, 1975) thus virulence genes in the pathogen may not occur. Whether *Sr2* or *Sr12* owe their stability to any of these mechanisms is unknown, and furthermore may be difficult to determine. If such information was available, the future stability could be predicted. Nevertheless, without this information a cautious approach to the use of these resistances is desirable. Assuming virulent pathogen variants can arise, it is suggested that *Sr2* and *Sr12* be combined with other genes in a single genotype. The probability of the simultaneous acquisition of two or more corresponding virulence genes should be significantly lower than that for the individual genes taken separately. Such combinations of genes should extend the effective life of the individual resistance genes, provided these genes are not deployed individually in cultivars grown simultaneously with the multiple resistance cultivars. A combination of *Sr2* and *Sr12* appears to be a very useful resistance, as illustrated by the performance of Newthatch, Yaqui 50 and further derivatives which presumably carry both genes. Macindoe (1948) suggested a breeding procedure for combining a seedling resistance such as *Sr12* with the adult-plant resistance, *Sr2*, by selecting resistant plants from F_3 families homozygous for *Sr2* but heterozygous for low seedling reaction. The low seedling reaction is made homozygous in the following generation.

9. SUMMARY

Epidemiological studies confirmed the effectiveness of adult-plant stem rust resistances in Hope and Marquillo and certain of their respective derivatives. Among tetraploid wheats selected for susceptibility as seedlings, some cultivars, namely, Glossy Huguenot, Marouani, Palestine 2649 and W2472 expressed adequate resistance which prevented measurable grain weight losses. However, disease progress studies proved time consuming, and observed differences in grain weights were confounded by maturity and variable reactions to additional diseases. Adequate data on stem rust reaction were obtained from two or three observations on small plots, between the stages of heading and dough ripeness.

The persistent adult-plant resistance in Hope and certain derivatives was attributed to recessive alleles, *Sr2Sr2*, located in the short arm of chromosome 3B. *Sr2* was incompletely hemizygous-effective. Analyses of one thousand grain weights demonstrated that *Sr2* in CS(Hope 3B) gave considerable protection against grain weight loss under a rust epidemic. Plants carrying *Sr2* were resistant at all times in the field, but permitted considerable rust development in greenhouse tests. Hence, classifications for *Sr2* were conducted only in the field. The amount of rust development on plants with *Sr2* was variable, and an inheritance study involving homozygous *Sr2Sr2* F_3 lines of Warigo/CS(Hope 3B) and F_4 lines of Gabo/Selkirk, suggested that *Sr2* was modified by alleles at an additional locus in each cross. In Warigo/CS(Hope 3B) and Gabo/Selkirk crosses there was no evidence that the expression

of *Sr2* was affected by alternative alleles at the *Sr6*, *Sr7b* and *Sr17* loci, when strains virulent on plants carrying the resistant alleles were used. Although *Sr23* is detectable in seedlings with all *P. graminis tritici* strains, there was no evidence that it affected adult-plant reaction. Hence, the modifiers of *Sr2* were not known genes for specific resistance. Recessive alleles at a locus completely or very closely linked with *Sr2* were shown to condition pseudo-black chaff development. Since genetic modification of pigmentation and rust reaction were independent, it was possible to select resistant plants relatively free of pigment.

Although Spica does not carry *Sr2*, it showed varying levels of adult-plant resistance compared with W3498 to certain strains with virulence for *Sr7b* and *Sr17* in greenhouse tests. This resistance, possibly determined by alleles at a single locus, was not detected in field studies involving crosses not possessing *Sr2*. However, in the Spica/Hopps cross, it was suggested that the Spica resistance may have additively interacted with *Sr2*.

The adult-plant leaf rust resistance of Hopps, apparently effective against all strains of *P. recondita* with which it has been tested, was shown to be governed by a single dominant allele, which segregated independently of *Sr2*.

A single dominant allele in CS(Hope 3B) conditioned seedling resistance to all strains of *P. recondita* including strains virulent on *Lr14a*. The origin of this resistance is unknown.

