THE UNIVERSITY OF
SYDNEY

HOST:PATHOGEN STUDIES OF WHEAT STRIPE RUST IN AUSTRALIA

by

COLIN ROSS WELLINGS, M.Sc.Agr.

A thesis submitted in fulfilment of
the requirements for the degree of
Doctor of Philosophy

Department of Agricultural
Genetics and Biometry

University of Sydney

March, 1986

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, is original and contains no material previously published or written by another person, except where due reference is made in the text.

ACKNOWLEDGEMENTS

It is with sincere appreciation that I acknowledge the professional skills, critical appraisal and continuing enthusiasm of my supervisor, Dr. R.A. McIntosh, during the course of these studies. My thanks are extended to Mr. J.D. Oates for supervising the construction of the stripe rust glasshouse and for his support and encouragement. I thank Mr. R.M. Pfeiffer for his valuable technical assistance.

I particularly wish to acknowledge the late Emeritus Professor I.A. Watson for critical comments on the final draft, and for the inspiration he engendered in the study of the cereal rusts. I am grateful to Dr. N.H. Luig and Dr. T.T. The for helpful discussions and to Mr. D.J.S. Gow for the preparation of photographic plates.

I also wish to thank Ms. J. Kaldor for help with statistical analysis, my wife Sue for diligently preparing manuscript drafts, Mrs. B. Gibson for dedication and skill in typing the final manuscript, and to the staff of the Plant Breeding Institute for their co-operation. The glasshouse facilities for stripe rust studies were provided through a grant to the Plant Breeding Institute by the Australian Wheat Industry Research Council.

I especially wish to thank my family, and in particular my wife and children, for their encouragement, support and enduring patience throughout these studies. Finally, I am grateful to God for the privilege and strength to observe and record his creation:

"His invisible attributes, that is to say
his power and deity, have been visible,
ever since the world began, to the eye of
reason, in the things he has made."

Romans 1,20

SUMMARY

Stripe rust of wheat was first detected in Australia in October, 1979. Although systemic fungicides offered a convenient means of disease control, consistent economic returns could not be demonstrated. It was apparent that under Australian conditions, host resistance was the best means of reducing the probability of epidemic development and associated crop losses. Studies undertaken with the pathogen, the host, and the host:pathogen interaction, provided a basis for the following conclusions.

1. Stripe rust, appearing in the eastern Australian wheat belt in each season from 1979 to 1985, is now regarded as endemic to the region. However, the disease has not been recorded in Western Australia.

2. The pathogens causing stripe rust in Australia were *Puccinia striiformis f.sp. tritici,* which mainly infected wheat, and *P. striiformis f.sp. dactylidis,* which exclusively infected cocksfoot grass. There was no evidence to suggest that *P. striiformis f.sp. hordei* and *P. striiformis f.sp. poae* are present in Australia.

3. The host range of *P. striiformis f.sp. tritici* included wheat, triticale, barley, cereal rye and some grasses in the Pooideae family.

4. The occurrence of different pathotypes in various locations in the early stages of epidemic development indicated that the pathogen over-summered at independent locations throughout the wheat belt. Southern New South Wales and Victoria were more regularly affected by the disease compared to other regions. However, the onset and development of epidemics in winter resulted in severe spring epidemics over large areas of eastern Australia.

5. Annual pathotype surveys in eastern Australia indicated that four variants of the pathogen developed from the initial single pathotype, 104 E137 A-, introduced in 1979. These pathotypes were presumed to have arisen as a consequence of mutations involving single genes for increased

virulence.

6. Pathogenic variation with respect to cultivar Avocet has not been described previously. The resistance of Avocet was tentatively designated *YrA*, and pathotypes virulent and avirulent with respect to *YrA* were designated as A+ and A- respectively. An addition to the accepted international system of pathotype nomenclature was proposed to accommodate this previously unrecognised variation.

7. Virulence with respect to *Yr5*, which was not present in commercial cultivars, was recorded at very low frequency. Although this resistance gene has been described, it is not represented in the international system of pathotype nomenclature. It was proposed that the differential tester for *Yr5, i.e. T. spelta album*, be added to the world set.

8. The pathogen found on wheat in New Zealand in 1980 was identical to that occurring in Australia. It was presumed to have been wind-borne from Australia. A second pathotype, virulent for *Yr7*, was identified in 1982 and has not been detected in Australia. This pathotype is believed to have arisen as a consequence of mutation in the original introduction.

9. The frequency and distribution of particular pathotypes in eastern Australia were related to cultivars with the corresponding genes for host resistance. The close association with a particular cultivar(s) was especially evident in the early spread of a new pathotype.

10. The pathotypes detected in Australia and New Zealand were used in experiments to identify the presence of *Yr6*, *Yr7*, *YrA* and the combination *Yr6 + YrA* in host stocks. The reported close linkage between *Yr7* and *Sr9g* was used to confirm the presence of *Yr7* in certain cultivars. Inheritance studies confirmed postulations based on pathotype tests and enabled the detection of the combination *Yr6 + Yr7* in Pavon 76 and Hermosillo 77.

11. *Yr7* was inherited as a single dominant gene and produced seedling infection types displaying little

variation with changes in environment. This gene was detected in Thatcher and several Canadian and U.S.A. wheats derived from Thatcher. *Yr7* showed linkage of 17.75% ± 5.20% with *Lr13*.

12. *Yr6* was inherited as a recessive gene. The resistant infection type responses produced by cultivars possessing *Yr6* were intermediate at 10C and low at 18C. This gene was common in Brazilian, Mexican and Australian wheats reflecting similarities in pedigree.

13. The *YrA* resistance was inherited as two complementary dominant genes. Stocks possessing *YrA* produced high infection types at low light intensities when tested with avirulent pathotypes. Under these conditions, this resistance could not be recognised. The *YrA* resistance was present in Mexican, Australian and one Indian wheat all of which were derived from material distributed by CIMMYT.

14. The combination *Yr6 + Yr7* was present in two Mexican wheats and possibly two Australian wheats. The combination *Yr6 + YrA* was found in one Mexican wheat and one selection of an Australian wheat derived from CIMMYT material.

15. On the basis of infection types, pathotype tests and genetic studies, the single dominant resistance factor of Nacozari 76 appeared to be previously uncatalogued.

16. Preliminary studies of triticale indicated that single dominant genes or a combination of a dominant gene and a recessive gene conferred stripe rust resistance in three cultivars.

17. Adult-plant resistance, in addition to resistances conferred by seedling genes, was evident in most wheats.

CONTENTS

1 INTRODUCTION

Wheat, and its associated pests and diseases, have been principal determinants in the often fluctuating history of man's civilisation (Baker, 1970). In the contemporary world scene, wheat is a staple food for nearly 40 percent of the population (Wiese, 1977). Most of the production occurs in the northern hemisphere where the major areas under cultivation are North America, Europe, the U.S.S.R., China and the Indian sub-continent.

Australian wheat production has an important role in the national economy and in world wheat trade. Statistics from the Australian Wheat Board (Anonymous, 1985) indicate that annual production over the five year period from 1979 to 1984 averaged 14.8 million tonnes harvested from 11.7 million hectares. A large proportion of Australia's grain is sold on the export market where the Middle East, U.S.S.R., China and Japan are the main purchasers. World trading in wheat approaches 100 million tonnes with the U.S.A. accounting for approximately 40 percent and Australia, Canada and the European Economic Community each contributing approximately 15 percent. In the Australian economy, wheat is a significant export earner, returning $2.9 billion in 1984-85.

Wheat belongs to the graminaceous genus *Triticum* which is composed of a polyploid series including wild and cultivated types. The diploid einkorn wheats, *T. monococcum* L., have 14 chromosomes comprising the AA genome. These wheats are found in the eastern Mediterranean, but are of negligible economic importance. Wild emmer, *T. turgidum* L., an allotetraploid with 28 chromosomes comprising the AABB genomes, is presumed to have originated by chromosome doubling of a hybrid (AB) between einkorn (AA) and an unknown species of *Aegilops* (BB). Durum is an important cultivated form of *T. turgidum* which produces semolina particularly suited to the manufacture of pasta products. Hexaploid bread wheat, (*T. aestivum* L.) with 42 chromosomes comprising the AABBDD genomes, is believed to have arisen as a chromosomally doubled hybrid

involving cultivated emmer (AABB) and goat grass, *Aegilops squarrosa* L. (DD) (McIntosh, 1976). Common bread wheat is the most widely cultivated form, and is grown on all continents except Antarctica (Baker, 1970).

Nearly 200 parasitic and non-parasitic diseases of wheat have been described, and approximately 50 are of economic importance. The rusts are an economically significant group of parasitic diseases of wheat. During the 1960's, the rusts were conservatively estimated to have reduced North American wheat yields by over 1 million tonnes annually (Wiese, 1977). Severe epidemics of stem rust in the U.S.A. in 1935 resulted in statewide yield losses of over 50 percent (Roelfs, 1978). More recently, the stem rust epidemic in eastern Australia in 1973 caused an estimated economic loss of $100-200 million (Anonymous, 1975). In Pakistan, a severe leaf rust epidemic in 1978 resulted in an estimated national loss of 830,000 tons valued at $US86 million (Hussain *et al.*, 1980). Green and Campbell (1979) calculated that resistant wheat cultivars sown in the stem rust liable areas of Canada provide crop protection valued at an estimated $217 million annually. In contrast, the annual cost of the main breeding program responsible for producing stem rust resistant cultivars was estimated to be $650,000. The general importance of the rusts was gauged by Wiese (1977) who noted that the majority of wheat disease literature was devoted to these diseases.

Stripe rust is an economically important disease in world wheat growing areas which are cool to mild and moist (Johnson *et al.*, 1967). Under these conditions, stripe rust is of greater incidence than either stem rust or leaf rust (Doling, 1967) and can reduce yields in certain cultivars by 50 percent or more (Stakman and Harrar, 1957). Roelfs (1978) reported that severe stripe rust epidemics in the western states of the U.S.A. in 1960 and 1961 resulted in crop losses of 20 to 75 percent.

Both genetic and chemical means, as well as strategies which combine both, have been used to control stripe rust. The availability of fungicides, such as triadimefon, which are systemic and residual in mode of action (Scheinpflug *et al.*, 1978) has made chemical control of this disease an attractive strategy. Under European conditions of high input, high return cereal production, chemical control is an effective, economic and routine operation (Brent, 1983). In situations of lower yield potential, chemical control has been occasionally used as an emergency measure in epidemics of unusually high intensity and severity; for example, the 1981 stripe rust epidemic in the Pacific Northwest U.S.A. (Line, 1983a). Increasing social awareness of potential problems associated with continued large-scale chemical application (Zadoks, 1979) and the possible occurrence of fungicide-resistant pathotypes (Wolfe, 1971) suggests that chemical control alone, even in high yielding situations, should not be a long term strategy.

The achievement of genetic control of stripe rust has been the goal of European wheat breeding groups since the early twentieth century. Due to rapid adjustments by pathogen populations, host resistance has not remained effective when based on single genes or on simple gene combinations (Johnson *et al.*, 1967). Such changes have led to a search for resistances which remain effective against all pathogen isolates, *i.e.* horizontal resistance *sensu* van der Plank (1968). However, Zadoks (1979) noted that despite advances in the elucidation of phenomena associated with horizontal resistance, such as partial resistance (Parlevliet, 1979), there have been no cultivars specifically developed and released with this reported type of resistance. Johnson (1978) proposed the concept of durable resistance to describe resistance which has remained effective after having been widely exposed to pathogen populations over considerable periods of time. Breeding strategies to incorporate sources of durable resistance to stripe rust into commercial wheats have been adopted by the Plant Breeding

Institute, Cambridge, England (Johnson, 1983a).

In Australia, stem rust and leaf rust of wheat have probably been present since the early days of colonisation (Wellings and Luig, 1984). The first record of stripe rust in Australia in October 1979 (O'Brien *et al.*, 1980) was met with concern among agricultural research and administrative personnel. A meeting of the National Wheat Rust Control Committee was convened in November, 1979, and an urgent request for funds to the Wheat Industry Research Council was made to allow construction of suitable facilities for the study of stripe rust. Approval was given in a joint grant to the N.S.W. Department of Agriculture and The University of Sydney (Watson and Butler, 1984). The author was seconded by the N.S.W. Department of Agriculture to The University of Sydney, Plant Breeding Institute, Castle Hill, in order to undertake studies on wheat stripe rust.

The results presented in this thesis represent the first host:pathogen studies involving wheat stripe rust in Australia. The first objective was to identify and monitor the pathotypes occurring in wheat-growing areas. Pathotype surveys are prerequisite to the selection of the most appropriate cultures for screening host materials and for predicting the response of cultivars in the field.

The second objective was to determine the genetic basis of resistance to stripe rust in Australian and exotic wheats. This especially included wheats of CIMMYT[1] origin as breeding programs in eastern Australia have incorporated CIMMYT-distributed germplasm since the early 1960's (Syme, 1983). Due to initial apprehension that the pathogen might not survive in Australia, and to subsequent restrictions placed on field work with stripe rust, the initial studies of host resistance concentrated on resistances which could be detected in glasshouse tests.

---

[1]Centro Internacional de Mejoramiento de Maiz Trigo (International Maize and Wheat Improvement Centre) Londres 40, Mexico, 6 D.F., Mexico

2 LITERATURE REVIEW

2.1 DISTRIBUTION AND ECONOMIC IMPORTANCE OF STRIPE RUST

The rust diseases have caused great concern to cereal cropping enterprises since the earliest recorded history. Reference to rust epidemics are found in the Mosaic books of the Old Testament, and in the writings of early scholars (Large, 1940). Rust epidemics reported in England in 1725 and Sweden in 1794, may have been due to stripe rust (Chester, 1946).

The incidence of stripe rust in wheat-growing areas of the world is correlated with prevailing environmental conditions, particularly mean temperatures of less than 20C and ample moisture. Hence this disease is considered to be of major importance in northern Europe (Zadoks, 1979), the Andean region of South America (Stakman and Harrar, 1957), north and north-west provinces of China (Johnson and Beemer, 1977), the northern hills and plains of India (Joshi *et al.*, 1976), Pakistan (Hassan *et al.*, 1979) and Japan (Leonard and Martin, 1967). In areas where climate is highly variable, the seasonal incidence of stripe rust appears to be more sporadic. Such areas include the western wheat areas of North America (Leonard and Martin, 1967) extending from California to Washington State, and in northern Canada and Alaska (Stakman and Harrar, 1957). Traditionally dry Mediterranean climates, as experienced in North Africa, Egypt and other near Eastern countries, have reported stripe rust epidemics in irrigated crops grown in winter (Zadoks, 1979).

Disease incidence is also related to the susceptibility of cultivars in common use. Thus in 1978, a combination of a long, cool spring and the widespread use of susceptible genotypes contributed to a severe epidemic in Italy where stripe rust is usually considered a minor disease (Vallega and Zitelli, 1979).

Until recently, Australia was the only continent remaining free of stripe rust. The combination of a suitable late winter/spring environment and the availability

of susceptible hosts, particularly cultivars Zenith and possibly Egret and Halberd, ensured rapid spread of the disease following its appearance in 1979 (McIntosh and Wellings, 1986). Subsequent re-appearance in each season has established stripe rust as an endemic disease in the eastern Australian wheat belt.

The principal effect of stripe rust epidemics is a reduction in yield and quality of grain. Initial studies undertaken by Bever (1937) in glasshouse experiments using a susceptible spring wheat cultivar indicated that yield losses could be as high as 65 per cent due to reductions in dry matter production, root growth, plant height, size and number of heads, and size and number of grain. These effects were more pronounced with infection at the seedling stage, although infection at anthesis was also associated with reduced root weight as well as yield loss. Water uptake was retarded in rusted plants, although water loss per unit area was greater than from disease-free controls. The response of reduced root growth, following infection over a range of growth stages, was confirmed by Martin and Hendrix (1967). Further glasshouse studies by Doodson *et al.* (1964) showed that yield losses due to leaf infection were twice those due to ear infection.

Field experiments on a range of cultivars have shown yield losses varying from 30 to 90 per cent following early infection (Kaidash *et al.*, 1976). The appearance of a new strain virulent for the adult-plant resistance in Joss Cambier caused yield losses in that cultivar estimated at 34 per cent (Mundy, 1973). Using backcross derivatives of Norin 10/Brevor// Burt, Allen *et al.* (1963) classified resistant and susceptible lines within plant height groups. Yield losses as high as 82 per cent in the short statured group suggested a greater vulnerability to stripe rust among the semi-dwarf genotypes. Doling and Doodson (1968) determined that yield losses ranged from eight to twenty per cent in both spring and winter wheat cultivars under field conditions in England.

It is evident, therefore, that yield losses due to stripe rust in the field will vary with cultivar response, pathotype of the pathogen, genetic background of the host, time of initial infection, and factors determining epidemic development, particularly temperature, moisture (Kaidash *et al.*, 1976) and crop nutrition (Russell, 1978).

Losses due to stripe rust in wheat are generally less than those attributed to stem rust (caused by *Puccinia graminis* Pers. *f.sp. tritici* Eriks and Henn.) which can cause widespread crop failure in particular years (Watson and Butler, 1984). However, the more frequent incidence of stripe rust in European and Mediterranean climates, combined with its capacity to cause significant losses, ensures that this disease will continue to be a potentially major limiting factor to world wheat production.

2.2 THEORETICAL BASIS FOR HOST-PARASITE STUDIES - THE GENE-FOR-GENE RELATIONSHIP

The recognition and exploitation of host resistances are the principal means of achieving control of diseases caused by obligate plant pathogens; this is particularly true of the rust diseases of temperate cereals. The theoretical framework in which the interactions of host and pathogen are studied was gradually elucidated during the first half of this century.

Biffen (1905) demonstrated that the resistance of cultivar Rivet to the wheat stripe rust fungus, *Puccinia striiformis* Westend., was inherited as a single recessive factor. J.H. Craigie, in 1927 (McIntosh and Watson, 1982), showed that sexual recombination occurs at the pycnial stage of the rust fungus life cycle, thus allowing the study of the inheritance of pathogenicity in the pathogen. However, it was not until Flor (1942) published results of his work with flax rust (*Linum usitatissimum*.L:*Melampsora lini* L. (Ehrenb) Lèv.), that concurrent inheritance studies in host and pathogen were undertaken. From this work, he concluded that "the range of pathogenicity of a physiologic

race is determined by pathogenic factors specific for each resistance factor possessed by the host" (Flor, 1942). Further work confirmed these early conclusions and resulted in the classic description of the gene-for-gene hypothesis: "for each gene conditioning rust reaction in the host there is a specific gene conditioning pathogenicity in the parasite" (Flor, 1956). In the flax:*M. lini* relationship, host resistance was dominant and pathogen virulence was recessive (Flor, 1971).

The general application of the gene-for-gene model is indicated by Day (1974) who listed 26 host-parasite interactions involving agricultural crop plants and fungi, nematodes, insects, bacteria and viruses, for which the hypothesis had been demonstrated or implied. However, some workers have concluded that host resistances which are polygenic and have not been overcome by the pathogen do not conform to the hypothesis. McIntosh and Watson (1982) noted that in many instances reported cases of non-specific host resistances were confounded by the concurrent presence of combinations of specific resistance factors. Day (1974) considered that polygenic control of pathogenicity was possible, although currently not confirmed, as quantitative characters in fungi have been shown to be polygenic in nature. Ellingboe (1975) stated that "non-specific resistance is that resistance which has not yet been shown to be specific", thus implying that an apparent lack of virulence for a host resistance does not exclude its discovery at a future date.

Thus the gene-for-gene hypothesis has wide application in the study of host-pathogen interactions. It allows the postulation of host and pathogen genotypes in the absence of formal genetic studies. Host resistance surveys may be conducted using standard pathogen cultures of known pathogenicity. Conversely, pathogenicity surveys may be conducted using standard host lines of known resistance. Experiments using both host lines and pathogen cultures of known genotype are used to investigate biochemical aspects of the host-pathogen interaction. This experimental

design is also known as the quadratic check (Watson and McIntosh, 1982).

A further experimental design allows the sorting of data generated from tests involving unknown host lines and unknown pathogen cultures. Thus Zadoks (1961) applied the principle of the gene-for-gene relationship in the interpretation of wheat stripe rust data obtained using 15 pathogen isolates and 17 wheat cultivars. He postulated that 14 corresponding pathogenicity:resistance gene pairs were involved. Lewellen *et al.* (1967) and Line *et al.* (1970) also concluded that the interaction of wheat genotypes and *P. striiformis* cultures conformed with the gene-for-gene relationship. However, due to the absence of a sexual stage in *P. striiformis*, the formal genetic proof of the gene-for-gene relationship cannot be established.

2.3 TERMINOLOGY

Terms used to describe features of host and pathogen are difficult to standardise, and vary according to the purpose of the investigation (Robinson, 1969). For genetic studies, the terminology of Loegering (1966) has been widely accepted (McIntosh and Watson, 1982). The terms reaction, pathogenicity and infection type, describe the phenotypes of the host, the pathogen and the host:pathogen interaction, respectively. The contrasting expressions for host reaction are resistant and susceptible, for pathogen pathogenicity are avirulent and virulent and for the infection type of the host:pathogen interaction are incompatible and compatible.

2.4 THE PATHOGEN

2.4.1 TAXONOMY

Amongst the graminicolous fungi, *Puccinia* is distinguished from five other major genera on the basis of teliospore morphology (Cummins, 1971). The stripe rust, or yellow rust, pathogen is one of three species of *Puccinia* which parasitise wheat (*Triticum* spp.). It is broadly contrasted with *Puccinia graminis f.sp. tritici* which causes stem or black rust, and *Puccinia recondita* Rob. ex Desm. *f.sp. tritici* Eriks and Henn. which causes leaf or

brown rust, by having uredinia in chlorotic streaks. Germ pore arrangement on urediniospores and the presence of paraphyses in uredinia are additional diagnostic features used in the identification of *Puccinia* spp. on wheat (J. Walker, pers. comm.). However, separation of *P. striiformis* from other *Puccinia* spp., especially on grass hosts, can be extremely difficult (Mulder and Booth, 1971).

The stripe rust fungus was described first by Schmidt as *Uredo glumarum* in 1827 (Mulder and Booth, 1971). During the 1850's and 1860's, the heteroecious nature of some rust fungi was demonstrated by deBarry and the Tulasne brothers (Large 1940). Thus a number of rust pathogens previously described as *Uredo* spp. or *Aecidium* spp. were shown to be genetically identical, and so new genera were used to accommodate these polymorphic fungi. Westendorp described *Puccinia striaeformis* in 1854 (Johnson and Newton, 1946). Eriksson and Henning (1894) revised a group of grass rust pathogens which resulted in the description of *Puccinia glumarum*. The latter name was in general use until Hylander *et al.* (1953) argued for the validity of Westendorp's earlier description. The accepted name for the stripe rust fungus at the species level is *Puccinia striiformis* Westend. (Bull. Roy. Acad., Belg. 21,235. 1854).

Within well defined morphological species of rust fungi there exist variants which can be distinguished on various physiological criteria. This was demonstrated by Eriksson and Henning (1894) who used the term *forma specialis* to describe entities which showed host-specialisation within the species of the wheat stem rust and stripe rust pathogens.

Eriksson (1894) recognised five *formae speciales* within *P. glumarum viz., f.spp. tritici* (specialised on wheat), *secalis* (rye), *elymi (Elymus arenarius* L.), *agropyri (Agropyron repens* L. Beauv.) and *hordei* (barley). He noted that some of these were not as specialised to particular host species as others. Gassner and Straib in 1934 (Manners, 1950) rejected Eriksson's *formae speciales* concept. They considered that host range was a variable criterion and preferred to regard

the variants as physiological races with varying pathogenicities on certain cereals and grasses. However, Zadoks (1961) re-evaluated these *formae speciales* and concluded that *f.spp. agropyri, dactylidis, hordei,* and *tritici* were useful designations. He noted that little or no work had been done with *f.spp secalis* or *elymi*. However, *f.sp. agropyri* was noted to have a host range that included wheat, barley and *Agropyron repens* (Zadoks, 1961) making it impossible to distinguish from *f.sp. tritici*.

Stripe rust on barley commonly occurs in Europe and pathogen isolates are unable to infect *Agropyron repens, Dactylis glomerata* L. and wheat (Zadoks, 1961; Manners, 1950). The National Institute of Agricultural Botany (N.I.A.B.), Cambridge, England, conducts an annual pathogenicity survey for barley stripe rust (Bayles and Thomas, 1983). It thus appears that *f.sp. hordei* is a widely recognised taxon that causes a distinctive disease known as barley stripe rust.

Manners (1960) described the pathogen causing stripe rust on cocksfoot (*Dactylis glomerata*) as morphologically and physiologically distinct from that causing stripe rust on wheat. The cocksfoot pathogen was described as *Puccinia striiformis* Westend. var *dactylidis* Manners and, in accordance with Article 26.2 of the International Code of Botanical Nomenclature (Voss, 1983) *P. striiformis* Westend. var *striiformis* was applied to the wheat stripe rust fungus. This nomenclature has gained some acceptance (Cummins, 1971). However, Saville (1984) considered that the cocksfoot pathogen may be a different species and that further taxonomic work was required. Tollenaar (1967) provided evidence that urediniospore and teliospore dimensions were too variable for reliable classification and consequently reduced Manners' variety to the level of *forma specialis*. Other workers investigating stripe rust of cocksfoot have drawn the same conclusion, *e.g.* Latch (1976).

Britton and Cummins (1956) reported stripe rust on seven species of *Poa* and two species of *Alopecurus*. Tollenaar and Houston (1967) designated the stripe rust pathogen occurring on Kentucky bluegrass (*Poa pratensis* L.) as a

forma specialis restricted to *Poa spp.* and with a higher optimum germination temperature than *f.sp. tritici.*

Mains (1933) noted the similarity of *P. glumarum* to three *Puccinia spp.* which produced aecia on species of *Mahonia* and *Berberis.* He suggested that these hosts may also support an aecial stage for *P. glumarum.* However, pycnial and aecial stages of *P. striiformis* remain unknown (Arthur, 1962; Mulder and Booth, 1971).

The current nomenclature of the stripe rust pathogen can be summarised as follows:

Puccinia striiformis Westend. 1854 *f.sp. tritici* Eriks. 1894. The pathogen of stripe rust of wheat and other cereals including barley, rye and triticale. Also reported on over 40 genera of grasses (Mulder and Booth, 1971). Optimum urediniospore germination at 10-13C (Manners 1960).

P. striiformis f.sp. hordei Eriks. 1894. Stripe rust pathogen of barley and certain other cereals, *e.g., T. turgidum* L. group *dicoccon* (Manners, 1950).

P. striiformis f.sp. dactylidis (Manners) Tollenaar, 1967. The stripe rust pathogen of cocksfoot which is compatible only with cocksfoot *(Dactylis glomerata).* Optimum urediniospore germination at 21-24C (Manners, 1950).

P. striiformis f.sp. poae Tollenaar 1967. The pathogen of stripe rust on Kentucky bluegrass *(Poa pratensis)* and certain other *Poa spp.* Optimum urediniospore germination at 15C (Tollenaar, 1967).

2.4.2 VARIATION IN PATHOGENICITY

Following Eriksson and Henning's (1894) demonstration of host range differences within *P. graminis* and *P. striiformis,* it was widely held that these *formae speciales* were the ultimate units of specialisation. However, Stakman and Piemeisel (1917) isolated a culture of *P. graminis f.sp. tritici* which could infect a range of hard spring and hard winter wheats that had been resistant to other cultures. Further work resulted in the detection of additional cultures of differing

pathogenicity within *f.sp. tritici*. These cultures were variously termed 'biologic forms', 'physiologic forms' and 'physiologic races' (Johnson and Newton, 1946). In Australia, Watson and Luig (1963) adopted the term 'standard race' to describe entities of *P. graminis f.sp. tritici* which could be distinguished using the differential host stocks proposed by Stakman *et al.* (1962). Pathogenic variation within the standard races was observed and Watson and Luig (1963) described them as 'strains'. A supplementary set of hosts was used to distinguish strains that were of importance in local wheat breeding activities.

Robinson (1969) noted that although 'physiologic race' was recognised at the Sixth International Botanical Congress, 1935, it is a term which can be applied to any physiological criteria used in species sub-division. The term 'pathotype' was proposed by Robinson (1969) to describe individuals which have the same features of pathogenicity. Russell (1978) also recognised pathotype as a more appropriate term.

2.4.2.1 Variation detected in seedling tests

Hungerford and Owens (1923) published large lists of cultivar responses to the stripe rust pathogen in the United States of America (U.S.A.). Rudorf (1929) noted that some cultivars resistant in the U.S.A. were susceptible in tests conducted in Germany. However, Allison and Isenbeck (1930) were the first to determine the presence of pathotypes within *P. striiformis f.sp. tritici*. Using 10 differential hosts, they described four different pathotypes. In 1931, Wilhelm (Manners, 1950) detected five pathotypes in collections from various locations in Europe using a group of 10 differentials that were different from those of Allison and Isenbeck. Thus various workers established pathotype variation within the wheat stripe rust pathogen, although there were no obvious relationships between the described pathotypes.

In the late 1920's, Gassner and Straib, at Braunschweig, Germany, began a systematic study of variation in European collections of the stripe rust pathogen. Extensive

seedling testing of approximately 1400 wheat cultivars resulted in the selection of 11 host testers for pathotype surveys (Gassner and Straib, 1932). These testers were selected on their ability to distinguish between collections of the pathogen that were available at the time (Zadoks, 1961). The cultivars were Michigan Amber, Blé rouge d'Écosse, Strubes Dickkopf, Webster, Holzapfels Früh, Vilmorin 23, Heines Kolben, Carsten $\underline{V}$, Spaldings Prolific, Chinese 166, and Rouge Prolifique Barbu. At various times, Straib subsequently added five additional testers which included *T.diccoccum* var *tricoccum*, three barley cultivars and Petkuser rye (Manners, 1950). Using these differentials, Gassner and Straib published six reports by 1939 describing 47 pathotypes of the stripe rust pathogen collected from Europe, Asia and the Americas (Manners, 1950).

The adoption of this standard group of 11 differential testers on an international basis allowed workers a means to communicate comparative observations on pathotype variability. Bever (1934b) reported that the previously resistant cultivar, Red Russian, was susceptible in a nursery at Idaho, U.S.A. Comparison of various rust collections inoculated on the differential set under standard conditions revealed that Red Russian and the differential, Chinese 166, were susceptible to the Idaho culture. Thus 'races' 19 and 28 were described, and this represented the first report of pathotype variation in the stripe rust pathogen in the U.S.A. Bawden *et al.*(1978) identified 10 pathotypes, five of which had not been previously recognised, in surveys of England and Wales from 1931 to 1933.

World War II caused an interruption of research on pathotype variability. Fang (1944) reported nine pathotypes from two provinces in China using the 11 European differentials. All Chinese cultures were found to be virulent on Chinese 166, in contrast to European cultures which were generally avirulent. Manners (1950) reported the isolation and distribution of twelve pathotypes in Great Britain during 1945 to 1948 using the Gassner and Straib differentials with the addition of cultivar Wilma.

A resurgence of interest in pathotype variability occurred during the mid-1950's following severe stripe rust epidemics in Europe (Zadoks, 1961) and in the U.S.A. (Line, 1983b). Dr. Eva Fuchs was appointed to the Federal Institute of Biology for Agriculture and Forestry, Braunschweig, to continue the investigations initiated by Gassner and Straib. The pathotype nomenclature was re-evaluated using fresh cultures as well as some cultures remaining from pre-war studies. Due to the environmentally sensitive responses of certain differentials, many of the pathotype designations were grouped under broader categories (Fuchs, 1960). Manners (1950) had previously recognised this feature and allocated biotype nomenclature to some cultures which were only slightly different from the standard pathotypes. Fuchs (1960) selected further cultivars which supported and extended the range of differentiation obtainable with the standard testers.

The severe epidemic in the Netherlands in 1955 resulted in the appointment of J.C. Zadoks to the Agricultural University, Wageningen, to undertake epidemiological studies of stripe rust (Zadoks, 1961). Zadoks, in co-operation with the Braunschweig group, established the "Yellow Rust Trials Project" in order to collect data on pathotype variation throughout Europe. This was extended to non-European locations and, as a result of a resolution of the First International Congress of Plant Pathology in 1968, an "International Survey of Factors of Virulence of *Puccinia striiformis* " was established. The group at the Institute for Phytopathological Research, Wageningen, under the direction of R.W. Stubbs, continue to co-ordinate and test materials for international pathotype surveys of the stripe rust pathogen.

In the U.S.A. there were no additional reports of pathotype variation from Bever's (1934b) report until interest was renewed following severe epidemics in the late 1950's and early 1960's. A pathotype, virulent on seedlings of cultivar Suwon 92, was detected in 1961 from different locations in the Pacific northwest (Purdy and

Allan, 1966). This pathotype, and its presumed progenitor which was avirulent on Suwon 92, were both virulent on Chinese 166. The original pathotype isolated by Bever (1934) was avirulent on Chinese 166 and was not detected in the later studies. In 1973, collections of stripe rust from the previously resistant cultivar Pitic 62, grown in California, were shown to involve a different pathotype (Line, 1976). The occurrence of this pathotype resulted in a severe epidemic on Pitic 62 which was grown on more than 50 per cent of the wheat area in the Sacramento Valley. The original pathotype described by Bever (1934b) was virulent on Heines Kolben which has the same resistance gene as Pitic 62 (Section 6.3.1). This suggests that virulence for the Pitic 62 resistance may have been present at a low level in the population and had remained undetected. Line (1983b) summarised the occurrence, distribution and relationships of 25 pathotypes detected in the U.S.A. from 1960 to 1982.

In the United Kingdom, an unexpectedly severe epidemic of stripe rust on cultivar Rothwell Perdix in 1966 (Johnson, 1983c) resulted in increased research emphasis on pathotype surveys and breeding for resistance. A direct result was the formation of the "United Kingdom Cereal Pathogen Virulence Survey Committee" to co-ordinate and publish annual pathogenicity survey data. These surveys currently involve 10 diseases of winter cereals.

Using the standard European differentials, pathotype surveys were reported from India in the late 1930's and 1940's with no further reports until the early 1960's (Ahmad *et al.*, 1970). In 1970-71, two cultures from the pathotype survey were virulent on Holzapfels Früh and were subsequently shown to be virulent on Sonalika (Sharma *et al.*, 1973). Sonalika was noted to be infected in the field in 1972, although the pathotype involved could not be determined. Singh *et al.*(1978) noted that this pathotype was not recovered in subsequent seasons, although it was reportedly prevalent in 1981 (Nagarajan, 1983) and 1982 (Nagarajan *et al.*, 1984).

Over the past 20 years, host differentials employed for pathotype surveys have varied internationally. The historic set of Gassner and Straib was widely used, but by the mid 1950's, it became increasingly apparent that individual differentials provided unreliable information due to variation of disease response with environment (Fuchs, 1960), and genetic heterogeneity in both agronomic type (Zadoks, 1961) and host response (Manners, 1950). Workers in the U.S.A. established a set of seven wheats for pathotype identification (Line *et al.*, 1970). This set was intended to be a means of communication between research groups and was designed to be dynamic in order to allow the addition or deletion of supplementary testers as required. However, Volin and Sharp (1973) found it necessary to modify the set by deleting four cultivars and adding five new ones. This immediately reduced the value of the standard set in communicating pathotype similarities or differences over time.

A meeting of European workers in 1970 agreed upon a differential set which , in combination with binary notation and decanary values, allowed pathotype nomenclature to be uniform and acceptably brief (Johnson *et al.*, 1972). An international host group was assembled which, it was anticipated, would distinguish most previously published global variation. A European supplementary set was similarly compiled to allow the detection and description of known pathotype variation within the region. The cultivars in the differential set were to remain fixed although supplementary differentials could be added to the international set, the European set, or to a local set. This system has been widely adopted among European workers and, due to the research conducted by Stubbs and co-workers at Wageningen, has been accepted internationally. A major advantage of this system was the inclusion of wheats possessing the 10 described genes for resistance to *P. striiformis f.sp. tritici*. This allows prediction of the response of cultivars with known resistance genes to known pathotypes. A similar system of pathotype description

using the same method of notation, but with variation in the differential cultivars and their order, is being evaluated in India (Nagarajan *et al.*, 1984).

Priestley *et al.* (1974) proposed a new classification system in which cultivars with major resistance factors are listed in numerical order. Pathotypes are described by corresponding virulence factors, in contrast to the pathogenicity formula method of Green (1965) which lists both avirulence and virulence factors. The advantage of these systems is the immediate recognition of the pathogenicity genotype. The main disadvantage is the unweildy size of the formulae when resistance factors are numerous.

In view of the international recognition of nomenclature proposed by Johnson *et al.* (1972), this system was adopted for preliminary use in Australia and will be used for pathotype description in this thesis.

A difficulty in pathotype analyses using seedling tests has been deciding between compatible and incompatible interactions. Line *et al.* (1970) considered that infection type "2" or greater on the Gassner and Straib (1932) scale should be classed as compatible. According to Johnson *et al.* (1972) European workers agreed with this, although British laboratories regarded infection types "3" or greater as compatible (Johnson *et al.*, 1972). These discrepancies between compatible and incompatible, in combination with the environmentally labile phenotypic responses of some differential cultivars, can lead to uncertain conclusions in pathotype surveys. Fuchs (1960) aptly stated: "Race identification in yellow rust, which reacts more sharply to environmental changes than all other rust species, demands great effort and critical self-restraint."

2.4.2.2 Variation in adult plant response

Despite intensive efforts since the 1930's to distinguish pathotypes using seedling tests in the glasshouse, study of the interaction of cultivar and pathogen genotypes in the field has not received the same attention. Manners

(1950) found differential interactions between 12 cultivars and seven pathotypes in field experiments, and noted that pathotypes obtained from a particular cultivar generally caused severe infection on that cultivar in the field. Zadoks (1961) demonstrated that variation of cultivar response in naturally-infected field nurseries, sown at many locations throughout Europe, could not be satisfactorily explained by pathotype analyses using the standard differentials in seedling tests. The pathotypes were thus named after the cultivar that was severely infected. For example, the 'Probus race' severely infected cultivar Probus in Switzerland in 1961 (Brönnimann, 1964). These field pathotypes were defined as being distinct entities capable of causing epidemics on some cultivars, but not on others (Zadoks, 1961).

Further work has shown that some of these field pathotypes are associated with seedling virulences; for example, the 'Opal race' was described by Ubels *et al.* (1965) and subsequently shown to be related to seedling virulence on Hybrid 46 and Suwon 92/Omar (Stubbs *et al.*, 1974).

In England in 1969, cultivars Joss Cambier and Maris Beacon became susceptible as seedlings to a new pathotype, 104 E137 (Chamberlain *et al.*, 1971). Joss Cambier was resistant as an adult plant in the field, and was grown on over 30 per cent of the winter wheat area in England until 1971 when commercial crops became severely infected. Rust collections from affected crops were identified as 104 E137 (Johnson and Taylor, 1972a). However, using techniques, including growth rate as a measure of agressiveness on seedling leaves (Priestley and Doling, 1974), urediniospore yield from seedling leaves (Johnson and Taylor, 1972a) and a differential response of adult plants in the field (Johnson and Taylor, 1972b), isolates from Joss Cambier were shown to be distinctive types of 104 E137. Johnson and Taylor (1972a) proposed 104 E137 Type 1 for the original pathotype from Maris Beacon and 104 E137 Type 2 for the Joss Cambier

pathotype. Similar variation occurred in pathotype 41 E136 with respect to Joss Cambier (Johnson and Taylor, 1972b).

As a further example, the winter wheat cultivar Maris Huntsman was first grown commercially in 1973 (Priestley, 1978). In 1974, this cultivar showed more than the expected level of infection (Anon, 1974). Cultures obtained from affected crops were identified as pathotypes 41 E136 and 104 E137 which were both previously known to produce susceptible infection types on Maris Huntsman seedlings (Johnson *et al.*, 1975 ; Priestley *et al.*, 1975). Comparisons of urediniospore yields on seedling leaves suggested that 41 E136 collected from Maris Huntsman was specifically adapted to Maris Huntsman (Johnson *et al.*, 1975). This was confirmed in field experiments which showed that cultures of 41 E136 obtained from Maris Huntsman produced up to three times the level of infection on Maris Huntsman compared to reference cultures of 41 E136 (Johnson and Taylor, 1976b). Despite the increased virulence of some pathotypes on Maris Huntsman, this cultivar continued to show adequate resistance in commercial crops (Johnson *et al.*, 1984).

Further adult-plant virulences have been detected in addition to the variation observed in seedling tests. At the Plant Breeding Institute, Cambridge, England, these adult-plant virulences within the standard pathotypes have been noted with a number, allocated in chronological order of detection, following the pathotype code (Johnson *et al.*, 1984). Table 2.1 summarises some of the pathotypes which have been shown to have additional adult-plant virulence.

Similarly, specificity for adult-plant resistances has been detected in the international surveys conducted by R.W. Stubbs and colleagues at Wageningen, The Netherlands. Results for the survey in 1969, 1970 and 1971 (Stubbs *et al.*, 1974) showed that field nurseries allowed the detection of 12 field variants within four standard pathotypes. For example, the following pathotypes were detected:

40 E8 Falco
40 E8 Etoile de Choisy
40 E8 2X/55

TABLE 2.1

Adult-plant virulences within standard pathotypes of *P. striiformis f.sp. tritici* ; nomenclature, chronological appearance and principal cultivars affected in England

Pathotype	First Record		References
	Year	Cultivar Affected	
104 E137 (1)	1969	Maris Beacon	Chamberlain *et al.*, 1971
104 E137 (2)	1971	Joss Cambier	Johnson and Taylor, 1972a
104 E137 (3)	1972	Maris Bilbo	Johnson *et al.*, 1975
41 E136 (1)	1968	Cama	Johnson *et al.*, 1975
41 E136 (2)	1972	Joss Cambier	Johnson and Taylor, 1972b
41 E136 (3)	1974	Maris Huntsman	Johnson and Taylor, 1977
41 E136 (4)	1979	Hobbit	Johnson *et al.*, 1984
108 E141 (2)	1975	Maris Kinsman	Johnson and Taylor, 1976b
108 E141 (3)	1981	Brigand	Johnson *et al.*, 1983

2.4.2.3 Alternative methodologies for pathogenicity surveys

Pathotype surveysare interpreted on thebasis of the gene-for-gene relationship using hosts of known genotype. The response of unknown pathogen cultures to the array of host testers (*i.e.* the differentials) allows the postulation of pathogen genotypes. The assessment of the host-pathogen interaction is usually determined qualitatively on the basis of infection type in seedling tests. However, minor differences in infection type, particularly in compatible interactions, are difficult to accommodate in pathotype nomenclature.

This difficulty has led some workers to examine the possibility of using quantitative measurements of host-pathogen interactions. Johnson and Taylor (1972a, 1976a) demonstrated that significant differences in spore production were due to minor differences in susceptible infection types. However, these differences in spore production on seedlings were correlated with differences in field responses (Johnson and Taylor 1972b). It was concluded that the quantitative assessment of spore production on seedlings was capable of distinguishing variation within a standard pathotype, in contrast to qualitative visual assessment (Johnson and Bowyer, 1974), and was able to predict adult-plant responses (Johnson and Taylor, 1972b). However, Johnson and Taylor (1976a) recognised that the laborious nature of this method will mean that visual assessment will continue to be important in large scale pathotype surveys.

Priestley and Doling (1974) measured hyphal growth rates following infection of seedling leaves as a means of assessing aggressiveness of isolates of *P. striiformis f.sp. tritici*. This method detected a number of differences within standard pathotypes, although the lack of comparative field work failed to substantiate the practical value of the differences. It was later concluded that the proposed aggressiveness of some isolates was actually associated with the presence of specific adult-plant virulences (Johnson and Taylor, 1975). Priestley and Doling (1976) proposed the "Wycombe Seedling Method" for the quantitative assessment of susceptible interactions on seedling leaves. Again, this method is time consuming and does not appear to have been adopted in pathotype surveys.

2.5 THE HOST

From 1901 studies of the inheritance of a variety of characters in agricultural crop plants were viewed from a new perspective with the independent discovery by DeVries, Correns and Tschermak of the work of Mendel which was originally presented to the Brünn Society in 1865 (Biffen, 1905). Farrer (1889) had earlier stated that

resistance to stem rust in wheat was heritable. R.H. Biffen (1905) presented data for a large range of characters in wheat which were inherited in accordance with Mendel's laws. He demonstrated that the resistant durum, Rivet, when crossed with the susceptible bread wheats, Michigan Bronze and Red King, produced F1 hybrids which were susceptible to stripe rust in the field. Further field tests in F2 and F3 confirmed that the rust resistance of Rivet was inherited as a recessive Mendelian factor.

Biffen recognised a number of significant implications in these results. Firstly, the evidence of inheritance of rust resistance added credence to Marshall Ward's data in refuting Eriksson's mycoplasm hypothesis. Secondly, resistance was inherited independently of morphological features: "immunity simply depended on the luck of the shuffle". Thirdly, and most importantly, Biffen saw the opportunity "of, so to speak, picking out the valuable characters from different varieties and building up an ideal type". Biffen thus laid the foundations for inheritance studies directed at the incorporation of disease resistance into commercial cultivars.

Further work on the inheritance of stripe rust resistance was conducted by Biffen, and other workers in England and Europe, using observations from field epidemics. However, Rudorf (1929) was the first to study inheritance in the glasshouse using artificial inoculations.

2.5.1 INHERITANCE OF RESISTANCE

The terminology used to categorise various types of host resistance has been approached from a number of discipline-related viewpoints. Two types of resistance are usually contrasted and the relationship between discipline and terminology is illustrated in Table 2.2 from terms discussed by Robinson (1969) and Russell (1978). The array of terms cannot be considered synonymous within groups; for example, durable resistance could be under single gene control as with *Sr26* which is used in Australia to provide resistance to stem rust (Johnson, 1984). For

TABLE 2.2

Contrasting terms used to describe the response of host plants to disease

Discipline	Contrasting Expressions	
genetics	major gene	minor gene
	oligogenic	polygenic
epidemiology	vertical	horizontal
pathology	specific	non-specific
	specific	general
	overall	adult-plant
physiology	hypersensitive	non-hypersensitive
breeding	qualitative	quantitative
	seedling	adult-plant
	transient	durable

convenience, the terms presented by Day (1974) will be used to discuss host resistance to *P. striiformis*.

2.5.1.1 Oligogenic resistance

Oligogenic resistance is defined in broad genetic terms as being effected by one or more genes that are relatively easy to detect in segregating populations. These genes are often conveniently detected in seedling tests in the glasshouse and remain effective to the same pathotype in the field (Manners 1950), hence the term "major gene" resistance. In the majority of cases, oligogenic resistances are, or will be, overcome by pathotypes with matching genes for virulence, hence the term "specific" resistance.

Research conducted over the last 60 years in various laboratories has been directed at understanding the mode of inheritance of stripe rust resistance, particularly in hybrid populations subjected to seedling tests. Results from certain of these studies are summarised in Table 2.3. Different workers frequently drew conflicting conclusions from what were apparently relatively similar studies; the reasons for such differences may be due to pathotype variation, the susceptible background employed

Table 2.3

Results of certain studies[1] investigating the inheritance of resistance to *P. striiformis*

Reference	Stocks	No. Resistance Factors	Inheritance	Type of Data
Allan and Vogel, 1961	Suwon 92	1	Dominant	Field;F1,F3
Allan *et al.*, 1966	Nord Desprez	2	Complementary	Glasshouse,Field;F2,
	Spinkota	2	Complementary	F3
	Dickson 114	2	Complementary	
Allan and Purdy, 1967	Kans. 587023	1	Recessive	Glasshouse;F1,F2,F3
	P.I.94349	1	Dominant	
Lewellen and Sharp, 1968	Rego	2	Complementary	Glasshouse;F1,F2
	P.I.178383	1	Dominant	
Allan and Purdy, 1970	P.I.178383	1	Dominant	Glasshouse;F2,F3
	Heines Kolben	1	Recessive	
	Spaldings Prolific	1	Recessive	
Helm and Allan, 1970	P.I.178383	1	Dominant	Glasshouse;F1,F2
	P.I.94349	1	Dominant	
	Suwon 92	1	Dominant	
Pal *et al.*, 1956	Frondoso	3	Dominant, Complementary	Field;F1,F2,F3
Ghosh *et al.*, 1958	Cometa Klein	1	Dominant	Field;F1,F2
Sikka *et al.*, 1960	La Prevision	2	Dominant +	Field;F1,F2,F3
	NP710	2	Recessive	
Nambisan and Kholi, 1961	Cometa Klein	2	Complementary	Glasshouse;F1,F2,F3
	Frondoso	1	Recessive	
	La Prevision	1	Dominant	
Rao *et al.*, 1963	St. 464 (*T. turgidum*)	1	Dominant	Field;F1,F2
Bakshi and Sawhney, 1965	La Prevision	1	Dominant	Glasshouse;F1,F2,F3
Sandhu and Singh, 1970	Lageahinho	2	Dominant	Field;F1,F2,F3
	E159, E259	1	Dominant	
	E255	2	Complementary	
Gandhi, 1971	Cometa Klein	2	Dominant	Field;F1,F2
	Frondoso	1	Dominant	

TABLE 2.3 Cont.

Reference	Stocks	No. Resistance Factors	Inheritance	Type of Data
Minhas and Singh, 1973	Kalyansona	1	Dominant	Field;F1,F2,F3
	Lerma Rojo	1	Dominant	
Grewal *et al.*, 1976	Chris	2	Dominant	Field;F1,F2,F3
	Justin	1	Dominant	
	UP307	1	Dominant	
	Crim	2	Complementary	
	WG586	2	Complementary	
	WG175	2	Complementary	
Omar *et al.*, 1970	Anza	1	Dominant	Field;F1,F2,F3
	Bowie	1	Dominant	
	Narino 59	1	Dominant	
	Bonza	3	Dominant	
Slovencikova, 1980	Benno	1	Dominant	Field;Glasshouse
	Orlando	1	Dominant	
	Zorba	1	Dominant	
Stubbs *et al.*, 1984	Gelderse Ris	1	Recessive	Glasshouse;F1,F2
Walther and Herdan, 1984	Alcedo	2	Complementary	Field;F1,F2
	Berseé	3	Dominant, Recessive	
Dutlu, 1984	Robin	2	Complementary	Glasshouse;F1,F2,F3
	Kavkaz	2	Dominant	

[1]additional examples are reviewed by Röbbelen and Sharp, 1978.

or to differences in the classification of resistant and susceptible progeny (Röbbelen and Sharp, 1978). However, the cataloguing of genes in different cultivars was not attempted until the early 1960's, and even at the present time is not as advanced as that for the other wheat rust systems.

Zadoks (1961) reported results indicating the responses of 17 cultivars to 15 pathogen isolates. He concluded that six genetic factors, or genes, determined seedling resistance and these were assigned alphabetic symbols. Further symbols were allocated to genes effecting adult plant resistance (Table 2.4). Almost simultaneously, Lupton and Macer (1962) reported on inheritance studies involving seedling tests of seven cultivars using four pathotypes. They identified four independent genes to which they assigned the symbols *Yr1* to *Yr4* in accordance with the International Code of Genetic Nomenclature (McIntosh, 1983). Macer (1966) described *Yr5* to *Yr7* inclusive, Riley *et al.* (1968b) designated *Yr8* and Macer (1975) added *Yr9* and *Yr10*.

The relationships between resistance gene symbols assigned by Zadoks (1961) and the *Yr* symbols in current use (McIntosh, 1983) are illustrated for a range of wheats in Table 2.4. It is evident that both systems are in complete agreement for the presence of unique resistances among the various hosts.

Yr1 was described as a dominant gene exhibiting a very low response to avirulent pathotypes (Lupton and Macer, 1962) and was located on chromosome 2A using a Red Bobs monosomic series (Macer, 1966). Chinese 166 has been known to have a single dominant resistance gene from studies by Favret and Vallega (1953) and was a differential cultivar selected by Gassner and Straib (1932).

Yr2 was designated as a single dominant gene in Heines VII and Soissonais-Desprez (Lupton and Macer, 1962). Labrum (1980) concluded that *Yr2* in cultivar Heines Peko was located in chromosome 7B.

TABLE 2.4

Relationships between genes assigned by Zadoks (1961) and the currently accepted *Yr* gene nomenclature in a range of European cultivars

	Overall Resistance	Adult Plant Resistance	Current Nomenclature
Heines Kolben	B		*Yr6*
Heines Peko	B	c	*Yr6*
Heines VII	U	d	*Yr2*
Chinese 166	L		*Yr1*
Vilmorin 23	M		*Yr3*
Nord Desprez	M	z	*Yr3*
Cappelle-Desprez	M	n	*Yr3*
Hope x Timstein	T		*Yr7*
Flamingo	X	e	

Lupton and Macer (1962) considered that cultivars Cappelle-Desprez, Hybrid 46 and Minister had distinguishable alleles at a single locus. These alleles were designated *Yr3a, Yr3b* and *Yr3c,* respectively. However, the F2 data that were presented for the intercrosses between these cultivars indicated segregation in some populations when tested with each pathotype; this suggested that allelism was not strictly demonstrated. Moreover, the only differential response among these cultivars to the 4 pathotypes was the susceptibility of Cappelle Desprez to pathotype 2B. A repetition of Lupton and Macer's work has never been published, despite the fact that the allocation of separate alleles does not appear to have wide acceptance. In this respect, Johnson *et al.* (1972) included Vilmorin 27 to be a tester for *Yr3* without specifying the presence of a particular *Yr3* allele.

Similarly, different resistance alleles at the *Yr4* locus were proposed with Cappelle-Desprez and Hybrid 46 being named as carriers of *Yr4a* and *Yr4b* , respectively (Lupton and Macer, 1962). The data that were presented

suggested allelism between the resistances in these cultivars with pathotype 2B distinguishing the susceptible Cappelle-Desprez from the resistant Hybrid 46.

Macer (1966) failed to chromosomally locate the genes in Heines VII, Hybrid 46 and Minister using conventional monosomic analysis. No reasons for the failures were presented.

The resistance in *Triticum spelta album* was noted to be inherited as a single dominant gene which was designated as *Yr5* (Macer, 1966). This gene remained effective to all known pathotypes (Macer, 1975) although Stubbs *et al.* (1974) noted unconfirmed reports of severe infection of *T. spelta album* in field nurseries in Iran and Tunisia, and Nagarajan (1983) reported a pathotype virulent with respect to *Yr5* in India. In 1976, Gaines reported the location of *Yr5* in the long arm of chromosome 2B (Johnson and Dyck, 1984).

The *Yr6* gene was inherited as a recessive factor in contrast to the dominance of other genes in the same study (Macer, 1966). However, El-Bedewy and Röbbelen (1982) observed dominance of *Yr6* in tests involving some pathotypes and suggested that the change of dominance was related to pathotype aggressiveness. El-Bewdey and Röbbelen (1982) located *Yr6* in the short arm of chromosome 7B, which agreed with the results of Labrum (1980).

The single dominant resistance gene in Thatcher was designated *Yr7* (Macer, 1966). Stubbs (1966) located *Yr7* in chromosome 2B of Thatcher (designated 2A in the paper). In 1976, Gaines confirmed that *Yr7* was located in the long arm of chromosome 2B (Johnson and Dyck, 1984). McIntosh *et al.* (1981) reported that *Yr7* was closely associated with the stem rust resistance gene *Sr9g*. In this study, recombination between *Yr7* and *Sr9g* was infrequent and was estimated at 1.6% in hybrid populations involving Chinese Spring substitution lines. The authors found that *Sr9g* and *Yr7* remained associated in many bread wheats that were derived from the durum wheat Iumillo, via Marquillo, Double Cross and Thatcher. They considered Lee to be a derivative of Thatcher rather than Hope. They also claimed that the durums Acme and Kubanka possessed *Sr9g* but not *Yr7*.

Johnson and Dyck (1984) further investigated the possibility of allelism between *Yr5* and *Yr7*, reported by Gaines in 1976. They established that these genes were non-allelic, and proposed that Thatcher (*Yr7*) has a dominant inhibitor of *Yr5* since Thatcher/*T. spelta album* was susceptible in F1 to a pathotype avirulent for *Yr5* and virulent for *Yr7*. The authors commented that data for segregating F3 families supported the inhibitor hypothesis.

Riley *et al.* (1968a, 1968b) produced an addition line in Chinese Spring following the use of the grass *Aegilops comosa* Sibth. and Sm. (2n = 14) as a stripe rust resistance donor. The addition line was crossed with *Aegilops speltoides* Tausch. to allow recombination between the alien 2M chromosome and its group 2 homoeologues in wheat (Riley *et al.*, 1966). The recombined chromosome showed characteristics of 2M and 2D and was designated 2M/D (Riley *et al.*, 1968b). The stripe rust resistance was inherited as a dominant gene and was catalogued as *Yr8* (Riley *et al.*, 1968a). McIntosh *et al.* (1982) reported a single gene for leaf rust resistance in certain translocation lines of similar pedigree to Compair. They suggested that this resistance, designated *Lr28*, was derived from *A. speltoides* as it was located in chromosome 4BL. On the other hand, a gene conferring resistance to certain cultures of *P. graminis f.sp. tritici* was noted in Compair and related lines with *Yr8*. This gene was designated *Sr34* and, being located in the 2M chromosome and 2M/D derivatives, was genetically associated with *Yr8*.

Zeller (1973) and Bartos *et al.* (1973) determined that a single dominant gene conferring resistance to stripe rust, stem rust and leaf rust was located in a translocated 1B/1R chromosome. Macer (1975) designated the gene for resistance to stripe rust as *Yr9*. The genes for stem rust and leaf rust resistance were catalogued as *Sr31* and *Lr26* (McIntosh, 1983). Stubbs *et al.* (1977) noted that pathotype 232 E137 with virulence for *Yr9* produced adult-plant responses ranging from resistant (*e.g.* Riebesel 47-51) to susceptible (*e.g.* Aurora, Clement) in field tests in a

range of cultivars possessing the 1R(1B) substitution or 1B/1R translocation chromosomes. Crosses between a number of cultivars possessing *Yr9* showed the expected allelism in F2 seedling tests with avirulent pathotypes but some crosses showed segregation in adult-plant response. The authors suggested hypotheses to explain the apparent ineffectiveness of *Yr9* in the adult-plant stage in some cultivars, but critical progeny tests in F3 were not reported. Variation in response to *Yr9*-virulent pathotypes among a range of wheats possessing *Yr9* was confirmed by Zeller and Fuchs (1983).

Lewellen *et al.* (1967) determined that seedling resistance to stripe rust in P.I.178383 was inherited as a single incompletely dominant gene. Metzger and Silbaugh (1970) noted that the gene for brown (red) chaff colour, located in chromosome 1B, was closely associated with the resistance gene in P.I.178383. Thus by implication, the resistance gene was located in chromosome 1B. Macer (1975) designated the resistance gene in cultivar Moro, a derivative of P.I.178383, as *Yr10*.

The genes designated *Yr1* to *Yr10* were allocated corresponding resistance numbers, R1 to R10, by Priestley (1978). Resistance numbers R11 to R14 were assigned to cultivars which had adult-plant resistances that could be neutralised by particular pathotypes. There have been no reports of genetic or cytogenetic studies in cultivars with these factors for adult-plant resistance. Their presence in cultivars has been demonstrated in replicated tests using appropriate pathotypes inoculated onto either hill plots in the field (Johnson and Taylor, 1977) or in polythene tunnels (Priestley *et al.*, 1984b). Designation of *Yr* symbols and representative host stocks for resistance factors R11 to R14 is currently under discussion (R.A. McIntosh, pers. comm.).

In contrast, Stubbs (1985) used the numbers 1 to 10 to describe *Yr1* to *Yr10*. Numbers 11 to 15 were used to designate the undescribed seedling resistances in Suwon 92/Omar (11) and Carstens V (12) and the adult-plant resistances in Heines IV (13), Alba (14) and Dippes Triumph

(15). It would appear that these resistances should also be considered for inclusion in the *Yr* gene symbols.

Israeli workers have studied stripe rust resistance occurring in *Triticum dicoccoides* Koern. populations. Gerechter-Amitai and Stubbs (1970) inoculated seedlings of 53 host accessions collected from 32 sites throughout Israel. Resistant selections were tested in field nurseries and found to be resistant as adult plants. One selection, *T. dicoccoides* var *aaronsohni* G-25, was noted to be resistant to all pathotypes from the Wageningen culture collection. The resistance in G-25 was transferred to durum (Gerechter-Amitai and Grama, 1974) and bread wheats (Grama and Gerechter-Amitai, 1974). Grama *et al.* (1983) concluded that resistances of 32 *T. dicoccoides* selections when crossed with *T. durum* were inherited as single dominant genes in 27 accessions, two dominant genes in three accessions, and one recessive gene in one accession. Amitai *et al.* (1985) reported that G-25 was resistant to all available pathotypes. In a cross involving *T. spelta album* and G-25, F2 seedlings segregated for two independent dominant genes; one was probably *Yr5* and the other a single dominant gene from G-25. Amitai *et al.* (1985) designated *Yr15* for the resistance gene in G-25.

Worland and Law (1985) located a single gene for adult-plant resistance to stripe rust in chromosome 2D of Cappelle-Desprez. Previous commentators had noted the absence of resistance genes to *P. striiformis* in the D genome (Röbbelen and Sharp, 1978). The gene in Cappelle-Desprez was designated *Yr16* and was found to be influenced by the closely associated gene *Ppd1* for daylength insensitivity, as earlier maturing lines carrying *Yr16* were more severely infected compared to their later maturing counterparts (Worland and Law, 1985).

Table 2.5 summarises the described genes for stripe rust resistance, their chromosome locations, and certain reference stocks that are in common use.

TABLE 2.5

Host resistance genes for reaction to *Puccinia striiformis f.sp. tritici*

Gene	Effectiveness	Mode of Inheritance	Chromosome Location	Reference Stock[4]	Literature Citation
Yr1	Seedling[2]	Dominant	2A	Chinese 166	Lupton and Macer, 1962
Yr2	Seedling	Dominant	7B	Heines VII	Lupton and Macer, 1962
Yr3a	Seedling	Dominant		Cappelle-Desprez	Lupton and Macer, 1962
3b	Seedling	Dominant		Hybrid 46	Lupton and Macer, 1962
3c	Seedling	Dominant		Minister	Lupton and Macer, 1962
Yr4a	Seedling	Dominant		Cappelle-Desprez	Lupton and Macer, 1962
4b	Seedling	Dominant		Hybrid 46	Lupton and Macer, 1962
Yr5	Seedling	Dominant	2BL	*T. spelta album*	Macer, 1966
Yr6	Seedling	Recessive	7BS	Heines Kolben	Macer, 1966
Yr7	Seedling	Dominant	2BL	Thatcher	Macer, 1966
Yr8	Seedling	Dominant	2M/2D	Compair	Riley *et al.*, 1968a
Yr9	Seedling	Dominant	1B/1R	Kavkaz	Macer, 1975 Bartos *et al.*, 1973
Yr10	Seedling	Dominant	1B	Moro	Lewellen *et al.*, 1967 Macer, 1975
Yr11[1]	Adult-plant[3]			Joss Cambier	Priestley, 1978
Yr12[1]	Adult-plant			Mega	Priestley *et al.*, 1984a
Yr13[1]	Adult-plant			Maris Huntsman	Priestley *et al.*, 1984a
Yr14[1]	Adult-plant			Hobbit	Priestley, 1978
Yr15	Seedling	Dominant		*T. dicoccoides* G-25	Amitai *et al.*, 1985
Yr16	Adult-plant	Dominant	2D	Cappelle-Desprez	Worland and Law, 1985

[1]allocated provisional *Yr* nomenclature (R.A. McIntosh, pers. comm.).

[2]resistances detected in the seedling stage are effective in adult plants when tested with the same avirulent pathotype.

[3]adult-plant resistances are ineffective in seedling tests.

[4]further stocks possessing *Yr1* to *Yr10* are listed in McIntosh (1983).

2.5.1.2 Polygenic resistance

Polygenic resistance is controlled by genes which individually contribute small phenotypic effects (Day, 1974) and which may be difficult to detect when alone in susceptible backgrounds. A synonymous term is minor gene resistance. However, combinations of minor genes can confer high levels of resistance upon some cultivars.

As early as 1911, polygenic resistance to *P. striiformis* in wheat was demonstrated by Nilsson-Ehle. He observed levels of field resistance that were better than the respective parents among progenies of crosses between susceptible and moderately resistant wheats (Röbbelen and Sharp, 1978). The phenomenon of transgressive segregation in disease response was noted by other workers, for example Allan *et al.* (1963) found resistant progenies among backcross populations based on the susceptible cultivars Norin 10/Brevor 14 and Burt.

Zadoks (1961) introduced the concept of minor gene resistance following investigations of wheat stripe rust. He used the term 'rest resistance' to describe the response of a cultivar to a pathotype which was virulent for the major genes in that cultivar. It was speculated that the minor genes modified the resistance expressed by major genes. In some instances, this resulted in lower responses than those resulting from the major genes alone. Although this was inovative in concept, Zadoks (1961) recognised that further genetic data were required to test the hypothesis.

Lewellen *et al.* (1967) found a single dominant gene for seedling resistance in each of cultivars Chinese 166 and P.I.178383. However, the infection types produced by progenies of selected, presumed homozygous, susceptible F2 plants ranged from resistant to susceptible. Resistant seedlings were more frequent at high temperatures (15C night/24C day) rather than at low temperatures (2C/18C). The resistant progeny were attributed to the action of three temperature-sensitive recessive minor

genes in the case of P.I.178383 and to a single minor gene in Chinese 166. Lewellen and Sharp (1968) similarly demonstrated the presence of minor genes in cultivar Rego, but in this instance the parent line and the derivatives with the minor genes were resistant at low temperatures and susceptible at high temperatures. They further demonstrated that the minor genes from P.I.178383 and Rego, showing contrasting temperature sensitivities, could be combined to provide resistance that was equal or better than the parents, and that was effective at both temperature profiles. Brown and Sharp (1969) showed that some minor genes were particularly sensitive to relatively short exposures to contrasting temperatures. Temperature regimes prior to inoculation were noted to influence minor gene expression in some lines (Brown and Sharp, 1969).

The work of Sharp and colleagues thus confirmed the hypothesis of Zadoks (1961). Minor gene lines developed by Sharp were tested by Stubbs (1977) in field trials conducted throughout Europe. It was concluded that the type of resistance in Sharp's materials were similar to that described as 'rest resistance' by Zadoks.

The value of minor genes in conferring resistance to *P. striiformis f.sp. tritici* was demonstrated with the occurrence of a pathotype virulent on cultivar Moro (Beaver and Powelson, 1969). Moro, a backcross derivative selected for the presence of the single dominant gene of P.I.178383, was susceptible in seedling tests to the new pathotype in contrast to P.I.178383 which maintained a moderately resistant response. Sharp and Volin (1970) further demonstrated that lines with additive minor gene resistances derived from P.I.178383 showed lower responses in seedling tests to the Moro-virulent pathotype than either Moro or P.I.178383.

Pope (1968) concluded that a large number of minor genes contributed to transgressive segregation for resistance in progenies of various crosses involving susceptible, moderately susceptible and moderately resistant

wheats. However, minor gene combinations in some wheats were highly resistant and phenotypically indistinct from wheats with dominant genes for resistance. Pope (1968) stated that these minor genes were "genes controlling functions in a gene complex that confers resistance" and concluded that conventional genetic analyses of resistance may not be appropriate. Henriksen and Pope (1971) found that the susceptible cultivars Baart 46, Dicklow and Federation each contributed minor genes enhancing the resistance of Hussar. The minor genes from the susceptible wheats contributed to resistance in an additive manner.

Krupinsky and Sharp (1978) found that additive minor genes were highly heritable. In later studies, Krupinsky and Sharp (1979) analysed crosses among a range of spring and winter wheats which were commercially and agronomically adapted, and which were intermediate to susceptible in response to stripe rust. Using seedling tests to select resistant progeny, transgressive segregation for resistance did not appear until F4 to F6. Similar results were obtained with winter wheats in the field. It was thus concluded that selection for improved resistance in crosses of wheats of intermediate response to *P. striiformis* would be a useful strategy in breeding for resistance.

Studies by Wallwork and Johnson (1984) involving English winter wheats confirmed the occurrence of transgressive segregation. They noted that some transgressive segregates possessed specific resistance derived from the parents. Thus choice of appropriate pathotypes may be critical in assessing the level of pathotype resistance in such lines.

Minor genes involved in transgressive segregation were reported in *T. aestivum* and *T. durum* (Reinhold *et al.*, 1983). Gerechter-Amitai *et al.* (1984) tested a range of *T. dicoccoides* selections in independent tests in Israel and Montana, U.S.A. Seedlings of some selections were more resistant when tested at high temperature profiles in seedling tests. The same selections showed increased resistance as adult-plants in field nurseries as temperatures increased in spring. Grama *et al.* (1984) demonstrated that transgressive segregation in crosses between selections of

T. dicoccoides was due to temperature sensitive minor genes.

Line *et al.* (1974, 1976) and Line (1980b) described eight types of host resistance in wheats grown in the Pacific Northwest, U.S.A. on the basis of epidemic development and pathotype specificity. Resistance Type 7 was noted to have remained effective for more than 20 years (Line *et al.*, 1983). This Type was characterised in field and controlled environment studies by conferring resistance in adult plants exposed at high temperature regimes of 10C night/35C day (Line, 1983c). Genetic studies showed that this form of resistance was heritable (Milus and Line, 1984a). Line *et al.* (1983c) and Milus and Line (1984b) reported two independent additive recessive genes in each of Gaines, Nugaines and Luke, with Gaines and Nugaines having one gene in common. Line (1980a, 1980b) cautioned that cultivars with this resistance were vulnerable to epidemics and crop losses when spring conditions remained cooler than expected.

Thus a number of forms of polygenic resistance in wheat to *P. striiformis* have been reported following both field and glasshouse studies. Much of the research was conducted by Sharp and colleagues who showed that this type of resistance is inherited through the additive effects of temperature-sensitive minor genes.

2.6 STRATEGIES FOR INCORPORATING STRIPE RUST RESISTANCE IN WHEAT

The relative ease of recognising and manipulating major genes for stripe rust resistance allowed the selection of these resistance genes alone, or in simple combinations, in breeding programs. Röbbelen and Sharp (1978) reviewed the classical pedigree method employed by early European wheat breeding groups which favoured the selection of major genes for rust resistance. This strategy was encouraged by studies showing that such genes conferred high levels of resistance to both seedlings and adult plants (Purdy and Allan, 1963). However, cultivars with these types of resistances were rapidly overcome by pathotypes with matching

genes for virulence. Macer (1975) noted that pathotypes with increasingly complex virulences had emerged in British crops since the mid-1960's. Presumably this was due to mutations detected following the introduction of specific resistances into breeding programmes during the 1950's and 1960's (Day, 1974). Priestley *et al.* (1974) reported that 16 pathotypes were identified between 1966 and 1972 and that these were highly specialised to particular cultivars.

The transient nature of major gene resistances led some workers to suggest that the incorporation of apparently non-specific minor genes would be desirable in breeding programs. Allan and Purdy (1970) reported that the breeding program at Pullman, Washington was being re-directed to incorporate major gene resistances in recurrent parents with adult-plant resistances conferred by minor genes. The use of adult-plant resistances, which remained effective in cultivars when seedling resistances were overcome by new pathotypes, were increasingly advocated, *e.g.* Jönsson (1978). However, Johnson and Taylor (1980) enumerated examples of British winter wheats which had specific factors for adult-plant resistance. Although adult-plant resistances may thus be as transient as major gene seedling types, some cultivars such as Maris Huntsman continued to display acceptable levels of resistance despite the presence of pathotypes which overcame some of the seedling and/or adult-plant resistance factors that were present. In 1972, Johnson proposed the term "durable" resistance to describe resistances that had remained effective in cultivars widely grown in stripe rust prone areas for a considerable period of time (Johnson, 1983b). In defining durable resistance, no suggestion of genetic control or the exclusion of the possibility of future pathotypes virulent for the resistance was intended (Johnson, 1981b).

Johnson and Law (1973) investigated the genetic basis of reputed durable resistance to stripe rust in the French cultivar Hybride de Berseé (abbreviated Berseé) which has a reciprocal translocation involving chromosomes

5B and 7B. Berseé plants monosomic for chromosome 5BS-7BS and nullisomic 5BS-7BS were noted to be more susceptible in the field compared to other monosomic stocks of Berseé. This was confirmed by measuring spore yields produced from seedlings (Johnson and Law, 1975). The results suggested that gene(s) in chromosome 5BS-7BS determined a large part of the reported durable resistance in Berseé. Johnson (1978) noted certain 1976 results of R. Gaines who demonstrated that chromosome 5BS-7BS also controlled adult-plant resistances in Vilmorin 27, Cappelle-Desprez and Caribo. Johnson and Law (1975) suggested that this chromosome could be transferred to cultivars in which the resistance might be difficult to detect. However, Johnson (1981a) indicated that the usefulness of the technique probably depended on the particular genetic background that was selected.

Johnson (1978, 1984) proposed that a breeding strategy for durable resistance should be based on the use of parental material with reported durable resistance. Pathotypes used to test and select progenies should be virulent for all the known specific resistance factors present in the parents. This method has been adopted by the Plant Breeding Institute, Cambridge, England (Lupton, 1982). Nevertheless, Johnson (1984) concluded that the products of such a program still require widespread testing to confirm the presence of durable resistance.

Breeders undoubtedly agree that the incorporation of durable resistance is highly desirable. However, the major limitation, particularly under Australian conditions, is the recognition and selection of parental materials combining durable resistance with other acceptable agronomic attributes (Wellings and McIntosh, 1982).

3 GENERAL MATERIALS AND METHODS

3.1 MATERIALS

3.1.1 HOST

Host materials for genetic investigation were selected on the basis of disease responses observed in seedling tests in the glasshouse. Australian and overseas wheats were selected and tested with the available pathotypes of *P. striiformis f.sp. tritici* and certain hypotheses of the gene or gene combinations involved in conferring resistance were proposed. To test the hypotheses, a range of cultivars was chosen for genetic analyses. Further details of these cultivars will be given in relevant sections of this thesis.

Pedigree designations were based on the nomenclature of Purdy *et al.* (1968).

3.1.2 PATHOGEN

Pathogen cultures used in the studies were detected in the course of Annual Pathotype Surveys (Section 4). The nomenclature of Johnson *et al.* (1972) was used for pathotype designation. However, it was necessary to add supplementary testers in order to distinguish pathotypes avirulent and virulent on cultivar Avocet. Such pathotypes were designated A- and A+, respectively. One or other of these designations was added as a suffix to the standard pathotype code.

Table 3.1 lists the *P. striiformis tritici* pathotypes used in the studies, together with culture numbers, details of origins and pathogenic characteristics. The use of pathotype 106 E139 A- was restricted only to occasions when accessions from New Zealand were being examined as part of the Annual Pathotype Survey.

In some instances, genetic linkages between genes conferring resistance to *P. striiformis tritici* and genes conferring resistance to *P. graminis tritici* and/or *P. recondita tritici* were examined. The Australian system for designation of *P. graminis tritici* pathotypes was described by McIntosh *et al.*

TABLE 3.1

Pathotypes of *P. striiformis f.sp. tritici* used in genetic studies; their pathogenic attributes and origins

Pathotype	Pathogenicity		Source of Isolate		
	Avirulent for	Virulent for	Survey Accession No.	Cultivar	Location
104 E137 A-	*Yr1,5,6,7,8,9,10,A*[1]	*Yr2,3,4*	821559	Flinders	Grafton, N.S.W.
104 E137 A+	*Yr1,5,6,7,8,9,10*	*Yr2,3,4,A*	821552	Avocet	Forbes, N.S.W.
106 E139 A-	*Yr1,5,6,8,9,10,A*	*Yr2,3,4,7*	821589	Oroua	Springston, N.Z.
108 E141 A-	*Yr1,5,7,8,9,10,A*	*Yr2,3,4,6*	832002	Millewa	Horsham, Victoria
108 E141 A+	*Yr1,5,7,8,9,10*	*Yr2,3,4,6,A*	831917	Millewa	Rutherglen, Victoria
360 E137 A-	*Yr1,6,7,8,9,10,A*	*Yr2,3,4,5*	841521	M2369	Forbes, N.S.W.

[1] *YrA* refers to a previous undocumented gene(s) in WW15 (synonyms Anza, Karamu, Mexicani) and to certain Australian cultivars.

(1983), and the system for *P. recondita tritici* pathotypes by Watson and Luig (1961) and Luig *et al.* (1985). Table 3.2 lists the pathotypes of *P. graminis tritici* and *P. recondita tritici* used in the present studies.

TABLE 3.2

Source of *P. graminis tritici* and *P. recondita tritici* pathotypes used in genetic studies

Pathotype	Source of Isolate		
	Survey Accession No.	Cultivar	Location
98-1,2,3,5,6[1]	781219	Unknown	Forbes, N.S.W.
343-1,2,3,5,6[1]	78128	Halberd	Werribee, Vic.
104-2,3,6,7[2]	76694	Gamut	Boggabri, N.S.W.

[1] *P. graminis tritici.*

[2] *P. recondita tritici.*

3.2 METHODS

3.2.1 HOST PREPARATION

3.2.1.1 Glasshouse studies

Seedling tests A potting mix of 90% washed dune sand and 10% spent mushroom compost was firmly pressed into 10cm diameter plastic pots and watered with a complete soluble fertiliser containing fungicide to inhibit damping-off diseases (Aquasol ®, 35g, phenyl mercuric acetate, 20mls, in three litres of water applied to 100 pots). After draining, seed was sown and covered with approximately 15mm potting mix. A nitrogenous fertiliser (Nitram ® at 25g/31 water/ 100 pots) was applied when seedlings attained a height of approximately four cm.

The method of sowing varied according to the purpose of the experiment. Parental materials were sown at two lines per pot with 10-15 seeds per line. Segregating F3 lines were sown at 20-25 seeds per line scattered evenly

in a single pot. For inoculum increase, 30-50 seeds were sown in a clump in the centre of a pot, and maleic hydrazide ($C_4H_4N_2O_2$, 3.0g/31 water/100 pots) was applied to the seedlings together with the nitrogenous fertilizer. The stripe rust susceptible cultivars used for inoculum increase were Sonora (W195[1]) which had some resistance to the powdery mildew pathogen (*Erysiphe graminis* DC.ex Merat. *f.sp. tritici* Marchal), and a Federation backcross derivative (McIntosh line 74.1221 = Federation *8/W804; hereafter designated Fed. 1221) possessing *Pm4b* for resistance to powdery mildew.

All sowing operations were carried out in an airconditioned sowing room kept at approximately 15-20C. Plants were ready for inoculation after 10-12 days when the second leaf was approximately 50% emerged. As far as possible, attempts were made to obtain infection on both the first and second seedling leaves.

Adult plant tests A soil mix of 80% washed dune sand plus 20% spent mushroom compost was supplemented with a slow release complete fertiliser (Osmocote ®, 500g/40 pots) and slow release micro-nutrients (Micromax ®, 70g/40 pots). Prior to inoculation, plants were grown in 15cm plastic pots in glasshouse rooms at 17±2C.

Pests and disease control Seedling and adult plant materials were maintained free of pests and unwanted diseases during all stages of preparation and testing. Powdery mildew and aphids (*Myzus persicae* Sulz. and *Metopolophium dihodium* (Walker)) were particularly troublesome during winter and spring months due to infestations in adjacent field areas. Milstem ® (0.5ml/1) was used to selectively control powdery mildew without significantly affecting subsequent rust development. Rogor ® (1ml/1) was used for aphid control.

3.2.1.2 Field Studies

Direct sowing Approximately 25 seeds of selected F3 lines were sown in 1m rows in the field in late autumn. Parental

---

[1]W numbers refer to the University of Sydney, Wheat Accession Register.

lines were included as controls. After every fifth row, a rust susceptible control was sown to ensure adequate and even distribution of inoculum.

Fertiliser was applied at sowing and pre and post-emergent herbicides were applied to assist in weed control.

Transplanting In 1981 and 1985, F1 and F2 populations were sown in the glasshouse and tested as seedlings with pathotype 104 E137 A-. When differences in infection type were noted, individual plants with a particular infection type were tagged with coloured bell wire loops. After a short period of hardening following removal from the glasshouse, seedlings were transplanted in distinctive seedling response groups in the field; individuals with particular responses were identified by the coloured loops. In other years, similar materials were sown in the glasshouse and transplanted without seedling tests to avoid the possibility of transferring the pathogen from the glasshouse to the field.

3.2.2 INOCULUM PREPARATION

3.2.2.1 Standard pathotype cultures

Several cultures considered to be the same pathotype were compared on the differential testers. One culture considered pure and typical of the pathotype was selected and then increased from a presumed single uredinium. However, due to the systemic nature of infection and sporulation of *P. striiformis*, it was not possible to assume that a particular sporulating area arose from a single infecting urediniospore.

3.2.2.2 Inoculum increase

Inoculum was increased on a susceptible cultivar (Sonora or Fed. 1221) or on a cultivar that was susceptible to the particular pathotype; for example, 108 E141 pathotypes were increased on cultivar Oxley. Seedling clumps treated with maleic hydrazide produced abundant sporulating uredinia. The urediniospores were shaken from leaves onto glassine paper and spread evenly before being placed in a temperature

and humidity controlled storage facility (10C 25% R.H.) for three to five days. Cyclone collection equipment is routinely used at the Plant Breeding Institute for collecting urediniospores of *P. graminis tritici* and *P. recondita tritici*. However, the method employed allows only one collection as mechanical damage is caused to plants. In contrast, the glassine paper technique allowed up to three collections which greatly improved the inoculum yield of *P. striiformis tritici*.

Following drying, urediniospores were placed in aluminium foil packets, labelled, heat-sealed and located in liquid nitrogen storage at -196C. Upon retrieval from storage, urediniospores were heat-shocked by placing foil packets in a water bath at 40C for four minutes. Using these techniques, *P. striiformis tritici* cultures have remained viable in liquid nitrogen storage for six years. After removal from liquid nitrogen, inoculum in foil packets has remained viable for up to four weeks when held at 4C.

3.2.3 INOCULATION

3.2.3.1 Glasshouse studies

Urediniospores were suspended in light mineral oil (Pegasol ® 3440 Special) at approximately 10mg/10ml/100 seedling pots and sprayed, using a Freon pressure pack, over the host material. The inoculating equipment was washed and rinsed in alcohol and running water between successive inoculations. Inoculation took place in an enclosed room which was thoroughly sprayed for 45 seconds following each inoculation using a series of automatic sprinklers. In order to monitor the effectiveness of the washing and sterilization procedures, checks of susceptible seedlings were sprayed with oil to which no inoculum had been added. The absence of infections confirmed the effectiveness of these methods.

Following inoculation, plants were incubated at 100% relative humidity at 9±1C with 12 hours dark and 12 hours incandescent light (15-20 foot candles) for 24 hours.

Plants were then moved to benches in a glasshouse held at 17±2C. Under these glasshouse conditions, the relative humidity was 77% (standard error 11) while midday light intensities ranged from 675 foot candles (s.e.300) in winter to 2000 foot candles (s.e.800) in summer. Infection types were recorded 14 days after inoculation in summer and at up to 19 days after inoculation in winter.

3.2.3.2 Field studies

In 1981, infected susceptible seedlings transplanted into spreader rows allowed adequate infection and subsequent spread of the disease. In 1982 and 1983, inoculum inadvertantly released from the glasshouse was sufficient to establish mild field epidemics. However, they were sub-optimal for experimental purposes because they were initiated relatively late in the season and, due to restrictions on field studies, rust spreaders were intentionally resistant to stripe rust.

In 1984 and 1985, entire field plots were inoculated with urediniospores suspended in mineral oil (approximately 2g/700ml/0.25 ha) using ultra low volume applicators ('Ulva 8' ®, Micron England). Inoculation on three evenings in mid-winter ensured excellent infections throughout the experimental areas.

3.2.4 DISEASE ASSESSMENT

3.2.4.1 Seedling tests

The host-pathogen interactions were assessed qualitatively using an infection type scale similar to that described by Gassner and Straib (1932). The broad divisions of the scale are illustrated in Plate 3.1 and are described as follows:

0; hypersensitive flecks just visible

; hypersensitive fleck

;C a larger area of affected leaf tissue showing chlorosis. No uredinia

1 uredinia small, scattered, not erupting through the epidermis and enclosed by chlorotic and/or necrotic tissue

2 uredinia larger, emerging through the epidermis and often associated with chlorosis and/or necrosis

3 large, freely sporulating uredinia associated with varying levels of chlorosis

4 large, freely sporulating uredinia

The main difference between the above system and that of Gassner and Straib was the adoption of the semicolon ";" to symbolize the hypersensitive fleck response in contrast to the symbol "0". The adoption of the ";" symbol was to maintain consistency with its use for the other wheat rust diseases at P.B.I., Castle Hill. In the cases of wheat stem rust and leaf rust "0" is used to designate plants that have escaped infection or have no visible symptoms of infection.

Composite infection types were used to indicate the range in response. When materials were genetically heterogeneous or segregating, individual infection types were noted in order from the most resistant to the most susceptible with each distinctive type separated by a comma. Infection type on first, second and subsequent leaves were separated in order by a slash "/". The symbols "+" and "-" were used to denote more and less, respectively, on the infection type scale, and the presence of unusual chlorosis or necrosis was noted using "C" and "N", respectively.

3.2.4.2 Adult plant tests

Assessment of disease responses on adult plants involved the use of two scales. The first, a measure of the severity of infection, assessed the percentage leaf area affected using the modified Cobb scale (Peterson *et al.*, 1948). The second scale was a qualitative assessment using infection type similar to that described by Line *et al.* (1974). The broad divisions of the infection type scale are illustrated in Plate 3.2 and can be described as follows:

OR	no obvious signs of infection
VR	hypersensitive flecks; chlorotic and/or necrotic blotches
R	small stripes; no sporulation
R-MR	small stripes; trace sporulation
MR	large stripes; light sporulation
MR-MS	large stripes; moderate sporulation
MS	large stripes; heavy sporulation
MS-S	abundant sporulation; chlorosis and/or necrosis
S	abundant sporulation; chlorosis
VS	abundant sporulation

Assessments were based on the responses of entire plants. Segregating or heterogeneous materials were noted by a similar manner to that used for seedling tests. Growth stage of the material was recorded using the decimal code illustrated by Tottman and Makepeace (1978).

The adequacy of field assessments varied between seasons. Restrictions imposed on release of the pathogen into field plots as part of National Wheat Rust Control policy resulted in late, sub-optimal epidemics in 1982 and 1983. Poor growth of direct-sown materials in 1983 was associated with problems of pre-emergent herbicide application. In 1984, there was unsatisfactory development of transplanted materials due to a combination of heavy aphid infestation, barley yellow dwarf virus and poor herbicide application. These problems resulted in inadequate assessments and the abandonment of some experiments.

3.3 ABBREVIATIONS

The following abbreviations will be used:

d.f.	=	degrees of freedom
cv.	=	cultivar
IT	=	infection type
Σ	=	sum of

Because of the lack of previous experience with stripe rust "a good deal of time was lost in obtaining the necessary know-how and finding out the best working conditions" (Zadoks, 1961).

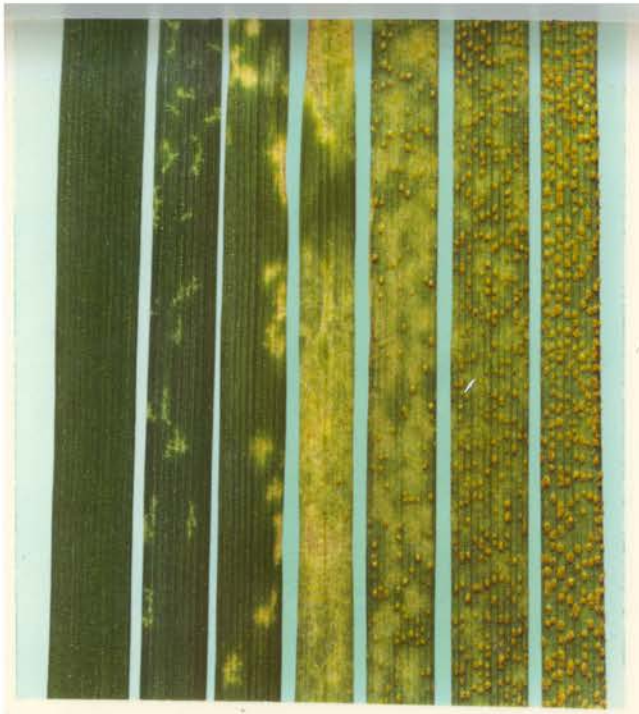
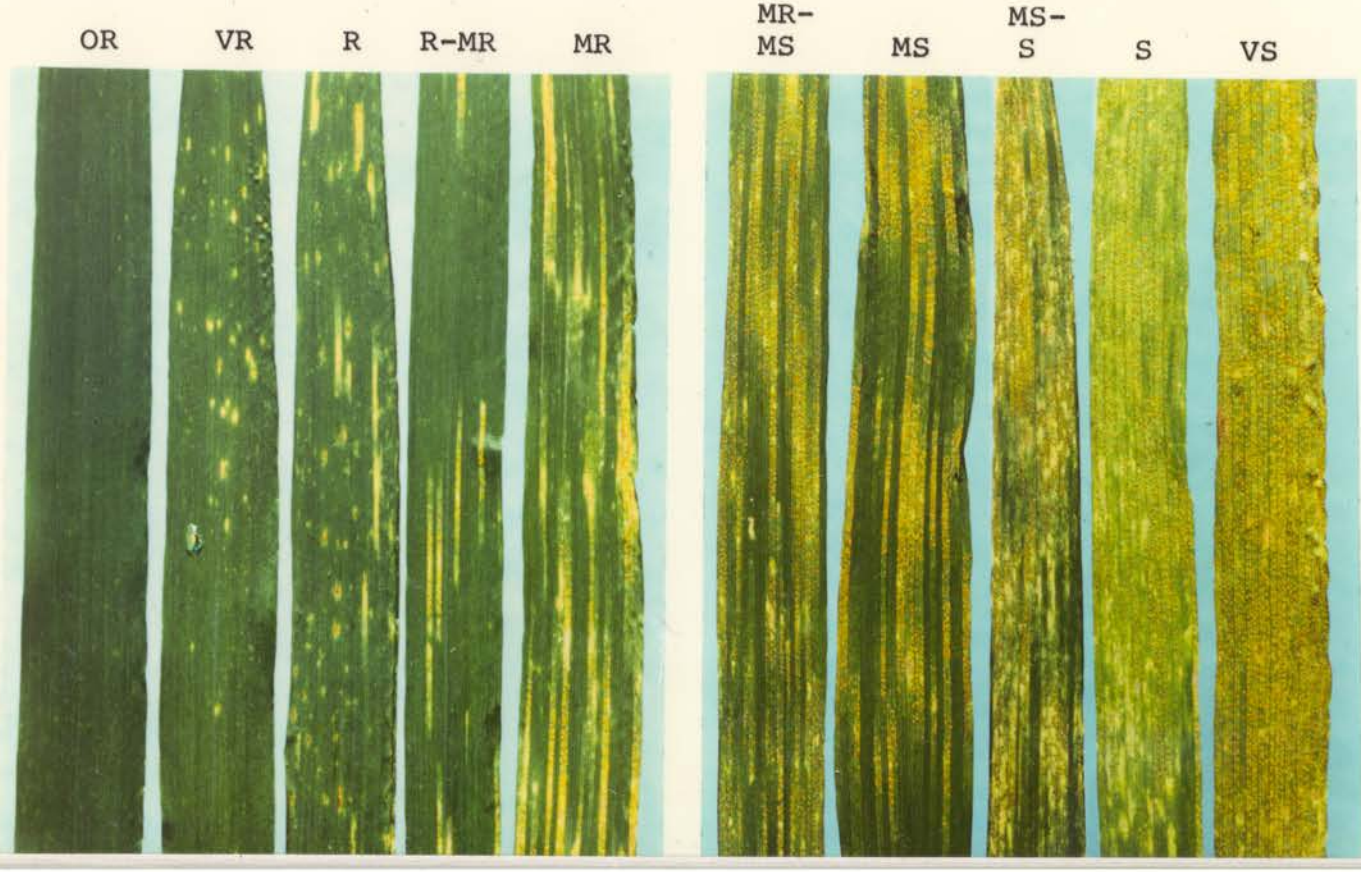


PLATE 3.1

Infection type responses of wheat seedlings inoculated with *Puccinia striiformis f.sp. tritici*

PLATE 3.2

Infection type responses of flag leaves inoculated with *Puccinia striiformis f.sp. tritici* in the field



4 PATHOGENICITY SURVEY AND EPIDEMIOLOGY OF STRIPE RUST IN AUSTRALASIA

4.1 INTRODUCTION

An effective breeding strategy directed at the achievement of resistance to stripe rust in wheat is dependent on an understanding of the nature of the pathogen as well as the host. Attributes of the pathogen, particularly its taxonomic status, host range and pathogenic specialisation, are prerequisite to the selection of the most appropriate cultures for screening breeding materials. Annual pathogenicity surveys provide a continual monitoring of these attributes in the pathogen population. Epidemiological information also assists the breeder in formulating the type of resistance required to effectively minimise crop loss and disease spread.

The early detection of new virulent pathotypes is a major objective of the survey. This allows agricultural advisors to adjust cultivar recommendations, in order to minimise crop losses to the farming community. The distribution of pathotypes is also used in predicting cultivar response in particular areas, and in recommending cultivar diversity to avoid the risk of crop loss following the occurrence of new pathotypes.

The occurrence of stripe rust in Australia resulted in an immediate need to establish the pathogenic spectrum involved in the initial epidemic and the continuing need to monitor possible pathogenic changes. Annual pathogenicity surveys of the rust diseases of wheat and oats in Australia have been conducted by the University of Sydney since the 1920's. It was thus a logical step to integrate the stripe rust pathogenicity survey with the Annual Rust Surveys of the Plant Breeding Institute.

4.2 MATERIALS AND METHODS

4.2.1 HOST RANGE EXPERIMENTS

Observations and collections from field surveys, and specimens sent to the laboratory by a range of co-operators, allowed the determination of the *formae speciales*

of the stripe rust pathogens, and of the host range of *f.sp. tritici* in eastern Australia. In some cases, stripe rust samples collected from hosts other than wheats were inoculated onto a wheat known to be susceptible to *f.sp. tritici*, or the wheat differential set, to confirm the presence of *f.sp. tritici*. Specimens deposited in the herbarium of the N.S.W. Department of Agriculture, Rydalmere (Herb DAR), were noted for date and location of collection and host and pathogen identification.

Inoculation experiments using pathotype 104 E137 A- were conducted using a range of grasses obtained from A. Jackson (Plant Breeding Institute, University of Sydney), R.D.B. Whalley (University of New England) and G. Lodge (N.S.W. Department of Agriculture, Tamworth). Methods of inoculation and incubation were previously described (Section 3.2.3). Tests were conducted on plant materials ranging from the two-leaf to flowering stages.