Seedling and adult-plant studies indicated that recessive alleles at a locus, designated *Sr12*, were primarily responsible for the adult-plant resistance in Marquillo and

Celebration. *Sr12* was located in the short arm of chromosome 3B. In a Marquillo cross, the dominant allele, *Sr9g*, enhanced the expression of adult-plant resistance attributed to *Sr12*, but alone, *Sr9g* produced no significant adult-plant resistance against the strains present. While Celebration was known to possess *Sr9g*, resistant lines carrying *Sr12* could not be further classified. CS(Thatcher 2B) and CS(Thatcher 3B) were resistant at both the seedling and adult-plant stages, but neither were as resistant as Thatcher. Presumably, an interaction between *Sr9g* and *Sr16* was responsible for the observed adult-plant resistance of CS(Thatcher 2B) while *Sr12*, and possibly *Sr16*, protected CS(Thatcher 3B). A Chinese Spring line carrying *Sr16* alone was susceptible.

Grain weight analyses substantiated adult-plant reactions and confirmed the effectiveness of *Sr12 Sr9g* combinations in comparison with lines carrying either gene alone. Using certain North American cultures it was shown that Celebration probably carries *Sr16*, whereas Marquillo does not. However Marquillo, but not Celebration, possesses dominant alleles for low seedling reaction at a locus closely linked in coupling with *Sr12*.

While the persistently effective *Sr2* resistance was considered to be 'non-specific', *Sr12* was believed to be specific mainly on the basis of the strain-variable seedling reactions associated with its presence.

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APPENDIX 1

1.1 CLASSIFICATION OF F_3 PLANTS IN SEGREGATING FAMILIES FROM THE CROSS HOPPS/YALTA WHEN TESTED IN 1975

Line designation	Resistant	Susceptible	$\chi^2_{1:3}$	P value
1201	3	19	1.64	>0.2
1202	6	19	0.003	>0.95
1203	5	14	0.02	>0.8
1209	7	11	1.85	>0.1
1210	5	12	0.18	>0.6
1216	5	16	0.02	>0.8
1226	4	11	0.04	>0.8
1227	3	21	2.00	>0.1
1231	5	12	0.14	>0.7
1236	3	19	1.52	>0.2
1240	5	10	0.56	>0.4
1242	5	15	0	
1243	6	19	0.01	>0.9
1247	5	18	0.13	>0.7
1249	6	17	0.01	>0.9
1260	5	17	0.06	>0.8
1262	5	15	0	
1263	2	18	2.40	>0.1
1264	7	21	0	
1265	3	11	0.10	>0.7
1267	3	16	0.86	>0.3
1295	9	10	5.07*	>0.02
1297	5	15	0	
1303	5	15	0	
1309	4	17	0.40	>0.5
1310	3	19	1.52	>0.2
1312	6	16	0.06	>0.8
1321	5	21	0.46	>0.4
1325	8	18	0.46	>0.4
1328	2	20	2.97	>0.05
1332	6	10	1.33	>0.2
1341	10	18	1.71	>0.1
1346	4	21	1.08	>0.2
1353	3	13	0.33	>0.5
Total segregation for all families	168	544	0.75	>0.3
Total χ^2 34 df			26.93	>0.8
Heterogeneity χ^2 33 df			26.18	>0.7

1.2 CLASSIFICATION OF F_3 PLANTS IN SEGREGATING FAMILIES FROM THE CROSS GABO/SELKIRK WHEN TESTED IN 1975

Line designation	Resistant	Susceptible	$\chi^2_{1:3}$	P value
831	6	16	0.06	>0.8
850	7	17	0.22	>0.6
852	3	18	1.29	>0.2
857	6	19	0.01	>0.9
872	7	16	0.36	>0.5
875	7	13	1.07	>0.3
881	7	12	1.42	>0.2
885	6	16	0.06	>0.8
889	8	12	2.40	>0.1
896	6	13	1.91	>0.1
900	5	23	1.83	>0.1
902	9	18	3.00	>0.05
903	3	19	1.52	>0.2
911	3	17	0.27	>0.6
913	4	21	1.08	>0.2
917	2	28	5.38*	>0.02
928	7	23	0.04	>0.8
929	7	15	0.55	>0.4
932	7	23	0.04	>0.8
939	10	16	2.51	>0.1
945	3	18	2.71	>0.05
961	8	16	0.89	>0.3
963	6	25	0.53	>0.4
968	7	15	0.55	>0.4
973	10	18	1.71	>0.1
983	7	17	0.22	>0.6
1000	5	12	0.18	>0.6
1002	9	19	0.76	>0.3
1004	4	18	0.55	>0.4
1008	8	12	2.40	>0.1
1009	5	17	0.06	>0.8
1030	9	12	3.57	>0.05
1031	6	21	0.11	>0.7
1032	9	17	1.28	>0.2
1038	6	22	0.19	>0.6
1042	10	18	1.71	>0.1
Total segregation for all families	232	632	1.58	>0.2
Total χ^2 36 df			42.44	>0.2
Heterogeneity χ^2 35 df			40.86	>0.2