4.2.2 COLLECTION OF FIELD SAMPLES

Diseased plant materials were collected from wheat-growing areas by various co-operators, including farmers, extension officers and research personnel. Co-operators were encouraged to send fresh material, with leaves folded together to avoid desiccation, placed in paper envelopes and despatched by surface mail.

Field surveys were conducted by motor vehicle, travelling along major roads throughout the eastern Australian wheat belt. Depending on the reported occurrence of rust, approximately 4,000 to 5,000 kilometers were covered each season by various members of the Plant Breeding Institute staff. Crop inspections were made at 20 to 40km intervals, depending on the availability of fields for inspection. Stripe rust samples were placed in paper envelopes, or on petri dishes containing water agar (Section 4.2.3). All samples were stored at 5C in a portable refrigerator while in transit to the Laboratory.

4.2.3 LABORATORY PROCEDURE

On arrival at the laboratory, diseased samples were accessioned with details of date collection, location, cultivar if known and the co-operator's name. Establishment of cultures from the accessioned material was poor in 1979 and 1980. A technique was devised to partially revive samples: accessioned materials, including glumes, stems and leaves cut to approximately 3cm lengths, were placed on agar (20g/l tap water) in petri dishes with the uredinia uppermost. Although the use of 20ppm kinetin in the agar reportedly delayed leaf senescence (Wolfe and Macer, 1964), this procedure proved unnecessary as material was kept for a maximum of only seven days prior to inoculation. Petri dishes were incubated at 5C in the dark for the one-to-seven day period. To avoid urediniospore germination prior to inoculation, care was taken to ensure that excess moisture in the petri dishes was minimal.

4.2.4 GLASSHOUSE PROCEDURES

Following incubation in petri dishes, uredinia generally produced abundant fresh urediniospores. The sporulating material was transferred to glass vials to which 2-5ml of Pegasol® were added to suspend urediniospores for inoculation. When inoculum was limited, due to small or poor quality samples, a modified spatula was used to mechanically transfer urediniospores to susceptible host seedlings. Methods of inoculation and incubation were described previously (Section 3.2.3).

4.2.5 SEEDLING TESTS

Seed of the differential cultivars proposed by Johnson *et al.* (1972) was imported from the Plant Breeding Institute, Cambridge, by R.A. McIntosh in 1979. Seed of these cultivars was increased at Castle Hill and Cowra (central-west N.S.W.). However, cultivars of winter habit produced small quantities of poor quality seed in these environments and were subsequently increased at the Department of Agriculture, Launceston, Tasmania, by W. Vertigan thereby greatly improving the quantity and

quality of seed.

The differential set is listed in Table 4.5. A supplementary group of three Australian cultivars was included. These comprised a susceptible check (Sonora W195 or Fed. 1221), a local cultivar with *Yr6* (Oxley, W3636) and a resistant selection of Avocet, referred to as Avocet R (AUS 95169).

When inoculum was limited, accessions were firstly inoculated on to susceptible cultivars for inoculum increase prior to testing on the differential set (Section 3.2.2 and 3.2.3). When accessions were obviously contaminated with leaf rust or stem rust, they were applied to seedlings of cultivar Agent. The combined resistances to these diseases conferred by *Lr24* and *Sr24* , respectively, together with the cool temperatures of incubation allowed uncontaminated cultures of *P. striiformis* to be established.

Inoculum from the accessions, either direct from petri dishes or initially increased as described above, was applied to seedlings of the differential set. The widespread epidemic of 1983 resulted in a large number of accessions. These were initially screened on a "short" set comprising Oxley *(Yr6)*, Lee *(Yr7)*, Avocet R (with gene(s) to be designated '*YrA*') and a susceptible check. Accessions were then stored in liquid nitrogen and re-tested at a later date on the complete differential host set. In 1985, accessions stored from the 1979 and 1980 surveys were re-examined for pathogenicity on Avocet R.

The pathotype nomenclature was that described by Johnson *et al.* (1972) with the addition of Clement (*Yr9*, decanery value 2^7= 128) to the world set as proposed by Johnson and Taylor (1976b). It is proposed that *Triticum spelta album (Yr5*, decanery value 2^8= 256) should also be added to the world set. Cultivar Avocet, and later a resistant selection of this cultivar, was used as a supplementary differential to distinguish cultures virulent and avirulent on the undescribed resistance present in some plants of Avocet and related cultivars. Virulence

and avirulence with respect to this resistance was described as A+ and A-, respectively, and were noted as a suffix following the international and European race designations, for example 104 E137 A+ and 104 E137 A-, respectively.

4.2.5.1 Mixed infections

Unlike the wheat stem rust and leaf rust pathogens, the stripe rust pathogen invades host material in a systemic manner with infection hypae proceeding slightly in advance of chlorosis and sporulation (Mares and Cousen, 1977). It was thus expected that mixtures of stripe rust pathotypes would be difficult to detect.

Experiment 4.1 Pathotypes 104 E137 A- and 104 E137 A+ were obtained from liquid nitrogen storage and tested for urediniospore germination according to the following procedure. A urediniospore suspension in mineral oil was spread on 2% water agar on a microscope slide and incubated in a petri dish with a moistened filter pad at 8C in the dark for 24 hours. Germination percentage was determined from observations of 100-130 random urediniospores. Both pathotypes were noted to have 100% germination.

Stock suspensions of 1.36×10^6 and 1.59×10^6 urediniospores/ml of 104 E137 A+ and 104 E137 A-, respectively, were made using a haemocytometer to adjust urediniospore concentration. Aliquots taken from the stock solutions were mixed in varying proportions to provide nine treatments. Three ml samples from each treatment were inoculated on to four pots of Avocet R and one pot of Sonora W195. Infection types were noted and urediniospores collected from Sonora in each treatment were used to inoculate a further four pots of Avocet R. Infection types were again noted and compared with those obtained from the original mixture.

4.2.5.2 Sub-cultures

During routine glasshouse testing, seedling infection types sometimes varied from those expected. This was due to sowing error, incorrect seed source, cultivars with environmentally-sensitive responses and genetically heterogeneous host lines. However, the

possibility of new mutant pathotypes occurring during glasshouse testing required investigation.

Experiment 4.2 The cv. VPM1 was originally developed in France as a source of eyespot (caused by *Pseudocercosporella herptrichoides* (Fron) Dei.) resistance derived from *Aegilops ventricosa* Host. (Doussinault *et al.*, 1983). However, it was initially selected locally as a source of stem rust resistance and was incorporated in the backcrossing project of the Australian National Wheat Rust Control Program. Subsequent observations indicated that this cultivar, and its stem rust resistant derivatives, possessed seedling and adult-plant resistance to stripe rust. Stripe rust testing of segregating third backcross F2 populations in the susceptible background of cv. Cook revealed seedling infection types higher than those recorded on the donor parent. Presumed single pustule isolates were transferred to Sonora W195 for inoculum increase. Each isolate was then inoculated onto a differential set, VPM1, and the source host line of the original isolate, and ITs noted.

4.2.6 ADULT-PLANT TESTS

Glasshouse testing of adult plants with stripe rust was not performed routinely. However, the detection of new pathotypes necessitated the assessment of the change in response of particular cultivars in the adult-plant stage. Preparation of plant material, inoculation procedure and assessment methods were described in Sections 3.2.1, 3.2.3 and 3.2.4, respectively.

Experiment 4.3 The Avocet-virulent pathotype, 104 E137 A+: Twenty seven accessions of known pathogenicity were compared for adult-plant response on cv. Avocet and an advanced breeding line, WW232, supplied by J. Fisher, Agricultural Research Institute, Wagga Wagga, N.S.W., as a stripe rust susceptible line of similar pedigree and maturity to Avocet.

Five accessions of known pathogenicity were compared on seven cultivars and eight advanced lines submitted by various wheat breeding centres.

Experiment 4.4 The *Yr6*- virulent pathotypes 108 E141 A-

and 108 E141 A+: Four isolates with all combinations of virulence and avirulence with respect to *Yr6* and *YrA* were compared on 13 cultivars. Temperature regimes of 15±2C and 20±2C were used to compare adult-plant responses.

Experiment 4.5 Adult-plant resistance of cv. Banks: Ten isolates were collected from commercial crops of Banks, some of which were reported to have unusually high levels of stripe rust. These were accessioned, the pathotype determined, and then inoculated on adult-plants of Banks and a susceptible control of either Warigal for Trial I or Teal for Trial II. Some accessions were used in both experiments.

4.3 RESULTS

4.3.1 HOST SPECIALISATION IN EASTERN AUSTRALIA

4.3.1.1 Occurrence and distribution of *Puccinia striiformis tritici*

Wheat stripe rust was first reported in Australia in October, 1979 (O'Brien *et al.*, 1980). First reports came from a farmer in the Charlton district of Victoria on or about 12 October and from Dooen, Victoria, on 16 October, 1979 (McIntosh, 1980). However, the first authenticated record of stripe rust in Australia was collected by J. Fisher, N.S.W. Department of Agriculture, at Darlington Point in southern N.S.W. on 18 October, 1979 (DAR 34382)[1]. Within six weeks of these early sitings, stripe rust was recorded in northern N.S.W. (DAR 34146), South Australia (DAR 34382) and Tasmania (DAR 33103). Assuming the focal centre of the initial outbreak was the Charlton district in Victoria, urediniospore dispersal in the first six weeks extended to a 900km radius.

In October, 1981, stripe rust was found in Queensland (811523)[2] and in later years it has been observed as far north as Theodore. The disease has not been recorded from the

---

[1]Specimen number lodged in Herb DAR.

[2]Accession number of the Annual Rust Survey, P.B.I., University of Sydney.

central highlands of Queensland. Isolates have been obtained from as far west as Ceduna (South Australia) on the eastern edge of the Great Australian Bight. This represents the western limit of stripe rust distribution in eastern Australia as the wheat growing areas of Western Australia have remained free of the disease.

4.3.1.2 Occurrence and distribution of *Puccinia striiformis f.sp. dactylidis*

Stripe rust of cocksfoot (*Dactylis glomerata* L.) was first recorded in Australia in December, 1979, at Narracorte in South Australia (DAR 34381). It was subsequently found in Tasmania in 1980 (DAR 33143) and N.S.W. in 1981 (DAR 35045). In N.S.W., the disease has been observed in the central and northern tablelands and the central western slopes. Glasshouse investigations confirmed that this pathogen could infect cocksfoot, but not wheat. Optimal temperatures for urediniospore germination and infection was found to be 15C in contrast to 10C for wheat stripe rust. These results correspond with similar observations in Europe (Manners, 1950).

4.3.1.3 Occurrence of *Puccinia striiformis f.sp. poae*

The pathogen of one specimen of rust on *Poa pratensis* L. from Canberra, Australian Capital Territory, was identified by J. Walker (Principal Research Scientist, N.S.W. Department of Agriculture) as *Puccinia striiformis* (DAR 35759). This rust pathogen was distinguished from *P. brachypodii* and *P. coronata* by the presence of large saccate paraphyses at the margins of uredinia. A sample was not obtained by this laboratory; consequently, infection studies to confirm the pathogen and its host range were not carried out. In the absence of these studies, the identity of *P. striiformis* infecting *Poa pratensis* was considered to be doubtful, and there was no evidence to suggest the presence of *f.sp. poae*.

4.3.1.4 Host range of *Puccinia striiformis f.sp. tritici*

A range of grasses representing each sub-family within the Poaceae (*sensu* Wheeler *et al.*, 1982) were inoculated under glasshouse conditions. Those showing no rust symptoms are listed in Appendix Table 1. The species which showed symptoms of infection are listed in Table 4.1. These belonged to three tribes of sub-family Pooideae and were often heterogeneous in response. Species in tribes Triticeae and Phalarideae which showed symptoms in inoculation experiments were confirmed as alternative hosts of *P. striiformis f.sp. tritici* by additional data from herbarium exsiccatae and by obtaining wheat-infecting isolates from these hosts in the course of the Annual Rust Surveys (Table 4.1).

Barley grass (*Hordeum leporinum* Link.), a common weed of wheat cultivation in Australia, was often found naturally infected with *P. striiformis f.sp. tritici* when the surrounding crop was severely rusted. From field observations, it was noted that the responses of individual barley grass plants ranged from resistant to susceptible, thus confirming inoculation experiments.

Commercial two-row barley (*Hordeum vulgare* L.) was variable in response to wheat stripe rust in inoculation tests. From 26 cultivars submitted by B. Read, Agricultural Research Institute, Wagga Wagga, 19 were highly resistant, 4 intermediate, 1 susceptible (cv. Dampier) and 2 were heterogeneous (cvs. Corvette and Ketch) in response ranging from resistant to susceptible. Certain entries in an international barley nursery (*e.g.*, International Barley Observation Nursery 7,259) were moderately susceptible in the field. In subsequent seedling tests they were shown to be susceptible. A commercial crop of cultivar Clipper was noted to be moderately infected when it was adjacent to a heavily rusted wheat crop (DAR 50465). One six-row barley accession (WU3138) was observed with stripe rust symptoms in the field at Wagga and was susceptible in glasshouse tests in this laboratory.

One entry in a 1981 triticale nursery that was

TABLE 4.1

Host range of *Puccinia striiformis f.sp. tritici* in eastern Australia

Host	Source of Data		
	Identified specimen[1]	Isolate from field sample[2]	Glasshouse Inoculation[3]
Sub-Family Pooideae			
Triticeae			
Agropyron scabrum (Labill.) Beauv	DAR51152	831579	S
Hordeum distichon L.	DAR35960	841776	R,S
Hordeum leporinum Link.	DAR33917	831580	R,S
Secale cereale L.	DAR52868		
Triticum aestivum L.	DAR34382		R,S
X *Triticale*	DAR43707	841518	R,S
Phalarideae			
Phalaris paradoxa L.	DAR35044	811752	I,S
Poeae			
Bromus diandrus Roth.			R,S
Bromus arenarius Labill.			R,S
Bromus japonicus Thumb.			S
Bromus rubens L.			R,S
Bromus unioloides Kunth			R,S
Lolium sp.	DAR33142		

[1]Specimens identified and lodged in Herb. DAR.

[2]Isolates established from survey accessions.

[3]R resistant, I intermediate, S susceptible.

highly susceptible in the field at Castle Hill was subsequently found to be susceptible at the seedling stage. Observations in nurseries and field trials at Castle Hill and in southern N.S.W. suggested that certain early-sown triticales suitable for grazing were prone to infection with wheat stripe rust. This predisposition to stripe rust probably reflected lineal relationships among the materials rather than being associated with a particular agronomic type.

Rough wheat grass (*Agropyron scabrum* (Labill.) Beauv.) was found to be an alternative host for wheat stripe rust, particularly in northern N.S.W. and southern Queensland where this summer-growing species is widespread. Naturally infected paradoxa grass (*Phalaris paradoxa* L.) was occasionally found in northern N.S.W., but was variable in response in glasshouse tests.

A number of *Bromus* spp. (tribe Poeae) were susceptible in inoculation tests. Prairie grass (*Bromus unioloides* Kunth) has been found to have stripe rust in the field, although isolates from this species could not be established on wheat and taxonomic identification of the pathogen was not obtained. The remaining *Bromus* spp. have not been observed with stripe rust under field conditions.

Lolium spp. tested in the glasshouse were resistant to stripe rust and isolates could not be established from suspected stripe rust infected field samples. In contrast, DAR 33142 was identified by J. Walker as *Puccinia striiformis* on *Lolium* sp. As a culture was not obtained by this laboratory, confirmation of *P. striiformis f.sp. tritici* infecting ryegrass could not be obtained, and must therefore be considered doubtful.

4.3.2 PATHOTYPE SURVEY

4.3.2.1 Techniques

Mixed Infection. Experiment 4.1 Infection type results are presented in Table 4.2. The differences between the infection types produced by the pure cultures on Avocet R

were very clear. One cycle of inoculum increase on Sonora did not appear to affect the infection type produced by either pathotype.

When the pathotypes were mixed, it was evident that increasing proportions of 104 E137 A+ gave slight, progressive increases in infection type. However, when the pathotype mixture was increased on Sonora prior to inoculation on to Avocet R, there was very little change in infection type between the mixtures except when 104 E137 A+ was the dominant pathotype.

Chlorosis, even with high infection types, suggested that a mixture of pathotypes was present. However, the relative proportion of pathotypes could not be predicted using infection type data. The need for inoculum increase of survey accessions did not cause inability to recognise mixed pathotypes, provided pure cultures were available to make comparisons.

Sub-Culture. Experiment 4.2 Homozygous resistant lines in a VPM1/4*Cook population showed responses ranging from ITs ;1C to 3-C when inoculated with 108 E141 A+. Urediniospores had been taken from single plants which displayed IT 3-C in each of three lines. All three isolates proved to be pathotype 108 E141 A+. The results of inoculations of these isolates on to VPM1 and the original lines from which the respective isolates were taken are shown in Table 4.3. Whereas VPM1 remained uniformly resistant to each isolate, variation in infection type was noted for each isolate-line combination. This variation was similar to that obtained in the original observations. Some plants within each line appeared to be more resistant than the VPM1 donor parent.

It was concluded that the variation in infection type produced by the Cook derivatives was due to variation in the host genotypes, rather than to a change in pathotype.

Agar Plate Method. The numbers of accessions received in each season, and the numbers of cultures successfully established on seedlings in the glasshouse are shown in Table 4.4. From 1981, there was a significant

TABLE 4.2

Infection types observed on Avocet R when inoculated with pure or mixed cultures of pathotypes 104 E137 A- and 104 E137 A+

Pathotype Mixture 104 E137 A-:104 E137 A+	Source of Inoculum	
	Original Mixed Inoculum	Mixed Inoculum Increased on Sonora
1:0	;N	1CN
0:1	3+	3+4
8:1	2C	3C
4:1	23C	3C
2:1	33-C	3C
1:1	3C	3C
1:2	3CN	3C
1:4	33+C	33+C
1:8	33+	3++C

TABLE 4.3

Infection types produced on seedlings of VPM1, and derived lines, by three selected isolates of *Puccinia striiformis tritici* obtained from the respective lines

Source of Isolate	Infection Type on VPM1	Original Line
VPM1/4*Cook:		
26352	1C	;N,12C
26355	1C	;N,1C
26356	11-C	;N,2+3C

TABLE 4.4

Numbers of stripe rust accessions received and numbers of viable cultures established in annual surveys from 1980 to 1984

Year	No. accessions	No. isolates	% establishment
1980	207	29	14
1981	371	225	61
1982	170	141	83
1983	656	520	79
1984	655	508	78

improvement in recovery of isolates due to the introduction of the agar plate method. Using this method, fresh urediniospores were produced from uredinia on diseased tissue. Recovery of cultures from accessions appeared to stabilise at around 80% of those received.

Material received in poor condition, particularly those with desiccated, senescent or decayed leaves, could not be satisfactorily revived using the technique.

4.3.2.2 Pathotype description

The infection types produced by *P. striiformis tritici* pathotypes determined in the Annual Rust Surveys from 1979 to 1984 are listed in Table 4.5. The responses for some of the differentials with various pathotypes determined in the eastern Australian wheat belt are illustrated in Plate 4.1

P. striiformis samples established from the initial epidemic in eastern Australia in 1979 were identified as 104 E137 (McIntosh, 1980). This identification was subsequently confirmed by R.W. Stubbs (1980). Pathotype 104 E137 was virulent for host resistance genes *Yr2, Yr3* and *Yr4*. In 1985, 17 isolates stored in liquid nitrogen from the 1979 and 1980 surveys were inoculated onto Avocet R. All were avirulent.

TABLE 4.5

Usual infection types produced by pathotypes of *P. striiformis tritici* detected in Australia

Differential Tester	Designated Resistance Gene	Decanery Value	Pathotypes					
			104 E137 A-	104 E137 A+	106 E139 A-	108 E141 A-	108 E141 A+	360 E137 A-
World Set								
Chinese 166	*Yr1*	1	0;	0;	0;	0;	0;	0;
Lee	*Yr7*	2	;N	;N	33+	;N	;N	;N
Heines Kolben	*Yr6*	4	;N1	;N1	;N1	3+	3+	;N1
Vilmorin 23	*Yr3*	8	3+	3+	3+	3+	3+	3+
Moro	*Yr10*	16	0;	0;	0;	0;	0;	0;
Strubes Dickkopf		32	3+4	3+4	3+4	3+4	3+4	3+4
Suwon 92/Omar		64	4	4	4	4	4	4
Clement	*Yr9*	128	0;	0;	0;	0;	0;	0;
Triticum spelta album[1]	*Yr5*	256	0;	0;	0;	0;	0;	4
		Total[2]	104	104	106	108	108	360
European Set								
Hybrid 46	*Yr4*	1	33+C	33+C	33+C	33+C	33+C	33+C
Reichersberg 42	*Yr7*	2	;N1-	;N1-	4	;N	;N	;N
Heines Peko	*Yr6*	4	;N1	;N1	;N1	3+	3+	;N1
Nord Desprez	*Yr3*	8	4	4	4	4	4	4
Compair	*Yr8*	16	0;	0;	0;	0;	0;	0;
Carstens V		32	;C	;	;	;C	;C	;C
Spaldings Prolific		64	;C	;C	;C	;C	;C	;C
Heines VII	*Yr2*	128	33+	3+	33+	33+	3+	33+
		Total[2]	137	137	139	141	141	137
Australian Set								
Avocet R[1]	*YrA*		;1	3+	;C	;;C	3+	;C
Oxley	*Yr6*		;N1	;N1	;N1	3+	3+	;N1
Sonora			4	4	4	4	4	4
			A-	A+	A-	A-	A+	A-

[1]proposed additions to the differential testers described by Johnson *et al.* (1972).

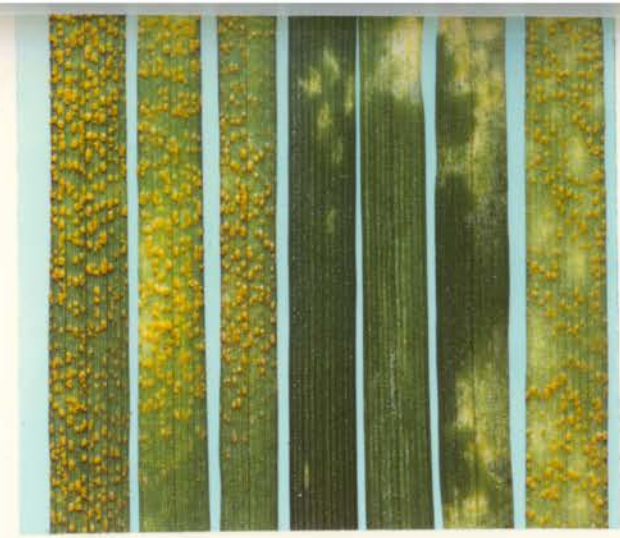
[2]totals obtained by adding decanery values of susceptible cultivars.

PLATE 4.1

Pathotypes of *Puccinia striiformis f.sp. tritici* detected in Australia from 1979 to 1984

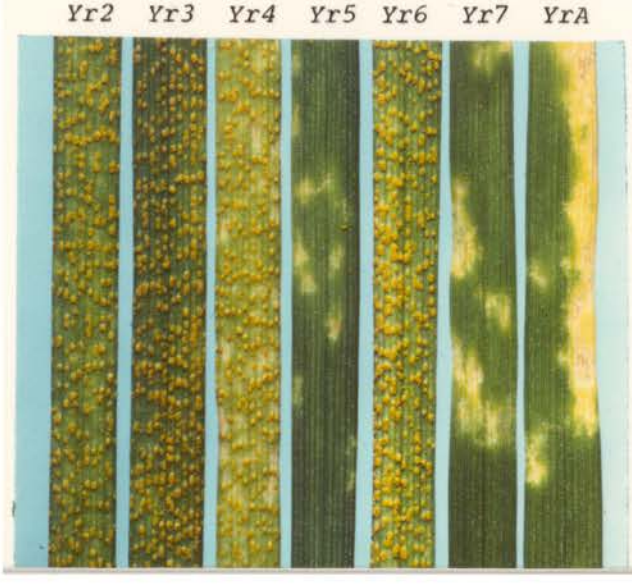
Yr2=Heines VII
Yr3=Vilmorin 27
Yr4=Hybrid 46
Yr5=*T. spelta album*
Yr6=Heines Kolben
Yr7=Lee
Yr8=Avocet R

104 E137 A-

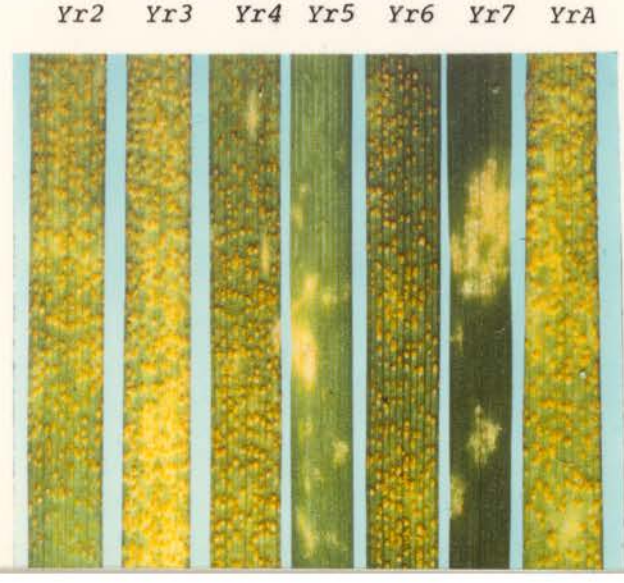


104 E137 A+

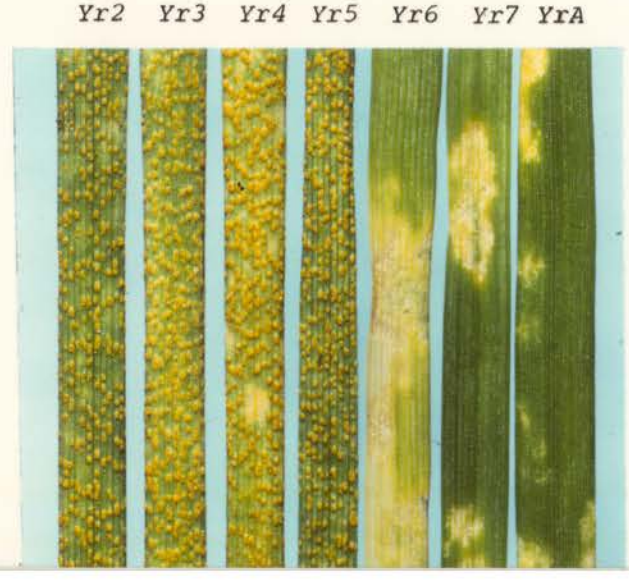
108 E141 A-



108 E141 A+



360 E137 A-



Thus, as far as can be ascertained, the original introduction in 1979 was pathotype 104 E137 A-. In 1980, stripe rust was discovered for the first time on wheat in the Southland region of New Zealand. Samples submitted to P.B.I., Castle Hill, were identified as pathotype 104 E137 A-. The occurrence of stripe rust in New Zealand is assumed to have resulted from the wind transport of urediniospores from eastern Australia. Wind has been repeatedly implicated in the movement of *P. graminis tritici* spores between Australia and New Zealand (Beresford, 1982; Luig, 1985).

In 1981, reports of unusually high incidence and severity of stripe rust on cultivar Avocet were received from irrigation districts in central and southern N.S.W. Field inspection by the author confirmed reports suggesting that resistance in this recently-released cultivar had become ineffective. Cultures established from rusted samples from the affected crops were shown to be pathotype 104 E137. However, in the overall survey, seedling infection types produced on cv. Avocet varied from resistant to susceptible. At first the nature of this variability was difficult to understand because of the small numbers of plants used in the tests. Further testing showed that Avocet gave a genetically heterogeneous response with infection types ranging from ;CN, ;12, 33+. Samples from severely rusted Avocet crops produced susceptible responses on all seedlings of Avocet. It was hypothesised that the increased rusting of Avocet was related to virulence for the seedling resistance present in some plants of Avocet and subsequently found in some, or all, plants of other cultivars (Section 5.4.1.1).

Two pathotypes were designated: 104 E137 A- and 104 E137 A+ which were avirulent and virulent, respectively, for the resistance in Avocet. The data in Table 4.6 were compiled to show the relationship between the respective pathotypes and the cultivars from which the diseased samples were taken. Clearly, in November 1981, there was an obvious association between the A+ pathotype and Avocet, despite large areas sown to cultivars

TABLE 4.6

Frequency of pathotypes in relation to source of rusted samples for November, 1981 in New South Wales

Source of Collection	Pathotype Frequency[1]	
	104 E137 A-	104 E137 A+
Avocet (*YrA*)	0.04	0.36
Other cultivars	0.40	0.20

[1]Based on 78 accessions.

TABLE 4.7

Frequency of pathotypes in relation to source of rust collection in eastern Australia in 1984

Source of Collection	Pathotype Frequency[1]	
	108 E141 A-	108 E141 A+
Millewa (*Yr6*)	0.53	0.27
Takari (*Yr6*)	0.05	
Oxley (*Yr6*)	0.02	
Bindawarra (*Yr6*)	0.02	0.02
Other cultivars	0.07	0.02

[1]Based on 42 accessions.

Egret and Condor which were later shown to be heterogeneous for the Avocet resistance. The latter cultivars were subsequently shown to possess greater levels of residual adult-plant resistance compared to Avocet. Occurrences of 104 E137 A+ collected from other cultivars were noted to increase late in the season. This was due to the availability of susceptible and/or moderately susceptible wheats such as Teal and Kewell.

In 1982, the cultivar Oroua became severely infected on the south island of New Zealand. Pathotype 106 E139 A- was identified from heavily rusted samples. This pathotype had additional virulence on the *Yr7* testers, Lee and Reichersberg 42, although in all other respects, it was identical with pathotype 104 E137 A-.

The 1983 epidemic was severe in extent and intensity, and resulted in the appearance of two new pathotypes late in the season. Pathotype 108 E141 had additional virulence on the *Yr6* testers Heines Kolben and Heines Peko. Variation on Avocet allowed the distinction between 108 E141 A- and 108 E141 A+ types. Although only ten isolates of these pathotypes were obtained (six A- and four A+) nine came from cultivars with the *Yr6* resistance, particularly Millewa and Bindawarra.

In 1984, 42 survey samples were identified as having virulence for *Yr6* (Table 4.7). Thirty eight were obtained from cultivars possessing *Yr6*. Eighty per cent of these samples came from Millewa crops, probably reflecting the greater areas sown to this cultivar. The 1983 and 1984 data again demonstrated the close association between a new pathotype and cultivars having the matching gene for resistance, especially in the early stages of pathotype establishment.

Two 1984 accessions were tentatively designated as pathotype 360 E137 A-. One of these came from Forbes (central-western N.S.W.) in October. These accessions were virulent for *Yr5* which is present in the proposed differential host *Triticum spelta album*. *Yr5* does not occur

in any commercial Australian cultivar. The field collections were routine samples and the crops from which samples of this pathotype were collected were not noted as having unusually high levels of rust. It is of interest that *Yr5*-virulent isolates of *P. striiformis* have not been reported from Europe. However, *Yr5*-virulent isolates have been reported in India (Sawhney and Luthra, 1970; Nagarajan, 1983) where the pathotypes appear to be distinctive from those present in Australia.

4.3.2.3 Pathotype distribution

The annual frequency distributions of pathotypes for various regions of Eastern Australia and New Zealand (Luig and Watson, 1970; see also Plate 4.2) are presented in Table 4.8. Stripe rust has not been found in Western Australia.

Following its introduction to eastern Australia in 1979, and to New Zealand in 1980, pathotype 104 E137 A- has been recorded from most wheat-growing areas in all seasons. Pathotype 104 E137 A+, first detected in 1981, has been frequently found in southern areas of eastern Australia. In 1984, it was detected in northern N.S.W. and was recorded for the first time in Queensland. One accession of pathotype 104 E137 A+ (811799) was recorded in New Zealand. Because of this single occurrence, and no subsequent detection, it may have been a laboratory contaminant that arose during processing at Castle Hill. In general, pathotypes 104 E137 A- and 104 E137 A+ have been predominant from 1981 to 1984.

Pathotype 106 E139 A- was first detected in New Zealand in 1982 and was recorded in each subsequent season. This pathotype has not been detected in eastern Australia, despite areas sown to commercial cultivars possessing the *Yr7* gene. In contrast, the *Yr6*-virulent pathotypes (108 E141 A- and 108 E141 A+) have been found in southern areas of eastern Australia and have not been recorded in Queensland or New Zealand. The apparent restricted distribution of *Yr6*-virulent pathotypes is probably largely influenced by

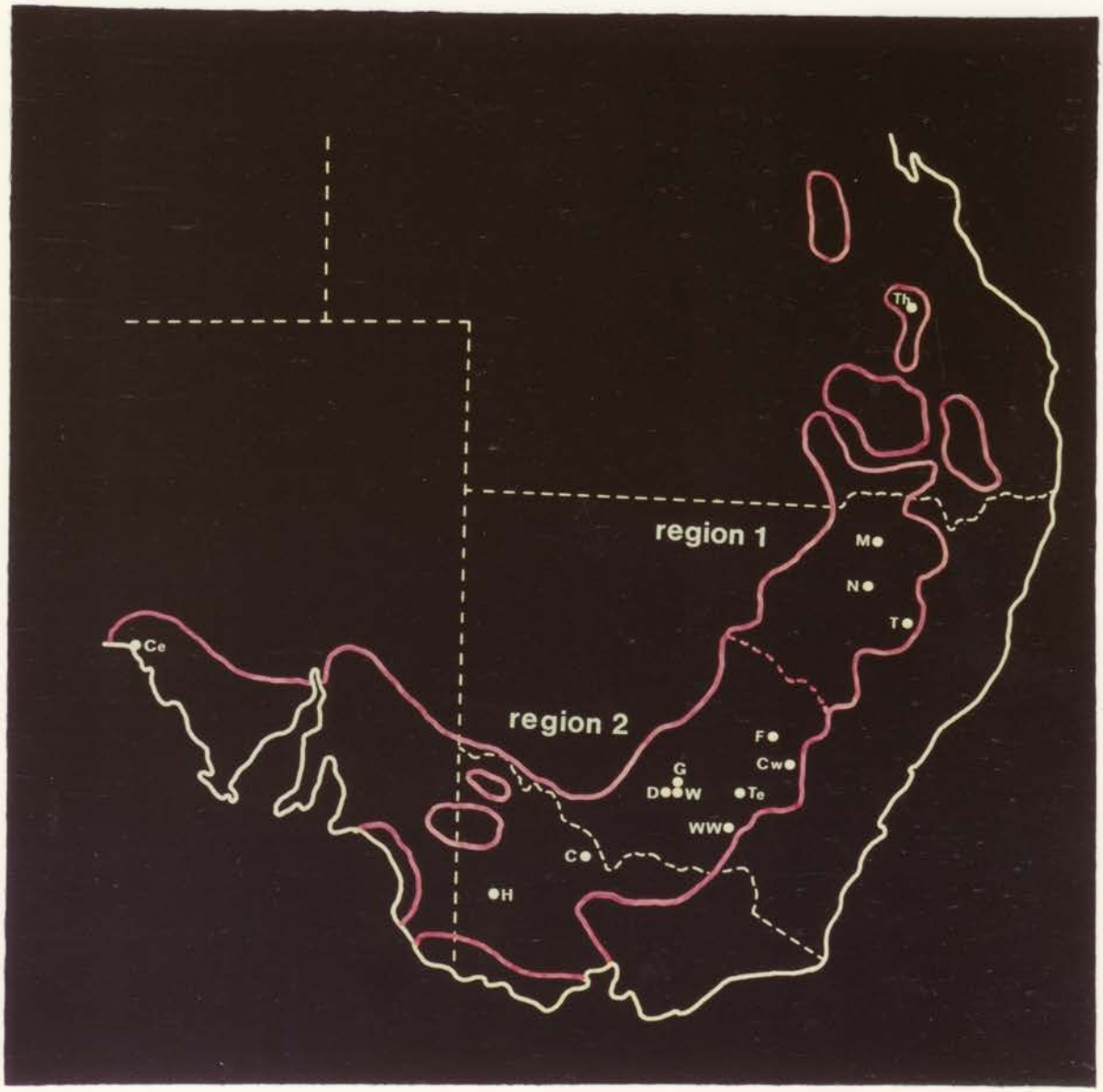


PLATE 4.2

Location of region 1, region 2 and certain towns in the eastern Australian wheat belt

C = Charlton	M = Moree
Ce = Ceduna	N = Narrabri
Cw = Cowra	T = Tamworth
D = Darlington Point	Te = Temora
F = Forbes	Th = Theodore
G = Griffith	W = Willbriggie
H = Horsham	WW = Wagga Wagga

TABLE 4.8

Frequency distributions of *Puccinia striiformis f.sp. tritici* pathotypes determined in Annual Rust Surveys from 1981 to 1984

Pathotype	Year	Region 1		Region 2				Region 4		Total	% of samples within years[1]
		Qld.	Nthn N.S.W.	Sthn N.S.W.	Vic.	Sth Aust.	Tas.	Nth N.Z.	Sth N.Z.		
104 E137 A-	1981	3	9	44	21	2	1	13	19	112	59
	1982		5	15			10	11	22	63	65
	1983	40	142	52	21	33	2	1	2	293	59
	1984	21	54	52	35	9	1	1	10	183	36
104 E137 A+	1981		2	64	7	1	4	1		79	41
	1982			28	4					32	33
	1983			99	59	34				192	38
	1984	5	23	127	63	26	1			245	48
106 E137 A-	1982								2	2	2
	1983								6	6	1
	1984							3	6	9	2
108 E141 A-	1983			2	2	2				6	1
	1984		1	13	12	3	10			39	8
108 E141 A+	1983			1	2	1				4	1
	1984		1	10	12		5			28	6
360 E137 A-	1984			2						2	0.4
Total		69	237	509	238	111	34	30	67	1295	

[1]total numbers for each year

1981	191
1982	97
1983	501
1984	506

the distribution in eastern Australia of cultivars possessing the *Yr6* gene.

Plate 4.3 maps the cumulative distribution of pathotypes as determined from Annual Survey samples, at the end of each calendar month in the eastern Australian wheat belt for the 1981 to 1984 seasons, inclusive. The maps show the progressive locations of pathotypes during each season and do not represent frequency distributions of rust incidence.

Stripe rust has been widely distributed throughout the eastern Australian wheat belt. Occurrences extended from Theodore, Queensland, in the north to Ceduna, South Australia, in the south-west (refer also to Plate 4.2). There were isolated occurrences of the disease in coastal areas of N.S.W. During the 1982 season, there was a severe drought throughout the eastern wheat belt resulting in relatively few stripe rust accessions. However, samples were received from crops grown under irrigation in central and southern N.S.W.

Plate 4.3 and Table 4.8 show that southern N.S.W. was the main area affected by stripe rust in each season, and also was the area from which the first isolates of new pathotypes were detected. Pathotype 104 E137 A- was the most widely distributed of the five pathotypes occurring in eastern Australia. In 1983, this pathotype was predominant in the epidemic that occurred in northern N.S.W. and Queensland. This suggested that 104 E137 A- survived the summer period in, and subsequently spread throughout, the region with little influence of inoculum from southern areas where both 104 E137 A- and 104 E137 A+ were present. Pathotype 104 E137 A+ was noted to spread rapidly after its first detection in November, 1981. It became common in southern areas in all seasons and appeared to spread to northern areas in 1984. The *Yr6* - virulent pathotypes showed less spectacular increases in southern areas following their initial detection in 1983. These two pathotypes also appeared to be moving northward in 1984. The two occurrences of 360 E137 A- in 1984 were widely separated in location and time of detection in southern N.S.W.

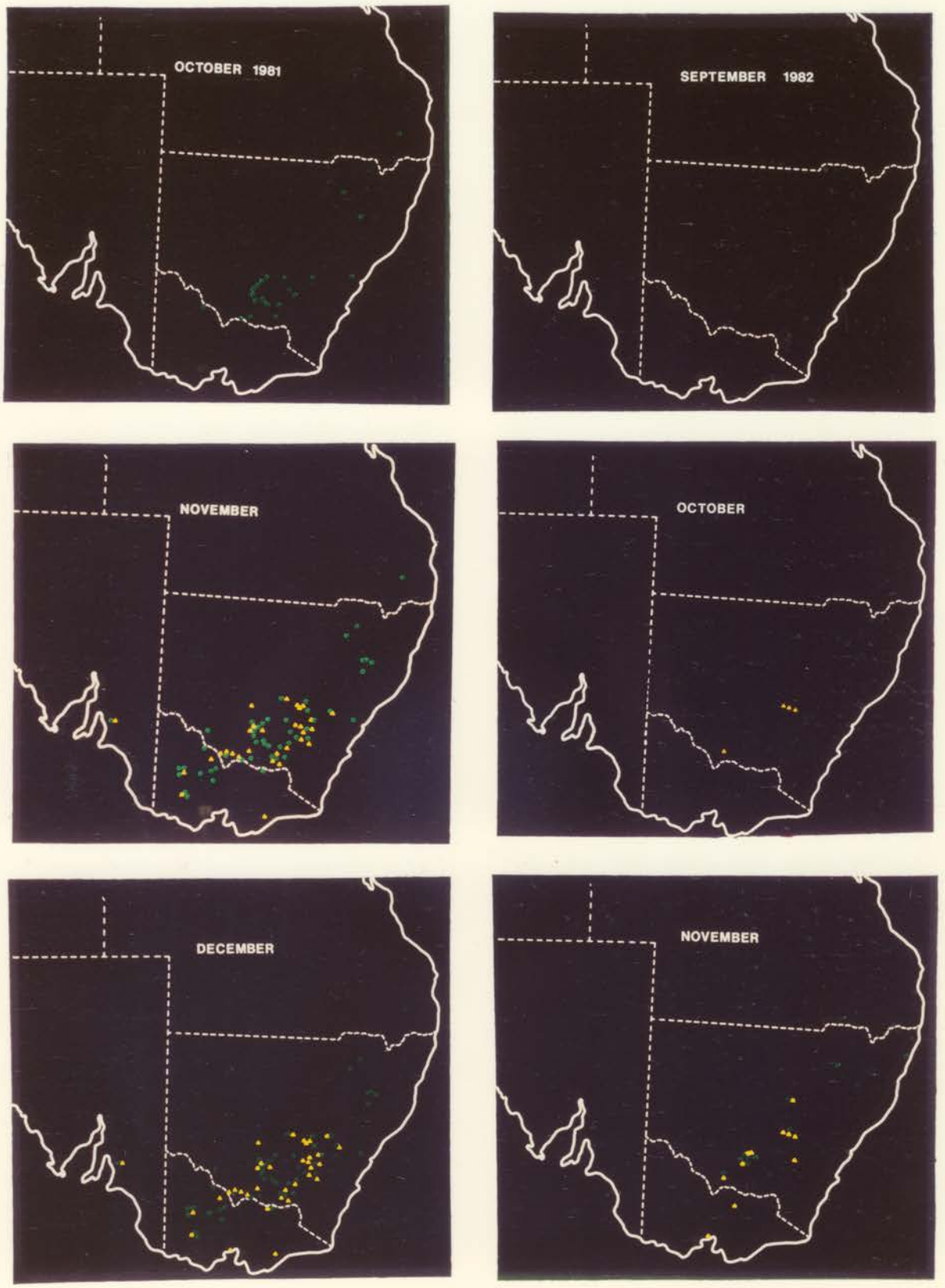
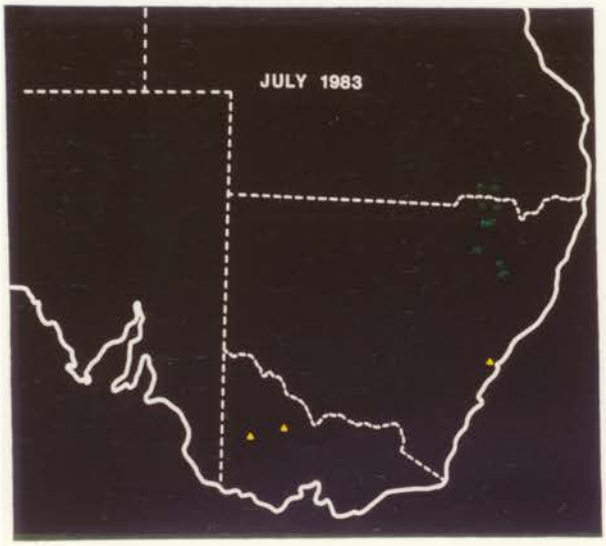


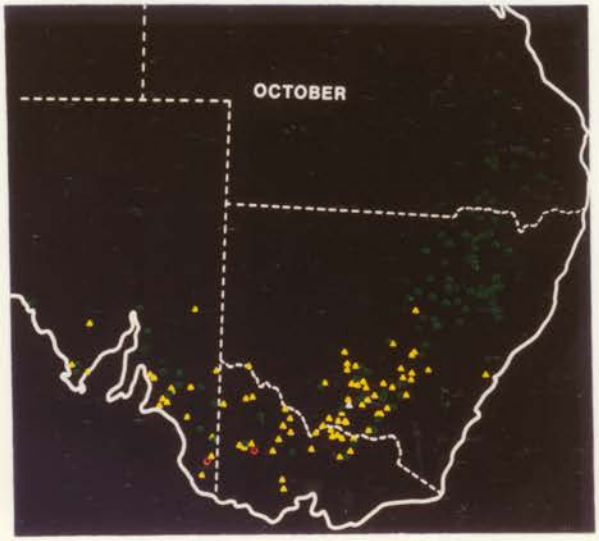
PLATE 4.3

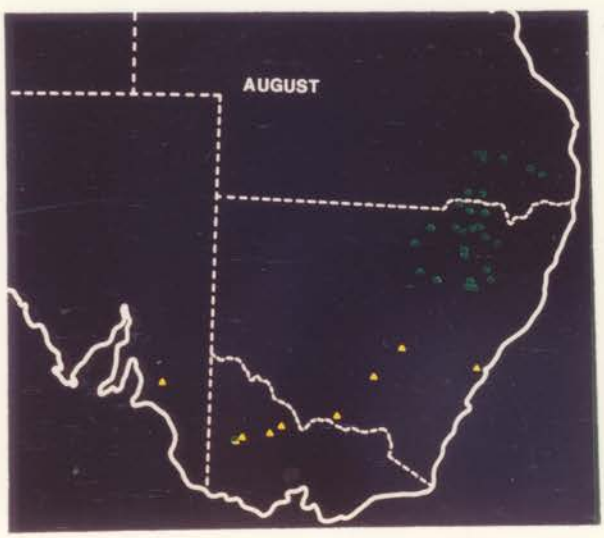
Cumulative distribution of pathotypes of *Puccinia striiformis f.sp. tritici* in eastern Australia in 1981,1982,1983 and 1984

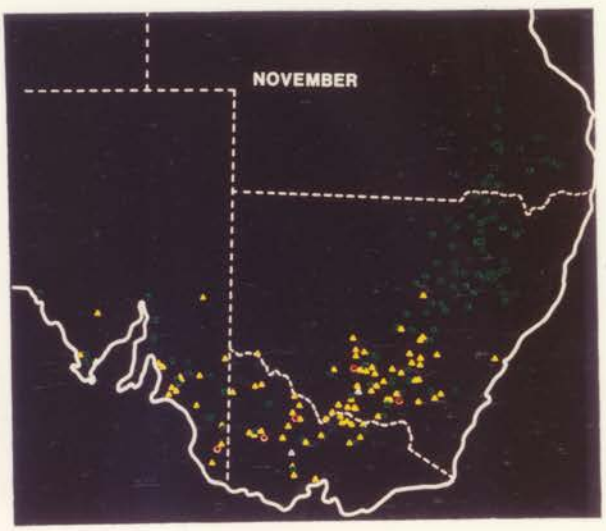
104 E137 A- = green dots

104 E137 A+ = yellow triangles









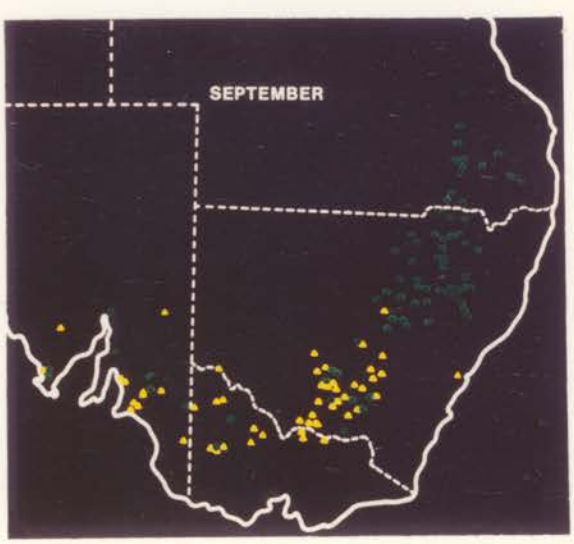
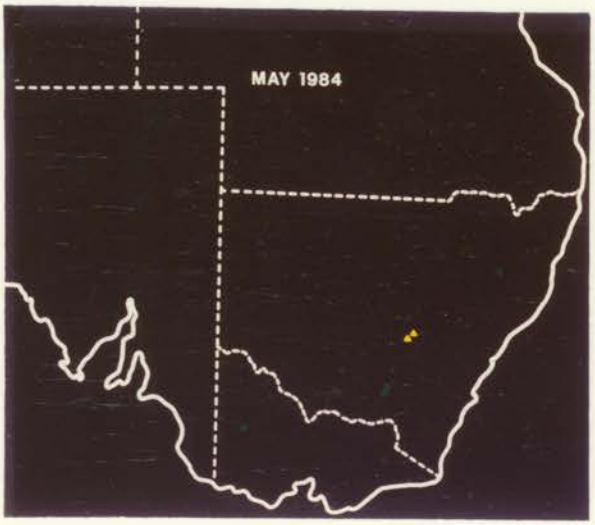
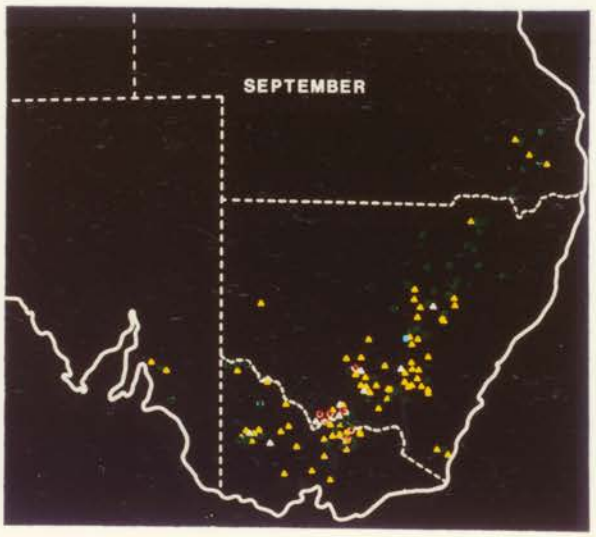


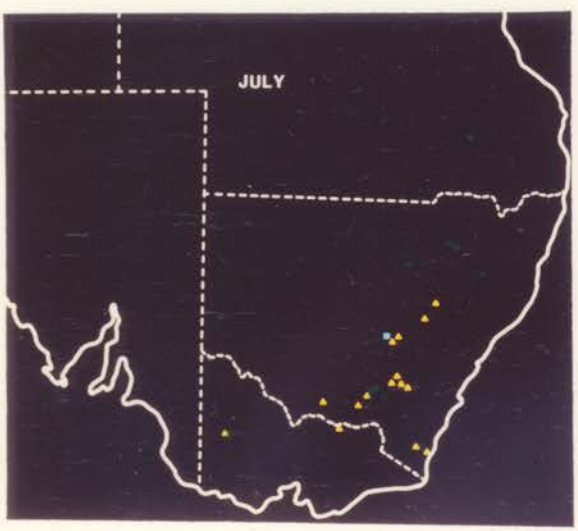
PLATE 4.3 Cont

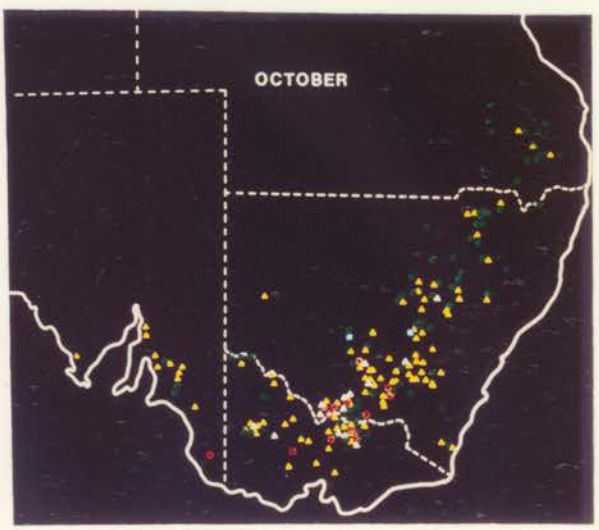
Cumulative distribution of pathotypes of *Puccinia striiformis f.sp. tritici* in eastern Australia in 1981, 1982, 1983 and 1984

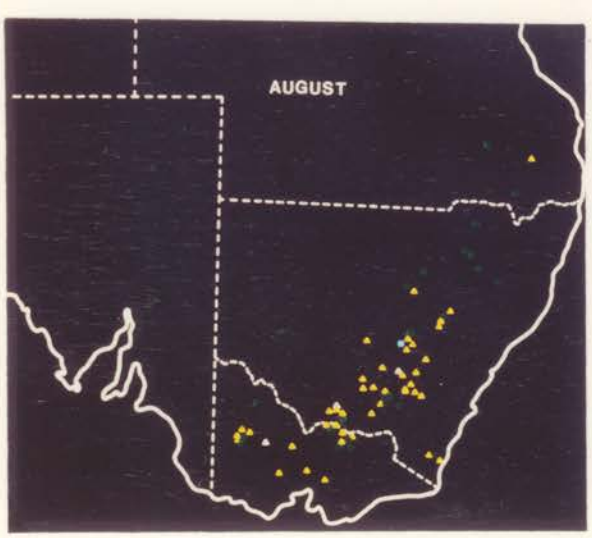
104 E137 A- = green dots
104 E137 A+ = yellow triangles
108 E141 A- = red circles
108 E141 A+ = white triangles











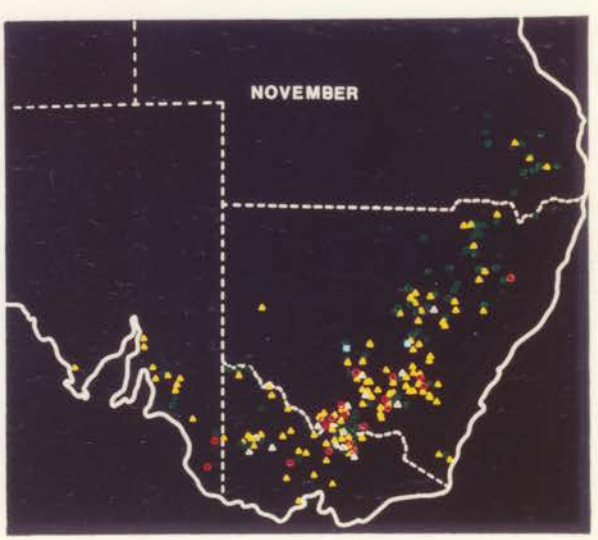


TABLE 4.3 Cont

Cumulative distribution of pathotypes of *Puccinia striiformis f.sp. tritici* in eastern Australia in 1981, 1982, 1983 and 1984

104 E137 A- = green dots
104 E137 A+ = yellow triangles
108 E141 A- = red circles
108 E141 A+ = white triangles
360 E137 A- = blue box

The progressive distribution of pathotypes suggested that the inoculum movement was generally localised within broad regions. This was particularly evident in the 1983 epidemic in northern N.S.W. and Queensland where 104 E137 A- was dominant, while southern N.S.W. and Victoria were mainly influenced by 104 E137 A+. However, movement of inoculum from south to north was evident in the distribution of pathotype 104 E137 A+ in 1984, and was possibly aided by increasing areas sown to cultivars with the '*Yr*A' (Avocet type) resistance, *e.g.* Sunstar.

The initial occurrences of different pathotypes at widely separated locations during early epidemic development in each year suggested that the pathogen survived independently at different locations in the wheat belt. Pathotypes first detected in an area were noted to reflect the predominant pathotypes found in that area during the previous season. The pathotypes first detected in a particular location also appeared to influence the subsequent pathotype composition during epidemic development.

4.3.2.4 Pathotype evolution

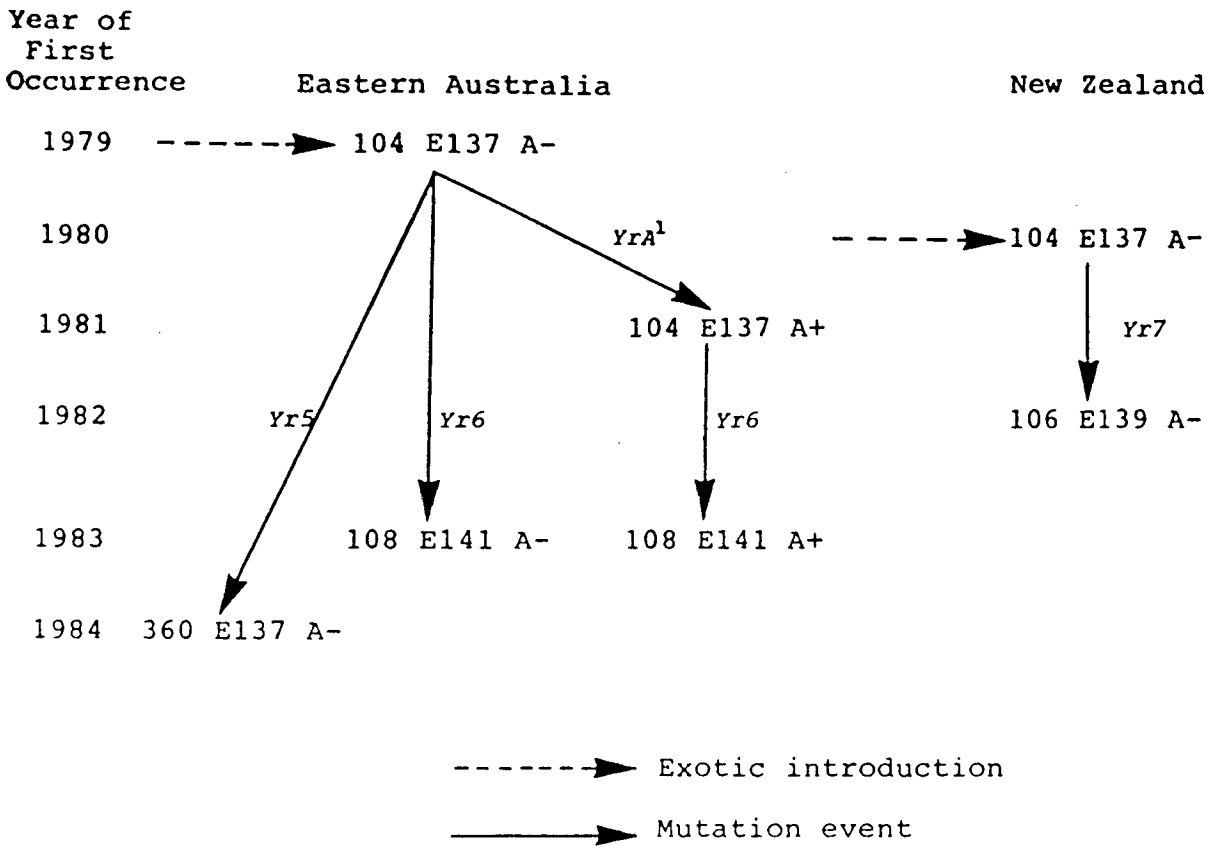
Since the introduction of pathotype 104 E137 A- to Australia in 1979, and to New Zealand in 1980, five variants have been found. This resulted in the presence of six pathotypes in the Australasian region after six seasons.

The six pathotypes recorded in 1984 appeared to be very similar. Figure 4.1 illustrates the sequence of their appearance in Australia and New Zealand. Each variant pathotype was assumed to have arisen as a consequence of mutation from avirulence to virulence with respect to a particular source of host resistance. The close step-wise relationships between pathotypes clearly indicated that single-gene mutational changes were occurring in the pathogen, and that further exotic introductions of *P. striiformis tritici* had not occurred during the period 1979 to 1984.

The increases in frequency of new pathotypes were related to the areas sown to cultivars possessing the matching genes for resistance. The relationships between

FIGURE 4.1

Chronology of the first occurrence, virulence change and probable origins of *Puccinia striiformis tritici* pathotypes in eastern Australia and New Zealand



[1]presumed mutation in the pathogen from avirulence to virulence with respect to a single source of resistance.

the areas sown to cultivars with *YrA*, or *Yr6*, and the frequencies of virulences for these resistances in the pathogen population in southern N.S.W. are illustrated in Figure 4.2. The rapid increase in the frequency of virulence for *YrA* was due to the large area sown to cultivars Avocet, Banks, Condor and Egret. It was also possible that virulence for *YrA* was present in 1980, but was undetected due to poor establishment of isolates in the glasshouse (Section 4.3.2.1) and inexperience of research personnel restricted to temporary growth cabinet facilities. For example, one report of a severely rusted Avocet crop was received from northern Victoria in 1980 some time after the crop had been harvested. Consequently, no disease sample was obtained.

The frequency of the *Yr6* virulence was also closely related to the area sown to cultivars Millewa and Oxley. (Figure 4.2).

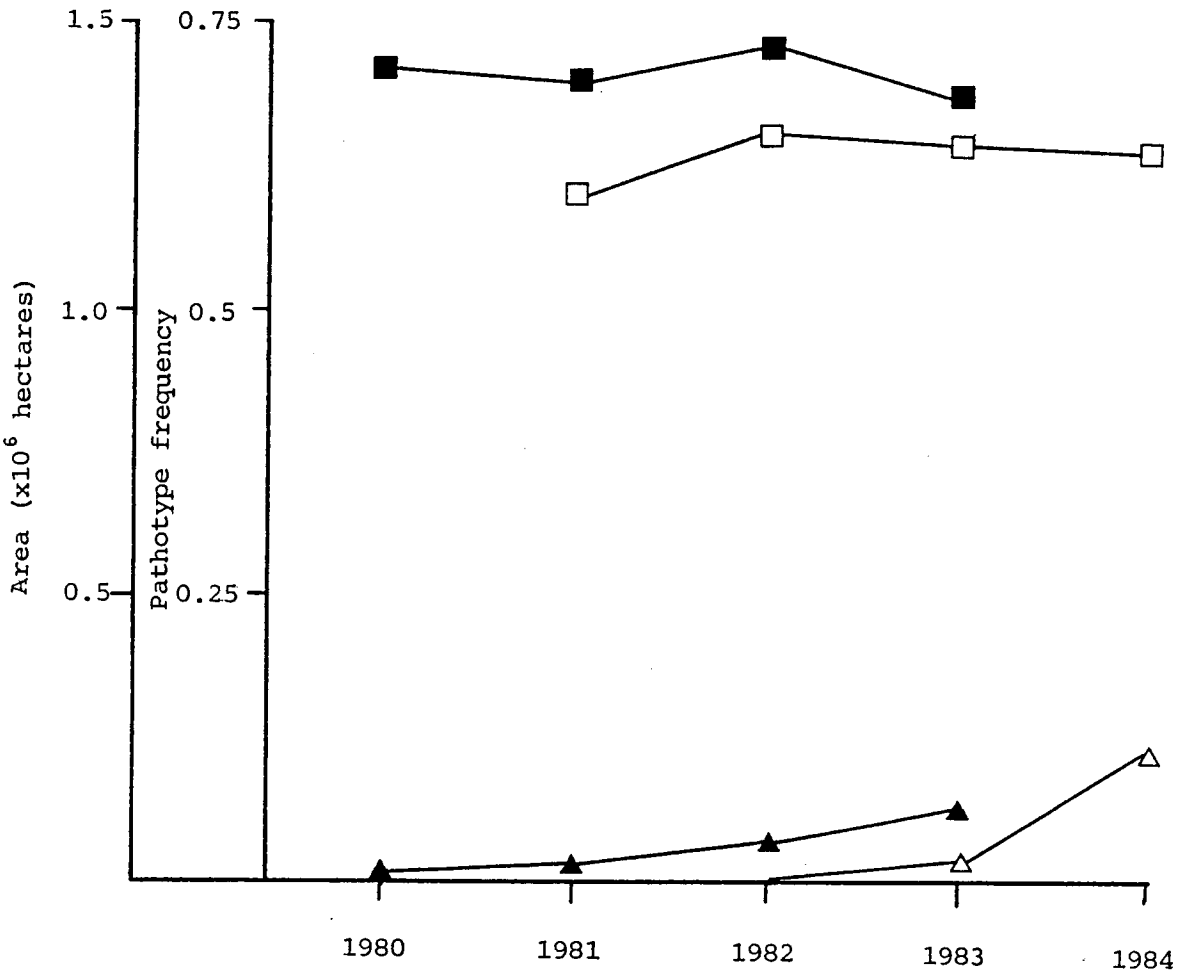
4.3.2.5 Adult-plant tests

Experiment 4.3. Twenty-three accessions of pathotype 104 E137 A+ produced responses on cv. Avocet ranging from 70MS-S to 100S. Four accessions of pathotype 104 E137 A- produced responses ranging from 40R-MR to 50MR-MS. The susceptible control, WW232, ranged in response from 80MS-S to 100S with both pathotypes. This clearly demonstrated that isolates avirulent or virulent on Avocet seedlings were correspondingly avirulent or virulent on adult plants of Avocet.

Five isolates of known pathogenicity were inoculated on to adult plants of a range of cultivars and advanced lines (Table 4.9). The responses to isolates of 104 E137 A+ was high, compared to 104 E137 A-, among wheats with the *YrA* resistance. The lines WW237 and DK2165 showed a similar differential response to the two pathotypes, suggesting the presence of *YrA* in these wheats. The *Yr6* and *Yr7* host resistance genes remained effective against all isolates. However, the response of Cook in this experiment was considerably higher than that experienced in the field (Section 5.4.3).

FIGURE 4.2

Relationship between total area sown to cultivars with particular resistances and the frequencies of pathotypes virulent for the corresponding resistance genes in southern N.S.W.



■ Σ area sown to cv.s Avocet, Banks, Condor and Egret.[1]

□ frequency of *YrA* virulence.

▲ Σ area sown to cv.s Millewa and Oxley.[1]

△ frequency of *Yr6* virulence.

[1] from information supplied by R.W. Fitzsimmons, N.S.W. Department of Agriculture.

TABLE 4.9

Adult-plant response of cultivars in the glasshouse to pathotypes of *Puccinia striiformis tritici* which were avirulent or virulent for *YrA*

			Accession, Pathotype				
	Cultivar[1]	Seedling Resistance[2]	811709 104 E137 A-	811602 104 E137 A+	811591 104 E137 A+	811595 104 E137 A+	811716 104 E137 A+
Group 1							
	Avocet R[3]	*YrA*	20MR	100S	90S	90S	90S
	Banks R	*YrA*	30MR-MS	80MS-S	80MR-MS	80MS	30MS
	Vulcan	*YrA*	30MR	70MS-S	90S	80MS-S	80MS
	WW15	*YrA*	10MR-MS	80S	80MS-S	80MR-MS	40MS-S
	Eagle	*Yr7*	10R	20R	30R-MR	10R	20R
	Cook	-	20MR-MS	60MS	60MR-MS	50MS	50MR-MS
	Kite	-	30MS	40MR-MS	50MR-MS	50MS	30MS
	WW237		5R	80MR-MS	50MS	50MR-MS	50MR-MS
	DK2165		30MS	80S	80S	60MS-S	70MS
Group 2							
	Egret R	*YrA*	5R-MR	60S	40MS-S	50MS	30MS-S
	Corella	*Yr7*	OR	OR	5R	OR	OR
	Oxley	*Yr6*	5R	5R	5R	5R	5R
	Harrier	-	40MS-S	80S	40S	50MS-S	70S
	M2397		30MS-S	30MS-S	50S	50MS-S	70S
Group 3							
	Osprey	-	30MS-S	30S	60MS-S	80S	40S
	M2391		30MS-S	50MS-S	60MS-S	50S	30MS-S

[1]Cultivars arranged in growth stages: Group 1 60-65, Group 2 40-45, Group 3 30-40.

[2]Seedling resistance genes indicated where known; "-" indicates no seedling resistance.

[3]R indicates progenies of resistant plants from genetically heterogeneous cultivars.

Experiment 4.4 The results for four pathotypes inoculated on to a range of cultivars at two temperature regimes are presented in Table 4.10. As expected, cultivars with *Yr6* were higher in response to pathotypes with *Yr6* virulence. Similarly, *YrA*-possessing cultivars were higher in response to *YrA*-virulent pathotypes. Cultivars without the *YrA* or *Yr6* resistances showed no obvious differential interaction with the four pathotypes. It was observed that responses of some cultivars in these experiments were higher than responses observed in the field. For example, Oxley showed higher responses to the *Yr6*-virulent pathotypes in this experiment compared to field responses to the same pathotypes (Section 5.4.3).

The higher temperature resulted in lower responses for most cultivar x pathotype combinations. However, the above trends of cultivar x pathotype interactions were unaffected by temperature.

Experiment 4.5 The results of glasshouse experiments using ten pathogen isolates collected from commercial crops of cv. Banks are present in Table 4.11. In this experiment, the responses of adult plants of Banks ranged from tVR to 5R-MR. The responses of the susceptible controls ranged from 5MS to 60MS-S. There was thus no evidence in these experiments of increased virulence with respect to the adult-plant resistance of Banks. However, the response of Banks to these pathotypes was extremely low compared to Experiment 4.3 (Table 4.9). This may have been due to lower levels of inoculum in this experiment, or to differences in environment between experiments.

These discrepancies between experiments and the observed differences in response of some cultivars between glasshouse and field tests emphasises the difficulties involved in the conduct and interpretation of glasshouse tests for adult-plant responses to *P. striiformis tritici*.

4.3.3 EPIDEMIOLOGY OF STRIPE RUST IN EASTERN AUSTRALIA

4.3.3.1 Epidemic onset

Table 4.12 lists the date, location and pathotype of the first reports of stripe rust in commercial crops

TABLE 4.10

Adult-plant responses, in the glasshouse, of cultivars to four *Puccinia striiformis tritici* pathotypes at two temperature regimes

	Accession		831795		821559		832002		831917	
	Pathotype		104 E137 A-		104 E137 A+		108 E141 A-		108 E141 A+	
	Temperature		15C	20C	15C	20C	15C	20C	15C	20C
	Cultivar[1]	Resistance								
Group 1										
	Sunkota	*Yr7*	OR	tR	5R	OR	tR	OR	tR	OR
	Avocet R	*YrA*	-	-	-	-	50MR-MS	20MR	80MS	50MS
	Sunstar	*YrA*	5R-MR	5R	40MR-MS	40MR	20MR	30MR-MS	40MS	20MR
	Millewa	*Yr6*	5R	5R	tR	5R	50MS	10MR	40MS	10MR
	Miling	*Yr6*	5R-MR	5R	5R	5R	80MS	10MR	60MS	30MS
	Takari	*Yr6*	5R	OR	tR	tR	30MS	10R-MR	20MR	20MS
	Kite	-	20MR-MS	10MR	30MR	10R	30MS	5R	20MR	5R
	Condor S	-	50MS-S	20MR-MS	60MS-S	30MR	80MS	40MS	60MS-S	50MS
	Torres	-	30MS-S	20MR-MS	90MS-S	40MR	90MS-S	30MS	70MS-S	50MS
	Bass	-	40MS	10MR	60MS	20MR-MS	80MS-S	20MR-MS	40MS	20MR
Group 2										
	Bindawarra	*Yr6*	20MR	5R	10R-MR	5R	40MS	10MR	60MS	30MR-MS
	Oxley	*Yr6*	5R	5R	5R	5R	50MS	20MS	30MR	30MS
	Jacup	*Yr6*	10R	-	10R	10R	20MS	-	30MR-MS	50MS

[1]Group 1 cultivars at growth stage 55-60.

Group 2 cultivars at growth stage 40-45.

TABLE 4.11

Adult-plant responses in the glasshouse for a selection of cultivar Banks and susceptible checks to *Puccinia striiformis tritici* isolates collected from commercial crops of Banks

Survey Accession Number	Pathotype	Response on: Banks S[1]	Susceptible
Trial I			Warigal:
831576	104 E137 A-	tVR	5MS
831792	104 E137 A+	tVR	30MS
831763	104 E137 A+	tR	20MS
831849	104 E137 A+	tVR	60MS-S
831866	104 E137 A+	tVR	5MS
831890	104 E137 A+	tVR	30MS-S
831891	104 E137 A+	tR	10MS
831924	104 E137 A-	tR-MR	20MS
831973	104 E137 A-	tVR	20MS
Trial II			Teal:
831737	104 E137 A+	5R-MR	50MS-S
831849	104 E137 A+	tR	40MS-S
831866	104 E137 A+	OR	30MS
831890	104 E137 A+	tR	40MS-S

[1]selection of cultivar Banks lacking the *YrA* resistance.

for each season since the initial introduction in 1979. The various locations are illustrated in Plate 4.2. In the first four seasons, stripe rust appeared in spring. A marked change occurred from 1983 when the disease was first reported in the winter months. The early occurrences in 1983 and 1984 resulted in increased incidence and severity of disease compared to previous seasons.

The data suggest that stripe rust survived at independent locations which varied between seasons. The development of different pathotypes at different locations clearly indicated that survival and early epidemic development

TABLE 4.12

Date, location and pathotype of the first reports and/or samples of stripe rust in eastern Australia from 1979 to 1984

Year	Date	Pathotype	Location	District
1979	18 October		Darlington Point	south, N.S.W.
	21 October		Cohuna	Victoria
	30 October	104 E137[1]	Tamworth	north, N.S.W.
1980	26 Sept.		Willbriggie	south, N.S.W.
	29 Sept.		Horsham	Victoria
	3 October		Temora	south, N.S.W.
1981	2 October	104 E137 A-	Griffith	south, N.S.W.
	2 October	104 E137[1]	Cowra	south, N.S.W.
	6 October	104 E137[1]	Wagga	south, N.S.W.
1982	11 October	104 E137 A+	Forbes	central, N.S.W.
	13 October	104 E137 A-	Narrabri	north, N.S.W.
1983	27 July	104 E137 A+	Horsham	Victoria
	28 July	104 E137 A-	Moree	north, N.S.W.
1984	16 May	104 E137 A+	Forbes	central, N.S.W.
	21 May	104 E137 A-	Wagga	south, N.S.W.
	22 May	104 E137 A-	Tamworth	north, N.S.W.

[1]virulence for *Yr*A not determined.

were localised. It was apparent that irrigation areas in central and southern N.S.W., centred around Griffith and Forbes, respectively, (Plate 4.2), were commonly associated with the first occurrences in most seasons. Summer irrigation in such areas presumably favoured the survival and out-of-season growth of wheat plants, thus providing suitable host materials for rust survival. However, stripe rust has not been identified in the over-summering phase in wheat-growing areas during the summer months ranging from January to April. Moreover, due to voluntary restrictions on the use of experimental stripe rust epidemics in wheat growing areas, no attempts have been made to encourage or study oversummering on an experimental basis.

4.3.3.2 Epidemic development

Regions 1 and 2 (Luig and Watson, 1970) represent

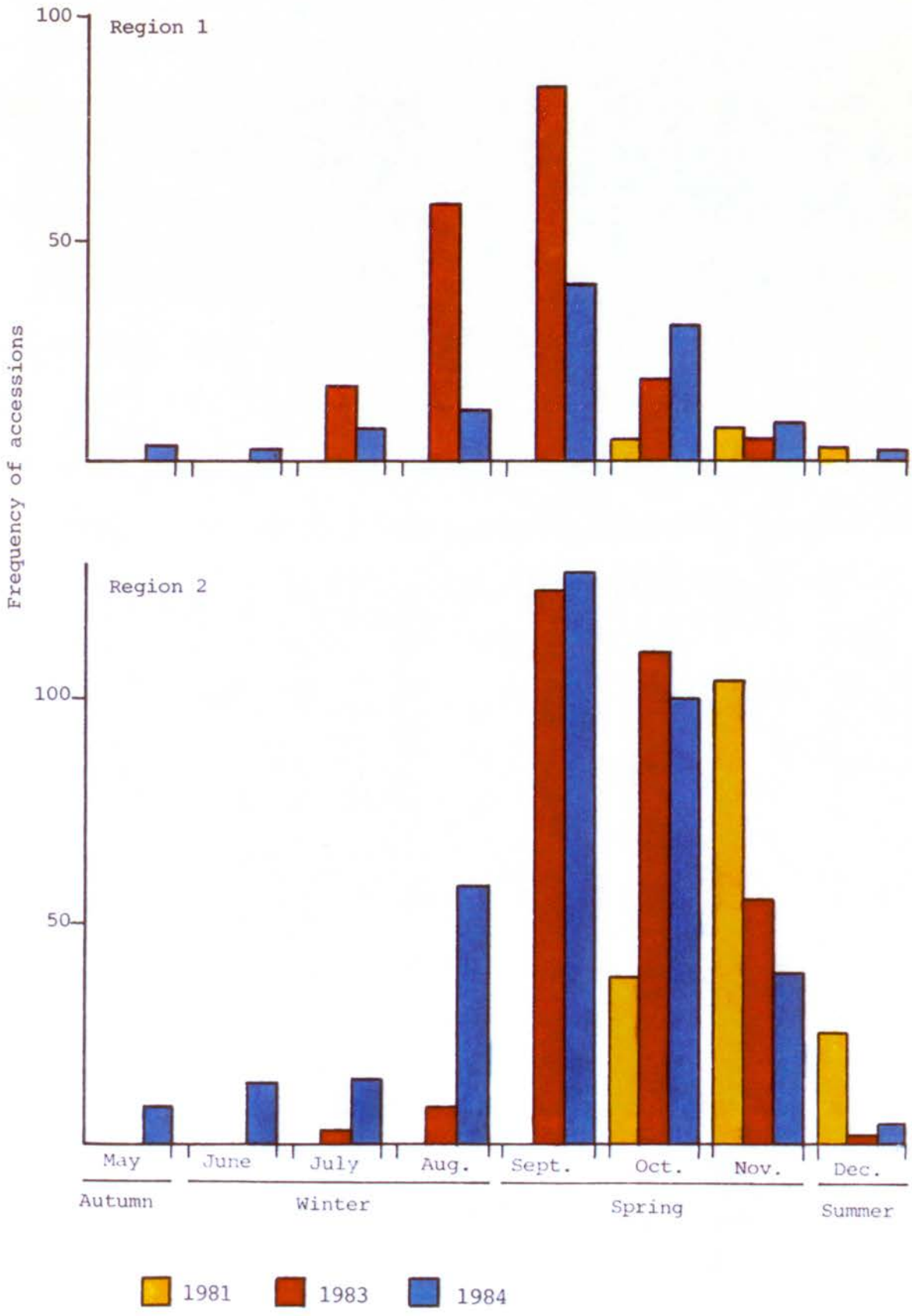
the main geographic divisions within the eastern Australian wheat belt and are illustrated in Plate 4.1. Region 1 is characterised by summer-dominant rainfall which requires a short (summer) or long (winter-spring-summer) fallow to allow moisture accumulation in the soil profile prior to autumn sowing. Region 2 has a more reliable winter-dominant rainfall pattern with cooler temperatures than Region 1. In general, time of sowing, crop development and time of harvest becomes increasingly delayed from Region 1 southward through Region 2.

The numbers of wheat stripe rust isolates for three seasons arranged according to date of accessioning and region of origin are illustrated in Figure 4.3. The 1981 season was typical of the seasons from 1979 to 1982. Epidemic onset and development occurred in the relatively short period of spring and early summer. Rust incidence in Region 1 was low and reported crop damage was negligible. Epidemics in Region 2 were regular and, in cultivars such as Zenith (Victoria, 1979 and 1980) and Avocet (N.S.W., 1981-83), crop losses in the order of 40-60% were reported (Murray *et al.*, 1985). The disease was recorded in Region 2 in all seasons from 1979, including the severe drought year of 1982 in which rust was found at low levels, particularly in irrigation areas. It was thus concluded that stripe rust was well-adapted to Region 2 and this disease is now regarded as endemic to the area.

The 1983 season, a wet year after drought conditions, was different. The epidemic began in mid-winter in both Regions 1 and 2. The rapid development of rust in Region 1 resulted in a peak in early spring which was similar in magnitude to the peaks in Region 2 in previous seasons (Figure 4.3). The epidemic appeared to decline quickly in spring in Region 1. Rapid disease development occurred in spring in Region 2 and then declined slowly in contrast to Region 1. The sudden peak in September in Region 2 may have been due to the introduction of inoculum from Region 1.

FIGURE 4.3

Frequency of stripe rust accessions for each month in Region 1 and Region 2 of the eastern Australian wheat belt in 1981, 1983 and 1984



The epidemic of 1983 was the most severe in extent and intensity since the introduction of stripe rust in 1979. In an attempt to protect crops, which in many cases were potentially the best for four years, farmers adopted the unprecedented strategy of large-scale application of fungicides. Estimates from Bayer Aust. Ltd. (R. Dawes, pers. comm.) and Ciba-Geigy Ltd. (J. Swain, pers. comm.) indicated that as much as 700,000 hectares were sprayed in N.S.W., representing approximately 20% of the area sown to wheat in N.S.W. in that year. This was the first occasion in which Australian farmers had used aerial applications of fungicide over large areas for disease control in wheat.

In 1984, the epidemic began in late autumn in both Regions (Figure 4.3). In contrast to 1983, Region 1 showed a slow rise in disease incidence through winter, a moderate peak in early spring and a slow decline. Region 2 experienced a greater incidence of disease through winter, resulting in a peak in spring which was similar to 1983. It was apparent that Region 1 contributed little inoculum to the epidemic in Region 2. Although disease incidence was similar to 1983, particularly in Region 2, fungicide applications were reduced to an estimated 100,000 hectares in N.S.W. (R. Dawes, pers. comm.). This was probably due to 1983 experiences which often failed to demonstrate an economic advantage from fungicide application (Marshall, 1984).

Under Australian conditions, wheat production is predominantly rain-fed with low fertiliser inputs compared with Europe. The relatively low yields, lack of expertise in large-scale spray application on wheat, and the absence of data for the relationship between disease response and expected yield loss in cultivars, were factors which led to variable experiences with fungicidal control strategies.

4.4 DISCUSSION

The introduction and rapid establishment of *P. striiformis f.sp. tritici* in eastern Australia in 1979

suggested that the pathogen may have been present, but undetected, earlier in the season or even in the previous season (Watson and Butler, 1984). The mode of entry of this pathogen into Australia is speculative. Luig (1977) reported the introduction of exotic pathotypes of *Puccinia graminis f.sp. tritici* in eastern Australia, and suggested that they may have originated in southern Africa. Comparative isozyme studies by Burdon *et al.* (1982) confirmed the hypothesis and Watson and de Sousa (1983) concluded that wind transport of inoculum from Africa to Australia was a distinct possibility. The initial appearance of stripe rust around Charlton (McIntosh, 1980), in contrast to multiple foci over a broad area resulting from an exogenous spore shower, does not favour inter-continental wind movement as a means of entry. The identification of 104 E137 as the pathotype present in Australia immediately suggested Europe as the source of inoculum. This pathotype was not present on the African continent or the Indian sub-continent (Stubbs *et al.*,1974) which were the most likely source if the initial inoculum had been wind-bourne. In confirming the pathotype identification, Stubbs (1980) reported that it was very similar to that found in southern Europe from 1977 to 1979. Hence it was suggested that the stripe rust pathogen was introduced into Australia from Europe due to urediniospore transport on man or his machines. Watson and Butler (1984) proposed that inoculum could have been retained in the undercarriage wheel bays of aircraft and released over wheat fields prior to landing at Melbourne. Considering the likelihood that the aircraft would have made previous landings prior to arrival in Australia, this hypothesis appears unlikely. The adherent nature of urediniospores (Rapilly, 1979) suggests that contaminated clothing or footwear worn or carried by a traveller from Europe is a more probable mode of entry of wheat stripe rust into Australia. It is clear that a person could be in contact with infected plants in Europe and non-infected plants in Australia within a period of less than 48 hours. An experiment demonstrated that urediniospores remain viable on clothing kept at room temperature for at least one week

(Wellings, unpublished). There is, however, no evidence of movement of possible rust-contaminated personnel from Europe to the Charlton area in 1978-79.

Following the initial detection in 1979, stripe rust was reported from widely separated locations within a short period of time. This suggested that the pathogen was transported over distances up to 900km in Australia. This potential for long distance dispersal was subsequently confirmed with the introduction of stripe rust into New Zealand, a distance of approximately 2,000km. Urediniospore movements have been reported to be variable. Zadoks (1961, 1967) observed inoculum transport up to 800km, while Hermansen and Stapel (1973) concluded that inoculum arrived in Denmark from England in 1971. Joshi *et al.* (1977) found stripe rust to be localised compared to the widespread nature of leaf rust in northern India. In contrast, Dubin and Stubbs (1985) observed the progressive movement of barley stripe rust over 6,000km in seven years following its introduction to Colombia, South America, in 1975. Rapilly (1979) suggested that relative humidity, which influenced the aggregation of spores into clumps, was an important determinant of the dispersal distance of *P. striiformis*. Thus, under relatively dry conditions in Australia and South America, urediniospores may not clump together and so could be capable of moving over long distances.

Stripe rust on cocksfoot was recorded in New Zealand in 1975, although it may have been introduced at some time in the previous 10 years (Latch, 1976). The recording of *P. striiformis f.sp. dactylidis* in Australia in 1979 was no doubt a consequence of heightened awareness of stripe rust following its occurrence on wheat. Thus the pathogen may have been present in Australia for a considerable period before detection. *Dactylis glomerata* is a perennial grass used in pastures in higher rainfall areas of the coast and adjacent tablelands. Stripe rust of cocksfoot does not appear to be an important disease under Australian conditions, although Latch (1976) noted

that responses of cocksfoot selections to this disease indicated that breeding for resistance could be worthwhile.

The remaining two forms of *P. striiformis, viz f.sp. hordeii* and *f.sp. poae*, have not been recorded or confirmed in Australia. These forms cause significant damage to their respective hosts overseas, and thus quarantine restrictions should not be relaxed in view of the presence of certain *f.sp.* of *P. striiformis* in Australia.

Experiments aimed at a determination of the host range of *P. striiformis tritici* on grasses in eastern Australia found that five genera in sub-family Pooideae could be involved. Overseas reports also indicated that the host range is limited to the Pooideae (Humphrey and Hungerford, 1924; Manners, 1950; Arthur, 1962). Saville (1979) notes that the leaf rust pathogen complex, which includes *P. coronata, P. striiformis, P. recondita, P. hordei* and *P. poae-nemoralis*, is largely confined to this grass sub-family. However, reports in the literature suggest a larger number of grass hosts than found in the present studies. These ranged from 59 species in 8 genera (Hungerford and Owens, 1923) to 230 species in 40 genera (Hassebrauk, 1965). This large range was often based on seedling inoculation tests in the glasshouse. Anikster (1984) warned that such studies result in wider host ranges than observed in nature. Zadoks (1961) noted that genetic variation in the grass hosts make conclusions difficult. Although the host range for *P. striiformis tritici* in Australia appears to be limited, the conclusions were based on field collections and observations in addition to inoculation experiments. However, in both glasshouse and field observations, relatively few genotypes within each species were assessed. Thus further experiments and observations will undoubtedly extend the host range for wheat stripe rust in Australia.

The host range of the wheat stripe rust pathogen on grasses has evolutionary and epidemiological significance. The first records of *P. striiformis* in the U.S.A. (Humphrey *et al.*, 1924) and Argentina (Humphrey and Cromwell, 1930) were collected on various grasses. Hassebrauk (1965)

speculated that *P. striiformis* was of Eurasian origin where, due to man's activities in plant domestication, it transferred from its initial wild grass hosts to cultivated forms, such as wheat and barley. Zadoks (1979) concluded that specialisation of the pathogen to cereals and/or grasses subsequently allowed the distinction of *formae speciales*.

The important practical implication of grass hosts is their potential role together with self-sown wheat in aiding the over-summer survival of the pathogen. Hendrix *et al.* (1965) found a range of grasses with actively sporulating stripe rust pustules during summer at high elevations in the Pacific Northwest. They established that the urediniospores were viable, but they did not confirm the pathogenicity of isolates on wheat. Tollenaar and Houston (1967) reported high altitude survival of the wheat stripe rust pathogen on grasses in California. Similar reports have come from Europe (Paichadze, 1974). However, Shaner and Powelson (1973) found that survival of the pathogen on grasses at high altitudes was less, and of shorter duration, than survival occuring on out-of-season wheat within the wheat-growing areas. The authors also suggested that pathotypes collected from grasses may be different from those occurring in wheat fields. Similarly, Zadoks (1961) concluded that grass hosts have a negligible role in the epidemiology of stripe rust in north-western Europe.

The means of over-summer survival of the stripe rust pathogen in Australia remains undetermined. R. Rees (pers. comm.) reported that the first record of stripe rust in Queensland for 1985 was on rough wheat grass, *Agropyron scabrum*. Although grass hosts are possible reservoirs of inoculum, it was evident from the present studies that a susceptible wheat is capable of producing greater quantities of inoculum than a susceptible grass. The first outbreaks of stripe rust in all seasons were located in wheat-growing areas often well removed from the cooler, higher slopes adjacent to the eastern wheat-

growing areas. Therefore, from overseas experience and from local observations, it is highly likely that over-summer survival occurs predominantly on out-of-season wheat plants. However, sporulating lesions have not been observed on wheat growing during summer. Stripe rust is known to remain as a latent infection in tissue during the severe winter conditions of the northern hemisphere (Burleigh and Hendrix, 1970). It is possible that mycelium may remain latent within infected wheat tissue during the Australian summer and sporulate when temperature and moisture are more favourable in autumn.

Similar studies have been conducted in examining the role of grasses in the epidemiology of wheat stem rust, *P. graminis tritici*, in Australia. Waterhouse (1929) demonstrated that a range of grass species were susceptible to this pathogen. Rees (1972) suggested that *Agropyron scabrum* a common perennial grass in southern Queensland, could aid the oversummer survival of *P. graminis tritici*. Luig and Watson (1977) noted that although this pathogen was frequently isolated from *A. scabrum*, the pathotypes detected were different, in frequency, from those obtained from wheat. They concluded that *A. scabrum*, and grasses in general, were not as important as wheat in the over-summer survival of *P. graminis tritici*. This confirmed earlier work (Luig and Watson, 1972) which also suggested that *A. scabrum* was an important host in allowing somatic hybridization between *P. graminis tritici* and *P. graminis secalis*.

The introduction of *P. striiformis tritici* into Australia has provided a unique opportunity to study the establishment and spread of an obligate pathogen in a new geographical area. The pathotype survey has provided one measure of these processes. The development of suitable techniques, in particular the agar-plate method, for processing field collections, greatly improved the procedural aspects of the national pathogenicity survey. The Australian survey averaged 412 accessions annually, from 1980 to 1984 with 75 per cent of accessions being recovered in a viable

condition using the agar-plate method. In comparison, Manners (1950) examined a total of 254 collections over a four year period with 88 per cent recovery, and the N.I.A.B., Cambridge, averaged 130 accessions annually, from 1974 to 1981, with 56 per cent recovery (data from U.K. Cereal Pathogen Virulence Survey reports, 1974-1982). An interesting feature of reported pathotype surveys is the universal failure to identify, or acknowledge, the problem of pathotype mixtures in diseased samples, even in cases where some single spore cultures have been established (Manners, 1950; Zadoks, 1961). However, Johnson and Taylor (1976b) noted that an imported culture consisted of a mixture of pathotypes, although no details were provided. The infection types noted in the present experiments with pathotype mixtures provided some basis for identifying mixtures encountered in surveys. Further experiments with other pathotype combinations would provide valuable information for use in survey work. Pathotype mixtures are commonly encountered with the other cereal rust diseases, but with the discrete localised pustule development typical of these diseases, mixtures are easier to identify.

A total of six pathotypes were detected in Australasia from 1979 to 1984. Except for variation with respect to '*YrA*', all pathotypes have been previously reported overseas (Nagarajan, 1983; Priestley *et al.*, 1974; Stubbs *et al.*, 1974; Johnson *et al.*, 1972). Cultures of pathotypes 104 E137 A-, 104 E137 A+ and 106 E139 A- have been sent to Wageningen, The Netherlands, where their identities were confirmed by R.W. Stubbs (pers. comm.). The tester for *Yr5, i.e. T. spelta album,* is not currently represented in the international system of pathotype nomenclature. It is proposed that this tester be added to the world set with a decanery value of $2^8 = 256$. The culture with virulence for *Yr5* in the present studies is thus designated pathotype 360 E137 A-. The identity of this pathotype was also confirmed by R.W. Stubbs (pers. comm.).

Virulence with respect to cv. Sonalika, which was

shown in the present studies to possess *YrA* (Section 5.4.1.1), was reported in India by Sharma *et al.* (1973). The latter virulence may thus be related to the *YrA* virulence reported in the present studies. The specificity in relation to *YrA* has been confirmed by R.W. Stubbs (pers. comm.), although R. Johnson, P.B.I., Cambridge, England, (pers. comm.) has not been able to reach the same conclusions. This may be due to virulence with respect to *YrA* in English cultures, difficulties in assessing seedling responses and the general absence of the *YrA* resistance in European wheats.

Because of the pathogenic variation in Australia with respect to the *YrA* resistance and the frequent occurrence of *YrA* in Australian wheats (Section 5) it was, and will continue to be, essential that Avocet R, or another wheat with this resistance, be included in rust surveys. This resistance is also relatively common in overseas wheats, especially spring types developed and distributed by the CIMMYT programme in Mexico. The additional tester could, therefore, be placed in the world set, but the assignment of a decanery value of 2^9,=512, to indicate virulence for this tester, could result in some pathotype numbers exceeding the three digit limit suggested by Johnson *et al.* (1972). It is therefore proposed that a third group, tentatively known as the "Australian" regional set, be added to the system proposed by Johnson *et al.* (1972). Avocet R, or an agronomically suitable alternative such as Inia 66, Sonalika or Anza (WW15), would be the first entry with a decanery value of $2^0 = 1$. This would result in the following changes to nomenclature for pathotypes reported in this thesis.