1.3 CLASSIFICATION OF F_3 PLANTS IN SEGREGATING FAMILIES FROM THE CROSS W3498/WARIGO WHEN TESTED IN 1975

Line designation	Resistant	Susceptible	$\chi^2_{1:3}$	P value
1858	5	11	0.33	>0.5
1867	3	8	0.03	>0.8
1887	7	18	0.12	>0.7
1898	5	17	0.06	>0.8
1902	5	13	0.08	>0.7
1903	8	16	0.89	>0.3
1918	8	18	0.46	>0.4
1926	5	14	0.02	>0.8
1933	4	9	0.23	>0.6
1940	5	10	0.56	>0.4
1962	6	15	0.14	>0.6
1972	5	12	0.18	>0.6
1981	5	18	0.13	>0.7
1987	5	17	0.06	>0.8
1988	3	14	0.49	>0.4
1990	6	14	0.27	>0.6
1994	5	12	0.18	>0.6
2001	5	10	0.56	>0.4
2002	4	10	0.10	>0.7
2009	5	15	0	
2030	4	14	0.08	>0.7
Total segregation for all families	108	285	1.29	>0.2
Total χ^2 21 df			4.97	>0.99
Heterogeneity χ^2 20 df			3.68	>0.99

APPENDIX 2

FREQUENCY DISTRIBUTIONS OF DISEASE INTENSITY DATA ON PLANTS IN F_3 FAMILIES FROM WARIGO/CS(HOPE 3B)

Disease intensity classes

% disease cover

Line No.	Replication 1					Replication 2					Family disease category
	0-2%	2-5%	5-10%	10-20%	20-30%	0-2%	2-5%	5-10%	10-20%	20-30%	
9436	2	1	13	1	0	1	6	9	12	0	Seg**
9437	5	14	11	4	0	9	9	2	4	0	Seg
9438	0	0	2	30	4	0	0	3	20	4	HH**
9439	0	9	12	2	0	2	4	5	9	1	Seg
9440	3	36	4	0	0	5	17	6	0	0	**HL**
9441	2	4	3	7	1	2	6	5	9	2	Seg
9442*	16	9	5	3	0	0	9	10	14	6	Seg
9443	2	14	4	0	0	8	11	0	0	0	HL**
9444	0	0	6	12	2	0	0	5	22	2	HH
9445	4	16	8	7	0	0	9	6	9	1	Seg
9446	7	15	1	0	0	2	18	0	0	0	HL
9447	3	9	10	3	0	0	9	12	2	0	Seg
9448	0	13	9	4	0	1	10	8	6	1	Seg
9449*	2	14	1	0	0	10	10	6	0	0	HL
9450	0	0	9	16	0	0	6	10	6	0	Seg
9451	0	5	13	12	2	0	2	6	15	7	Seg
9452	0	0	6	17	1	0	0	3	13	6	HH
9453	0	0	5	8	4	0	0	6	19	1	HH
9454	4	16	9	2	0	13	4	5	1	0	Seg
9455	0	0	1	16	1	0	0	2	16	0	HH
9456	0	9	12	0	0	3	10	17	2	0	Seg
9457	7	12	0	0	0	8	9	0	0	0	HL
9458	0	6	8	5	2	0	4	10	7	0	Seg
9459	0	1	8	7	0	1	3	5	15	5	Seg
9460	6	19	0	0	0	11	19	2	0	0	HL
9461	0	0	11	4	0	0	7	7	8	0	Seg
9462	0	6	10	8	0	2	9	14	6	0	Seg

APPENDIX 2 (CONT)

Line No.	Replication 1					Replication 2					Family disease category
	0-2%	2-5%	5-10%	10-20%	20-30%	0-2%	2-5%	5-10%	10-20%	20-30%	
9463	0	0	5	8	0	0	0	1	21	13	HH
9464	7	6	2	0	0	5	19	6	0	0	**HL**
9465	4	13	12	0	0	1	4	16	6	0	Seg
9466	7	5	6	0	0	5	13	6	0	0	**HL**
9467	6	12	0	0	0	7	16	0	0	0	HL
9468	5	12	10	4	0	1	8	10	4	0	Seg
9469	5	8	2	3	0	4	6	7	10	0	Seg
9470	0	6	14	8	0	4	12	10	4	0	Seg
9471	4	4	2	26	0	3	18	5	2	0	Seg
9472	0	0	11	24	0	0	0	17	17	0	HH
9473	7	10	7	3	0	3	11	10	3	0	Seg
9474	5	6	12	1	0	1	10	7	2	0	Seg
9475	2	5	9	3	0	0	6	15	3	1	Seg
9476	4	26	3	0	0	12	12	1	0	0	HL
9477	0	0	7	9	4	0	0	10	22	2	HH
9478	2	10	15	6	0	1	3	11	10	4	Seg
9479	30	3	0	0	0	30	0	0	0	0	HL
9480*	12	8	6	0	0	6	12	0	0	0	HL
9481	24	1	0	0	0	12	2	0	0	0	HL
9482	0	7	25	4	0	1	6	15	6	0	Seg
9483	23	2	1	0	0	22	7	0	0	0	HL
9484	12	12	1	0	0	16	4	0	0	0	HL
9485	1	14	2	7	0	7	16	3	4	0	Seg
9486*	3	19	2	0	0	11	2	3	0	0	HL
9487	1	8	13	4	0	0	4	4	13	0	Seg
9488	0	0	4	24	0	0	0	9	7	0	HH
9489	0	2	10	6	0	6	11	5	3	0	Seg
9490	0	4	10	8	0	1	6	10	2	2	Seg
9491	0	0	2	18	0	0	0	4	23	7	HH
9492	0	0	4	15	0	0	0	0	14	4	HH
9493	11	9	0	0	0	19	9	0	0	0	HL
9494	5	16	2	0	0	1	30	1	0	0	HL
9495	0	15	0	0	0	5	18	1	0	0	HL
9496	6	11	0	0	0	23	3	0	0	0	HL

APPENDIX 2 (CONT

Line No.	Replication 1					Replication 2					Family disease category
	0-2%	2-5%	5-10%	10-20%	20-30%	0-2%	2-5%	5-10%	10-20%	20-30%	
9497	0	0	3	8	4	0	0	1	2	13	HH
9498	0	0	7	10	0	0	0	5	12	2	HH
9499	5	8	2	0	0	3	13	9	4	0	Seg
9500	0	1	14	4	0	0	0	5	23	2	Seg
9501	0	2	12	4	0	1	6	4	4	0	Seg
9502	12	7	6	0	0	1	3	4	14	0	Seg
9503	1	7	11	2	0	1	5	8	12	0	Seg
9504	7	16	6	6	0	2	3	7	3	0	Seg
9505	0	6	20	3	0	0	4	2	15	3	Seg
9506	0	0	12	10	0	0	0	3	11	11	HH
9507	11	8	0	0	0	2	19	1	0	0	HL
9508	0	13	8	9	0	1	13	13	14	0	Seg
9509	1	4	7	3	0	0	2	13	8	0	Seg
9510	6	13	1	0	0	4	18	2	0	0	HL
9511	15	8	6	0	0	12	14	0	0	0	HL
9512	7	12	3	0	0	7	17	0	0	0	HL
9513	0	3	3	16	4	4	4	11	8	1	Seg
9514	0	5	17	7	0	0	1	8	7	1	Seg
9515	7	20	2	0	0	9	11	0	0	0	HL
9516	0	0	2	6	17	0	0	0	19	8	HH
9517	7	9	6	2	4	0	7	8	6	3	Seg
9518	0	0	2	7	3	0	0	5	6	14	HH
9519	12	2	0	0	0	5	12	3	0	0	HL
9521	3	8	4	0	0	0	3	9	6	0	Seg
9522	6	14	2	0	0	12	12	0	0	0	HL
9523	0	0	5	7	0	0	0	1	5	16	HH
9524	7	19	2	0	0	21	5	0	0	0	HL
9525	0	4	14	2	0	1	3	7	7	0	Seg
9526						0	0	5	10	5	HH
9527	0	6	3	8	0	0	4	3	3	0	Seg
Warigo	29	8	0	0	0	27	3	0	0	0	L
CS(Hope 3B)	0	0	5	25	7	0	0	16	20	6	H