Current	Proposed
104 E137 A-	104 E137 A0
104 E137 A+	104 E137 A1
106 E139 A-	106 E139 A0
108 E141 A-	108 E141 A0
108 E141 A+	108 E141 A1
360 E137 A-	360 E137 A0

New pathotypes were closely associated with particular cultivars, especially in the early stages of establishment and

spread. As pathotypes became established, they were increasingly recovered from cultivars of unrelated host genotypes. This sequence of events was noted by Priestly *et al.* (1974) for stripe rust and Watson and Luig (1968) for stem rust of wheat in Australia. However, the rapid and widespread occurrence of 104 E137 A- in Region 1 in 1983 was not related to unique pathogenic attributes. This particular situation resulted from a combination of susceptible host material and a conducive environment, and it is assumed that the chance establishment and spread of any of the Australian pathotypes would have led to an identical event.

The unique situation in Australia where a presumed single pathotype was introduced gives an excellent supportive picture of the pathogenic development of the other cereal rusts. Long term surveys over 65 years with stem rust and leaf rust have shown that in the absence of the sexual host, dramatic changes in pathogenicity can occur with the arrival of exotic pathotypes followed by single gene mutations for virulence. This sequence of events has clearly been repeated in stripe rust.

Pathotypes were identified using conventional seedling tests and were generally shown to cause predicted responses to adult-plants in glasshouse experiments. However, there is an increasing need to develop skills to monitor pathogenic variation with respect to the adult-plant resistances in Australian wheats, *e.g.* Banks and Cook. Ideally, methods should be developed to allow convenient monitoring within the season of sample collection. The need to conduct polythene tunnel trials as described by Priestley *et al.* (1984a, 1984b) or replicated field experiments (Johnson and Taylor, 1977) may lead to delays of 12 months for results and considerably restricts the number of cultures that can be examined.

The occurrence of some new pathotypes of *P. striiformis* has been attributed to somatic recombination involving whole nuclei (Little and Manners, 1969a, 1969b; Goddard, 1976; Wright and Lennard, 1980). Cytological studies indicated

the absence of diploid nuclei in somatic hyphae, thus precluding the possibility of recombination of virulence genes in a parasexual cycle (Little and Manners, 1969b; Wright and Lennard, 1978; Wright *et al.*, 1978). Johnson and Taylor (1976b) presumed that mutation was the most plausible explanation for the occurrence of a new pathotype virulent on Compair. They suggested that if virulence were a recessive character, then avirulent heterozygotes must occur quite frequently in order to allow new pathotypes to emerge as a consequence of single mutations or nuclear exchanges. The sequential detection of pathotypes, varying only in respect of single attributes of increased virulence, in Australasia suggests that mutation is the most likely mechanism involved. The apparent simultaneous detection of 108 E141 A+ and 108 E141 A- pathotypes was attributed to two independent mutations rather than to some other mechanism.

Contrary to the early predictions of Waterhouse (1936), observations throughout south eastern Australia clearly indicate that the environment is suitable for the development of stripe rust epidemics. A comparison of daily mean maximum and minimum temperatures between Cambridge, England, and various locations in N.S.W. is presented in Table 4.13. It is apparent that temperatures in late autumn, winter and spring in much of the eastern Australian wheat belt, are similar to those occurring during late spring at Cambridge. The May-June period at Cambridge, which is the usual time of maximum stripe rust development, corresponds with June to September at Moree and May to October at Temora.

Although the climate is suitable for *P. striiformis tritici* during most of the wheat crop year in Australia, epidemics of long duration have only been observed in 1983 and 1984. The epidemics of relatively short duration experienced in the spring in the years 1979 to 1982 may have been a function of restricted oversummer survival, together with inadequate rainfall. The recent long season epidemics resulted in significantly greater incidences of stripe rust in spring. In comparison, stripe rust epidemics

long term

TABLE 4.13

Comparison of mean daily maximum and minimum temperatures (c) between Cambridge, England and two locations in New South Wales

Location		Autumn			Winter			Spring			Summer		
Cambridge,[1]		Sept.	Oct.	Nov.	Dec.	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.
England	Max.	18.6	14.8	9.5	6.9	6.1	6.7	9.3	12.2	16.3	19.8	21.2	21.1
	Min.	9.2	6.6	3.1	1.3	0.5	0.6	1.4	3.8	6.5	9.1	10.9	10.7
Moree, N.S.W.[2]		Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Jan.	Feb.
Region 1	Max.	30.9	27.4	21.7	19.0	17.8	19.2	23.0	27.3	30.1	32.4	33.5	33.2
	Min.	16.7	12.3	7.3	4.8	3.1	5.4	8.2	12.6	14.7	18.2	19.5	19.3
Temora, N.S.W.[2]		Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Jan.	Feb.
Region 2	Max.	21.7	23.0	17.2	14.3	13.2	14.5	17.8	22.6	25.7	29.5	31.4	31.2
	Min.	12.8	8.5	4.6	2.3	1.4	3.0	3.9	7.8	9.8	13.6	15.9	16.3

[1]H.J.B. Lowe, Plant Breeding Institute Annual Report 1980, p124.

[2]Commonwealth Bureaux of Meteorology.

in the northern hemisphere are noted to be severe in winter wheats when autumn infection is followed by a mild winter favouring widespread survival, and a cool spring favouring infection and spread (Coakley, 1978), whereas spring wheats are apparently less frequently affected due to their late development. It is apparent from these observations that host resistance in Australian wheats needs to be operative at a very early stage of crop development in order to reduce the potential for widespread spring epidemics.

It was also noted that epidemics in Region 2 (Plate 4.2) of the eastern wheat-belt, were of greater incidence and severity compared to Region 1. The apparently consistent over-summer survival and the original location of new pathotypes were also features of Region 2. Hence wheat breeding programs for Region 2 should include resistance to stripe rust as an important objective. Due to higher temperatures and lower probability of winter rainfall, epidemics of stripe rust in Region 1 should be less frequent and less damaging than those occurring in Region 2. In contrast, Region 1 has been traditionally considered the stem rust-prone area, and breeding efforts to incorporate resistance to this disease have been successful in minimising losses for many years (Watson and Butler, 1984).

After several years of experience, it was finally concluded by the Australian plant pathology community that the stripe rust pathogen was endemic in south-eastern Australia. As a consequence, certain voluntary restrictions imposed on the use of *P. striiformis* cultures by breeding establishments were relaxed. While this will increase the possible risks associated with escape of the pathogen from research establishments, it will also permit breeders greater scope for epidemic induction in disease nurseries, and this will increase the opportunities for selection.

5 SURVEY OF HOST RESISTANCE TO STRIPE RUST

5.1 INTRODUCTION

The occurrence of a single pathotype of *P. striiformis tritici* in Australia between 1979 and 1981 initially restricted investigations of host resistance to classical genetic studies involving the analysis of hybrid populations. These studies usually require a minimum of 12 months, and often two years, or longer before meaningful results indicating the numbers of resistance factors and their linkage relationships are obtained.

Experiments using a range of pathogen cultures of known pathogenicities to test host stocks with unknown resistance genes yield relatively rapid information permitting postulation of the identity and number of resistance factors. The hypotheses thus formulated can be tested in appropriately designed genetic analyses.

The detection and characterisation of new pathotypes in 1981 and 1983 in Australia, enabled experiments to be designed to survey host stocks for resistance. These experiments were arranged and interpreted on the assumption that the wheat: *P. striiformis tritici* interaction followed the gene-for-gene relationship. In addition, the reported close linkage between genes for resistance to stripe rust and stem rust (McIntosh *et al.*, 1981) was exploited to confirm the presence of *Yr7* or to obtain information pertaining to *Yr7* when the appropriate *P. striiformis tritici* pathotype was either unavailable or, after its detection in New Zealand in 1982, of restricted availability. The limited range of pathogenicity among current Australian pathotypes will require a continuation of genetic studies, particularly in host stocks of undetermined resistance.

5.2 HOST MATERIALS

Table 5.1 lists the host materials used in the survey of resistance to stripe rust. The cultivars were broadly divided into Australian and exotic bread wheats,

TABLE 5.1

Source, origin and pedigree of selected host materials

Cultivar	Source of Seed	Country of Origin	Pedigree[2]
Australian Wheats			
Avocet	AUS 20601[3]	Australia	WW119/WW15/Egret
Banks	AUS 20599	Australia	PWTH/Condor sib//2*Condor
Bindawarra	J. Gyarfas	Australia	Mexico 120/Koda//Raven
Celebration	R.A. McIntosh	Australia	Double Cross/Dundee//Dundee
Cocamba	T.T. The	Australia	AUS 10894/4*Condor
Condor	AUS 16036	Australia	WW80/2*WW15
CSP44	W.M. Hawthorn	Australia	WW80/2*WW15
Corella	J. Fisher	Australia	Huelguen/4/Mayo/Norin 10//Yaktana 54/3/ Kenya/Lincoyan/5/2*Egret
Eagle	W3219[4]	Australia	Thatcher/*Ag. elongatum* deriv.//3*Falcon
Egret	AUS 16037	Australia	Heron/2*WW15
Gatcher	W3201	Australia	Charter/3*Gabo/3/Santa Catalina/Thatcher// Mayo
Jacup	T.T. The	Australia	Bencubbin/3/Charter//Sword/Kenya C6041/ 4/Mexico/5/Gamenya
Miling	T.T. The	Australia	As for Jacup
Millewa	W.M. Hawthorn	Australia	Sonora 64/Yaqui50E//Gaboto/Mexico 8156
Oxley	W3636	Australia	WW80/2*WW15
Sunkota	J. Gyarfas	Australia	Timson/IRN 67-451
Sunstar	J. Gyarfas	Australia	Condor/4/WW15 2*/3/Steinwedel/WC356// La Prevision

TABLE 5.1 Cont.

Cultivar	Source of Seed	Country of Origin	Pedigree[2]
Torres	T.T. The	Australia	3Ag3/3*Condor
Takari	J. Gyarfas	Australia	Kite/3/Frocor/Kentana//2*Festiguay
Vulcan	J. Gyarfas	Australia	Condor/Pitic 62//Condor sib
WW80	W.M. Hawthorn	Australia	Penjamo 62/4*Gabo 56//TZPP/Nainari 60
WW15[1]	AUS 12274	Australia	Lerma Rojo//Norin 10/Brevor 14/3/3*Andes E.
Exotic Wheats			
Atlas 66	W.M. Hawthorn	U.S.A.	Frondoso//Redhart 3/Noll 28
Cajeme 71	R.A. McIntosh	Mexico	Ciano sib/3/Sonora 64/Klein Rendidor// Siete Cerros sib
Chris	W.M. Hawthorn	U.S.A.	Frontana/3*Thatcher/3/Kenya 58/Newthatch// 2* Thatcher
Ciano 67	R.A. McIntosh	Mexico	Pitic 62/Chris sib//Sonora 64
FKN	W.M. Hawthorn	U.S.A.	Complex cross involving Frontana, Kenya and Newthatch
Frontana	W.M. Hawthorn	Brazil	Frontiera/Mentana
Frontiera	W.M. Hawthorn	Brazil	Polysu/Alfredo Chaves 6
Hermosillo 77	R.A. McIntosh	Mexico	Jaral 66/Napayo/3/Lerma Rojo 64// Tezanos Pintos Precoz/3*Andes/4/Bluebird/ Norteno 67//Ciano sib/Candeal/3/Calidad
Inia 66	R.A. McIntosh	Mexico	Lerma Rojo 64/Sonora 64
Manitou	W.M. Hawthorn	Canada	Thatcher *7/Frontana//Canthatch/3/PI170925/ 6*Thatcher
Colotana	W255	Brazil	Colonista/Frontana

TABLE 5.1 Cont.

Cultivar	Source of Seed	Country of Origin	Pedigree[2]
Nacozari 76	R.A. McIntosh	Mexico	Tezanos Pintos Precoz/Paloma//Candeal
Nainari 60	R.A. McIntosh	Mexico	Supremo/Mentana//Gabo/3/Thatcher/ Queretaro//Kenya/Mentana/Gabo
Norquay	W.M. Hawthorn	Canada	Lerma Rojo/Sonora 64//Justin
Nuri 70	W.M. Hawthorn	Mexico	Ciano sib/3/Sonora 64/Klein Renidor// Siete Corros 66
Oroua	F.R. Sanderson	New Zealand	1966, I.S.W.R.N.395[5]/Skemer
Pavon 76	R.A. McIntosh	Mexico	Vicam 71//Ciano 67 sib/Siete Cerros 66/ 3/Kalyansona/Bluebird
Penjamo 62	R.A. McIntosh	Mexico	Frontana/Kenya 58//Newthatch/3/Norin 10/ Brevor
Pitic 62	R.A. McIntosh	Mexico	Yaktana 54//Norin 10/Brevor
Rio Negro	W.M. Hawthorn	Brazil	Supreza/Centenario
Romany	W.M. Hawthorn	Kenya	Colotana 261-51/Yaktana 54A
Shoshi	W.M. Hawthorn	Unknown	Unknown
Sinton	W.M. Hawthorn	Canada	Manitou/3/Thatcher*6/Kenya Farmer//Lee*6/ Kenya Farmer
Sonalika	W.M. Hawthorn	India	388/Andes//Pitic 62 sib/3/Lema Rojo 64
South Africa 43	W.M. Hawthorn	South Africa	Agel/Kenya B256G//39W73/3/39W23
Surpreza	W.M. Hawthorn	Brazil	Polysu/Alfredo Chaves 6
Tanori 71	R.A. McIntosh	Mexico	Sonora 64//Ciano 67/Inia 66
Tezanos Pintos Precoz	W.M. Hawthorn	Unknown	Unknown
Thatcher	R.A. McIntosh	U.S.A.	Marquis/Iumillo//Marquis/Kanred
Tobari 66	W.M. Hawthorn	Mexico	Tezanos Pintos Precoz/Sonora 64

TABLE 5.1 Cont.

Cultivar	Source of Seed	Country of Origin	Pedigree[2]
Durum Wheats			
Kamilaroi	R.A. Hare	Australia	Durati sib/Leeds
820528	R.A. Hare	Australia	Kamillaroi sib/Guillemot
820889	R.A. Hare	Australia	Kamillaroi sib/Guillemot
820911	R.A. Hare	Australia	Kamillaroi sib/Guillemot
820946	R.A. Hare	Australia	Kamillaroi sib/Guillemot
8211539	R.A. Hare	Australia	Kamillaroi sib/Guillemot
Triticales			
Ningadhu	N.L. Darvey	Australia	Inia/rye/2*//Armadillo 'S'
Coorong	N.L. Darvey	Australia	Inia/Armadillo 'S'//1648
Dua	N.L. Darvey	Australia	Maya 11/Armadillo 'S'

Footnotes

[1]WW15 synonyms

Anza	AUS 15042	U.S.A.
Karamu	AUS 18637	New Zealand
Mexicani	10RCB,1979-80,6[6]	Sudan
SNA-1	AUS 19665	Chile
T4	AUS 14092	South Africa

[2]pedigrees derived from Zeven and Zeven-Hissink (1976) and Skovmand and Rajaram (1978).

[3]accession number of the Australian Wheat Collection, Tamworth.

[4]accession number of the University of Sydney Wheat Collection.

[5]International Spring Wheat Rust Nursery.

[6]Regional Crossing Block-Breadwheat, 1979-80.

durum wheats and triticales. Details of source, origin and pedigree were noted.

The various wheats listed as WW15 synonyms (Table 5.1) are apparently derived from the same CIMMYT cross II8739-4R-aM-1R (Qualset *et al.*, 1984; Skovmand and Rajaram, 1978). Qualset *et al.* (1984) listed the pedigree of Anza as Lerma Rojo//Norin 10-Brevor/4/Yaktana 54/Norin 10-Brevor/3/3*Andes in contrast to the abbreviated form presented by Skovmand and Rajaram (1978) which is listed in Table 5.1. "Andes E." was synonymous with Andes Enano which translates from Spanish to "Andes dwarf" (R.A. Fischer, pers. comm.). It was presumed that the pedigree of Andes E. was Yaktana 54//Norin 10/Brevor/3/3*Andes.

5.3 HETEROGENEITY WITHIN CULTIVARS

When tested with pathotype 104 E137 A-, some cultivars were clearly heterogeneous in response. Seedlings with different infection types were tagged, transplanted and harvested for progeny testing. Results for certain of these selections are summarised in Table 5.2. The results clearly confirmed that cv.s Avocet, Egret, Banks, Condor, Gatcher and Penjamo 62 were genetically heterogeneous in response to stripe rust. Plants exhibiting low infection types were referred to as resistant selections and noted as R, *e.g.* Avocet R. In contrast, plants with high infection types were noted as susceptible, *e.g.* Avocet S. It was concluded that selected plants of Nainari 60 and Inia 66 did not show significantly different responses.

Heterogeneity in response to stripe rust was also noted in the field. During a survey of commercial wheat areas in southern N.S.W. and Victoria in 1980, individual plants within crops of cv. Egret frequently showed variable responses. Single plants with different responses were selected and harvested prior to progeny testing in the field and glasshouse. The results for 10 selected plants are presented in Table 5.3. Progenies of four plants selected as resistant in four crops at three locations were shown to be resistant in field and glasshouse tests. Similarly, three susceptible selections produced susceptible progenies.

TABLE 5.2

Seedling infection types produced by certain wheat cultivars and their selected progenies following inoculation with pathotype 104 E137 A-

Cultivar	Progenies		
	Unselected Population	Selected for low infection type	Selected for high infection type
Avocet	;N1,2C,3+	;C12-	3+
Egret	;1=,2, 3+	12C	3+
Condor	;1, 3+	;12C	3+
Banks	;N1-,33+	;N1-	3+
Gatcher	12C,3+	12-C	3++
Penjamo 62	;N,2C,3+C	;N1-	3+
Inia 66	;N1,23=C	;N12	12-C
Nainari 60	;N,2-C,3-	;N12	12C

TABLE 5.3

Progeny tests of cultivar Egret selections made during a field survey, 1980, using pathotype 104 E137 A-

Field Survey Location	Selection	Progeny Tests	
		Field 1981	Glasshouse
Jerilderie, N.S.W.	R	20R	;12
Temora, N.S.W. 1	R	10R	;12
2	R	10R	;12
Wallenbeen, N.S.W.	R	10R	;12
	S	20MR-MS	3+
Cootamundra, N.S.W.	R	5R,20MS	;1,33+
	S	40MS	3+
Horsham, Victoria	S	20MR-MS	3+
Boort, Victoria	R	20MR-MS	33+
	S	10R,20MR-MS	12,33+

However, some discrepancies occurred. Plants designated Cootamundra R and Boort S gave progenies with heterogeneous responses. This indicated that either selection on a single plant basis was not always effective or that the selections were not single plants. A plant selected at Boort, Victoria, as resistant produced susceptible progeny. This plant may have been uninfected rather than resistant at the time of selection. Despite these discrepancies, it was concluded that much of the variation in field response of cv. Egret was associated with variation in its seedling response.

The extent of genetic heterogeneity in each of the Australian cv.s Avocet, Egret, Condor and Banks was determined from seedling tests on foundation seed samples deposited with the Australian Wheat Collection, Tamworth, N.S.W., at the time of official registration, and samples from the breeding centres which produced them. The seedling response data in Table 5.4 indicated that genetic heterogeneity was present in most of the samples. However, two of three samples from Temora were homogeneous for seedling response. The development of these cultivars in the absence of selection for stripe rust resistance would have permitted genetic variation to remain undetected. Clearly, each of these cultivars was genetically heterogeneous in stripe rust response at the time of release. With further seed increases, random drift or plant-to-row maintenance procedures led to drastic changes in gene frequencies, *e.g.* the homogeneous samples from Temora.

5.4 HOST-PATHOGEN SPECIFICITY

5.4.1 SEEDLING TESTS

5.4.1.1 Cultivars showing the *YrA* resistance

The seedling and adult-plant resistance, which was present in certain plants of several Australian cultivars, was overcome by a virulent pathotype in 1981 (Section 4.3.2.2). The genetic basis for this resistance was tentatively designated *YrA* because its occurrence was associated with markedly increased levels of stripe rust in cv. Avocet which was released in 1980. Progenies

TABLE 5.4

Seedling response of various sources of certain Australian cultivars to pathotype 104 E137 A-

Cultivar	Seed Source	Resistant Plants		Susceptible Plants		% Resistant Plants
		Number	IT	Number	IT	
Avocet	AUS 20601[1]	41	;C1=	53	3+4	43.6
	Temora, 1984[2]	55	;C1			100.0
	J. Fisher[3]	66	;C1	38	3+4	63.5
Egret	AUS 16037	46	;C1	32	3+4	60.0
	R. Martin[1]	80	;C1			100.0
Condor	AUS 16036	54	;C1	61	3+4	50.0
	R. Martin[1]	19	;C1	80	3+4	19.1
Banks	AUS 20599	66	;CN1-	39	3+4	62.8
	J. Syme[4]	61	;C1-	33	3+4	64.9

[1]Foundation seed sample lodged by the breeder at the time of registration.

[2]Agricultural Research Station, Temora, N.S.W.

[3]Agricultural Research Institute, Wagga Wagga, N.S.W.

[4]Queensland Wheat Research Institute, Toowoomba, Queensland.

of Avocet plants known to possess *YrA* from tests with the original pathotype were clearly susceptible with the new pathotype. In 1983, A+ and A- variants which were virulent for *Yr6* became available.

Various experiments from 1980 provided increasing evidence that the resistance identified in some plants of cv. Avocet was also present in other Australian and several exotic wheats. As the Australian wheats were all closely related to WW15, it seemed appropriate to test various sources of named cultivars selected from WW15 itself, or from the same CIMMYT cross.

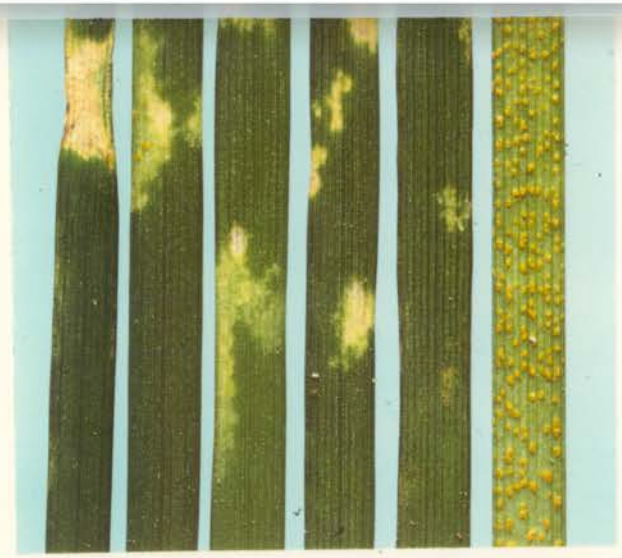
The data in Table 5.5 are from the most recent experiments using the paired A- and A+ pathotypes, with and without virulence for *Yr6*. Certain responses are illustrated in Plate 5.1. As expected, R selections of various wheats gave low infection types, whereas S selections were susceptible. Of four cultivars released after the occurrence of stripe rust in Australia, Sunstar, Cocamba and Vulcan were resistant, whereas Torres was susceptible. These wheats are all closely related to Condor which was heterogeneous. The CIMMYT wheats Sonalika, Nainari 60, Inia 66, Nuri 70, Tanori 71 and WW15, and its derivatives, showed the same response patterns as Avocet R.

The recorded low ITs produced with *YrA*-avirulent pathotypes varied from ;(Avocet R) to 2C(Egret R). However, in repeated tests and in single plant progenies from these wheats, there appeared to be no significant genetic variation, either between or within wheats, which possessed *YrA* (see also Table 5.2). The ITs produced on second-emergent leaves were always distinctly lower than those produced by the primary leaf. This consistent observation was sometimes taken into account when ITs on the first leaves were intermediate. Pathotype 108 E141 A- produced ITs that were distinctly lower on resistant host selections than those produced by 104 E137 A-. Because of IT variability encountered with *YrA*-avirulent isolates, comparisons with *YrA*-virulent isolates were sometimes

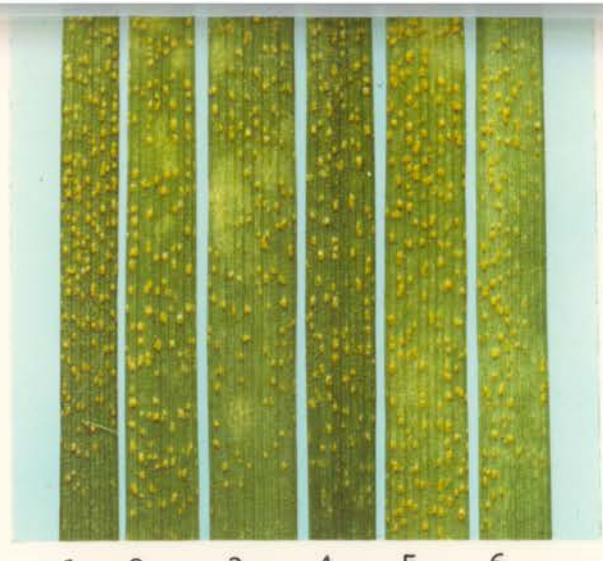
TABLE 5.5

Infection types produced by a range of wheats infected with *P. striiformis tritici* pathotypes avirulent and virulent with respect to the *YrA* resistance

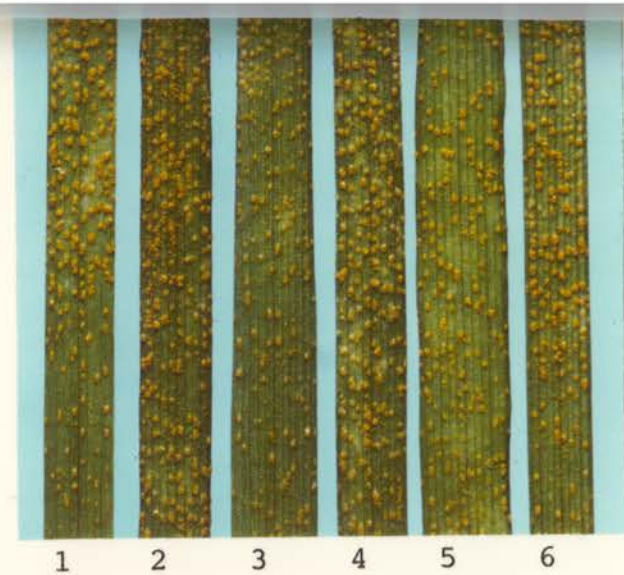
Cultivar	104 E137 A-	104 E137 A+	108 E141 A-	108 E141 A+
Controls				
Avocet R *(YrA)*	;C1-	4	;	4
Avocet S	3+	4	3+4	3+4
Federation	4	4	4	4
Australian				
Banks R	1C	33+	;C1=	33+C
Banks S	3+	4	4	4
Egret R	2C	3+4	;C	4
Egret S	3+4	3+	3+4	4
Condor R	12C	4	;C	4
Condor S	3+4	4	4	4
Sunstar	12C	4	;C	4
Cocamba	;1C	3+	;1C	4
Vulcan	12C	4	;1=	4
Torres	4	4	4	4
Exotic				
Sonalika	;C	3+	;CN	3+
Nainari 60	;C1-	3+	;C	3+
Inia 66	;C1=	3+	;C	4
Nuri 70	;1C	3+4	;1C	33+
Tanori 71	1+C	3+	1+C	33+
WW15 and synonymous wheats				
WW15	;12	3C	;C	33+
Anza	1C	3+	;;N	3+
Karamu	;N1	3+		
T4	;N12-	4		
SNA 1	;N1	3+C	;;N	3+
Mexicani	;12	33+C	;;N	3+



108 E141 A-, high light



108 E141 A-, low light



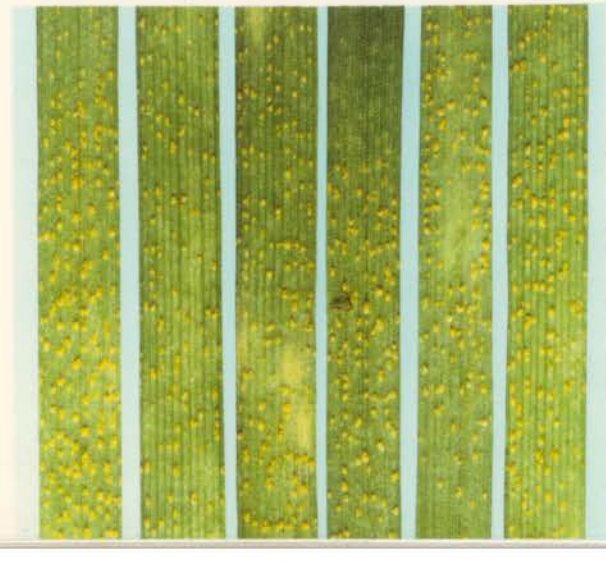
104 E137 A+, high light

PLATE 5.1

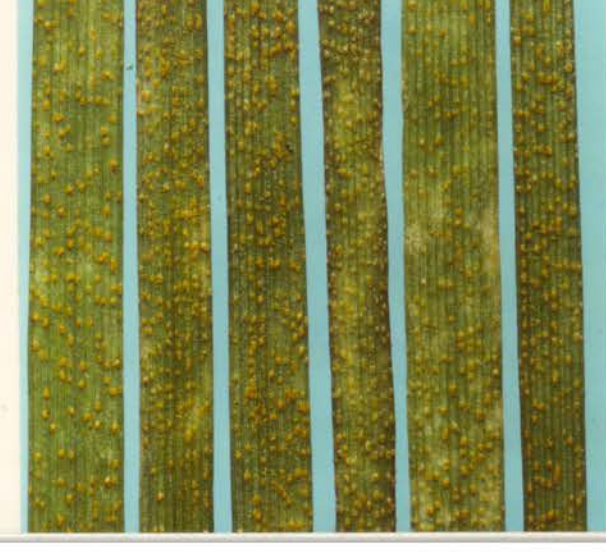
Infection types of wheats postulated to possess *YrA* in response to variation in pathotype and light intensity
1=BanksR 2=CondorR 3=EgretR 4=Sunstar 5=AvocetR 6=Federation 7=AvocetR 8=WW15 9=Nainari60 10=Inia66 11=Sonalika 12=Federation

108 E141 A-, high light

108 E141 A-, low light



104 E137 A+, high light



necessary in order to more clearly substantiate decisions regarding the presence, or absence, of the *YrA* resistance.

The recognition of the *YrA* resistance in Avocet R has been confirmed by R.W. Stubbs, Wageningen, The Netherlands (pers. comm.). R. Johnson, Cambridge, England (pers. comm.) suggested that Avocet R showed the same response as cultivars possessing *Yr2*, such as Heines VII. This cultivar, together with Merlin and Cleo, which were also reported to carry *Yr2* (McIntosh, 1983), plus wheats with other gene combinations, were compared with Avocet R using pathotypes 104 E137 A- and 104 E137 A+. The results, listed in Table 5.6, indicated that Avocet R was the only cultivar that differentiated the two pathotypes. Thus, if Avocet R does carry *Yr2*, it must also carry an additional factor distinguishing Australian A- and A+ pathotypes.

5.4.1.2 Cultivars showing the *Yr6* resistance

The seedling IT responses of Australian and introduced wheats to pathotypes avirulent (104 E137 A-, 104 E137 A+) and virulent (108 E141 A-, 108 E141 A+) on seedlings of cv. Heines Kolben, which carries *Yr6* (Macer, 1966), are presented in Table 5.7. Certain of these responses are illustrated in Plate 5.2. This data represents the culmination of several individual experiments leading to the postulation that various wheats possessed *Yr6*.

The low ITs obtained in these studies were less variable both within and between experiments, and within and between cultivars, than those obtained for wheats possessing the *YrA* resistance. Again, in contrast to wheats possessing *YrA*, the low ITs on second-emergent leaves of wheats possessing *Yr6* were similar to, or sometimes slightly higher than, those produced on seedling leaves.

Penjamo 62S was susceptible to all pathotypes (Section 5.3, Table 5.3). The remaining bread wheats displayed similar responses to Heines Kolben which is the international differential tester for *Yr6* (Johnson *et al.*, 1972). It was therefore concluded that these wheats possessed *Yr6*.

TABLE 5.6

Seedling infection type responses of cultivars with reported resistance genes after inoculation with two pathotypes of *P. striiformis tritici*

Cultivar	Genotype	104 E137 A-	104 E137 A+
Heines VII	*Yr2*	3+	3+
Merlin (AUS27148)	*Yr2*	3+	3+4
Minister (AUS3032)	*Yr3c*	3+	3+
Cleo (AUS14955)	*Yr2+3c*	33+C	
Cappelle-Desprez (W3055)	*Yr3a+4a*	33+C	3+C
Hybrid 46	*Yr3b+4b*	33+	33+
Avocet R	*YrA*	;C1=	3+C

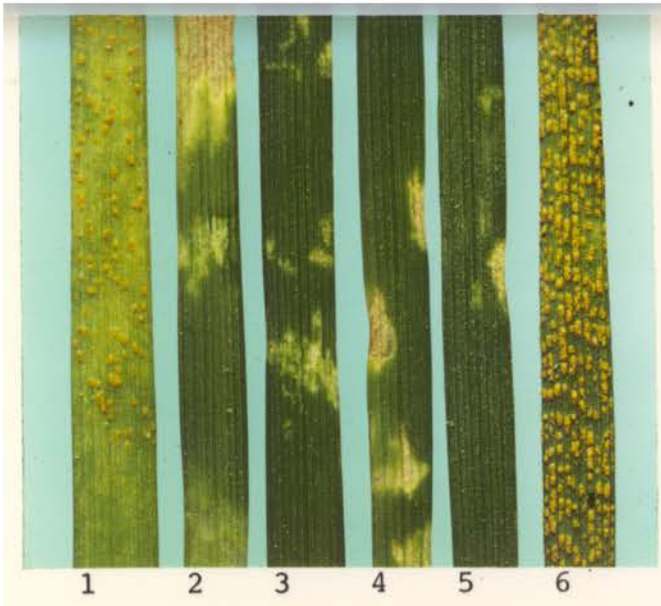
Similarly, certain durum wheats were found to produce differential responses indicative of the presence of *Yr6*. Lines 820528 and 820889 displayed intermediate infection types in response to some *Yr6*-virulent pathotypes. Since all breeding lines were selected from a cross involving Kamilaroi, it is likely that the intermediate responses observed in lines 820528 and 820889 were due to gene(s) inherited from Kamilaroi. Two lines (*i.e.* 820946 and 8211539) were genetically heterogeneous for the postulated *Yr6* resistance, although progeny tests were not performed to confirm the observation.

An interesting feature from these experiments was the consistently lower ITs with avirulent pathotypes, shown by durum wheats postulated to carry the *Yr6* resistance. Bread wheats with this gene produced higher ITs with the same avirulent pathotype. T. The (1976) noted progressive increases in IT responses as stem rust resistance genes were transferred

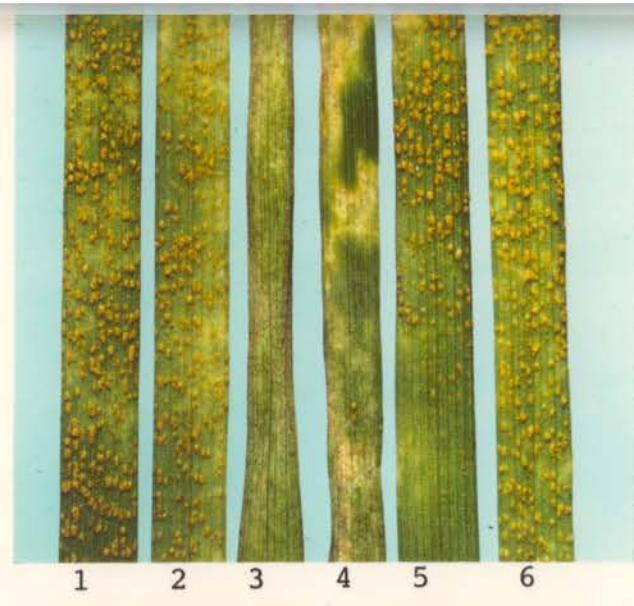
TABLE 5.7

Infection type responses of bread wheat and durum wheat cultivars to *P. striiformis tritici* pathotypes avirulent and virulent for *Yr6*

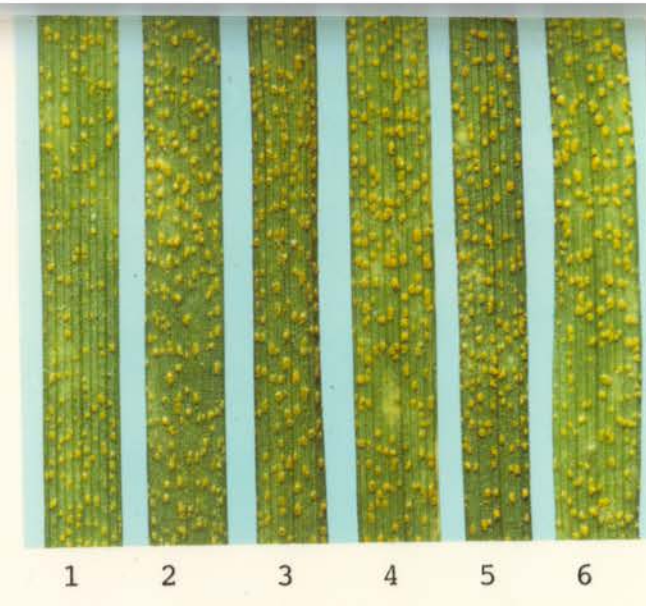
Cultivars	104 E137 A-	104 E137 A+	108 E141 A-	108 E141 A+
Controls				
Heines Kolben *(Yr6)*	;N1-	;N1=	3+	3+
Sonora	4	4	4	4
Australian				
Oxley	;;N	;;N	3+4	3+
Jacup	;C	;C	3+4	3+4
Miling	;CN	;C1=	3+C	4
Bindawarra	;1C	12C	3+4	4
Millewa	;C	;C	3+C	4
Takari	;C	;N	33+C	33+
WW80	;	;;N	4	4
Exotic				
Frontana	;	;	4	4
Romany	;1=C	;N1=	3+	3+
Colotana	;C	;N	33+	33+
Frontiera	;CN	;C	4	4
TZPP	;CN	;N1	3+	33+
Shoshi	;1=C	;N	33+	3+4
Surpreza	;N	;1=C	3+	3+
Rio Negro	;C	;C	3+	4
Tobari 66	;N	;N	3+4	3+4
Atlas 66	;11-C	;N	4	4
Pitic 62	;C	;N	3+	33+
Ciano 67		;1=C		3+4
Penjamo 62R	;1-C	;C	3+C	3+C
Penjamo 62S	4	3+	3+4	4
Durum Wheats				
Kamilaroi	22+C		1+2C	1C
820528	O;;		22+	2++
820889	O;;		2+	3-
820911	O;;		33+	3+
820946	O;;,3+4		2+3,3+	3-,3+
8211539	O;;,3+4		3-,3+	3,3+



104 E137 A+,17C



104 E137 A+,10C

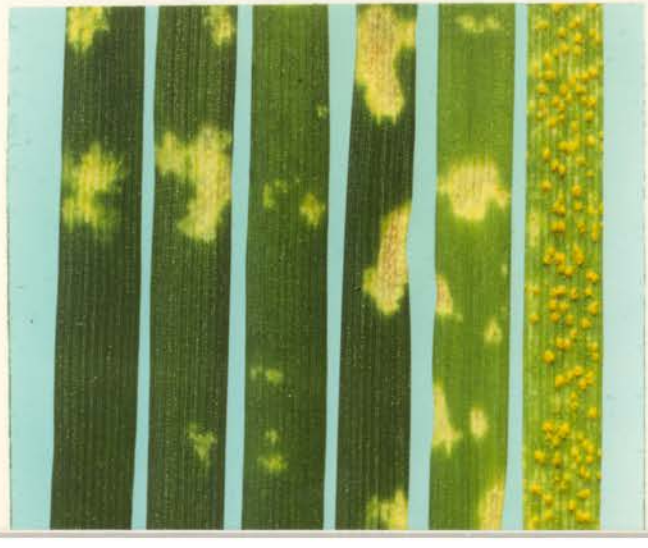


108 E141 A-,17C

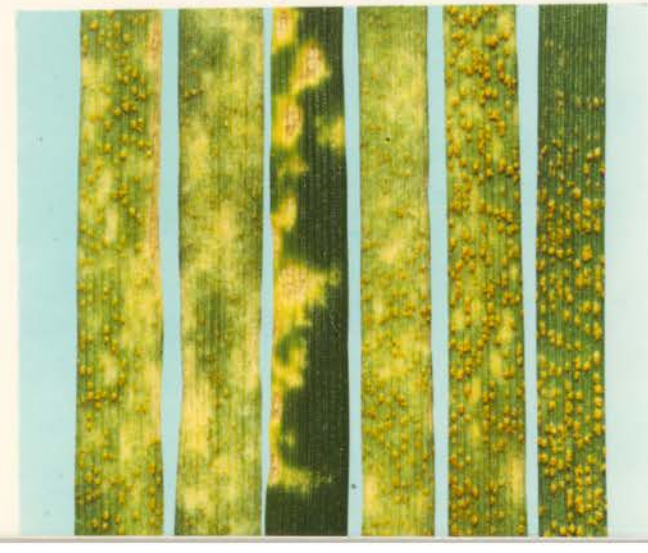
PLATE 5.2

Infection types of wheats postulated to possess *Yr6* in response to variation in pathotype and temperature
1=Bindawarra 2=Miling 3=Millewa 4=Takari 5=Oxley 6=CondorS 7=Penjamo62P 8=Pitic62 9=Frontana 10=Shoshi 11=Tobari66 12=CondorS

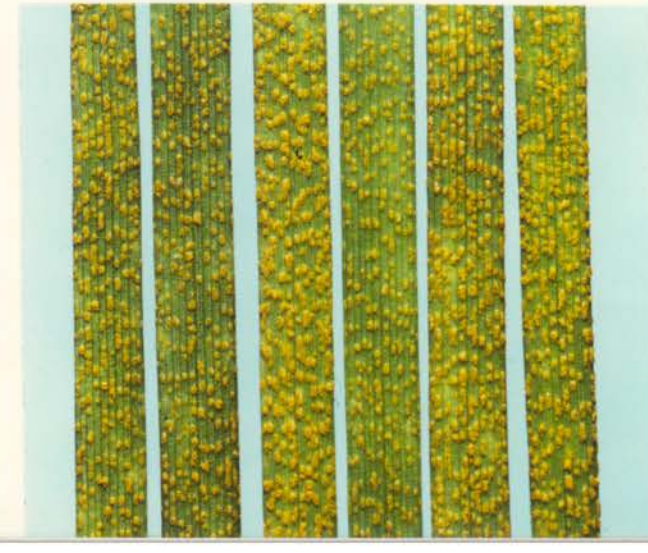
104 E137 A+,17C



104 E137 A+,10C



108 E±41 A-,17C



from diploid wheats to durum and bread wheat derivatives.

5.4.1.3 Cultivars showing the *Yr7* resistance

The IT responses produced by a series of Australian and exotic wheats when inoculated with *P. striiformis tritici* pathotypes avirulent (104 E137 A-, 104 E137 A+, 108 E141 A-, 108 E141 A+) and virulent (106 E139 A-) with respect to cv.s Lee and Celebration, which carry *Yr7* (McIntosh, 1983), are listed in Table 5.8. All entries, with the exception of the susceptible control and Gatcher S, showed similar responses to the five *P. striiformis tritici* pathotypes. Since the response patterns were similar to those of the Lee and Celebration controls, it was suggested that each of the Australian and exotic wheats possessed *Yr7*. The actual low ITs for the wheats with *Yr7* were more intermediate and generally distinctive from those produced by cultivars with *YrA* and *Yr6*.

McIntosh *et al.* (1981) reported that many hexaploid wheats carrying *Yr7* also carried *Sr9g* for resistance to stem rust. The genes *Yr7* and *Sr9g* were closely linked. In order to examine this relationship in the wheats listed in Table 5.8, and thus provide more evidence for the *Yr7* postulations, simultaneous inoculations were performed using *P. graminis tritici* pathotypes 343-1,2,3,5,6 and 98-1,2,3,5,6 which differ only in avirulence and virulence, respectively, for *Sr9g*. In most instances where *Yr7* was postulated, the infection types produced by 343-1,2,3,5,6 were distinctly lower than those produced by 98-1,2,3,5,6, thus indicating the presence of *Sr9g*. The Australian cv. Eagle gave ITs lower than those produced by other wheats when inoculated with pathotype 343-1,2,3,5,6. Moreover, a similar response was obtained with 98-1,2,3,5,6. This was attributed to the presence of *Sr26*. Thus stem rust data provided no evidence supporting or refuting the presence of *Yr7* in Eagle. However Luig (pers. comm.), working with segregating populations, found evidence for the presence of *Sr9g* in this cultivar. South Africa 43 gave a heterogeneous response to both *P. graminis tritici* pathotypes, suggesting that an additional factor(s) conferred resistance to both pathotypes.

TABLE 5.8

Infection type responses of cultivars to *P. striiformis tritici* and *P. graminis tritici* pathotypes with contrasting pathogenicities with respect to *Yr7* and *Sr9g*

Cultivar	*P. striiformis tritici*					*P. graminis tritici*	
	104 E137 A-	104 E137 A+	108 E141 A-	108 E141 A+	106 E139 A-	343- 1,2,3,5,6	98- 1,2,3,5,6
Control							
Lee *(Yr7)*	;N1=	;N1=	;N1=	;N1=	3+4	2-	3+4
Celebration *(Yr7)*	;N	;N	;N	;N	3++	2-	3+
Sonora	4	4	4	4	4	4	4
Australian							
Eagle	;N	1-N	;N	;CN	3+4	;12=	;12=
Sunkota	;N	;N	;N	;CN	3+4	12=	2-2
Gatcher R	1+C	1+2C	1C	1-C	33+C	;1-	3
Gatcher S	3+	3++	3+	3+	3+	33+	33+
Exotic							
South Africa 43	;N	1=CN	;N1-	;CN	3+4	;1,12=	;1,3
Oroua	;N1=	;N	;N1	;N1	3+4	2=	4
Chris	;N1-	1=C	;N1-	;N1-	3+	12=	2+3
Norquay	;N	;N1=	;N1=	;1=C	3++	;1=	3C
Sinton	1C	;N1=	1C	1=CN	3+	;12-	23C
Thatcher	1+2C	;1C	1+C	1=C	3+	;12=	3
Manitou	;1-	1-C	1C	1=C	3+4	1=	2+

5.4.1.4 Wheat and triticale cultivars with unknown genes or gene combinations

The IT responses for a range of wheat and triticale cultivars when inoculated with *P. striiformis tritici* and *P. graminis tritici* pathotypes were compared with standard testers of known genotype (Table 5.9). "Resistant" selections taken from CSP44 (tested with 108 E141 A-) and Cajeme 71 (tested with 104 E137 A+) appeared to have the resistance gene combination *Yr6 + YrA*. "Susceptible" selections taken from CSP44 apparently possessed *Yr6*, whereas "susceptible" selections from Cajeme 71 possessed *YrA*. It was thus concluded that CSP44 was genetically homogeneous for *Yr6* and heterogeneous for *YrA*, whereas Cajeme 71 was homogeneous for *YrA* and heterogeneous for *Yr6*. The remaining wheats and triticales were resistant to all *P. striiformis* pathotypes; consequently, postulations of genetic factors for host resistance could not be based on response data alone.

Tests with *P. graminis tritici* pathotypes 343-1,2,3, 5,6 and 98-1,2,3,5,6 indicated that *Sr9g* was present in Corella. Thus, assuming close linkage of *Sr9g* and *Yr7*, as reported by McIntosh *et al.* (1981) and indicated in earlier phenotypic data (Section 5.4.1.3, Table 5.8), the present results suggest the presence of *Yr7* in Corella. If *Yr7* is present, there must be a second gene conferring resistance to pathotype 106 E139 A-. Of the genes present in Australian wheats, this second gene in Corella could be either *Yr6* or *YrA*. As this cultivar was a backcross derivative of Egret, it was likely that the second gene was *YrA*. However, ITs observed in tests with pathotype 106 E139 A- suggested the presence of *Yr6* as the second leaf IT was slightly higher than the seedling leaf.