* Lines in which replicates were not consistent. ** HL: homozygous low, seg: segregating, HH: homozygous high

APPENDIX 3

FREQUENCIES OF F_3 SEEDLINGS IN INFECTION TYPE CLASSES IN WARIGO/CS(HOPE 3B) WHEN TESTED WITH 122-Anz-1,2,3

F_2 line designation	Low reaction (it";")	High reaction (it"3+")	$\chi^2_{3:1}$	P
9437	14	10	3.56	>0.05
9441	14	2	1.33	>0.2
9442	15	4	0.16	>0.6
9443	18	9	1.00	>0.3
9444	10	5	0.47	>0.3
9449	11	5	0.33	>0.5
9450	19	5	0.22	>0.6
9451	20	3	1.75	>0.1
9459	12	2	0.86	>0.3
9464	18	7	0.12	>0.7
9473	9	8	4.23	<0.05
9474	20	3	1.75	>0.1
9475	13	4	0.02	>0.8
9484	18	4	0.55	>0.4
9486	20	6	0.05	>0.8
9487	16	3	0.86	>0.3
9488	15	4	0.16	>0.6
9496	21	5	0.46	>0.4
9497	6	10	12.00	<0.01
9498	18	3	1.29	>0.2
9499	26	11	0.44	>0.5
9500	21	4	1.08	>0.3
9502	18	8	0.46	>0.4
9505	20	5	0.33	>0.5
9506	19	3	1.51	>0.2
9507	19	8	0.31	>0.5
9508	17	8	0.33	>0.5
9512	12	2	0.86	>0.3
9516	14	3	0.49	>0.4
Total	473	154	0.06	>0.95
Total χ^2 over all families	29 df		37.29	>0.1
Heterogeneity χ^2	28 df		37.23*	>0.1

* The deletion of family 9497 reduces this value to 25.23 which for 27 d.f. has P>0.5.

APPENDIX 4

FREQUENCIES OF F_3 SEEDLINGS IN INFECTION TYPE CLASSES IN CELEBRATION/W3498 WHEN TESTED WITH 126-Anz-6,7,11 OR 34-Anz-2,4,5,7,11

F_2 line designation	Strain	Low reaction (it";x") ;x-	High reaction (it"3+,3+") 3+	$\chi^2_{1:3}$	P
Celebration W3498					
6706	126-Anz-6,7,11	11	17	3.05	>0.05
6712		8	12	2.40	>0.1
6715		4	17	0.42	>0.5
6716		6	23	0.29	>0.5
6717		9	19	0.38	>0.5
6725		8	11	2.96	>0.05
6729		3	23	2.49	>0.1
6731*		1	18	3.96	<0.05
6732		7	17	0.22	>0.6
6735		6	19	0.01	>0.9
6736		4	14	0.07	>0.7
6737		4	24	1.72	>0.1
6742		7	16	0.36	>0.5
6743		6	24	0.40	>0.5
6745		8	17	0.65	>0.4
6746		3	20	1.76	>0.1
6748		7	18	0.12	>0.7
6750		8	18	0.46	>0.4
Total segregation for all families		110	327	0.01	>0.9
Total χ^2 18 df				21.72	>0.2
Heterogeneity χ^2 17 df				21.71	>0.2

* line 6731 was repeated and the segregation classification was confirmed.

APPENDIX 4 (CONT)

Celebration		(it"x+3") x+3	(it"33+,3+") 3+		
W3498					
6759	34-Anz-2,4,5,7,11	3	13	0.33	>0.5
6760		8	22	0.04	>0.8
6762		5	12	0.18	>0.6
6764		5	18	0.13	>0.7
6769		7	25	0.17	>0.6
6770		9	16	1.61	>0.2
6788		5	22	0.61	>0.4
6790		3	16	0.22	>0.6
6810		5	12	0.18	>0.6
Total segregation for all families		50	156	0.92	>0.3
Total χ^2 9 df				3.46	>0.9
Heterogeneity χ^2 8 df				2.54	>0.95

APPENDIX 5

FREQUENCIES OF F_3 SEEDLINGS IN INFECTION TYPE CLASSES IN CELEBRATION/W3498 WHEN TESTED WITH 326-Anz-1,2,3,5,6

Celebration it";x2-": W3498 it"3+"

Line designation	F_2 genotype	Infection type classes				$\chi^2_{3:9:1:3}$	P
	Sr12sr12Sr9gsr9g	"x2-,;x2"	"2-2,2"	"x+3,x3"	"33+,3+"		
6760		6	20	3	6	0.40	>0.9
6762		3	8	2	3	1.11	>0.7
6764		4	16	1	4	0.67	>0.8
6770		5	16	0	3	2.77	>0.3
6788		4	13	1	5	0.27	>0.95
6807		1	9	3	4	5.27	>0.1
Total segregation for all families		23	82	10	25	0.78	>0.8
Total χ^2 18 df						10.49	>0.9
Heterogeneity χ^2 17 df						9.71	>0.9
	Sr12Sr12Sr9gsr9g					$\chi^2_{3:1}$	
6766		13		10		4.19	<0.05
6776		14		3		0.49	>0.4
6789		14		3		0.49	>0.4
6791		14		7		0.79	>0.3
6794		17		4		0.40	>0.5
6798		20		4		0.89	>0.3
Total segregation for all families		92		31		0.002	>0.95
Total χ^2 6 df						7.25	>0.2
Heterogeneity χ^2 5 df						7.25	>0.2

APPENDIX 5 (CONT)

Line designation	F_2 genotype	Infection type classes			$\chi^2_{3:1}$	P
	sr12sr12Sr9gsr9g "x2-,;x2"	"2-2,2"	"x3,x+3"	"33+,3+"		
6773		16		4	0.27	>0.6
6792		19		4	0.71	>0.3
6804		6		7	5.77	<0.01
Total segregation for all families		41		15	0.09	>0.7
Total χ^2 3 df					6.75	>0.05
Heterogeneity χ^2 2 df					6.66	<0.02

APPENDIX 6

FREQUENCIES OF F_3 SEEDLINGS IN INFECTION TYPE CLASSES IN CELEBRATION/W3498 WHEN TESTED WITH CULTURE 56-E-1

Celebration it";x-": W3498 it"3+"

Line designation	F_2 genotype	Infection type classes				$\chi^2_{3:9:1:3}$	P
	Sr12sr12Sr16sr16	";x-,;x"	"2-,2"	"x3"	"3+"		
6704		6	18	2	4	0.58	>0.9
6712		8	13	4	5	4.37	>0.2
6713		13	15	1	4	9.50	<0.05
6715		7	15	1	6	0.98	>0.8
6716		5	15	4	6	2.71	>0.4
6722		7	22	2	7	0.07	>0.99
6725		3	12	2	4	0.74	>0.8
6729		5	14	3	4	1.50	>0.6
6730		6	19	2	5	0.26	>0.95
6760		6	21	3	3	2.39	>0.4
6799		4	12	2	4	0.30	>0.95
6806		3	16	4	6	3.80	>0.2
6812		3	12	2	4	0.74	>0.8
Total segregation for all families		76	204	32	62	4.81	>0.1
Total χ^2 39 df						27.94	>0.9
Heterogeneity χ^2 38 df						23.13	>0.95

APPENDIX 6 (CONT)

Line designation	F_2 genotype	Infection type classes				$\chi^2_{1:3}$	P
	Sr12sr12Sr16Sr16	";x-,;x"	"2-,2"	"x3"	"3+"		
6708		6	16			1.91	>0.1
6769		4	20			0.89	>0.3
6788		4	16			0.27	>0.6
6800		7	16			0.36	>0.5
6801		7	20			0.01	>0.9
Total segregation for all families		28	88			0.01	>0.9
Total χ^2 5 df						3.44	>0.5
Heterogeneity χ^2 4 df						3.43	>0.4
	Sr12Sr12Sr16sr16					$\chi^2_{3:1}$	
6702		26		8		0.04	>0.8
6704		14		4		0.08	>0.7
6733		18		4		0.55	>0.4
6738		10		5		0.56	>0.4
6747		17		6		0.02	>0.8
6791		13		4		0.02	>0.8
6811		14		4		0.08	>0.7
Total segregation for all families		112		35		0.11	>0.9
Total χ^2 7 df						1.35	>0.98
Heterogeneity χ^2 6 df						1.24	>0.98