Pavon 76 showed a stripe rust response similar to Corella, again with evidence of *Sr9g* for resistance to stem rust. In this case, a genetic analysis established that the host resistance genotype was *Yr6 + Yr7* (Section 6.3.5).

TABLE 5.9

Infection type responses of wheat and triticale cultivars to *P. striiformis tritici* and *P. graminis tritici* pathotypes

Cultivar	*P. striiformis tritici*						*P. graminis tritici*	
	104 E137 A-	104 E137 A+	108 E141 A-	108 E141 A+	106 E139 A-	360 E137 A-	343- 1,2,3,5,6,	98- 1,2,3,5,6
Controls								
Avocet R *(YrA)*	;C1=	4	;	4	;N1=	;C1		
T. spelta *(Yr5)*	0;;	0;	0;	0;	0;	3+4		
H. Kolben *(Yr6)*	;N1-	;N1=	3+4	3+	;N1	;N1-		
Lee *(Yr7)*	;N1=	;N1=	;N1=	;N1=	3+4	;N1-	2-	3+4
Mentana	4	4	4	4			3+	3+
Australian Wheats								
CSP44	;	;	;,4	4				
CSP44 R	;	;	;	3+4				
CSP44 S	;C	;C	3+4	3+4				
Corella	;	;	;N	;N	;C1-/1C	;	2=	33+
Exotic Wheats								
Cajeme 71	;N1	;N,3+	;N1	3+				
Cajeme 71 R	;	;C	;C	3+4				
Cajeme 71 S	;1-	3+4	1C	3+4				
Pavon 76	;	;	;;N	;;N	;C1=/1C	;	;1	2
Hermosillo 77	;	;	;N	;N	;N/1-C	;	12=	2
Nacozari 76	2C	12-	1+C	2+C	12-		2-2	2
Triticales								
Ningadhu	0;	0;;	0;	0;				
Coorong	;N	;;N	;N	;				
Dua	;N	;	;	;N				

Similarly, the IT response of Hermosillo 77 to pathotype 106 E139 A- suggested the presence of *Yr6*. The responses to the stem rust pathotypes were interpretted to indicate the absence of *Sr9g*, although tests with stripe rust pathotypes virulent for *Yr6* showed ITs similar to those observed for *Yr7* in Pavon 76.

The intermediate response of Nacozari 76 to all *P. striiformis tritici* and *P. graminis tritici* pathotypes did not allow the postulation of the resistance genes *YrA*, *Yr6*, *Yr7* or *Sr9g* in this cultivar. Similarly, the incompatible ITs in three triticale cultivars to all pathotypes of *P. striiformis tritici* did not allow the postulation of the genetic basis for seedling resistance.

5.4.2 EFFECT OF ENVIRONMENT ON SEEDLING RESPONSE

5.4.2.1 Temperature

Two independent experiments were conducted under glasshouse conditions to examine the effects of temperature on the low ITs produced in certain host:pathogen interactions. Infection types were recorded 12 to 23 days after inoculation, depending on temperature.

The results presented in Table 5.10 indicated that ITs produced by cultivars possessing the *YrA* resistance were unaffected by temperature. There were no consistent differences in response between the pathotypes, although relatively high ITs for Avocet R and Egret R were noted with respect to 104 E137 A-. It was previously noted (Section 5.4.1.1) that this pathotype generally produced low ITs that were slightly higher than those with 108 E141 A-. However, this feature was not apparent from the results of the present experiments.

The data in Table 5.11 indicated that the IT responses displayed by cultivars possessing *Yr6* varied with temperature. Certain of these responses are illustrated in Plate 5.2. The cultivars Heines Peko, Miling, Millewa, Bindawarra, Tobari 66, Atlas 66, Pitic 62, Romany and Shoshi showed distinctly higher ITs at low temperatures. This tendency was also noted in Takari, WW80, Penjamo 62R and Jacup in

TABLE 5.10

Infection type responses for cultivars possessing *YrA* when inoculated with avirulent pathotypes and incubated at three temperatures

	Experiment1, 104 E137 A-			Experiment 2, 108 E141 A-		
	12 days[1] 21C±2C	14 days 16C±2C	23 Days 11C±2C	18 Days 17C±2C	20 Days 10C±2C	20 Days 11±2C/18±2C
Avocet R	22+C	;1C	23-	;N1-	;	;C
Egret R	23C	;1	23-	1-C	;C	;C
Banks R	;N	;N1-	;N	;N1-	;N1-	;N
Condor R	;N	;N	;N1=	;N1-	;N12	;C
Sunstar	;N1	;N	;N	1C	;N1-	;N
Vulcan	;N1-	;N1-	;N	;1C	;N1=	;C1=
WW15	;N	;N1	;N	;N1-	;N1-	;
Inia 66	;C	;C	1-C	;C1	;C1	;C1
Nainari 60	;N	;	;N	;1C	;1=	;C1=
Tanori	;N	;C	1=N			
Sonalika				;N1-	;N	;C
Sonora	3+	3+4	4	3+4	4	4

[1]interval from inoculation to recording notes.

TABLE 5.11

Infection type responses produced by cultivars possessing *Yr6* when inoculated with avirulent pathotypes and incubated at various temperatures

Cultivar	Experiment 1, 104 E137 A-			Experiment 2, 104 E137 A+		
	12 days[1] 21±2C	14 days 16±2C	23 days 11±2C	18 days 17±2C	20 days 10±2C	20 days 11±2C/18±2C
Heines Peko	;	;	33+	;CN	2+	1-
Miling	;;N	;N	23-	;N	23=	;C1=
Millewa	;	;	3	;	1	12C
Bindawarra	;	1=	3	12C	33+	1-
Tobari 66	;	;C	33+	;N	23-	;
Atlas 66	;	;	33+	;N	1	2
Pitic 62	;	;C	2+	;C	11-	;C1=
Romany	;N1-	;	33+	;N	1	;N
Shoshi	;;N	;N	2+	;CN	1	;C
WW80	;;N	;N	3CN	;N	;N	;;N
Penjamo 62R	;C	;N	33-	;C	1	;C
Jacup	;;N	;C	23-	;N1-	;N1	;C
Oxley	;	;N		;N	;N1+	;C
Frontana	;	;	;N	;	;1=	;
Takari	;N	;N	23-CN	;N	;N1	;C1=
Condor S	3+	3+4	4	3+4	4	4

[1]interval from inoculation to recording notes.

Experiment 1, but was not as clear in Experiment 2. In contrast, Oxley and Frontana showed only slight variation in IT with temperature.

Infection types produced by cultivars possessing *Yr7* were not significantly affected by pathotype or temperature (Table 5.12). However, Gatcher R appeared to have slightly higher infection types to all pathotypes when exposed to periods of low temperature.

5.4.2.2 Light intensity

An experiment was conducted in a controlled environment cabinet maintained at 17±3C with a 12 hour light period. Light was provided by fluorescent[1] and incandescent lamps. Two light intensity regimes were imposed by using shade cloth to cover one replicate of a

[1]Sylvania ®, cool white powertube.

TABLE 5.12

Infection type responses produced by cultivars possessing *Yr7* when inoculated with avirulent pathotypes and incubated at various temperatures

Cultivar	17±2C, 18 days[1]				10±2c, 20 days		11±2C/18±2C, 20 days	
	104 E137 A-	104 E137 A+	108 E141 A-	108 E141 A+	104 E137 A+	108 E141 A-	104 E137 A+	108 E141 A-
Sunkota	;N	;N	;1=	;	;N	;N	;N	;N
Norquay	;N1-	1=C	1-C	;N	1-	1-	;C1-	1-
Gatcher R	1+C	1+C	1C	2C	2	2	2	2
Gatcher S	3++	3+	3	2C	3-	33+	33+	33+

[1]interval from inoculation to recording notes.

duplicated set of materials. This reduced light intensity at plant level by approximately 60 per cent.

The results are presented in Table 5.13. Infection types for compatible host-pathotype interactions at the higher light intensity were slightly higher than those exposed at the lower light intensity. The interactions of *YrA*-possessing cultivars with the avirulent pathotype 108 E141 A- resulted in distinctly higher ITs at low light intensity. This resulted in inability to recognise incompatibility associated with *YrA* under such conditions. Selected results are illustrated in Plate 5.1. In addition, at the higher light intensity, the IT responses with 108 E141 A- were higher than generally experienced with this pathotype under glasshouse conditions (Section 5.4.1.1). This suggested that light intensities of 740 foot candles were beginning to affect the expression of the low IT in cultivars possessing *YrA*. In comparison to the controlled environment cabinet, glasshouse light intensities ranged from 675 foot candles on winter days to 2000 foot candles on summer days (Section 3.2.3.1).

Cultivars possessing *Yr6* gave only slightly higher ITs with the avirulent pathotype 104 E137 A+ at the lower light intensity. This was particularly apparent with Bindawarra, although this cultivar produced an intermediate response at the higher light intensity.

Results for cultivars possessing *Yr7* (Table 5.13) indicated that low ITs produced at lower light intensity were only slightly higher than those produced at the higher light intensity.

It was concluded that variation in light intensity did not affect the ability to recognise the *Yr7* or the *Yr6* host:pathotype specificities. On the other hand, it was impossible to recognise *YrA* at low light intensity.

5.4.3 ADULT PLANT TESTS

The responses of adult plants to various pathotypes were examined in field experiments, conducted in 1985. Cultivars were sown in hill plots (25 seeds at 30cm centres), each

TABLE 5.13

Infection types of cultivars inoculated with two pathotypes of *P. striiformis tritici* and exposed at two light intensities at 17±3C

Cultivar	740±78 foot candles		255±52 foot candles	
	104 E137 A+	108 E141 A-	104 E137 A+	108 E141 A-
YrA Cultivars				
Avocet R	3	;N1-	3	3
Egret R	3+	22+	3	3-
Banks R	3+	;N	33+	3
Condor R	3+	22+	3	3
Sunstar	3+	22+	3	3
Vulcan	3+	;1	33+	3
WW15	3+	22+	3	3
Inia 66	3	;N12	3	2+
Nainari 60	3+	;N1-	3	3
Sonalika	2+3	;N	3-	3-
Yr6 Cultivars				
Oxley	;N	3+	1+	3
Jacup	;N	4	3-	3+
Miling	;N	4	;N1	3+
Millewa	;N	4	;	3
Takari	;N	33+	;N	3
Bindawarra	2+	3+4	3-	3+
Heines Peko	;N	4	1-	3
Pitic 62	;N	33-	1-	3
Penjamo 62R	;N	33-	;C	33+
Frontana	;	4	1=	3
Tobari 66	;N	4	;	3
Atlas 66	;N	3+4	1-	3
Shoshi	;N	33-	;C	2+
Romany	;N	3	1-	3
Yr7 Cultivars				
Sunkota	;N	;N	1=	1-
Gatcher R	1	1-	2	2-
Gatcher S	3	3+	3-	33-
Norquay	;N	;N	1	1-
Control				
Sonora	3+4	3+4	3+	3+

surrounded on four sides by comparable plots of the susceptible cultivar, Zenith. Three experimental areas, individually sown as randomised complete blocks with three replications, were separated by 1m blocks of tall, stripe rust resistant *Secale cereale*. Pathotypes 104 E137 A+, 108 E141 A- and 108 E141 A+ were separately inoculated into each experimental area.

Plots were assessed for per cent leaf area affected by rust from early to mid-September on three occasions, although the data did not vary greatly between dates. Data collected on the 9th September, when plants were between growth stages 35 and 50, were used for analysis.

For the purpose of statistical analyses, cultivars were divided into the following groups according to seedling responses:

Group A -cultivars displaying *YrA* specificity
Group B -cultivars displaying *Yr7* specificity
Group C -cultivars displaying *Yr6* specificity
Group D -cultivars displaying *Yr6 + YrA* specificity
Group E -cv.s Pavon 76 and Hermosillo 77
Group F -cv. Corella
Group G -cv. Nacozari 76

An analysis of variance was applied to each experiment using per cent leaf area affected by rust which was log transformed prior to analysis.

5.4.3.1 Results

The raw data from each experiment are presented in Appendix Table 2. Analyses of variance for the three experiments are presented in Table 5.14. Significant differences were noted between groups in all experiments.

Means and standard errors for respective groups in each experiment, arranged in order of increasing response, are presented in Table 5.15. Group F was the most resistant in contrast to Group B which produced the highest mean response to each pathotype. Groups D,E and G showed responses which were not significantly different ($p>0.05$) to the most

TABLE 5.14

Analyses of variance for adult-plant response of cultivar groups to pathotypes of *P. striiformis tritici*

Source of Variation	104 E137 A+			108 E141 A-			108 E141 A+		
	df	Mean Square	F Ratio	df	Mean Square	F Ratio	df	Mean Square	F Ratio
Replicates	2	0.1103	1.829	2	0.0392	0.936	2	0.0367	0.604
Between Groups	6	0.2680	4.444**[1]	6	0.2712	6.473**	6	0.3567	5.867**
Group A	13	0.1612	2.673**	13	0.1187	2.833**	13	0.1218	2.003*
Group B	3	0.3858	6.398**	3	0.3043	7.263**	3	0.2944	4.842**
Group C	17	0.2015	3.342**	17	0.2622	6.258**	17	0.2575	4.235**
Group D	1	0.0379	0.629	1	0.0379	0.905	1	0.0151	0.248
Group E	1	0.0151	0.250	1	0.0677	1.616	1	0.0000	0.000
Error	70	0.0603		75	0.0419		74	0.0608	
Total	113			118			117		

[1]Significant F Ratio p<0.05*
p<0.01**

resistant group F in all experiments. Although differences between experiments could not be contrasted statistically, qualitative comparisons suggested that Group A showed no change in response to the three pathotypes. It was expected that this group may have shown a low response to 108 E141 A- and higher responses to 104 E137 A+ and 108 E141 A+. Group C appeared to show a slightly lower response to 104 E137 A+ (13.75% leaf area affected) compared to 108 E141 A- (18.28%)although this was less clear with 108 E141 A+ (14.75%).

Significant differences in response occurred between cultivars within Groups A, B and C in each experiment. Means and standard errors for individual cultivars in Group A, arranged in order of increasing response within each experiment, are presented in Table 5.16. Cultivar Vulcan was the most resistant and Sonalika, Avocet S and Egret S consistently the most susceptible to each pathotype. With pathotype 108 E141 A-, the resistant selections of cultivars Avocet, Banks, Condor and Egret showed the expected lower

TABLE 5.15

Mean leaf area affected (log_{10}) of cultivar groups when infected with *P. striiformis tritici* pathotypes and assessed as three independent field experiments

	104 E137 A+			104 E141 A-			108 E141 A+		
Group[1]	Mean		Standard Error	Group	Mean	Standard Error	Group	Mean	Standard Error
F	0.699	a[2]	0.142	F	0.716 a	0.118	F	0.699 a	0.142
E	0.849	ab	0.100	G	0.900 ab	0.118	E	0.799 ab	0.101
D	0.937	ab	0.100	D	0.979 abc	0.083	G	0.799 ab	0.142
G	1.000	ab	0.142	E	1.022 abc	0.091	D	0.950 ab	0.101
A	1.058	ab	0.043	A	1.114 bc	0.032	C	1.169 b	0.035
C	1.139	ab	0.035	C	1.262 bc	0.029	A	1.205 b	0.040
B	1.292	b	0.071	B	1.278 c	0.059	B	1.234 b	0.071
experiment mean	0.996		0.037		1.038	0.034		0.979	0.037

[1]Group A *(YrA)*
Group B *(Yr7)*
Group C *(Yr6)*
Group D *(Yr6 + YrA)*
Group E (Pavon 76, Hermosillo 77)
Group F (Corella)
Group G (Nacozari 76)

[2]Duncan's multiple-range test; means with the same letter are not significantly different ($p>0.05$).

TABLE 5.16

Mean leaf area affected (log_{10}) of cultivars showing *YrA* resistance inoculated with *P. striiformis tritici* pathotypes and assessed as three independent field experiments

Cultivar	104 E137 A+ Mean[1]		Standard Error	Cultivar	108 E141 A- Mean		Standard Error	Cultivar	108 E141 A+ Mean		Standard Error
Banks R	0.648	a	0.175	Vulcan	0.874	a	0.121	Vulcan	0.865	a	0.177
Vulcan	0.659	a	0.145	Banks R	0.907	a	0.146	Sunstar	1.001	a	0.146
WW15	0.799	ab	0.175	Inia 66	0.975	a	0.121	Nainari 66	1.002	a	0.181
Condor S	0.799	ab	0.175	Condor R	0.975	a	0.121	Avocet R	1.060	ab	0.146
Condor R	0.860	ab	0.145	Egret R	0.975	a	0.121	Condor S	1.074	ab	0.177
Egret R	0.860	ab	0.145	Nainari 60	0.991	ab	0.121	Banks S	1.101	ab	0.146
Sunstar	0.860	ab	0.145	Banks S	1.033	ab	0.121	Egret R	1.160	ab	0.146
Avocet R	0.960	abc	0.145	Sunstar	1.033	ab	0.121	WW15	1.240	abc	0.181
Nainari 60	1.119	bc	0.146	Avocet R	1.075	ab	0.121	Condor R	1.261	abc	0.146
Avocet S	1.230	bcd	0.176	Condor S	1.092	ab	0.121	Banks R	1.302	abc	0.146
Egret S	1.241	bcd	0.181	Egret S	1.134	ab	0.121	Inia 66	1.319	abc	0.146
Banks S	1.241	bcd	0.181	WW15	1.175	ab	0.121	Avocet S	1.334	abc	0.146
Sonalika	1.379	cd	0.148	Avocet S	1.367	bc	0.121	Egret S	1.493	bc	0.146
Inia 66	1.602	d	0.245	Sonalika	1.641	c	0.121	Sonalika	1.668	c	0.146

[1]Duncan's multiple-range test; means with the same letter are not significantly different ($p>0.05$).

responses than the corresponding susceptible selections, although the differences were not significant ($p>0.05$). Differences between these selections with the *YrA*-virulent pathotypes were not consistent. Qualatitive comparisons between experiments indicated that Inia 66 was distinctly low in response to 108 E141 A- (9.43% leaf area affected) compared with its response to 104 E137 A+ (40.00%) and 108 E141 A+ (20.86%). However, other cultivars did not show the expected response differences between pathotypes virulent and avirulent with respect to *YrA*.

The responses of Group B cultivars with *Yr7* to the three pathotypes were not significantly different (Table 5.17). However, Norquay and Sunkota were consistently lower in response compared to Gatcher R. The absence of *Yr7* in Gatcher S resulted in higher responses to the three pathotypes, although the response difference between Gatcher R and Gatcher S was significant ($p<0.05$) in only one of the three experiments. The relatively high response of Gatcher S caused the mean response of Group B (Table 5.15) cultivars to be higher than would otherwise be expected for cultivars with *Yr7*.

The responses of Group C cultivars (Table 5.18) to 104 E137 A+ were generally low compared with the other two pathotypes. Cultivars Jacup, Tobari 66 and Millewa gave unexpectedly high responses with 104 E137 A+. Examination of raw data revealed that an unusually high response was noted in one of the three replicates for each cultivar, suggesting that some contamination with *Yr6*- virulent pathotypes may have occurred in the experimental area. Penjamo 62S, selected for the absence of *Yr6*, was significantly ($p<0.05$) higher in response to 104 E137 A+ than Penjamo 62R. However, the R and S selections of Penjamo 62 were not significantly different in response to the *Yr6* - virulent pathotypes, 108 E141 A- and 108 E141 A+.

The responses of Group C cultivars to the *Yr6*-virulent pathotypes showed variation ranging from low (5.01% for Oxley with 108 E141 A+) to high (59.59% for Millewa with the same pathotype). Cultivars Oxley, Frontana, Shoshi,

TABLE 5.17

Mean leaf area affected (log_{10}) for cultivars with *Yr7* when inoculated with three pathotypes of *P. striiformis tritici* and assessed as three independant field experiments

104 E137 A+				108 E141 A-				108 E141 A+			
Cultivar	Mean		Standard Error	Cultivar	Mean		Standard Error	Cultivar	Mean		Standard Error
Norquay	0.860	a[1]	0.1456	Sunkota	0.975	a	0.1212	Norquay	0.901	a	0.1462
Sunkota	1.061	ab	0.1456	Norquay	1.033	ab	0.1212	Sunkota	1.102	a	0.1462
Gatcher R	1.438	bc	0.1456	Gatcher R	1.334	ab	0.1212	Gatcher R	1.302	ab	0.1462
Gatcher S	1.654	c	0.1456	Gatcher S	1.668	c	0.1212	Gatcher S	1.636	b	0.1462

[1]Duncan's multiple-range test; means with the same letter are not significantly different ($p<0.05$)

TABLE 5.18

Mean leaf area affected (log_{10}) for cultivars with *Yr6* when inoculated with pathotypes of *P. striiformis tritici* and assessed as three independant field experiments

104 E137 A+				108 E141 A-				108 E141 A+			
Cultivar	Mean		Standard Error	Cultivar	Mean		Standard Error	Cultivar	Mean		Standard Error
CSP44[1]	0.649	a[2]	0.1758	Frontana	0.812	a	0.1502	Oxley	0.700	a	0.1462
Atlas 66	0.760	a	0.1456	Atlas 66	0.819	a	0.1465	Frontana	0.801	ab	0.1462
Shoshi	0.819	a	0.1456	Shoshi	0.874	ab	0.1212	WW80	0.901	abc	0.1462
WW80	0.860	a	0.1456	Oxley	0.933	abc	0.1212	Romany	0.901	abc	0.1462
Heines Peko	0.860	a	0.1456	WW80	0.970	abc	0.1465	CSP44[1]	0.960	abc	0.1462
Frontana	0.860	a	0.1456	Heines Peko	0.975	abc	0.1212	Tobari 66	1.001	abcd	0.1814
Oxley	0.960	a	0.1456	CSP44[1]	1.033	abc	0.1212	Shoshi	1.001	abcd	0.1814
Takari	1.019	a	0.1456	Tobari 66	1.075	abc	0.1212	Penjamo 62R	1.102	abcde	0.1462
Romany	1.061	a	0.1456	Takari	1.134	abcd	0.1212	Pitic 62	1.102	abcde	0.1462
Pitic	1.178	b	0.1456	Penjamo 62R	1.234	bcde	0.1212	Heines Peko	1.102	abcde	0.1462
Bindawarra	1.193	b	0.1456	Romany	1.334	cdef	0.1212	Atlas 66	1.160	bcde	0.1462
Miling	1.220	b	0.1456	Penjamo 62S	1.494	defg	0.1212	Frontiera	1.201	bcde	0.1814
Penjamo 62R	1.262	b	0.1456	Miling	1.526	fg	0.1212	Takari	1.261	cdef	0.1462
Frontiera	1.299	b	0.1758	Jacup	1.541	fg	0.1212	Miling	1.435	defg	0.1462
Jacup	1.330	bc	0.1806	Pitic 62	1.567	fg	0.1212	Bindawarra	1.461	efg	0.1462
Tobari 66	1.338	bc	0.1758	Frontiera	1.572	fg	0.1465	Penjamo S	1.527	efg	0.1766
Millewa	1.420	bc	0.1456	Millewa	1.668	g	0.1212	Jacup	1.668	fg	0.1462
Penjamo 62S	1.695	c	0.1456	Bindawarra	1.700	g	0.1212	Millewa	1.775	g	0.1462

[1]CSP44 selection homogeneous *Yr6*

[2]Duncan's multiple-range test; means with the same letter are not significantly different ($p < 0.05$)

Heines Peko, Tobari 66, WW80 and CSP44 consistently showed responses of less than 13% leaf area affected to pathotypes virulent for *Yr6*. In contrast, Millewa, Bindawarra, Jacup, Miling and Penjamo 62S consistently showed responses which were greater than 28% leaf area affected. The remaining cultivars were variable or intermediate in response to these pathotypes.

Cultivars in Groups D,E,F and G were consistently low in response to all three pathotypes (Table 5.19). Analyses of variance indicated that there were no significant differences between cultivars within these groups.

5.4.3.2 Conclusions

These field experiments demonstrated that cultivars showing similar pathotype specificities in seedling tests were also resistant to the appropriate avirulent pathotype/s as adult plants under field conditions. However, tests with virulent pathotypes frequently indicated the presence of adult-plant resistances in addition to the corresponding seedling resistance factors. For example, Vulcan with *YrA* and Oxley with *Yr6* were more resistant than other wheats possessing the same genes.

A number of difficulties were encountered in the experiments. The experimental design did not allow statistical comparisons between pathotypes. Thus cultivar x pathotype interactions could not be studied to determine possible specificities for adult-plant resistances. In addition, it appears that increased replication may be necessary to increase statistical sensitivity in distinguishing between the adult-plant responses of cultivars with the same designated seedling resistance factors.

Infection levels of pathotypes 104 E137 A+ and 108 E 141 A+ on a range of cultivars, including Avocet plants with the *YrA* resistance, was usually less than 50%. This contrasts with observations indicating that the response of Avocet can be as high as 100% in other experiments or in commercial field conditions. Relatively low infection levels may have been due to sub-optimal plant growth, although

TABLE 5.19

Mean leaf area affected ($\log_{10}$) of cultivars to *P. striiformis tritici* pathotypes in three independent experiments

Cultivar	104 E137 A+ Mean[2]		Standard Error	Cultivar	108 E141 A- Mean		Standard Error	Cultivar	108 E141 A+ Mean		Standard Error
Corella	0.659	a	0.145	Corella	0.691	a	0.1466	Corella	0.700	a	0.146
Pavon 76	0.760	a	0.145	Cajeme 71	0.874	ab	0.1212	Pavon 76	0.800	a	0.146
CSP44[1]	0.818	a	0.145	Hermosillo 77	0.874	ab	0.1212	Hermosillo 77	0.800	a	0.146
Hermosillo 77	0.860	a	0.145	Nacozari 76	0.874	ab	0.1212	Nacozari 76	0.800	a	0.146
Nacozari 76	0.960	a	0.145	CSP44	1.033	ab	0.1212	Cajeme 71	0.901	a	0.146
Cajeme 71[1]	0.977	a	0.145	Pavon 76	1.113	b	0.1502	CSP44	1.001	a	0.146

[1] selections homogeneous *Yr6 + YrA*

[2] Duncan's multiple-range test; means with same letter are not significantly different ($p>0.05$).

susceptible spreader plots were uniformly and totally infected. It is possible that the pathotypes chosen were inappropriate as variation in virulence with respect to adult plants may have occurred independently of virulence detected with seedlings. Current methods used in pathotype analysis and culture selection did not take account of possible variation with respect to adult plant virulence.

5.5 PEDIGREE ANALYSIS

The source and distribution of *Yr7* appeared to be separate and distinct from *Yr6* and *YrA*. The North American wheat, Thatcher, was the key contributor to the pedigrees of various cultivars possessing *Yr7*. Cultivar Lee was considered to be a derivative of Thatcher (McIntosh *et al.*, 1981) rather than Hope/Timstein (Zeven and Zeven-Hissink, 1976).

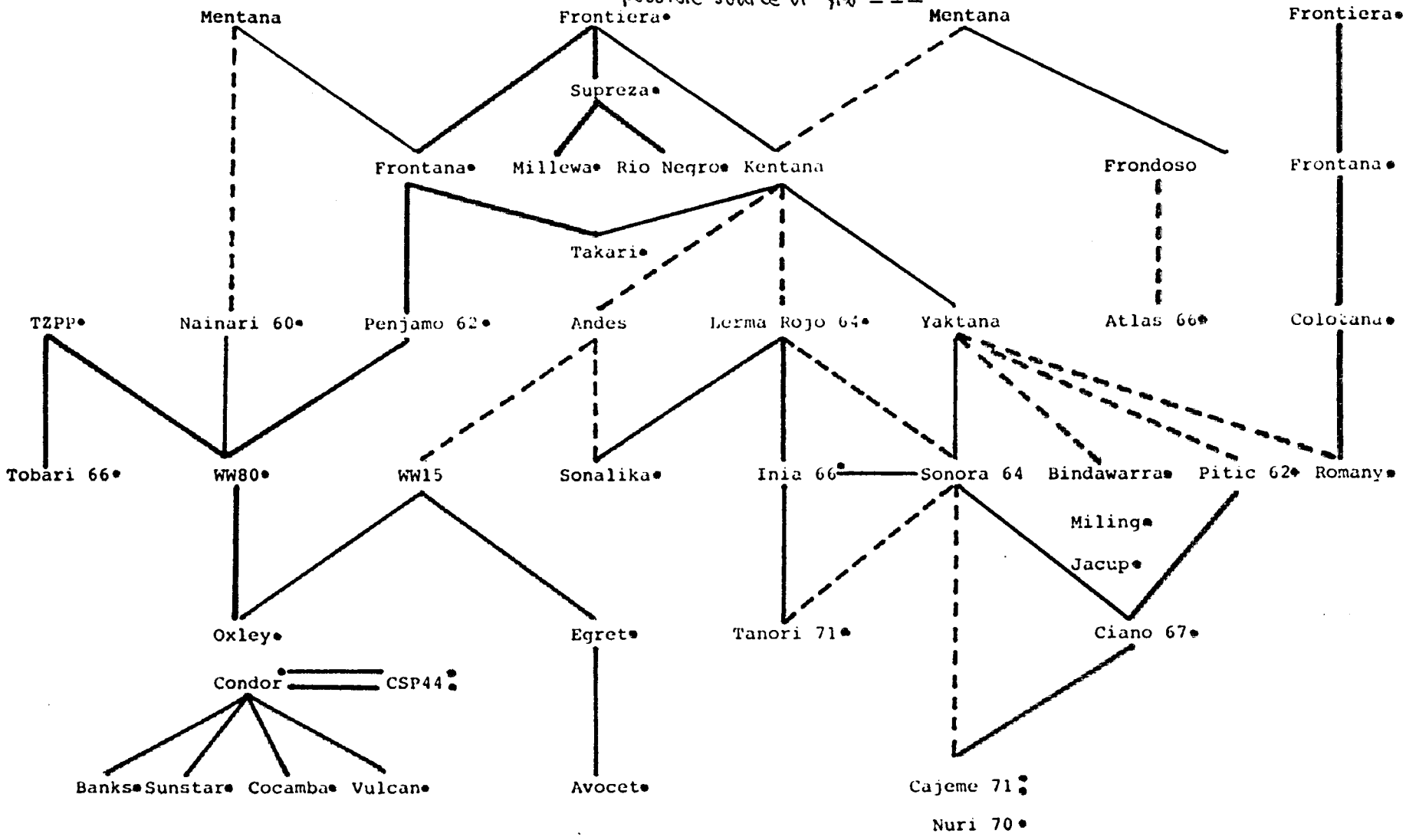
In contrast, the pedigrees of cultivars possessing *Yr6* or *YrA* were closely interrelated (Figure 5.1). Indeed, it was surprising that only two wheats appeared to have both resistances in combination. The close pedigree relationship between *Yr6* and *YrA* was exemplified in the breeding and selection of Australian wheats. The selection WW15, a red-seeded semi-dwarf wheat, was crossed with WW80, a white-seeded wheat of normal stature. The white-seeded, semi-dwarf progenies of this cross were selected and ultimately released as cv.s Condor and Oxley. The *YrA* and *Yr6* resistances, contributed by WW15 and WW80 respectively, were apparently randomly distributed in the resulting breeding populations as Oxley was shown to possess *Yr6* and Condor was shown to be heterogeneous for *YrA*. However, a single plant selection of Condor taken by N.H. Luig, Plant Breeding Institute, University of Sydney, and designated CSP44, was shown to possess *Yr6* and to be heterogeneous for the *YrA* resistance. The IT responses of WW15, WW80, Condor R and CSP44 to a range of pathotypes is illustrated in Plate 5.3.

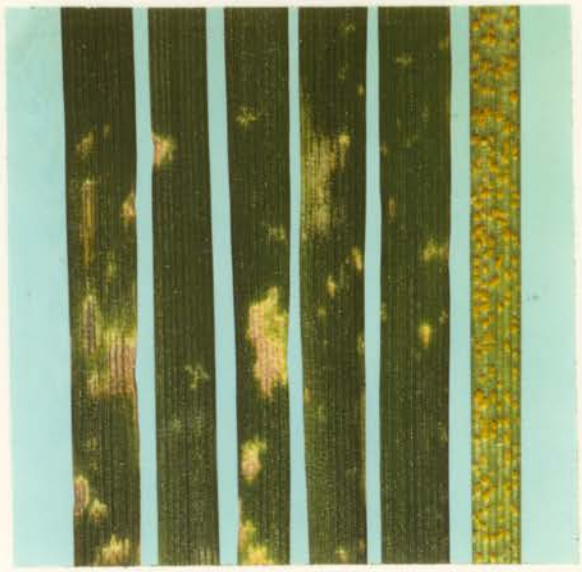
The Brazilian wheat, Frontiera, appeared to be a likely source of *Yr6* among the materials studied. The

FIGURE 5.1

Pedigree relationships and possible pathways for host resistance genes *Yr*A and *Yr*6

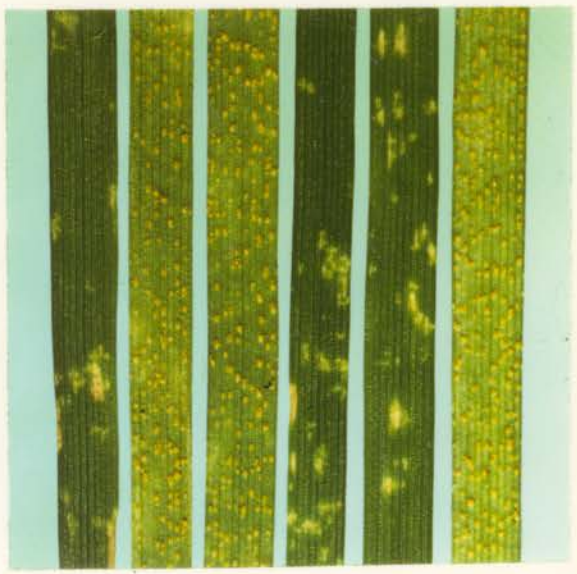
pedigree relationships —— possible source of *Yr*A - - - confirmed presence of *Yr*A •
presumed source of *Yr*A —— presumed source of *Yr*6 ═══ confirmed presence of *Yr*6 •
possible source of Yr6 - - -





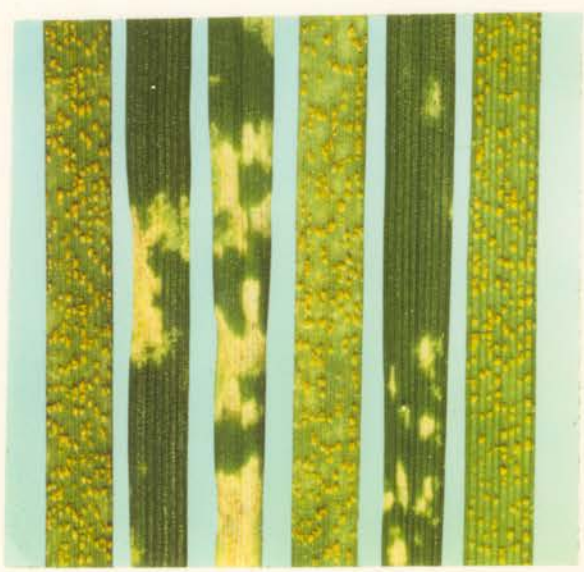
1 2 3 4 5 6

104 E137 A-



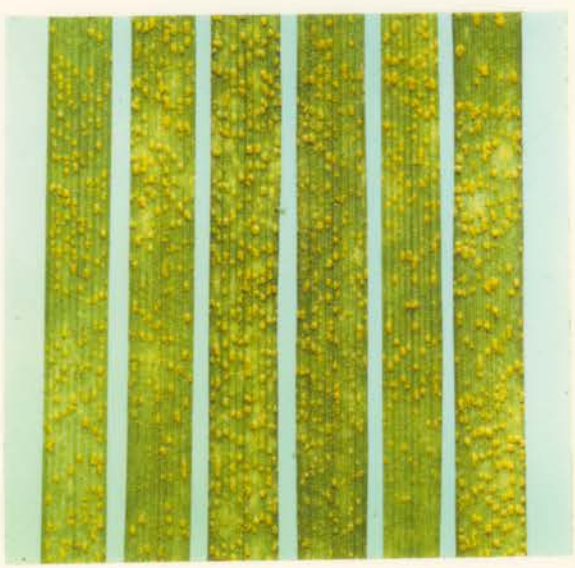
1 2 3 4 5 6

104 E137 A+



1 2 3 4 5 6

108 E141 A-



1 2 3 4 5 6

108 E141 A+

PLATE 5.3

Infection types of related wheats in response to inoculation with *Puccinia striiformis f.sp. tritici*

1=WW80 2=WW15 3=Condor R 4=CSP44*(Yr6)* 5=CSP44*(Yr6+YrA)*

6=Federation

origins of *YrA* among these materials were less clear as seedling tests of some sources of Mentana, Kentana, Andes and Yaktana have failed to confirm the presence of the *YrA* resistance. However, a recent test confirmed the presence of *YrA* in one source of Lerma Rojo 64. Although this requires confirmation, it appears that this cultivar could be the source of *YrA* in Sonalika, Inia 66, Cajeme 71 and Tanori 71. The origins of *YrA* in Nainari 60 and WW15, and related genotypes, remains undetermined.

5.6 DISCUSSION

Reported surveys of host resistance to *P. striiformis tritici* pathotypes are few. Whereas early progress, initiated by Zadoks (1961) and Lupton and Macer (1962), was made in cataloguing certain resistance genes and establishing standard testers for those genes, many later investigators did not attempt to relate their pathotypes and their proposed host resistances to those already described *e.g.* Purdy and Allan, 1963; Sandhu *et al.*, 1977; Singh and Sethi, 1978; Gill *et al.*, 1978. While such reports may have provided useful information for local breeding programs, they did not provide information that was useful or interpretable on an international basis.

In the current studies, pathotypes of *P. striiformis tritici* were characterised with respect to the international and European differential sets. With this information, pathotype pairs varying in avirulence and virulence with respect to particular host resistance factors were selected to assay for the presence or absence of the corresponding resistance genes in an array of cultivars and advanced lines.

The *Yr7* gene was detected in cv.s Chris, Gatcher R, Manitou, Thatcher, Lee and Celebration, agreeing with the report of McIntosh *et al.* (1981). The presence of *Sr9g*, and thus *Yr7*, in cv. Eagle (McIntosh, 1983) was confirmed on the basis of specificity using pathotype 106 E139 A-. The proposed *Yr7-Sr9g* genotype of cv.s Sunkota, Oroua, Norquay, Sinton and South Africa 43 have not been previously reported. The cv.s Norquay, Sunkota and Gatcher R.

were shown to have adult plant resistance effective against *Yr7*-avirulent pathotypes in the field. The *Yr7* gene has been reported in cultivars from Pakistan (Perwaiz and Johnson, 1985) and India (Sawhney and Luthra, 1970), but does not appear to be frequent in European wheats (Taylor *et al.*, 1981; Kema *et al.*, 1984).

The *Yr6* gene was detected in a number of Australian and exotic wheats. With the exception of Heines Kolben, the 20 bread wheat cultivars listed in Table 5.7 have not been previously reported to possess *Yr6*. Resistance attributed to *Yr6* was noted to be temperature sensitive in seedling tests, and was effective in adult-plants inoculated in the field with pathotypes with the corresponding avirulence. There was a wide range of adult-plant responses among cultivars possessing *Yr6*. The Australian cv. Oxley was rated as resistant to *Yr6*-virulent pathotypes, whereas cv.s Millewa and Bindawarra were susceptible.

The presence of *Yr6* in Ciano 67 reported in these studies differs from the suggestion of *Yr7* given by McIntosh *et al.* (1981). The Ciano 67 stock used in the present studies was obtained by R.A. McIntosh from C.N. Law, Plant Breeding Institute, Cambridge, England. However, Perwaiz and Johnson (1985) noted some stocks of Ciano 67 as having *Yr6* and *Yr7* in combination. In view of these discrepancies, further work is necessary to examine a range of Ciano 67 stocks before the resistance genotype is confirmed.

Yr6 appears to be common in a wide range of wheats from many countries. This gene has been reported in cultivars from Pakistan (Perwaiz and Johnson, 1985), India (Sawhney and Luthra, 1970) and England (Taylor *et al.*, 1981). The present studies suggested that *Yr6* is common in CIMMYT-produced wheats, particularly among their early cultivars such as Pitic 62 and Penjamo 62 (Briggle and Vogel, 1968). The use of CIMMYT populations by Australian breeding programs explains the occurrence of *Yr6* in certain Australian commercial wheats.

Stripe rust resistance in durum wheats has not received the degree of attention given to bread wheats. Joshi *et al.* (1985) observed that durum wheats were frequently infected by stripe rust in southern India, but gave no details of cultivar responses. Rao *et al.* (1963) concluded that a single dominant gene in the durum line, St.464, conferred adult-plant resistance. Results reported here indicated that *Yr6* may be present in some durum lines. The range and frequencies of resistance genes present in other durums remain unknown. It is highly likely that a number of catalogued resistance genes are present in durum cultivars as six of seven resistance genes (viz. *Yr1,Yr2,Yr5,Yr6,Yr7,Yr10,Yr15*) have been located in chromosomes of the A or B genomes. For example, Iumillo durum contributed *Sr9g-Yr7* to the breadwheat cultivar, Thatcher (McIntosh *et al.*, 1981).

Pathogenic variation with respect to wheats with the tentatively designated *YrA* resistance have not been reported previously. Using local pathotypes, this resistance was detected in a large range of Australian and exotic wheats. Those selected and released from CIMMYT-distributed populations frequently possessed *YrA*, *e.g.* Sonalika, Inia 66, WW15. In Australia, the presence of *YrA* reflected the widespread use of WW15 following its introduction in the 1960's. Several derivatives of WW15 were released in the mid-1970's (Syme, 1983) and these cultivars have dominated the eastern Australian wheat areas over the past decade. The presence of *YrA* in these wheats was fortuitous, since many of them were developed prior to 1979 and were thus not selected for stripe rust resistance. This is reflected in genetic heterogeneities of cultivars, such as Egret, Condor, Banks and Avocet, for the presence of *YrA*.

Adult-plant responses among cultivars possessing *YrA*, when tested in the field with *YrA*-virulent pathotypes, varied over a wide range of resistant and susceptible responses. In some cultivars, high levels of resistance were detected (*e.g.*, c.v. Vulcan). However, the nature and potential

durabilities of such resistances will require further study. Anza has been reported as a possible source of durable resistance (Johnson, 1983c) and this suggests that some Australian cultivars may have inherited additional resistances from WW15 which is synonymous with Anza. However, there have been reports of high levels of stripe rust on Anza from Chile, West Germany and China (Anon., 1982).

Genetic heterogeneity within cultivars and advanced lines for response to various pathotypes was relatively frequent. This feature was very common among wheats possessing *YrA*, and less frequent in cultivars with *Yr6* or *Yr7*. Reports of cultivar heterogeneity in response to *P. striiformis* are comparatively rare. Manners (1950) noted that some wheats in the standard differential set of Gassner and Straib gave heterogeneous responses, but did not comment as to whether this represented microenvironmental effects or variation in the host genotypes.

Studies of the effects of environment on seedling host responses showed that wheats with *Yr7* were not affected by variations in temperature or light intensity. In contrast, wheats with *Yr6* showed higher ITs with avirulent pathotypes at lower temperatures. Manners (1950) drew attention to the early reports of Gassner and Straib (1932) who observed the same pattern of temperature sensitivity in relation to Heines Kolben, in which the gene responsible was later designated *Yr6*. In other examples of temperature sensitive resistances, the general trend was also for increasing compatibility with decreasing temperature (Zadoks, 1961). However, Sharp (1965) found that cv. Rego was resistant at low temperatures and susceptible at high temperatures, in contrast to P.I. 178383 and Itana, which remained resistant and susceptible, respectively, at both temperature regimes. Strobel and Sharp (1965) demonstrated that the responsiveness and non-responsiveness of cultivar resistance to temperature variations was related to the presence of protein bands in electrophoretic tests. They speculated that the additional protein in some cultivars, when grown at high temperatures, may aid pathogen growth, thus increasing

the degree of compatibility.

Resistance attributable to *YrA* was shown in the present studies to be sensitive to changes in light intensity. Responses to avirulent pathotypes were lower at a higher intensity and were high at the lower light intensity that was imposed. A review of early work with a range of cereal rusts indicated that high light intensities permitted optimum growth of pathogens (Bever, 1934a). Mares and Cousen (1977) measured colony growth in leaves of cv. Nord Desprez inoculated with *P. striiformis* and found higher growth rates at higher light intensities. Bever (1934a) studied the compatible interaction between Pannier barley and *P. striiformis*, and found that light intensity had a negligible influence on IT. In contrast, he found that day lengths exceeding 12 hours resulted in a change of IT from susceptible to resistant. Manners (1950) found similar responses in wheats inoculated with a number of avirulent pathotypes. He demonstrated higher infection types at short compared to long days. Stubbs (1967) found three cultivars which produced susceptible responses at low light intensities and resistant responses at high light intensities. The opposite trend was noted in the responses of Topper barley. These cultivars were thus classified as photolabile. In contrast, some wheats showed no change in infection type at different light intensities and were classified photostable. Slovenvcikova (1974) found that cv. Chambord was more susceptible at low light intensity and demonstrated that in inheritance studies, high light intensities resulted in increased numbers of resistant F2 plants. However, the data suggested that the recessive mode of inheritance was not affected by light intensity.

It was concluded from the present studies that resistances conferred by *Yr6* and *Yr7* were photostable. In contrast, resistance conferred by *YrA* was photolabile. However, the interaction of daylength and light intensity was not investigated in the present study nor in previously published studies.

The resistance evident in cultivars Corella, Pavon 76, Nacozari 76 and Hermosillo 77 could not be determined with the available *P. striiformis* pathotypes. However, responses to *P. graminis tritici* pathotypes suggested that *Sr9g*, and, by implication, *Yr7*, was present in Corella and Pavon 76.

The triticale cultivars Ningadhu, Coorong and Dua possessed unknown resistances. Johnson and Taylor (1976b) also failed to observe differential interactions between triticale cultivars and a range of *P. striiformis* pathotypes which included virulence with respect to *Yr9*. Genetic studies will be required to determine the mode of inheritance of these resistances and their relationship to previously catalogued resistance genes in wheat.

6 INHERITANCE OF RESISTANCE TO STRIPE RUST

6.1 INTRODUCTION

Hypotheses concerning the presence of resistance genes in an array of hosts can be drawn from studies using selected pathotypes. Where previously documented genes are postulated, genetic experiments can be designed to test for allelism using standard stocks. The mode of inheritance, including dominance or recessiveness, and linkage relationships with other genes can be investigated in segregating populations. The information obtained may be useful in breeding where an objective may be to incorporate such resistance genes into agronomically suitable genotypes.

When the available pathotypes are not capable of identifying the resistance factors in certain hosts, investigations of segregating populations, using the available range of pathogenic variation, allow the deduction of the numbers of genes, their modes of inheritance and their linkage relationships with respect to other genes. Appropriate tests of allelism can then be designed in order to confirm the deductions.

Although seedling tests in the glasshouse are convenient for inheritance studies, it is important to relate the results to the responses of such lines under field conditions with the same pathotype(s). Generally, it is expected that genes conferring resistance in seedling tests will also confer resistance to juvenile and adult-plants in the field when tested with the same pathotype. The degree of resistance may vary with the particular gene, the growth stage and the prevailing environmental conditions. Moreover, the detection and study of adult-plant resistance additional to seedling resistance should be an important aspect of genetic investigations.

Thus inheritance studies complement and extend the results of host resistance surveys in providing genetic knowledge for use when incorporating resistance to stripe rust in breeding programs.

Responses of a range of cultivars and advanced lines from various breeding programs in Australia suggested that *Yr6* and *Yr7* may be common (J. Syme, 1980, unpublished report). Because of the initial lack of pathogenic variation, inheritance studies were undertaken to investigate the nature and relationships of seedling resistances in Australian and exotic wheats. The subsequent detection of pathotypes allowed the confirmation and extension of these studies.

6.2 MATERIALS AND METHODS

6.2.1 ADDITIONS TO HOST MATERIALS

The following stripe rust susceptible wheats were used in crosses with resistant stocks: Line E
Teal
Gabo

6.2.2 INHERITANCE STUDIES

6.2.2.1 Populations

For inheritance studies, approximately 50 unclassified F2 seedlings in each cross were transplanted in the field. They were classified as adult-plants for response to stripe rust by marking individuals with coloured paint which persisted on the dry straw until the plants were threshed. Larger populations were separately tested as seedlings to obtain F2 segregation data.

F3 lines were generally classified as homozygous resistant (HR), segregating (Seg) or homozygous susceptible (HS) on the basis of seedling infection type, or the combination of infection type and per cent-leaf-area affected in adult tests in the field. Certain populations were examined as F3 rows in the field at Castle Hill in 1981, 1982 and 1984, and at Horsham (Victoria) in 1981 and 1983. Some populations were duplicated between sites and between years. Where discrepancies occurred in classification for individual F3 lines, the most frequent classification was adopted.

6.2.2.2 χ^2 analyses

χ^2 analyses were applied in testing the goodness of fit of the segregation data to postulated genetic ratios.

Individuals with intermediate F2 responses were pooled with the resistant classes unless otherwise noted. Adult F2 plants with intermediate responses were pooled with either resistant or susceptible classes, depending on the responses of the intermediate group in comparison to parental controls.

Pooling of data and/or Yates correction for continuity (Steele and Torrie, 1980) were not applied to classes of low frequency. This allowed the additive nature of χ^2 to be employed in computing homogeneity χ^2 values, *i.e.*

$$\chi^2 \text{ total} = \chi^2 \text{ individuals} + \chi^2 \text{ homogeneity}$$

This parameter was used as a measure of the similarity of all segregating F3 lines in conforming to the proposed ratio.

6.3 RESULTS

6.3.1 CULTIVARS WITH *Yr6*

6.3.1.1 Glasshouse studies

Results of inheritance studies conducted with cultivars and advanced lines which, in pathotype tests, behaved in similar manner to wheats known to possess *Yr6* are presented in Table 6.1. For comparison, data for the inheritance of *Yr6* in cv.s Heines Kolben and Heines Peko are included, but kept separate from the overall analysis among cultivars. F1 plants from four of the 17 crosses listed in Table 6.1 were tested with pathotype 104 E137 A-. In each case, they produced IT 3+ or 4, in contrast to the respective resistant parental responses of ;N, indicating that resistance was recessive. Segregation in F2 populations and in segregating F3 lines indicated a single recessive gene in each cultivar. Within segregating lines, ITs among plants classified as resistant ranged from ;C to 1 in Pitic 62/Line E, to ;1C or 12 to 3-C in Bindawarra/Line E. Susceptible plants in the same lines were always clearly distinctive with ITs 33+ and 3+.

In crosses where F3 lines were tested, the ratios of line behaviour conformed satisfactorily with the hypothesised ratios of 1HR:2Seg:1HS. The pooled ratios of resistant and susceptible plants within segregating lines conformed satisfactorily with 1:3 ratios in 15 of the 19 crosses; the pooled data for three crosses deviated from the expected at the $p = 0.05$ level, and one deviated at $p = 0.01$. All of these deviations were restricted to different populations within two crosses, *viz.* Line E/Millewa and Jacup/Gabo. However, χ^2 homogeneity values were not significant for these populations, indicating consistent bias in the results. In Line E/Millewa, both the F2 and F3 segregations tended toward excesses of resistant segregates. It appeared that this could have had some biological significance. On the other hand, the deviations in the Jacup/Gabo cross could have been random, since the F2 segregation tended towards excessive resistant segregates in contrast to the F3 populations. The χ^2 homogeneity value for segregating F3 lines in the Romany/Teal population was significant at the $p = 0.05$ level, although the data for individual lines were generally consistent for a single recessive gene.

Test of allelism Cultivars Heines Kolben and Heines Peko, European standards possessing *Yr6*, were crossed with Oxley. The results of stripe rust tests on F1, F2 and F3 progenies are presented in Table 6.2. Low ITs on F1 plants and the lack of F2 and F3 segregates with High ITs confirmed that Oxley possessed *Yr6* . This cultivar was then used as a standard for *Yr6* in further tests of allelism (Table 6.2).

Infection types on seedlings from the crosses involving Oxley varied from ;N to 23-. However, ITs 3 or 3+ were noted in five crosses. If the resistance factors in the respective wheats were allelic, then the susceptible plants were either

1. misclassified resistant plants
2. progenies of occasional outcrosses between F1 plants and susceptible genotypes such that the progeny plants were susceptible due to the recessive nature of the gene

TABLE 6.1

F1, F2 and F3 seedling data for crosses of cultivars possessing *Yr6* following inoculation with *P. striiformis tritici* pathotype 104 E137 A-

Cross [1]		F1	F2			F3				Segregating F3 Lines		
		IT	ITs	R+I :S	$\chi^2_{1:3}$	HR	Seg	HS	$\chi^2_{1:2:1}$	Pooled R+I :S	$\chi^2_{1:3}$	χ^2_{homog}
Heines Peko/Line E	1	3+	;1CN:33+	6:16	0.06	13	15	10	2.16	77:269	1.39	8.08
	2		;N1:33+	5:22	0.46	9	23	9	0.61	129:391	0.01	15.70
Heines Kolben/Line E		33+	;C:3+	49:134	0.38							
Pitic 62/Line E	1	4	;C:33+	23:51	1.46	16	18	10	3.09	154:457	0.01	6.94
	2		;;N:3+	28:61	1.98	11	29	14	0.63	175:529	0.01	5.92
Bindawarra/Line E			12,3-C:3+	9.32	0.20	19	18	14	5.39	101:295	0.05	10.72
Bindawarra/Teal		4	;C1:3+	23:72	0.03							
Line E/Millewa[3]	1		;N2-C:3+	38:79	3.50	21	31	10	4.03	197:441	11.75**[2]	23.28
	2					12	27	9	1.13	160:396	4.23*	28.62
Jacup/Gabo	1	3+	;;N:3+	48:108	2.77	7	18	7	0.50	75:293	4.18*	7.95
	2					3	18	12	5.18	70:288	5.66*	7.77
Jacup/Line E			;N,12C:3+	84:230	0.51							
Romany/Teal[3]						5	14	5	0.73	102:297	0.08	21.27*
Line E/Oxley			;N,2=:3+	18:50	0.08	16	29	21	1.52	130:475	3.98	16.16
Miling/Teal			;N,1C:3+	58:220	2.53	20	33	23	1.55	200:554	0.93	35.74
Takari/Teal		4	;C1-:3+	24:62	0.39							
WW80/Line E	1		;N,2=:3+	18:39	1.32	14	22	11	0.57	104:293	0.30	12.12
	2			20:41	1.97	19	20	11	4.56	88:275	0.11	8.68

TABLE 6.1 cont.

Cross [1]		F1	F2			F3				Segregating F3 Lines		
		IT	ITs	R+I :S	$\chi^2_{1:3}$	HR	Seg	HS	$\chi^2_{1:2:1}$	Pooled R+I :S	$\chi^2_{1:3}$	χ^2_{homog}
Shoshi/Teal[3]	1					10	22	6	1.79	124:378	0.02	19.92
	2					10	29	13	1.04	184:563	0.05	26.64
Frontana/Line E[3]	1					11	15	6	1.68	97:344	2.12	13.12
	2					14	26	9	1.20	139:345	3.57	14.76
Tobari 66/Line E[3]						12	22	14	0.45	133:464	2.35	17.97
Atlas 66/Line E[3]	1					18	27	15	0.90	158:502	0.40	28.90
	2					15	23	13	0.66	137:473	17.34	15.24
Penjamo 62R/Teal			;C1-:33+	32:67	2.29							
Penjamo 62R/Line E	1		;N,12C:3+	38:118	0.03							
	2		;C,1:33+	30:95	0.07							
Total				490:1325	3.86	253	441	223	3.29	2528:7662	0.20	363.57

[1]numbers 1 and 2 refer to separate families within each population.

[2]* significant p = 0.05
** significant p = 0.01

[3]populations provided by R.A. McIntosh and W.M. Hawthorn.

TABLE 6.2

F1, F2 and F3 results for intercrosses of wheats postulated to carry *Yr6* when seedlings were inoculated with *P. striiformis tritici* pathotype 104 E137 A-

Cross		F1	F2		F3		
		IT	ITs	R+I :S	HR	Seg	HS
Heines Peko/Oxley	1	;N1	;N,23=C	53:0	33	0	0
	2		;N,23=C	59:0			
Heines Kolben/Oxley	1	;1-CN	;N,12C,23-C	141:0	67	0	0
	2		;N,23-C	54:0	58	0	0
Millewa/Oxley		;1+CN	;;N	162:0			
Bindawarra/Oxley		;C	;CN1=	261:0			
WW80/Oxley			;;N	230:0			
Romany/Oxley			;N1=,11+	78:0			
Frontana/Oxley	1		;N1=,11+C	74:0	75	0	0
	2		;N	173:0			
Penjamo 62R/Oxley		;	;;N	58:0			
Rio Negro/Oxley			;C,;C1:3	107:6			
Tobari 66/Oxley	1		;N,12-C:33+	64:1	56	2	0
	2		;N1-,12:3+	151:5			
Atlas 66/Oxley	1		;N,;N1-:3+	145:2	51	0	0
	2		;N1=,11+C	74:0			
Pitic 62/Oxley	1	;;N	;N;1:3	66:2	20	0	0
	2		;;N	161:0	39	0	0

or 3. contaminant plants introduced at the time of machine threshing or at sowing.

Data for the progenies of the six F2 plants with IT3 in Rio Negro/Oxley are not available and independent F3 tests could not be conducted. Five of the six plants with high seedling reactions in Tobari 66/Oxley were scored susceptible as adult plants in the field, indicating that they could not be homozygous *Yr6Yr6*. In family 1 of this cross, 2 of 58 F3 lines segregated for a single recessive gene. Since both Tobari 66 and Oxley were each shown earlier to carry single recessive genes for resistance, it was concluded that the two segregating lines must have been the consequence of outcrossing or of contamination of the F1 with heterozygous seeds. The two susceptible F2 plants in Atlas 66/Oxley were susceptible as adults in the field; one of these was homozygous susceptible in F3, indicating that it must have been a contaminant; the other plant was not progeny tested. In Pitic 62/Oxley, both plants with IT3 were awnless in an otherwise awned population. Because the awned character is recessive, these plants must have resulted from outcrossing or contamination. Fifty nine F3 lines from this cross were scored homozygous resistant.

Conclusions The postulation of *Yr6* in a range of Australian and exotic wheats was based on several criteria, including IT, temperature sensitivity, pedigree, recessiveness of gene action and allelism. The occurrence of pathotypes virulent with respect to *Yr6* allowed confirmation of these conclusions.

Segregations in crosses of Oxley, Millewa, Bindawarra, Miling, Jacup, Takari, WW80, Shoshi, Romany, Frontana, Tobari 66, Atlas 66, Pitic 62 and Penjamo 62R conformed satisfactorily to those expected for a single recessive gene. This was in agreement with the recessive behaviour of *Yr6* in populations derived from the standard cv.s Heines Kolben and Heines Peko. The allelism for resistance found in crosses of Oxley with Heines Peko and Heines Kolben confirmed that the gene present in Oxley was *Yr6*. Resistance in 10 wheats was shown to be allelic with that in Oxley.

Occasional susceptible plants occurring in crosses involving five of these wheats were attributed to either outcrossing or contamination. The probable allelism in crosses of Oxley with Jacup, Miling and Takari could not be confirmed because the F1 hybrids were sterile grass-clump dwarfs. Resistance in Shoshi, which segregated for a single recessive gene when crossed with a susceptible wheat, was not confirmed in an allelism test with *Yr6*. Conversely, the Rio Negro resistance, which appeared to be allelic with *Yr6* in a cross with Oxley, was not examined to determine the mode of inheritance. However, Shoshi and Rio Negro responded identically with Oxley when infected with *Yr6*-virulent pathotypes (Section 5.4.1.2).

Linkage with *Lr13* Several cultivars shown to possess *Yr6* also possessed *Lr13* for resistance to *P. recondita tritici* (Hawthorn, 1984). The relationship between *Yr6* and *Lr13* was examined using *Lr13* data obtained from either W.M. Hawthorn or R.A. McIntosh. F2 progeny data for two families of Millewa/Line E were examined for joint segregation (Table 6.3). The χ^2 analyses indicated that the respective single genes were inherited independently. Segregation data for five additional crosses tested with the same pathotypes are presented in Table 6.4. Again, the χ^2 analyses demonstrated that *Yr6* and *Lr13* were inherited independently.

6.3.1.2 Relationships between seedling and adult-plant responses

Oxley

F2 seedlings were classified for IT, tagged and transplanted to the field in one of three response groups. The individual F2 adult-plant responses were noted and progeny tests of F3 lines were conducted in the glasshouse and the field. The relationships between seedling and adult plant responses were examined (Table 6.5).

The relationship between F2 seedling and F2 adult-plant responses (Table 6.5A) indicated that plants resistant

TABLE 6.3

Distribution of F2 genotypes derived from F3 seedling tests of Line E/Millewa when tested with *P. striiformis tritici* pathotype 104 E137 A- and *P. recondita tritici* pathotype 104-2,3,6,(7)

	Family 1				Family 2			
	Lr13Lr13	*Lr13lr13*	*lr13lr13*	Total	*Lr13Lr13*	*Lr13lr13*	*lr13lr13*	Total
Yr6Yr6	2	11	8	21	3	7	2	12
Yr6yr6	5	17	9	31	2	18	7	27
yr6yr6	2	7	1	10		5	4	9
Total	9	35	18	62	5	30	13	48

		d.f.		d.f.
$\chi^2_{1:2:1}$	(*Lr13* vs *lr13*)	3.65	2	5.67	2
$\chi^2_{1:2:1}$	(*Yr6* vs *yr6*)	4.03	2	1.13	2
χ^2 linkage		3.42	4	3.03	4
$\chi^2_{1:2:1:2:4:2:1:2:1}$		11.10	8	9.83	8

TABLE 6.4

Distribution of F2 genotypes in various crosses segregating for *Lr13/lr13* and *Yr6/yr6*

	Yr6Yr6			*Yr6yr6*			*yr6yr6*			$X^2_{1:2:1}$	$X^2_{1:2:1}$	X^2	$X^2_{1:2:1:2}$
	Lr13Lr13	*Lr13lr13*	*lr13lr13*	*Lr13Lr13*	*Lr13lr13*	*lr13lr13*	*Lr13Lr13*	*Lr13lr13*	*lr13lr13*	(*Yr6* vs *yr6*)	(*Lr13* vs *lr13*)	linkage	:4:2:1:2:1
Shoshi/Teal 1	3	4	3	5	8	9		2	4	1.79	6.00	1.63	9.58
2	3	4	3	7	15	7	4	8	1	1.04	0.42	2.00	3.46
Romany/Teal	3	2		4	8	2	2	2	1	0.73	3.00	1.60	5.33
Frontana/Line E 1	6	5	3	5	17	4	4	2	3	1.20	1.04	6.63	8.87
Tobari 66/Line E	3	7	2	7	8	7	1	10	3	0.45	0.13	4.92	5.50
Atlas 66/Line E 2	1	6	8	4	14	5	2	6	5	0.66	4.76	5.14	10.56

TABLE 6.5

Relationships between F2 plant and F3 line classifications based on tests performed in the glasshouse (seedling) and field (adult) in Line E/Oxley inoculated with pathotype 104 E137 A-.

A.

F2 Seedling		F2 Adult			F3 Seedling		
		R	I	S			
IT	No.	OR	20MR	50S	HR	Seg	HS
;N	6	6			6		
2=C	12	11	1		10		2
3+	50	21	10	19		29	19
Total		38	11	19	16	29	21

B.

F3 Adult	F3 Seedling			Total
	HR	Seg	HS	
HR	11	3		14
Seg	2	21	16	39
HS			2	2
Total	13	24	18	55

and intermediate as seedlings were also resistant as adult-plants in the field. However, of 50 plants classified susceptible in seedling tests, 21 displayed a high level of resistance as adult plants. This suggested that adult-plant resistance in addition to *Yr6* conferred resistance in Oxley. The F2 seedling responses were highly correlated (96.9%) with the F3 line classifications (Table 6.5A). Two plants were apparently misclassified as 2=C in F2, as their F3 progenies proved HS (IT 3+). Apart from this exception, there was close agreement between the F2 seedling phenotype and the F3 line behaviour.

The relationship between F3 seedling and F3 adult-plant responses also demonstrated the presence of additional adult-plant resistance (Table 6.5B). Sixteen lines scored HS in seedling tests segregated to high levels of resistance. However, no line was scored as HS in seedling tests and HR as adult plants. This may have been a chance effect due to the small population of HS lines examined in the field.

Other cultivars

Other crosses involving cultivars postulated to possess *Yr6* were also tested in both the seedling and adult-plant stages, and the relationships between the different tests are discussed under the following headings:

Association of F2 adult-plant and F3 seedling responses

The relationships between F2 adult-plant and F3 seedling responses for four crosses are presented in Table 6.6. Generally, the F2 plants classified as resistant gave HR seedling progeny; however, there were several discrepancies in Pitic 62/Line E. The plants classified intermediate as F2 adults gave variable results as F3 seedlings. In contrast, those classified intermediate in Bindawarra/Line E were predominantly HR. In WW80/Line E and Miling/Teal, the progenies of F2 plants with intermediate responses were predominantly segregating or HS, whereas in Pitic 62/Line E, they were distributed across all three seedling response groups.

Plants classified susceptible as F2 adults almost

TABLE 6.6

Relationship between F2 adult-plant and F3 seedling responses in four crosses inoculated with *P. striiformis tritici* pathotype 104 E137 A-

Cross (parent response)	F2 Adult response		F3 seedling HR	Seg	HS	Total
Pitic 62/Line E	20R-MR	R	18	9	3	30
(20R-MR,80MS-S)	40MR-MS	I	9	17	12	38
	70MS-S	S		21	9	30
	Total		27	47	24	98
Bindawarra/Line E	tVR	R	8			8
(tVR,80MS-S)	20MR	I	10	1	2	13
	70MS	S	1	17	12	30
	Total		19	18	14	51
WW80/Line E 1	10R	R	14	2		16
(5R,80MS-S)	80MS-S	S		20	11	31
	Total		14	22	11	47
2	15R	R	19	1		20
	40MR-MS	I		3	1	4
	70MS-S	S		16	10	26
	Total		19	20	11	50
Miling/Teal	5R	R	17	2	2	21
(OR,40MS-S)	40MR-MS	I	3	18	9	30
	60MS-S	S		13	12	25
	Total		20	33	23	76

invariably gave segregating or susceptible seedling progeny; one plant in Bindawarra/Line E was scored HR. These results provided little evidence for adult-plant resistance additional to that conferred by *Yr6* in WW80, Bindawarra and Miling. However, additional adult-plant resistance may have been present in Pitic 62.

Association of F3 adult-plant and F3 seedling responses

Comparisons of F3 adult-plant and F3 seedling responses for crosses involving five cultivars with *Yr6* are presented in Table 6.7. The distributions of F3 lines in Line E/ Millewa suggested segregation at single loci for both seedling and adult-plant tests. The close association between the two sets of results suggested that adult-plant resistance was conferred by *Yr6*. There was little evidence for additional adult-plant resistance in Millewa.

The frequencies of F3 lines tested at the adult-plant stage in Atlas 66/Line E suggested segregation for resistance at a single locus. However, the association between seedling and adult-plant responses was not close, mainly because 17 of 27 lines scored HS as seedlings segregated as adult-plants. However, if additional adult-plant resistance was present, it would have been expected that some HR lines would have been observed in the field among the 31 lines classified as segregating in seedling tests.

The results for crosses involving Frontana, Shoshi and Romany showed greater evidence for adult-plant resistance in addition to that conferred by *Yr6*. This was based on variation in adult-plant responses among lines classified HS in seedling tests.

Adult-plant responses of plants selected for seedling susceptibility Susceptible segregates from F2 populations of several crosses tested as seedlings were transplanted into the field for assessment of adult-plant response. The numbers of plants scored adult-plant resistant in crosses involving Pitic 62, Jacup and Miling were relatively small (Table 6.8) suggesting that no additional adult-plant resistance was present. In contrast, the results did indicate

TABLE 6.7

Relationship between F3 adult-plant and F3 seedling responses in hybrid populations inoculated with *P. striiformis tritici* pathotype 104 E137 A-

Cross (parent response)	F3 Adult	F3 Seedling HR	Seg	HS	Total
Line E/Millewa (60S,OR)	HR	22	2		24
	Seg		31	3	34
	HS			10	10
	Total	22	33	13	68
Atlas 66/Line E (tVR,70S)	HR	16			16
	Seg	1	29	17	47
	HS		2	10	12
	Total	17	31	27	75
Frontana/Line E (OR,80S)	HR	8	4	1	13
	Seg		16	2	18
	HS			1	1
	Total	8	20	4	41
Shoshi/Teal (5R,50MS-S)	HR	3	1	3	7
	Seg		3	11	14
	HS				1
	Total	3	4	14	22
Romany/Teal (5VR,40MS-S)	HR	1	1	1	3
	Seg	1	1	4	6
	HS				
	Total	2	2	5	9

TABLE 6.8

Responses of adult plants grown from selected susceptible seedlings in six wheat crosses tested with pathotype 104 E137 A-

Cross (parent response)	F2 Adult-plant Response	
	R+I (response)	S (response)
Pitic 62/Gabo (30R-MR,70MS-S)	1(20MR)	60(60MS-S)
Pitic 62/Line E (15R-MR,90S)	2(30MR)	41(50MS-S,90S)
Jacup/Line E (20MR,100S)	6(30MR-MS)	50(80MS-S)
Miling/Gabo (20R,80MS-S)	8(20R-MR)	25(80MS-S)
Takari/Teal (tR,50MS-S)	9(tR) + 24(20MR)	12(50MS-S)
Penjamo 62 R/Teal (30R-MR,60MS-S)	12(10R) + 31(40MR-MS)	15(60MS-S)

segregation for a degree of adult-plant resistance in Takari and possibly Penjamo 62R crosses. In both of these cases, the susceptible parent was Teal, which was not as susceptible as either Gabo or Line E. Thus, the possible genetic contributions of Teal may have added to the higher incidence of resistant plants in the Takari and Penjamo 62R cross populations.

Conclusions It was concluded that cv.s Oxley, Frontana, Romany, Shoshi, and Penjamo 62R carried gene(s) for adult-plant resistance additional to *Yr6*. Oxley showed 5% leaf area affected when inoculated with pathotype 108 E141 A+ in field experiments in 1985 (Section 5.4.3). The remaining above cultivars showed responses which were not significantly different from Oxley.

The cv.s Millewa, Jacup, Miling and Bindawarra appeared to have no detectable adult-plant resistance in addition to *Yr6* . In 1985 field tests with pathotype 108 E141 A+, these cultivars did not differ significantly from Millewa, which had 60% of leaf area affected by stripe rust.

The results for populations involving Takari suggested additional factor(s) for adult-plant resistance. In contrast, Atlas 66 and Pitic 62 crosses showed little evidence for adult-plant resistance. However, the responses of these three cultivars to 108 E141 A+ was intermediate between Oxley and Bindawarra (Section 5.4.3). Although crosses involving WW80 showed no evidence of additional adult-plant resistance in the present genetic studies, tests with pathotype 108 E141 A+ in the field showed that it was indistinguishable from Oxley (Section 5.4.3).

6.3.2 CULTIVARS WITH *Yr7*

6.3.2.1 Glasshouse studies

Results of inheritance studies of seedling resistance to stripe rust in cultivars displaying the *Yr7* specificity (Section 5.4.1.3) are summarised in Table 6.9. The F1 responses of the two hybrids that were tested were low, indicating the dominance of resistance. The tendency towards intermediate infection types characteristic of wheats

TABLE 6.9

F1, F2 and F3 seedling data for crosses of cultivars possessing *Yr7* following inoculation with *P. striiformis tritici* pathotype 104 E137 A-

Population	F1	F2			F3				Segregating F3 Lines		
	IT	ITs	R+I :S	$\chi^2_{3:1}$	HR	Seg	HS	$\chi^2_{1:2:1}$	Pooled R+I :S	$\chi^2_{3:1}$	$\chi^2_{homog.}$
Teal/Sinton[1]		1-CN,3C:3+	41:19	1.42	10	30	20	3.33	469:186	4.03*	27.06
Line E/Gatcher R					25	36	18	1.86	502:133	5.57*	14.67
Teal/Sunkota		1C,3-C:3+	36:10	0.26	12	27	23	4.93	380:131	0.11	16.26
Sunkota/Teal	23=C	;N,1C:3+	35:11	0.03							
Nuri 70/Avocet S		;C,1C:3+	98:37	0.42							
Chris/Teal[1]					9	18	4	2.42	395:134	0.03	18.14
South Africa 43/Teal[1] 1		;C12:3+	89:26	0.35	19	30	11	2.13	577:179	0.71	36.56
2			101:42	1.46							
Manitou/Line E[1]		;C,12-C:3+	72:34	2.83	10	17	3	3.80	338:110	0.05	14.83
Line E/Norquay[1]					13	22	21	5.21	318:118	0.99	16.41
Total			472:179	2.16	98	180	100	0.88	2979:991	0.01	151.51

[1]F2 populations provided by W.M. Hawthorn

carrying *Yr7* (Section 5.4.1.3) did not cause difficulties in recognising the resistant phenotype in F2 and F3 hybrid populations involving very susceptible wheats such as Avocet S and Line E.

Segregation ratios in F2 and F3 were generally in agreement with the hypothesis that resistance was determined by a single dominant gene. The χ^2 value for the pooled ratio of resistant and susceptible plants from segregating F3 lines in Teal/Sinton and Line E/Gatcher R were significant ($p<0.05$). Despite these discrepancies, the overall results were interpreted to indicate that resistance in each of these nine wheats was attributable to a single dominant gene.

Tests of allelism Cultivar Thatcher was adopted as a standard source for *Yr7* (Macer, 1966; McIntosh *et al.*, 1981). F2 and F3 populations of Thatcher/Sunkota failed to segregate when tested with pathotype 104 E137 A- (Table 6.10). This confirmed that Sunkota carried *Yr7*. F2 populations derived from crosses of Sunkota with Sinton and Norquay failed to show segregation (Table 6.10) indicating a common gene for resistance.

Conclusions Dominant, single gene inheritance in hybrid populations involving this group of cultivars was consistent with the reported behaviour of *Yr7* (Macer, 1966). This was supported by tests of allelism involving some cultivars, and the demonstration of the *Yr7* specificity in each instance (Section 5.4.1.3).

6.3.2.2 Relationship between seedling and adult-plant responses

The relationship between F2 adult-plant and F3 seedling responses in two populations are presented in Table 6.11. Twelve of 49 resistant F2 plants in Teal/Sunkota and 15 of 54 resistant F2 plants in Teal/Sinton were scored HS in F3 seedling tests. This clearly indicated the presence of adult-plant resistance, in addition to *Yr7*, in both cultivars.

A comparison of F3 seedling and F3 adult-plant

TABLE 6.10

F1, F2 and F3 seedling results for crosses involving Thatcher and Sunkota inoculated with pathotype 104 E137 A-

Cross		F1	F2		F3		
		IT	ITs	R+I :S	HR	Seg	HS
Thatcher/Sunkota		1-C	;;C1=	124:0	28	0	0
Sinton/Sunkota		;C	;C1-	100:0			
Norquay/Sunkota	1	;C	;;C	88:0			
	2		;;C	113:0			

TABLE 6.11

Relationship between F2 adult and F3 seedling responses in various crosses inoculated with *P. striiformis tritici* pathotype 104 E137 A-

Cross (Parent response)	F2 Adult Response		F3 seedling HR	Seg	HS	Total
Teal/Sunkota (80MS-S,tVR)	OR	R	12	25	12	49
	20MR	I		2	7	9
	50MS-S	S			4	4
	Total		12	27	23	62
Teal/Sinton (60S,tR)	tR	R	10	29	15	54
	40MS-S	S		1	5	6
	Total		10	30	20	60

data for South Africa 43/Teal (Table 6.12) again indicated the presence of adult-plant resistance in addition to *Yr7*. F3 lines of genotype *yr7 yr7* in Teal/Sunkota and Line E/ Norquay also showed evidence of adult-plant resistance.

In contrast to the above crosses, all 19 F3 lines in Line E/Gatcher R that were HS in seedling tests were scored HS as adult-plants. On the other hand, four HR plants of genotype *Yr7 Yr7*, included as controls, were clearly HR as adult-plants. This suggested that Gatcher R did not possess additional genes for adult-plant resistance.

Conclusions Adult-plant resistance, in addition to the resistance conferred by *Yr7*, was evident in hybrid populations derived from Sunkota, Sinton, South Africa 43 and Norquay. The selection Gatcher R lacked additional adult-plant resistance. This finding was not unexpected, because the related selection, Gatcher S, which lacks *Yr7* , responded with 40 to 50% leaf area affected in field tests with pathotypes avirulent with respect to *Yr7* (Section 5.4.3).

6.3.3 CULTIVARS WITH *YrA*

6.3.3.1 Glasshouse studies

A range of primary leaf seedling responses was obtained for F2 populations and segregating F3 lines, derived from crosses of wheats possessing the *YrA* resistance and cultivars lacking seedling resistance. However, ITs on second seedling leaves were distinctly lower than those on primary leaves on plants with intermediate responses. In some instances, the second leaf infection type was critical when deciding to place individual seedlings in an intermediate or a susceptible response group. ITs on resistant plants were also distinctly lower on the second leaf, as previously noted for the parental stocks (Section 5.4.1.1). Susceptible plants produced high responses on both the primary and secondary leaves.

Results for F2 populations derived from crosses involving standard susceptible cultivars are presented in Table 6.13. Plants with intermediate infection types were pooled with resistant plants for testing the goodness-of-

TABLE 6.12

Relationship between F3 seedling and F3 adult responses in hybrid populations inoculated with *P. striiformis tritici* pathotype 104 E137 A-

Cross	F3 Adult	F3 Seedling			Total
		HR	Seg	HS	
South Africa 43/Teal	HR	3	1	3	7
	Seg		1	6	7
	HS			2	2
	Total	3	2	11	16
Teal/Sunkota	HR			11	11
	Seg			5	5
	HS			6	6
	Total			22	22
Line E/Norquay	HR				
	Seg		2	12	14
	HS			7	7
	Total		2	19	21
Line E/Gatcher R	HR	4			4
	Seg				
	HS			19	19
	Total	4		19	23

TABLE 6.13

Frequencies of F2 seedlings in response groupings from crosses of cultivars possessing the *YrA* resistance following inoculation with *P. striiformis tritici* pathotype 104 E137 A-

Cross		ITs	R+I :S	$\chi^2_{3:1}$	$\chi^2_{9:7}$	Experiment[1]
Avocet R/Teal	1	;N,1CN/1C:3+4	47,52:76	31.70**	0.01	1
	2	;C1-,23/12C:3+	11,77:38	1.79	9.46**	2
Egret R/Teal		;;CN,23-/12C:3+4	62,100:68	2.56	18.80**	1
Condor R/Teal	1	;N,12CN/1C:3+4	43,67:84	34.60**	0.02	1
	2	;C1,3C/12C:3+	24,77:27	1.04	26.70**	2
Banks R/Teal	1	;N1=,23C/12C:3+4	50,50:86	44.74**	0.47	1
	2	;C1-,3C/12C:3+	25,84:35	0.04	22.12**	2
WW15/Teal	1	;N1=,23C/2C:3+4	45,49:80	40.83**	0.35	1
	2	;C1,33+C/;12:3+	17,57:42	7.77**	2.68	2
Nainari 60/Gabo	1	;;N,23/1C:3+	30,73:55	1.24	27.90**	1
	2	;C,12C/;1-C:3+	16,121:27	6.37*	49.61**	2
Nainari 60/Line E	1	;;N,23/1C:3+	16,59:59	25.88**	0.00	1
	2	;C,12/;C1:3+	38,77:39	0.01	21.24**	2
Inia 66/Gabo		;C1,3/;C1:3+	25,85:39	0.11	18.70**	2
Inia 66/Line E		;C1-,3/;1C:3+	28,94:40	0.01	23.91**	2
Tanori 71/Line E	1	;N,23/12C:3+4	20,77:68	23.13**	0.43	1
	2	;C,23/;C1:3+	33,76:25	2.88	34.29**	2

[1]Experiment 1 conducted in June, 1984

Experiment 2 conducted in June, 1985

fit of the observed data with postulated segregation ratios. Two models for the inheritance of resistance were proposed, *viz.*, a single dominant gene with segregation of 3 resistant: 1 susceptible, and two complementary dominant genes segregating 9 resistant: 7 susceptible. Neither model satisfactorily explained all the data, although individual populations conformed with one or other of the models.

These F2 populations were first tested in June, 1984. When the resistant and intermediate classes were pooled and contrasted with the susceptible class, the observed ratios for the Egret R and Nainari 60 populations conformed with segregation for a single dominant gene, whereas the remaining six populations conformed with the two complementary gene model. When nine populations were tested in 1985, seven conformed with 3:1 and two with 9:7. Populations in five of the seven crosses tested on both occasions showed changes in model conformity for 9:7 in 1984, to 3:1 in 1985. The segregations in WW15/Teal conformed with 9:7 and not 3:1 in both experiments. One population of Nainari 60/Gabo showed significant deviations when tested for goodness-of-fit to both models, however, deviations from the 9:7 model were much more extreme than for the 3:1 model.

Notes made during the course of the 1985 tests indicated that infection on the secondary leaves was uneven and plants were classified as susceptible only when both primary and secondary leaves were distinctly high in response. Thus some plants, which may have been susceptible, but were not infected on the second leaf, were classified as intermediate.

In order to clarify these discrepancies, all populations appearing in Table 6.13 were again tested in a single experiment in December, 1985, with particular attention being given to the establishment of uniform infection on the secondary leaves. The results are presented in Table 6.14. Segregation ratios for 13 of the 15 populations conformed with 9 resistant : 7 susceptible. Both populations, where the data differed significantly ($p<0.05$) from the expected, had excessive numbers of susceptible segregates,

TABLE 6.14

Frequencies of F2 seedlings in response groups derived from crosses of cultivars possessing the *Yr*A resistance following inoculation with *P. striiformis tritici* pathotype 104 E137 A-

Cross		ITs	R+I :S	$\chi^2_{9:7}$
Avocet/Teal	1	;N1=,2+3-/;C1:3+	13,59:67	1.12
	2		16,76:68	0.10
Egret R/Teal	1	;N1=,2++/;2C:3+4	25,111:76	5.38*
Condor R/Teal	1	;N1-,2++/1C:3+	12,80:91	2.66
	2		9,98:89	0.22
Banks R/Teal	1	;N1=,2++C/12C:3+4	39,86:74	3.48
	2		21,89:88	0.04
WW15/Teal	1	;CN1=,2+/12C:3+4	4,83:56	1.22
	2		12,85:72	0.09
Nainari 60/Gabo	1	;CN1,23/;1C:3+	10,58:47	0.39
	2		2,81:56	0.68
Inia 66/Gabo		;C1=,2++/1C:3+	12,70:58	0.31
Inia 66/Line E		12+/1C:3+	98:62	1.63
Tanori 71/Line E	1	;CN1-,2++/12C:3+	1,50:41	0.02
	2	23C/1C:3+	40:51	5.58*
Total			176,1164:996	1.18

		d.f.
$\Sigma\chi^2$	22.92	14
χ^2(1340:996)	1.18	1
$\chi^2_{homogeneity}$	21.74	13

hence there was no confusion with the possible single gene segregation. The overall pooled data were homogeneous and showed segregation in accordance with the complementary gene model.

Studies of these crosses in F3 indicated that homozygous resistant lines were only infrequently recovered (Table 6.15). Repeat tests were performed on 497 of the 538 F3 lines in Table 6.15, using pathotype 108 E141 A- as well as 104 E137 A-. The results of tests on individual F3 lines showed agreement in greater than 90% of instances. For statistical analysis, data were taken from the results of a single experiment performed on all available F3 lines in each cross. Classification of F3 lines indicated the

TABLE 6.15

Frequencies of F3 lines in seedling response classes from crosses of cultivars possessing the *YrA* resistance following inoculation with 104 E137 A-

Cross[1]		F3 HR	F3 Seg	F3 HS	$\chi^2_{1:8:7}$
Avocet R/Teal	1	6	20	18	4.10
Egret/Teal	1	2	19	22	1.03
Condor R/Teal	1	2	16	14	0.00
Banks R/Teal	1	2	16	6	3.43
WW15/Teal	1	0	17	17	2.43
	2	4	15	25	4.51
WW15/Federation	1	2	17	6	5.69
	2	3	16	5	5.71
Tanori 71/Line E	1	2	13	16	0.84
	2	1	21	26	2.90
Nainari 60/Gabo	1	3	15	18	1.07
Nainari 60/Teal	1	2	13	17	2.03
	2	7	19	18	7.07*
Inia 66/Teal	1	5	17	14	3.61
	2	2	15	24	3.65
Total		43	249	246	4.42

[1]some of these are the same populations derived from F2 plants described in Table 6.14.

segregation of complementary genes, *i.e.* 1HR:8Seg:7HS. The results for one population of Nainari 60/Teal showed a significant deviation ($p<0.05$) from the postulated ratio due to an excess of HR lines. Nevertheless, the overall results were consistent with those expected on the basis of complementary dominant genes.

Assuming complementary genes for resistance, individual segregating F3 lines should segregate in either 3:1 (F2 genotype *AaBB* or *AABb*) or 9:7 (F2 genotype *AaBb*) ratios in equal proportions. Whereas the minimum population size to distinguish the alternate possibilities at $p = 0.05$ was 67 seedlings (Hanson, 1959), populations

of this size were not available. Lines were therefore placed in one or other segregating class on the basis of the lower χ^2 value when tested for goodness-of-fit to both ratios. On this basis, results pooled for all segregating F3 lines showed that 130 segregated 3:1 and 119 segregated 9:7 ($\chi^2_{1:1}(130:119) = 0.49$; $p>0.5$). In addition, the pooled results for lines classified 3:1 or 9:7 showed good agreements with the expected ratios (Table 6.16).

F2 segregations for crosses between resistant and susceptible selections of cultivars that were found to be phenotypically heterogeneous in respect of the *YrA* resistance, are summarised in Table 6.17. Again, second leaf ITs were important in classification of the intermediate response. Segregation ratios for 22 of 25 populations were in accordance with that expected for variation at a single locus. The results for three populations deviated ($p<0.05$) from those expected. When pooled, the overall results showed good agreement with the postulated 3:1 ratio indicating segregation for a single dominant gene. The results for all 25 populations showed very significant ($p<0.01$) deviations from those expected for segregation of complementary dominant factors.

The results for 223 F3 lines from four crosses, involving resistant and susceptible selections of certain Australian cultivars, are given in Table 6.18. The line distributions for crosses, and overall pooled data, conformed with the ratio 1 HR : 2 Seg : 1 HS. Within segregating lines, a total of 1,626 plants were resistant and 512 were susceptible, conforming with the postulated segregation of a single dominant gene. The ratio of the pooled frequencies of resistant and susceptible plants in Avocet S/Banks R deviated from the postulated ratio ($p<0.05$).

Tests of allelism Intercrosses between cultivars selected for the presence of the *YrA* resistance failed to produce a susceptible segregate in 12 F2 populations from 11 crosses involving a total of 1461 seedlings (Table 6.19). In some crosses, a proportion of seedlings displayed intermediate responses. Eleven of 360 F3 lines from seven crosses were scored as segregating. Since the parents of such crosses were known

TABLE 6.16

Pooled frequencies of seedling plants from segregating F3 lines in crosses of parents possessing the *YrA* resistance

Cross		F3 lines segregating 3:1				F3 lines segregating 9:7			
		No. Lines	Pooled R+I :S	$\chi^2_{3:1}$	$\chi^2_{homog.}$	No. Lines	Pooled R+I :S	$\chi^2_{9:7}$	$\chi^2_{homog.}$
Avocet R/Teal	1	12	193:67	1.65	1.53	8	113:79	0.53	2.13
Egret R/Teal		12	237:78	0.01	8.59	7	132:84	2.07	2.17
Condor R/Teal	1	10	149:56	0.59	1.22	6	78:52	0.74	0.92
Banks R/Teal	1	9	127:45	0.12	2.47	7	56:53	1.05	7.34
WW15/Teal	1	10	174:67	1.01	3.44	7	99:77	0.00	3.43
	2	6	109:41	0.44	1.50	9	135:105	0.00	6.17
WW15/Federation	1	8	113:33	0.45	3.81	9	108:95	0.77	4.96
	2	11	122:47	0.71	1.38	5	64:46	0.17	4.62
Tanori 71/Line E	1	6	111:52	4.14*	0.74	7	91:76	0.21	1.61
	2	9	150:55	0.37	5.72	12	145:121	0.33	6.46
Nainari 60/Gabo	1	8	117:46	0.90	1.41	7	85:75	0.63	4.58
Nainari 60/Teal	1	3	51:16	0.04	0.38	10	112:120	5.99*	9.01
	2	14	256:75	0.96	6.23	5	61:50	0.06	0.99
Inia 66/Teal	1	6	101:38	0.41	3.11	11	146:118	0.10	1.61
	2	6	83:32	0.49	0.56	9	99:90	1.15	4.43
Total		130	2093:748	2.68	51.70	119	1524:1241	1.44	72.79

TABLE 6.17

Frequencies of F2 seedlings in response groups, derived from crosses between cultivars selected for the presence or absence of the *YrA* resistance, following inoculation with *P. striiformis tritici* pathotype 104 E137 A-

Cross		ITs	R+I :S	$\chi^2_{3:1}$
Avocet S/Avocet R	1	1CN,23C/2C:3+	18,81:35	0.09
	2	;N,12C/1-C:3+4	150,29:58	0.04
Egret S/Avocet R		;N,23C/1C:3+	30,99:25	6.31*
Condor S/Avocet R	1	;N1-,23/1-C:33+	35,67:45	2.47
Avocet S/Egret R	1	;,12C/1C:33+	70,108:48	1.71
	2	23C:33+	79:15	4.09*
Banks S/Egret R		;,23=/1-C:33+	26,84:37	0.00
Avocet S/Condor R		;;N,23C/1C:3+	20,101:33	1.05
Egret S/Condor R		;N,23/1C:33+	20,110:31	2.83
Condor S/Condor R	1	;C1,3/12C:3+	19,84:35	0.01
	2	;;N,12-C/1-C:3+4	129,10:49	0.11
Banks S/Condor R		;N,23/12C:3+	16,88:40	0.59
Avocet S/Banks R	1	;;N,23/1C:3+	34,83:43	1.08
	2	;N1-,23C/;C1:33+	37,50:19	2.83
Egret S/Banks R		:N,23/;1C:33+	31,73:41	0.83
Condor S/Banks R		;;N,23/1-C:3+	4,51:17	0.07
Banks R/Banks S		;CN,12C:3+4	150,19:53	0.15
Avocet S/WW15		1CN,23C:3+	5,90:46	4.37*
Egret S/WW15		;;N,23C/1C:3+	13,103:31	1.20
Condor S/WW15		;;N,23-C/1-C:3+	28,123:57	0.64
Banks S/WW15		;;N,23-C/1-C:3+	37,83:45	0.45
Vulcan/Avocet S		;C,3C/12C:3+4	32,30:16	0.84
Cocamba/Avocet S		;C1-,3-C/1C:3+4	66,14:30	0.30
Sonalika/Avocet S	1	;C,12C/;C:3+	37,12:15	0.08
	2	;C,12C/;C:3+	23,9:7	1.03
Total			1030,1680:871	33.17

		d.f.
$\Sigma\chi^2$	33.17	24
χ^2 (2710:871)	0.88	1
$\chi^2_{\text{homogeneity}}$	32.29	23

TABLE 6.18

Seedling response data for F3 lines derived from crosses of cultivars selected for the presence or absence of the *YrA* resistance, following inoculation with *P. striiformis tritici* pathotype 104 E137 A-

Cross	HR	Seg	HS	$\chi^2_{1:2:1}$	Segregating F3 lines Pooled R+I :S	$\chi^2_{3:1}$	χ^2_{homog}
Condor S/Condor R	14	30	10	1.26	484:148	0.84	18.55
Condor S/Banks R	13	16	12	2.02	248:66	2.65	4.93
Avocet S/Banks R	10	24	17	2.09	354:144	4.07*	13.88
Egret R/Egret S	25	37	15	2.71	540:154	2.92	18.48
Total	62	107	54	0.94	1626:512	1.26	65.06

TABLE 6.19

F2 and F3 results for intercrosses of cultivars with and without the *YrA* resistance following inoculation with pathotype 104 E137 A-

Cross	F2 ITs	F2 R+I :S	F3 HR	F3 Seg	F3 HS
WW15/Egret R	;;C,1-C/;C	150:0	44	0	0
Banks R/Egret R	;;C	109:0	32	3	0
Condor R/Egret R	;;C,;1-C/;C	99,10:0	49	2	0
Avocet R/Egret R	;;C	136:0			
Avocet R/Condor R	;;C	142:0	56	0	0
Banks R/Condor R	;;C	122:0	63	5	0
WW15/Condor R	;;C	129:0	47	0	0
Avocet R/WW15	;,;CN	4,109:0	58	1	0
WW15/Avocet R	;;C,12/12C	95,45:0			
Vulcan/Avocet R	;C,3/23=C	82,24:0			
Sonalika/Avocet R	;C,3/;12	97:0			
		107,4:0			

to possess a single dominant gene or complementary dominant genes for resistance, the observed numbers of segregating F3 lines were too small to support postulations of segregation for genetically independent dominant genes. Moreover, the records showed that all 11 plants, which subsequently segregated, were tall and awnless. This clearly showed that outcrossing or contamination had been involved. Thus, the Australian cultivar selections Avocet R, Egret R, Condor R, Banks R and Vulcan, and the Mexican breeding line, WW15, share a common gene for resistance. This gene was also present in Sonalika.

Linkage relationships F2 populations of Tanori 71/Line E segregated for progressive necrosis, the symptoms of which became apparent between tillering (growth stage 30) and flowering (growth stage 60). Based on the work of Hawthorn (1984) indicating complete linkage between the *Lr13* and *Ne2m* genes, it was concluded that Tanori 71 probably possessed *Lr13* in common with related Mexican wheats. The linkage between the *YrA* gene(s) and *Lr13* was investigated using two F3 populations of Tanori 71/Line E. The pooled data in Table 6.20 showed a single gene inheritance ratio for the assumed *Lr13*/*lr13* locus that was independent of the segregation for stripe rust response determined by complementary dominant genes.