APPENDIX 6 (CONT)

Line designation	F_2 genotype	Infection type classes			$\chi^2_{3:1}$	P
	sr12sr12Sr16sr16	";x-,;x"	"2-,2"	"x3"	"3+"	
6714			23		6	0.29	>0.5
6720			23		8	0.01	>0.9
6721			20		6	0.05	>0.8
6749			16		3	0.86	>0.3
Total segregation for all families			82		23	0.14	>0.7
Tatal χ^2 4 df						1.21	>0.8
Heterogeneity χ^2 3 df						1.07	>0.7
	Sr12sr12sr16sr16					$\chi^2_{1:3}$	
6706				9	23	0.17	>0.6
6717				5	16	0.02	>0.8
6740				3	12	0.20	>0.6
6743				7	20	0.01	>0.9
6746				4	12	0	
Total segregation for all families						0.01	>0.9
Total χ^2 5 df						0.4	>0.99
Heterogeneity χ^2 4 df						0.39	>0.98

APPENDIX

CLASSIFICATION OF F_3 SEEDLINGS IN SEGREGATING FAMILIES OF MARQUILLO/W3498 WHEN TESTED WITH 54-Anz-2,4,5,7,11 AND 126-Anz-6,7,11

Line designation	54-Anz-2,4,5,7,11 Infection types		$\chi^2_{1:3}$	P	126-Anz-6,7,11 Infection types		$\chi^2_{1:3}$	P
	(it";0,;x=")	(it"33+,3+")			(it";x=,;x-,;x")	(it"33+,3+")		
Marquillo	";0"				";x="			
W3498		3+				3+		
11375	3	14	0.39	>0.5				
11376	5	16	0.02	>0.8	2	19	2.72	>0.05
11377	4	12	-	-	6	15	0.40	>0.5
11388					7	18	0.09	>0.7
11389	4	20	0.88	>0.3	7	15	0.55	>0.4
11390	10	12	5.00	<0.05	4	11	0.03	>0.8
11391					5	12	0.17	>0.6
11392	7	13	1.18	>0.2	9	19	0.75	>0.3
11402	9	9	6.00	<0.05	6	13	0.44	>0.5
11406	6	22	0.16	>0.6	9	16	1.61	>0.2
11408					14	31	0.90	>0.3
11417	4	12	-	-				
11440	9	14	1.80	>0.1				
11441	4	8	0.44	>0.5				
11442	8	19	0.31	>0.5				
11445	8	12	2.40	>0.1	2	16	1.85	>0.1
11449					9	18	0.99	>0.3
11452	7	9	3.00	>0.05	7	21	-	-
11455	4	19	0.69	>0.4	7	27	0.36	>0.5
11456	4	13	0.03	>0.8	9	28	0.12	>0.7
11463	6	11	0.94	>0.3				
11468	6	14	0.27	>0.6	12	27	0.68	>0.4
11470	4	13	0.03	>0.8	5	31	2.37	>0.1
11480	1	15	3.00	>0.05				
11486	3	19	1.52	>0.2				
11488	6	12	0.67	>0.4				
11490	6	12	0.67	>0.4				

APPENDIX 7 (CON'

Line designation	34-Anz-2,4,5,7,11 Infection types (it";0,;x=")	(it"33+,3+")	$\chi^2_{1:3}$	P	126-Anz-6,7,11 Infection types (it";x=,;x-,;x")	(it"33+,3+")	$\chi^2_{1:3}$	P
11495	8	14	1.52	>0.2				
11498	4	16	0.27	>0.6				
11501	4	17	0.40	>0.5				
11504	8	12	2.40	>0.1				
11514	7	15	0.64	>0.4				
11517	4	16	0.27	>0.6				
11521	2	12	0.85	>0.3				
11522	4	14	0.07	>0.7				
11525	3	15	1.17	>0.2				
11530	3	15	1.17	>0.2				
11532	6	13	0.34	>0.5				
Total segregation for all families	181	479	2.07	>0.1	120	337	0.39	>0.5
Total χ^2 34 df			38.50	>0.2	17 df		14.03	>0.5
Heterogeneity χ^2 33 df			36.43	>0.2	16 df		13.64	>0.5