Conclusions It was concluded that the *YrA* resistance was determined by the interaction of two dominant complementary genes which segregated independently of the *Lr13*/*lr13* locus. Tests of allelism indicated that the *YrA* resistance was present in certain Australian wheats, their CIMMYT-derived source, WW15, and Sonalika. Additional inheritance and specificity studies have demonstrated the presence of this resistance in further Mexican wheats, including Nainari 60, Inia 66 and Tanori 71.

Since hybrid populations, derived from crosses of lines selected for the presence or absence of the *YrA* phenotype, segregated in F2 and F3 for a single dominant gene, it was concluded that one of the complementary loci was homozygous in all of the parents selected for the absence

	No. F3 lines
Egret S/Avocet S	53
Egret S/Condor S	60
Egret S/Banks S	55
Avocet S/Banks S	42
Condor S/Banks S	38
	248

TABLE 6.20

Distribution of F2 genotypes in Tanori 71/Line E determined from seedling tests of F3 lines with *P. striiformis tritici* pathotype 104 E137 A- and *P. recondita tritici* pathotype 104-2,3,6,7

		Lr13Lr13	*Lr13lr13*	*lr13lr13*	Total
YrA	HR	1	1	0	2
	Seg	10	18	9	37
	HS	7	17	17	41
	Total	18	36	26	80

		d.f.
$\chi^2_{1:2:1}$ (*Lr13* vs *lr13*)	2.40	2
$\chi^2_{1:8:7}$ (*YrA*)	3.05	2
χ^2 linkage	5.19	4
$\chi^2_{1:2:1:8:16:8:7:14:7}$	10.64	8

of the *YrA* resistance. If resistance were determined by complementary genes, it would be reasonable to assume that fixation of one or other of the complementary factors in genetically heterogeneous cultivars would have been at random. Hence it would have been reasonable to expect segregation for resistance in certain crosses between such susceptible selections. However, all 248 F3 lines from five crosses between various selections lacking the *YrA* phenotype failed to segregate for resistance. Therefore, some doubt must be expressed in regard to the complementary gene model. Alternatively, genetic linkage with some other character may be involved.

An alternative procedure to confirm the presence of complementary genes will be to follow the example of

Baker (1966) with crown rust resistance in Bond oats, and of Singh and McIntosh (1984a,b) with leaf rust resistance in Gatcher wheat. From crosses segregating for the postulated complementary genes, the respective workers isolated susceptible F4 plants which, when intercrossed, were shown to possess the alternative single dominant resistance gene.

The postulation of complementary genes is consistent with other experiences at Plant Breeding Institute, Castle Hill,. In attempting to chromosomally locate gene(s) determining the *YrA* resistance, McIntosh (pers. comm.) found a much reduced rate of return to homozygosity for resistance in monosomic series being generated in Egret R and CSP44. This delay was consistent with the present finding that the basis for resistance was more complex than a single dominant gene. McIntosh found evidence of a similar lack of homozygotes in an Inia 66 monosomic series which had undergone five or six backcrosses to Inia 66 before being imported from R. Pienaar, University of Stellenbosch, South Africa.

6.3.3.2 Relationship between seedling and adult-plant responses

The possibility of adult-plant resistance in addition to resistance detected in seedling tests was investigated by transplanting susceptible F2 seedlings from various crosses into the field. Results, presented in Table 6.21, indicated that adult-plant resistance was present in crosses involving Avocet R, Egret R, Condor R, Banks R, WW15, Nainari 60 and Inia 66.

These results agree with data indicating that selections of Australian cultivars possessing *YrA* showed some resistance when tested in the field with *YrA*-virulent pathotypes (Section 5.4.3.1, Table 5.15). However, experience in commercial fields indicated that Avocet crops are very susceptible to A+ pathotypes and demonstrate no obvious signs of resistance. It is possible that variation for virulence with respect to adult-plant resistances occurs, and that pathotypes used in field experiments were not virulent

TABLE 6.21

Responses of adult plants grown from selected susceptible seedlings in nine wheat crosses tested with pathotype 104 E137 A-

Cross (parent response)	F2 Adult-plant Response R+I (response)	S (response)
Avocet R/Teal (30R,50MS-S)	28(20R)	8(30MS)
Egret R/Teal (20R,50MS-S)	23(20R-MR)	14(50MS-S,80S)
Condor R/Teal (15R,50MS)	10(5R) + 11(30MR-MS)	6(80MS-S)
Banks R/Teal (10R,50MS-S)	6(10R) + 20(30MR)	7(50MS-S)
WW15/Teal (10R,50MS)	9(10R) + 19(30MR-MS)	14(80MS-S,S)
Nainari 60/Teal (10R,40MS-S)	24(10R-MR)	7(30MS)
Inia 66/Teal (20R,30MR-MS)	9(20R-MR)	12(30MS), 27(80S)
Inia 66/L32 (30MR,90S)	4(30MR)	35(80S)
Inia 66/Gabo (20MR,80MS)	12(20MR)	23(80S)

for the apparent adult-plant resistance in Avocet.

6.3.4 CULTIVARS WITH UNKNOWN GENES OR GENE COMBINATIONS

6.3.4.1 Glasshouse studies

Pavon 76 and Hermosillo 77 Triplicate sowings of F3 lines were simultaneously inoculated with different pathotypes. Each F3 line was classified HR, Seg or HS with respect to each pathotype. The responses of an individual F3 line to all pathotypes allowed postulations of the individual F2 genotype.

The results, presented in Table 6.22, for Pavon 76/Line E illustrate the method of analysis. Previous data (Section 5.4.1.4) indicated that Pavon 76 possessed *Yr7* and possibly *Yr6*, and thus appropriate pathotypes were chosen to detect these genes in the segregating populations. With pathotype 104 E137 A+, which was avirulent for both *Yr6* and *Yr7*, the lines were classified 23 HR : 26 Seg : 5 HS showing satisfactory agreement to the expected distribution, assuming segregation at two genetically independent loci.[1] Tests with pathotypes 106 E139 A-, which was virulent for *Yr7*, and 108 E141 A+, virulent for *Yr6*, indicated segregation for a single recessive gene and a single dominant gene respectively. χ^2 analyses showed satisfactory agreements with the respective genetic models (Table 6.22). Lines that were HR with either 108 E141 A+ or 106 E139 A- were HR with 104 E137 A+, indicating that *Yr7* or *Yr6*, respectively, conferred resistance to 104 E137 A+. Similarly, individual and pooled results for F3 lines segregating with 104 E137 A+ were tested for goodness-of-fit to 13:3, 3:1 or 1:3 where the specificity data suggested F2 genotypes of *Yr7yr7 Yr6yr6*, *Yr7yr7 yr6yr6 or yr7yr7 Yr6yr6*. There was satisfactory agreement in each case.

The same methods of analysis were applied to F3 data for Hermosillo 77. The Hermosillo 77 crosses also segregated for one dominant and one recessive gene. These genes appeared to be *Yr7* and *Yr6*, respectively. A summary of results for both Pavon 76 and Hermosillo 77 is presented in Table 6.23. The pooled F2 genotypes for both crosses conformed to the expected 7(*Yr7Yr7 Yr6Yr6*) : 4(*Yr7yr7 Yr6yr6*):

[1] $\chi^2_{7:8:1}$ (23:26:5)= 0.84 p = 0.6

TABLE 6.22

Seedling responses for individual F3 lines in Pavon 76/Line E inoculated with pathotypes of *P. striiformis tritici*, *P. graminis tritici* and *P. recondita tritici*

Plant Number	*P. striiformis tritici*									*P. graminis tritici*			*P. recondita tritici*	
	104 E137 A+ ITs R+I :S	$\chi^2_{13:3}$	$\chi^2_{3:1}$	$\chi^2_{1:3}$	108 E141 A+ ITs R+I :S	$\chi^2_{3:1}$	106 E139 A- ITs R+I :S	$\chi^2_{1:3}$	F2 Stripe Rust Genotype	343- 1,2,3,5,6 ITs	98- 1,2,3,5,6 ITs	F2 Stem Rust Genotype	104-2,3, 6,7 ITs	F2 Leaf Rust Genotype
Pavon 76	;				;;N		;N1-			;2=	2-		XX+	
Line E	4				4		4			3+4	3+		3+	
	;N,1C:3+4				;N1:3+4		;N,1C:3+4							
1	19:0				17:0		2:18	2.40	*Yr7Yr7,Yr6yr6*	;12=	22+,3,3+	*Sr9gSr9g*	XX+	*Lr13Lr13*
2	20:0				0:13		23:0		*yr7yr7,Yr6Yr6*	3+	33+	*sr9gsr9g*		
4	20:0				18:0		23:0		*Yr7Yr7,Yr6Yr6*	;12	2+	*Sr9gSr9g*		
5	16:0				16:2	1.85	24:0		*Yr7yr7,Yr6Yr6*	;12=,2+	22+	*Sr9gsr9g*	X+,3+	*Lr13lr13*
6	20:0				18:6	0.00	18:0		*Yr7yr7,Yr6Yr6*	2=,2+	2+	*Sr9gsr9g*	3+	*lr13lr13*
8	17:0				16:0		21:0		*Yr7Yr7,Yr6Yr6*	2=2-	3C	*Sr9gSr9g*	XX+	*Lr13Lr13*
9	20:0				25:0		18:0		*Yr7Yr7,Yr7Yr7*	2=2-	3C	*Sr9gSr9g*	X,3+	*Lr13lr13*
10	13:0				10:0		3:6	0.33	*Yr7Yr7,Yr6yr6*					
11	16:0				28:8	0.15	24:0		*Yr7yr7,Yr6Yr6*					
12	18:0				11:0		25:0		*Yr7Yr7,Yr6Yr6*					
13	16:0				16:4	0.27	19:0		*Yr7yr7,Yr6Yr6*	2-,3+	3C	*Sr9gsr9g*	X,3+	*Lr13lr13*
14	12:5	1.27			6:3	0.33	5:3	6.00*	*Yr7yr7,Yr6yr6*					
15	18:0				0:12		16:0		*yr7yr7,Yr6Yr6*					
16	15:0				0:16		19:0		*yr7yr7,Yr6Yr6*	33+	3C3+	*sr9gsr9g*	3+	*lr13lr13*
17	17:0				10:5	0.56	17:0		*Yr7yr7,Yr6Yr6*	2=,2+	2+3-	*Sr9gsr9g*	3+	*lr13lr13*
18	18:0				11:1	1.78	14:0		*Yr7yr7,Yr6Yr6*	;2=,3+	33+	*Sr9gsr9g*	X+,3	*Lr13lr13*
19	12:6	2,51			12:1	2.08	4:11	0.02	*Yr7yr7,Yr6yr6*					
20	18:4		0.55		10:5	0.56	0:18		*Yr7yr7,yr6yr6*	;2=,2,3	2+3-	*Sr9gsr9g*	X,3+	*Lr13lr13*
21	19:0				18:0		0:18		*Yr7Yr7,yr6yr6*	2-	3C	*Sr9gSr9g*	X-X	*Lr13lr13*
22	17:4	0.00			17:8	0.65	8:24	0.00	*Yr7yr7,Yr6yr6*	;2=,2,3	22+,33+	*Sr9gsr9g*	X,3+	*Lr13lr13*
23	14:4		0.07		11:7	1.85	0:18		*Yr7yr7,yr6yr6*	2=,2,3	3	*Sr9gsr9g*	X-,3+	*Lr13lr13*
25					9:5	0.86	3:11	0.10	*Yr7yr7,Yr6yr6*	2-,3+	3	*Sr9gsr9g*	XX+,3+	*Lr13lr13*
26	13:4	0.25			13:5	0.07	0:14		*Yr7yr7,yr6yr6*				3+	*lr13lr13*
28	12:6		0.67		7:5	1.78	0:16		*Yr7yr7,yr6yr6*	2-,3-C	22+	*Sr9gsr9g*	XX+	*Lr13Lr13*
29	16:0				19:0		9:18	1.00	*Yr7Yr7,Yr6yr6*	;2-,2-	3-	*Sr9gSr9g*	X+	*Lr13Lr13*

TABLE 6.22 Cont.

Plant Number	*P. striiformis tritici*								*P. graminis tritici*			*P. recondita tritici*	
	104 E137 A+ ITs R+I :S	$\chi^2_{13:3}$	$\chi^2_{3:1}$	$\chi^2_{1:3}$	108 E141 A+ ITs R+I :S	$\chi^2_{3:1}$	106 E139 A- ITs R+I :S	$\chi^2_{1:3}$	F2 Stripe Rust Genotype	343- 1,2,3,5,6 ITs	98- 1,2,3,5,6 ITs	F2 Stem Rust Genotype	104-2,3, 6,7 ITs	F2 Leaf Rust Genotype
30	13:4		0.19		9:6	1.80	0:18		*Yr7yr7,yr6yr6*	2-,3+	3C	*Sr9gsr9g*	X+,3	*Lr13lr13*
31	16:0				16:0		0:20		*Yr7Yr7,yr6yr6*	2-	3C	*Sr9gSr9g*	XX-	*Lr13Lr13*
32	15:0				14:0		3:9		*Yr7Yr7,Yr6yr6*	2-	22+,3	*Sr9gSr9g*	X,3+	*Lr13lr13*
33	13:1	1.24			12:2	0.86	5:6	2.45	*Yr7yr7,Yr6yr6*	2-,3	23	*Sr9gsr9g*	X,3+	*Lr13lr13*
34	22:0				5:0		0:10		*Yr7Yr7,yr6yr6*					
35	27:7	0.75			12:4		8:20	0.19	*Yr7yr7,Yr6yr6*	2-,2+3	3	*Sr9gsr9g*	X,3+	*Lr13lr13*
36	4:13			0.02	0:16		5:12	0.18	*yr7yr7,Yr6yr6*	3-3+	33+	*sr9gsr9g*	3+	*lr13lr13*
37	10:2	0.03			10:2	0.44	3:12	0.20	*Yr7yr7,Yr6yr6*	2-3+	2+3-	*Sr9gsr9g*	3+	*lr13lr13*
38	14:3	0.01			18:1	3.95*	5:11	0.33	*Yr7yr7,Yr6yr6*					
41	0:17				0:5		0:7		*yr7yr7,yr6yr6*					
42	16:6		0.06		9:4	0.23	0:12		*Yr7yr7,yr6yr6*	2=,2,3	3	*Sr9gsr9g*	X,4	*Lr13lr13*
43	15:6		0.14		14:7	0.78	0:20		*Yr7yr7,yr6yr6*				XX-	*Lr13Lr13*
44	13:6		0.44		10:2	0.44	0:17		*Yr7yr7,yr6yr6*	2-,3	2+3-	*Sr9gsr9g*	X-	*Lr13Lr13*
45	11:3	0.07			16:2	1.85	5:10	0.55	*Yr7yr7,Yr6yr6*	2,3	3-3	*Sr9gsr9g*	XX+	*Lr13Lr13*
46	18:0				18:0		0:14		*Yr7Yr7,yr6yr6*	2-	33+	*Sr9gSr9g*	XX+	*Lr13Lr13*
47	16:7	2.06			17:3	1.07	4:13	0.02	*Yr7yr7,Yr6yr6*	2-,3	2+3+	*Sr9gsr9g*	X,3+	*Lr13lr13*
48	14:0				13:0		3:13	0.33	*Yr7Yr7,Yr6yr6*	2-	22+	*Sr9gSr9g*		
49	16:5		0.02		14:8	1.51	0:18		*Yr7yr7,yr6yr6*	2-,3-	3-	*Sr9gsr9g*	33+	*lr13lr13*
52	0:10				0:11		0:15		*yr7yr7,yr6yr6*	2+3+	2+3+	*sr9gsr9g*	X-X,3+	*Lr13lr13*
53	15:6		0.14		13:3	0.33	0:15		*Yr7yr7,yr6yr6*	2-,3+	3	*Sr9gsr9g*		
54	0:16				0:14		0:17		*yr7yr7,yr6yr6*	33+	2+3	*sr9gsr9g*		
55	19:4	0.03			4:1	0.07	4:6	1.2	*Yr7yr7,Yr6yr6*					
57	0:15				0:3		0:3		*yr7yr7,yr6yr6*					
58	5:13			0.07	0:14		5:24	0.93	*yr7yr7,Yr6yr6*	3+		*sr9gsr9g*	3+	*lr13lr13*
59	0:16								*yr7yr7,yr6yr6*					
60	2:8			0.13	0:15		4:7	0.76	*yr7yr7,Yr6yr6*					
61	4:11			0.02	0:19		3:16	0.86	*yr7yr7,Yr6yr6*	3	3	*sr9gsr9g*	3+	*lr13lr13*
62	11:3	0.07			11:5	0.33	2:17	2.12	*Yr7yr7,Yr6yr6*	2-,3-		*Sr9gsr9g*	X+,3	*Lr13lr13*
63	5:18			0.13	0:18		3:15	0.67	*yr7yr7,Yr6yr6*	2+3	2+3	*sr9gsr9g*	3+	*lr13lr13*
64	3:18			1.29	0:23		3:15	0.67	*yr7yr7,Yr6yr6*					
F3 line classification (HR:Seg:HS)					13:28:13		13:23:18			10:21:8			10:16:10	
		$\chi^2_{1:2:1}$			0.07		2.11			0.44			0.44	

TABLE 6.23

F2 and F3 seedling results for Pavon 76 and Hermosillo 77 crosses inoculated with *P. striiformis tritici* pathotype 104 E137 A-

Cross	F2			F3			
	ITs	R+I :S	$\chi^2_{13:3}$	HR	Seg	HS	$\chi^2_{7:8:1}$
Pavon 76/Line E	;N,1+C:3+4	96:28	1.19	22	28	5	0.90
Hermosillo 77/Gabo	;N1-,1C:3+	118:32	1.06	19	17	1	1.36
Hermosillo 77/Line E	;N1-,2C:3+	116:35	1.94	25	25	4	0.34
Total		330:95	3.62	66	70	10	0.28

Cross	Segregating F3 Lines											
	No. lines	Pooled R+I :S	$\chi^2_{13:3}$	χ^2 homog.	No. lines	Pooled R+I :S	$\chi^2_{3:1}$	χ^2 homog.	No. lines	Pooled R+I :S	$\chi^2_{1:3}$	χ^2 homog.
Pavon 76/Line E	12	165:49	2.42	5.87	10	148:55	0.47	2.70	6	23:81	0.46	1.20
Hermosillo 77/Gabo	11	144:35	0.08	7.44	4	70:21	0.18	2.49	3	15:49	0.08	1.72
Hermosillo 77/Line E	11	203:51	0.29	6.95	8	107:38	0.11	3.56	5	29:80	0.15	2.43
Total	34	512:135	1.90	21.15	22	325:114	0.22	9.29	14	67:210	0.10	5.94

2(*Yr7yr7 yr6yr6*): 2(*yr7yr7 Yr6yr6*): 1(*yr7yr7 yr6yr6*); $\chi^2_{7:4:2:2:1}$ (66:34:22:14:10) = 2.07, p>0.7.

Crosses between Pavon 76, Hermosillo 77 and selected cultivars produced no susceptible segregates in F2 or F3 when tested with pathotype 104 E137 A- (Table 6.24). The failure of crosses with Pitic 62, Tobari 66 and Cajeme 71 to segregate was interpreted to indicate allelism with *Yr6*. The resistance of all F2 plants and F3 lines in the cross Hermosillo 77/Pavon 76 confirmed that cultivars shared at least one resistance gene. In another experiment, the same F3 lines and a further 139 F2 plants from Hermosillo 77/Pavon 76 were tested with pathotype 108 E141 A+. All F2 plants were resistant (IT;N) and all F3 lines were HR. This was interpretted to indicate the presence of *Yr7* in both parents.

A further means of confirming that one of the segregating genes in Pavon 76 was *Yr7* was to exploit the knowledge that in bread wheats, *Yr7* is usually associated in coupling with *Sr9g* (McIntosh *et al.*, 1981). In Section 5.4.1.4, it was shown that Pavon 76 probably possessed *Sr9g*, but the results for Hermosillo 77 were inconclusive.

Available F3 lines from crosses involving Pavon 76 and Hermosillo 77 were simultaneously inoculated with *P. graminis tritici* pathotypes avirulent (343-1,2,3,5,6) and virulent (98-1,2,3,5,6) with respect to *Sr9g*. The ITs were noted and lines were classified homozygous or segregating. In the Hermosillo 77 populations, the parental responses (IT 12- and 2, respectively) to the two pathotypes were not recovered in 77 F3 lines. ITs for each line ranged from 3 to 3+ to 4. The higher responses among the progeny, compared to Hermosillo 77, could not be explained, and it was concluded that *Sr9g* was not present. Thus, if the dominant gene in Hermosillo 77 is *Yr7*, as inheritance studies suggested, then this cultivar represents a rare genotype where *Yr7* is not associated with *Sr9g*.

However, segregation for *Sr9gsr9g* was apparent in the Pavon 76/Line E cross (Table 6.22). The presence

TABLE 6.24

Summary of F2 and F3 results for crosses of Pavon 76 and Hermosillo 77 with cultivars possessing *Yr6* and tested with *P. striiformis tritici* pathotype 104 E137 A-

Cross	F2		F3		
	ITs	R+I :S	HR	Seg	HS
Pitic 62/Hermosillo 77	;;C	223:0	58	0	0
Pavon 76/Pitic 62	;;C	196:0	52	0	0
Tobari 66/Hermosillo 77	;,;N1=	123:0	49	0	0
Tobari 66/Pavon 76	;;N,;1=	204:0	60	0	0
Cajeme 71/Hermosillo 77	;;CN	147:0	50	0	0
Pavon 76/Cajeme 71	;,12C	170:0	83	0	0
Hermosillo 77/Pavon 76	;	176:0	81	0	0

of the dominant *Sr9g* allele was clearly indicated by the lower responses of seedlings tested with *P. graminis tritici* pathotype 343-1,2,3,5,6 compared with 98-1,2,3,5,6. There was complete coupling between *Sr9g* and *Yr7*. F2 genotypes with respect to *Sr9g* were postulated on the basis of the response of F3 progenies.

Segregation for progressive necrosis was observed in the F2 population of Pavon 76/Line E. Hawthorn (1984) demonstrated that *Lr13* and *Ne2m* in many genotypes, including Pavon 76, were very closely associated in chromosome 2B. Thirty six F3 lines from Pavon 76/Line E were tested with *P. recondita tritici* pathotype 104-2,3,6,7. The results, presented in Table 6.22, indicated that *Lr13* was inherited as a single dominant gene.

The relationship between the respective rust resistance genes detected in the F3 population of Pavon 76/Line E were examined using χ^2 analyses for joint segregation. Results of these analyses are summarised in Table 6.25. Whereas *Yr6* was inherited independently of *Lr13* and *Sr9g-Yr7*, there was evidence for linkage between

TABLE 6.25

Tests of genetic independence for various segregating resistance genes in Pavon 76/Line E

Analysis	*Sr9g* vs *Yr7*	*Sr9g-Yr7* vs *Yr6*	*Sr9g-Yr7* vs *Lr13*	*Lr13* vs *Yr6*
$\chi^2_{1:2:1}$	*Sr9g* 0.44	*Sr9g* 0.49	*Sr9g* 1.13	*Lr13* 0.42
$\chi^2_{1:2:1}$	*Yr7* 0.44	*Yr6* 1.72	*Lr13* 0.31	*Yr6* 2.00
χ^2 linkage	53.26**	1.87	18.60**	3.47
$\chi^2_{1:2:1:2:4:2:1:2:1}$	54.14**	4.08	20.04**	5.89

Lr13 and *Sr9g-Yr7*. Recombination between *Lr13* and *Sr9g-Yr7* was estimated at 17.75±5.20%, using the maximum likelihood expression for linkage in coupling (Allard, 1956).

CSP44 and Cajeme 71 From previous results (Section 5.4.1.4) it was postulated that CSP44 possessed *Yr6* and was genetically heterogeneous for *YrA*. In contrast, Cajeme 71 was postulated to have *YrA* and was genetically heterogeneous for *Yr6*. Because results were obtained late in the investigations, it was not possible to construct hybrid populations to test the inheritance in all desired cross combinations. However, populations that were available allowed some studies to be carried out.

The Cajeme 71 parent plants used in the crosses listed in Table 6.26 were shown to have the combination *Yr6 + YrA* on the basis of tests with pathotypes 104 E137 A+, 108 E141 A- and 108 E141 A+. F2 segregation in Cajeme 71/Line E tested with 104 E137 A- showed good agreement with an expected ratio of 43 resistant : 21 susceptible based upon the joint independent segregations of a single recessive gene (*Yr6*) and a pair of complementary dominant genes (*YrA*). Similarly, the F3 line classification showed good agreement with the expected ratio of 19 HR : 38 Seg : 7 HS. When this same set of F3 lines was tested with pathotype 104 E137 A+, the line classification conformed with those expected for segregation at a single locus. The recessive nature of this resistance in segregating lines was consistent with the postulation of *Yr6*. Similarly, by testing F3 lines with pathotype 108 E141 A-, the segregation pattern indicated the presence of complementary dominant genes.

The F2 and F3 data for Cajeme 71/Oxley (Table 6.26) tested with pathotype 104 E137 A- demonstrated allelism for *Yr6*. When the same F3 lines were tested with 108 E141 A-, the line ratios indicated the involvement of a single gene, rather than complementary dominant genes. The 26 segregating F3 lines in Cajeme 71/Oxley tested with 108 E141 A-, produced a total of 440 resistant and 139 susceptible plants, thus conforming satisfactorily with

TABLE 6.26

F2 and F3 results for Cajeme 71 and CSP44 hybrid populations tested with *P. striiformis tritici* pathotypes

Cross	Pathotype	F2			F3			
		ITs	R+I :S	χ^2	HR	Seg	HS	χ^2
Cajeme 71/Line E	104 E137 A-	;;N,23/1-:3+4	73:33	0.14[1]	12	33	10	3.79[3]
	104 E137 A+				12	26	17	1.09[4]
	108 E141 A-				2	35	18	4.17[5]
Cajeme 71/Oxley	104 E137 A-	;;N,12C/2C	131:0		59	0	0	
	108 E141 A-				14	26	19	1.68[4]
CSP44/Oxley	104 E137 A-	;;N	177:0					
CSP44/Line E	104 E137 A-	;N,12C:3+	40:86	3.05[2]	29	33	18	5.47[4]
	108 E141 A-				0	0	48	
CSP44/Condor	104 E137 A-				88	0	0	
	108 E141 A-				88	0	0	
	104 E137 A+	;N:3-3,3+	33:80	3.27[2]	30	40	16	4.97[4]

F2 ratio tested [1] 43:21

[2] 1:3

F3 ratio tested [3] 19:38:7

[4] 1:2:1

[5] 1:8:7

segregation at a single locus ($\chi^2_{3:1} = 0.30$, $p>0.6$).

An F2 population of CSP44/Oxley was uniformly resistant to pathotype 104 E137 A-, indicating allelism for *Yr6* (Table 6.26). As the remaining two crosses, CSP44/Line E and CSP44/Condor, were obtained from W.M. Hawthorn as F2 populations, the actual CSP44 and Condor parents that were used had not been comprehensively tested with a range of pathotypes to determine their resistance genotypes. F2 and F3 populations of CSP44/Line E tested with 104 E137 A- segregated for a single recessive gene (Table 6.26) indicating that the particular CSP44 parent plant possessed only *Yr6*. All 48 F3 lines tested with 108 E141 A- were classified fully susceptible, again indicating that only *Yr6* was present.

All 88 F3 lines in CSP44/Condor, tested with pathotypes 104 E137 A- and 108 E141 A-, were resistant (Table 6.26). This indicated allelism for *YrA* and showed that both parents possessed the *YrA* resistance. Seventy six of these F3 lines were tested with 104 E137 A+, and the resulting single gene segregation was attributed to *Yr6* present in the CSP44 parent. Thus the CSP44 parent used in this cross possessed both *Yr6* and *YrA*.

The selection of appropriate pathotypes to test hybrid populations involving Cajeme 71 and CSP44 confirmed the presence of the recessive *Yr6* and the complementary dominant genes contributing to *YrA* in at least some plants of these cultivars. These genetic data supported the conclusions that were drawn from tests of these wheats with different pathotypes (Section 5.4.1.4).

Nacozari 76 A summary of F2 and F3 data from two crosses involving Nacozari 76 is presented in Table 6.27. In crosses with the susceptible parents Gabo and Line E, resistance segregated as a single dominant gene. Although the low ITs were intermediate, the distinction between resistant and susceptible segregates was clear in both F2 populations and F3 lines. However, the second leaf ITs were again important in aiding the identification of intermediate phenotypes. F3 lines in Nacozari 76/Gabo

TABLE 6.27

F2 and F3 results for crosses involving Nacozari 76 tested with *P. striiformis tritici* pathotype 104 E137 A-

Cross		F2 ITs	F2 R+I :S	F2 $\chi^2_{3:1}$	F3 HR	F3 Seg	F3 HS	F3 $\chi^2_{1:2:1}$
Nacozari 76/Gabo	1	3C/3C:4	94:42	2.51	10	24	6	2.40
	2	;C1,23C/1C:3+	106:23	3.54	6	19	2	5.67
Nacozari 76/Line E	1	12C,3/1C:3+	70:27	0.42				
	2	3C/3C:3+	92:39	1.59				
Total			362:131	0.65	16	43	8	7.30*
				$\chi^2_{61:3}$				$\chi^2_{37:26:1}$
Nacozari 76/Pavon 76		;,1-C/1-C:3	150:7	0.02	40	30	3	3.15
Hermosillo 77/Nacozari 76		;,1C/1-C:3	179:6	0.86	47	17	2	6.54*
Total			329:13	0.60	87	47	5	5.82
				$\chi^2_{13:3}$				$\chi^2_{7:8:1}$
Nacozari 76/Oxley		3C/3C:3+	92:39	10.44**	25	29	3	0.10
Pitic 62/Nacozari 76		;C,12C:3+	163:24	4.30*	15	27	3	2.03
Total			255:63	0.24	40	56	6	0.99

were retested with 108 E141 A-. There was an 87% agreement with the classifications based on 104 E137 A-. The pooled F3 line results deviated from the single gene hypothesis at $p<0.05$.

Intercrosses of Nacozari 76 with Pavon 76 and Hermosillo 77 segregated in both F2 and F3. The segregation ratios for both generations were consistent with those expected on the basis of two dominant and one recessive factors. However, a significant deviation ($p<0.05$) was noted for F3 line classifications in Hermosillo 77/Nacozari 76.

F2 and F3 data indicated that the single dominant gene in Nacozari 76 segregated independently of the recessive *Yr6* gene in Oxley and Pitic 62 (Table 6.27). Significant deviations were obtained with both F2 populations, but the pooled data showed good agreement with the postulated 13:3 ratio.

It appeared from ITs, pathotype tests and genetic studies that the single resistance factor in Nacozari 76 was previously uncatalogued.

Corella The F2 results are presented in Table 6.28. The cross Corella/Avocet R was produced as the pedigree of Corella suggested that *YrA* may have been derived from Egret. When two F2 populations from this cross were tested with 104 E137 A-, there was obvious segregation and the observed ratio was consistent with that expected on the basis of segregation of two dominant and one recessive genes, *i.e.* 61 resistant : 3 susceptible, or two complementary dominant genes plus one dominant and one recessive genes *i.e.* 235 resistant :21 susceptible ($\chi^2_{61:3}$ 220:17 = 3.99, $p>0.1$; $\chi^2_{235:21}$ 220:17 = 0.33 $p>0.6$). The population of 237 was too small to statistically distinguish between the two hypothesised models. Simultaneous inoculation of one of these populations with pathotype 104 E137 A+ reduced the apparent segregation to a single dominant and a single recessive gene. The F2 population from Corella/Sunkota failed to produce a susceptible segregate indicating allelism for *Yr7*.

TABLE 6.28

F2 results for Corella crosses tested with *P. striiformis tritici* pathotype 104 E137 A-

Cross		Pathotype	F2 ITs	F2 R+I :S	χ^2
Corella/Avocet R	1	104 E137 A-	;,;C1=:33+	133:9	0.86[1]
	2	104 E137 A-	;C1=:3+	87:8	2.97[1]
	1	104 E137 A+	;C,1C:33+	93:15	1.68[2]
Total		104 E137 A-		220:17	3.99[1]
Corella/Sunkota		104 E137 A-	;;C	125:0	

F2 ratio tested [1] 61:3
[2] 13:3

It was concluded that Corella had two genes for resistance. The dominant gene was allelic with *Yr7*. The recessive gene was probably *Yr6* since this was the only catalogued gene which was observed to be inherited in a recessive manner. The dominant pattern of segregation occurring in Corella/Avocet R was probably due to one or both of the complementary genes determining the *YrA* resistance.

Further support for the postulated combination of *Yr6 + Yr7* conditioning resistance in cv. Corella was received from R. Johnson, Cambridge, England (pers. comm. to R.A. McIntosh, 1985). Using pathotypes virulent with respect to *Yr6* (108 E141), *Yr7* (171 E138) or *Yr6 + Yr7* (39 E134) IT responses for Corella were low, low and high respectively. In addition, tests with stem rust pathotypes virulent and avirulent with respect to *Sr9g* identified this gene in Corella (Section 5.4.1.4). This is further evidence for the presence of *Yr7* in this cultivar.

Conclusions Inheritance studies, including tests of allelism, and the responses of parental stocks to various pathotypes (Section 5.4.1.4) led to the following conclusions:

1. Resistances in Pavon 76, Hermosillo 77 and probably Corella were determined by *Yr6* and *Yr7*. Hermosillo

77 represented a rare instance in which *Yr7* and *Sr9g* were not linked in coupling.

2. Cajeme 71 and CSP44 were genetically heterogeneous. CSP44 was homogeneous for *Yr6* and heterogeneous for the *YrA* resistance, whereas Cajeme 71 was homogeneous for *YrA* and heterogeneous for *Yr6*.

3. Nacozari 76 possessed a single dominant gene which had not been reported.

6.3.4.2 Relationships between seedling and adult-plant responses

Susceptible F2 seedlings from various crosses were transplanted in the field to test the possibility that adult plant resistance factors may be present in addition to the resistance genes detected in seedling tests. Results presented in Table 6.29, suggest that of the four resistant parents, only Hermosillo 77 appeared to lack adult-plant resistance additional to that determined by genes effective in the seedling stage.

These results agree with field experiments in which adult plants of CSP44 and Cajeme 71, selected for *Yr6 + YrA*, were resistant to pathotype 108 E141 A+ which has the combined virulence with respect to these genes (Section 5.4.3.1).

The relationship between F3 seedling and F3 adult-plant response for two populations of Nacozari 76 are presented in Table 6.30. Lines classified as HR in seedling tests were generally HR as adult-plants, indicating that the gene conferring seedling resistance also conferred resistance in adult plants. However, not all lines found to be HS as seedlings were HS as adult plants. Results for these discrepant lines need to be confirmed since 25 of 27 lines that segregated as seedlings were scored segregating as adult plants.

6.3.5 INHERITANCE IN TRITICALE CULTIVARS

Commercial triticales were developed from amphiploids combining the genomes of tetraploid wheat and cereal rye. Further hybridizations between primary

TABLE 6.29

Responses of adult plants grown from selected susceptible seedlings in six wheat crosses tested with pathotype 104 E137 A-

Cross (parent response)	F2 Adult-Plant Response	
	R+I (response)	S (response)
Pavon 76/Line E (10R,60S)	9(20R-MR) + 32(40MS)	9(80S)
Hermosillo 77/Gabo (10VR,50MS-S)	2(30MS-S)	22(80MS-S)
Hermosillo 77/Line E (5R,80S)	1(5R)	24(60MS-S,80S)
Cajeme 71/Line E (15R-MR,80S)	5(30MR)	15(60MS-S,80S)
Cajeme 71/Gabo (20R-MR,60MS-S)	2(15MR)	19(40MS-S)
CSP44/Line E (10R,80S)	5(10R) + 8(40MR-MS)	12(70S)

TABLE 6.30

Relationship between F3 adult and F3 seedling responses in hybrid populations inoculated with *P. striiformis tritici* pathotype 104 E137 A-

Cross (parent response)	F3 Adult	F3 Seedling			Total
		HR	Seg	HS	
Nacozari 76/Line E (OR, 60S)	HR	14	1	2	17
	Seg	1	21	5	27
	HS			8	8
	Total	15	22	15	52
Nacozari 76/Gabo (OR,60MS-S)	HR	11	1	1	13
	Seg		4		4
	HS			7	7
	Total	11	5	8	24

hexaploid triticales and hexaploid wheats have resulted in integrated hexaploid triticales where certain rye chromosomes have been replaced by chromosomes of the D genome. Integrated hexaploid triticales were often agronomically superior and produced higher quality grain than primary triticales.

Early assumptions of adequate levels of durable, multifactor resistances in triticales to a range of diseases, including the rusts, were questioned by McIntosh *et al.* (1983) who reported the sudden loss of stem rust resistance in a large proportion of triticale cultivars. Their work indicated that triticale may be highly vulnerable to infection by rust diseases, especially if relatively simple pathogenic changes were to occur in pathogen populations. For example, a single mutation to virulence with respect to *Sr27* in the stem rust pathogen in Australia resulted in severe losses in crops of cv. Coorong.

The basis of resistance in triticales to stripe rust has not been previously investigated. In the present studies, a stripe rust susceptible line was found in a triticale field nursery grown by N.L. Darvey, at PBI, Castle Hill. This line, designated NLD Sus. proved highly susceptible in subsequent glasshouse and field tests. It was used as a susceptible parent in crosses with commercial triticale cultivars in order to obtain preliminary genetic data.

6.3.5.1 Glasshouse studies

Seedling segregation results are presented in Table 6.31. Resistance in cv. Dua appeared to be determined by a single dominant gene. Seedlings with intermediate ITs were grouped in the resistant class.

The segregation data for Coorong/NLD sus were ambiguous. The F2 results marginally deviated from those expected for segregation of 3 resistant:1 susceptible. The F3 line behaviour showed a very significant ($p<0.01$) deviation from expectations for a 1 HR:2 Seg: 1 HS ratio. Despite these discrepancies, a single gene for resistance

TABLE 6.31

F2 and F3 seedling results for three triticale crosses following inoculation with *P. striiformis tritici* pathotype 104 E137 A-

Cross	F2			F3				Segregating F3 lines			
	ITs	R+I :S	χ^2	HR	Seg	HS	χ^2	No. lines	Pooled R+I :S	χ^2	$\chi^2_{homog.}$
Dua/NLD Sus.	;;CN,12C:3+4	121:52	2.36[1]	18	23	8	4.26[4]	23	420:116	3.22[1]	13.25
Coorong/NLD Sus.	;;CN,12C:3+4	127:58	3.98*[1]	20	12	5	16.73**[4]	12	222:77	0.09[1]	5.22
Ningadhu/NLD Sus.	;;CN,;12:3+4	112:19	1.55[2]	15	16	1	0.57[5]	5	37:20	3.09[1]	1.63
								8	99:18	0.86[2]	0.98
								3	16:17	9.71**[3]	0.06

Ratio tested by χ^2 analysis [1] 3:1 [4] 1:2:1

[2] 13:3 [5] 7:8:1

[3] 1:3

best explained the observed results.

Resistance in Ningadhu was determined by two genes, one recessive and one dominant. The F2 segregation conformed satisfactorily with the proposed 13:3 ratio. Segregating F3 lines were examined for goodness-of-fit to the expected ratios 13:3, 3:1 or 1:3. Eight lines segregated 13:3 and five lines segregated 3:1. Only three lines were possible candidates for the 1:3 category, and the pooled results for these differed significantly from the expected ($p<0.01$). Clearly, a larger population of F3 lines is required to further examine the postulated independent segregation of a dominant and a recessive gene in this cultivar.

The relationships between the resistance genes detected in these cultivars were not investigated. However, the dominant gene in all three cultivars could have been derived from Armadillo 'S' which was common to the pedigrees of all three cultivars.

6.3.5.2 Relationships between seedling and adult-plant responses

The responses of F2 adult plants and their seedling F3 progenies were studied in order to examine the relationship between seedling and adult-plant responses. The results presented in Table 6.32, suggest that adult-plant resistance in the Dua/NLD sus population was correlated with the seedling response. However, adult plants noted as intermediate in response gave variable progenies.

In Coorong/NLD sus there was evidence for adult-plant resistance additional to that determined by the gene effective at the seedling stage. Four of five plants scored HS as F3 lines were resistant or intermediate as adult plants.

In the F2 of Ningadhu/NLD sus, the most susceptible segregates were scored 40 MS as adult plants and this was considerably lower than the NLD sus parent. Only one F3 line, however, was scored HS in the seedling stage. Gene(s) detected in the seedling stage in this cross apparently

TABLE 6.32

Relationship between F2 adult and F3 seedling responses in various triticale crosses inoculated with pathotype 104 E137 A-

Cross (parent response)	F2 Adult response		F3 seedling HR	Seg	HS	Total
Dua/NLD Sus. (OR,80S)	OR	R	16	17		33
	20MR	I	2	6	3	11
	80S	S			5	5
	Total		18	23	8	49
Coorong/NLD Sus. (OR,80S)	OR	R	20	11	2	33
	40MR	I		1	2	3
	90S	S			1	1
	Total		20	12	5	37
Ningadhu/NLD Sus. (OR,80S)	OR	R	9	1		10
	10R-MR	I	4	9		13
	40MS	S	2	6	1	9
	Total		15	16	1	32

permitted the development of considerable levels of disease.

Conclusions Relatively small populations were used in these preliminary studies of stripe rust resistance in triticales. Larger populations, including intercrosses between resistant cultivars, will be required to confirm the present results and to examine the relationships between these resistances. The results with triticale suggested the presence of factors for adult-plant resistance additional to those detected at the seedling stage.

6.4 DISCUSSION

Yr6 and *Yr7* which occur separately or combined in several Australian and exotic wheats, were observed to be inherited as recessive and dominant Mendelian genes, respectively.

Macer (1966) described *Yr6* as being recessive whereas *Yr7* was dominant. *Yr6* was genetically independent of *Lr13* whereas *Yr7* showed linkage of 17.75 ± 5.20% with *Lr13* which is located in chromosome 2BS (McIntosh, 1983). These results were as expected, since *Yr7* was previously known to be located in chromosome 2BL (Johnson and Dyck, 1984) and *Yr6* was located in chromosome 7BS (El-Bewdey and Röbblen, 1982).

Kochhar *et al.* (1983) crossed cv. Tobari 66 with Bon Fermier (*Yr3a*) and Nudif TP 250 (*Yr1 + Yr6*) and failed to detect a susceptible segregate when F2 populations were inoculated with avirulent pathotypes. They concluded that Tobari 66 carried *Yr1 + Yr3a + Yr6*. In the present studies it was clear that Tobari 66 possessed *Yr6*. Because Australian pathotypes are virulent for *Yr3a*, no confirmatory data could be obtained in respect of the presence of this gene. However, *Yr1* is not present in local stocks of Tobari 66 as pathotype 108 E141 A-, which was virulent for *Yr6* and avirulent for *Yr1*, produced a high IT. The failure of Tobari 66/Nudif TP 250 to segregate in the study of Kochhar *et al.* (1983) is therefore attributed to allelism for *Yr6*. The conclusion that *Yr1* was present appears incorrect, and exemplifies the need to conduct tests of allelism only when the numbers of genes concerned have been established. The use of lines with single genes will be more efficient, in this respect, than the use of lines with gene combinations.

Again, the report of Kochhar *et al.* (1983), indicating that Sonalika and Tanori 71 carry *Yr1 + Yr3a + Yr4b + Yr6*, appears to be incorrect. These, and a range of Australian and exotic wheats, have been shown to carry *YrA*. It was concluded from the present studies that the *YrA* resistance was determined by two complementary dominant genes. One of these genes appeared to occur in several 'S' selections taken from WW15 derivatives which were genetically heterogeneous for the *YrA* resistance. The reason why one, and not one or the other, of the genes occurred in the 'S' selections was not clear. The presence of complementary genes is yet to be confirmed by intercrossing appropriately selected

susceptible wheats in order to reproduce the resistance. In one cross involving Tanori 71, the complementary genes determining stripe rust resistance segregated independently of *Lr13*. Results from the present studies suggest that local stocks of Sonalika and Tanori 71 do not possess the genes *Yr1* or *Yr6*, as proposed by Kochhar *et al.* (1983).

Reports of complementary genes have been relatively common in the stripe rust literature. In India, Nambisan and Kholi (1961) reported that F2 and F3 ratios in hybrid populations of Cometa Klein conformed with those expected for two segregating complementary dominant genes when tested as seedlings with two avirulent pathotypes. Earlier (Gosh *et al.*, 1958) and subsequent (Gandhi, 1971) studies found single dominant genes in Cometa Klein, but both of these studies related to adult plant responses in the field. Field studies of F1, F2 and F3 suggested complementary genes for stripe rust resistance in cv. Frondoso (Pal *et al.*, 1956) and the advanced line E255 (Sandhu and Singh, 1970). In Australian studies, local accessions of Frondoso were concluded to possess *Yr6* on the basis of response to selected pathotypes (Wellings, unpublished).

In the U.S.A., Allan *et al.* (1966) observed F2 and F3 ratios in populations of the resistant Nord Desprez crossed with the susceptible Norin 10-Brevor 14. They concluded that two complementary dominant genes conditioned resistance in both seedling and adult plant tests. Lewellen and Sharp (1968) found evidence for complementary genes conditioning the temperature-sensitive seedling resistance of cv. Rego. F2 ratios obtained at a high temperature regime (15C/24C) gave a good approximation to the expected distribution of 9 resistant : 7 susceptible, whereas data from the low temperature regime (2C/18C) were significantly different from those expected for either 9:7 and 3:1 models. The authors concluded that the latter results were due to the modifying influence of temperature-sensitive minor genes.

In Europe, Walther and Herdam (1984) claimed

evidence for complementary genes in cv. Alcedo after testing segregating populations in the field. However, segregation in the reciprocal cross suggested a single recessive gene. Dutlu (1984) tested seedlings of two different populations involving the Mexican cv. Robin with two pathotypes. He concluded that the F2 and F3 ratios were consistent with a model of two complementary dominant genes.

Thus, complementary genes for resistance to stripe rust have been reported on several occasions following studies in both glasshouse and the field. Further reports of complementary genes conferring rust resistance in the winter cereals were listed by Singh (1983). However, there were only two examples in which the complementary gene hypothesis had been convincingly demonstrated. Baker (1966), working with crown rust resistance in Bond oats, and Singh and McIntosh (1984a), working with leaf rust resistance in Gatcher wheat, demonstrated that the respective complementary genes could be isolated in susceptible lines. Intercrosses of such lines reproduced the resistance which segregated in the expected ratios in F2 and F3. Singh and McIntosh (1984b) located one leaf rust resistance gene in chromosome 3BS, whereas the second gene was located in 4Aß.

Grewal *et al.* (1976) noted that hybrids between the stripe rust susceptible wheats Crim and WG586 produced resistant progeny in F2. They subsequently showed F2 and F3 segregations that were consistent with complementary gene behaviour. This is possibly the best published evidence for complementary genes conditioning resistance to stripe rust in wheat. In the present studies the inheritance of resistance in several wheats possessing the *YrA* resistance was consistent with a complementary gene model. Crosses between susceptible selections of different, genetically heterogeneous wheats failed to confirm the hypothesis. Thus if complementary genes were involved, then susceptible sibling genotypes must have either lacked both of the genes for resistance, or must have carried the same gene. As

both possibilities appear unlikely, further work is necessary to confirm the presence of complementary genes in wheats with the *YrA* resistance.

Tests with the available Australian pathotypes indicated that Cajeme 71 and CSP44 combined *Yr6* and the *YrA* resistance. This was confirmed in inheritance studies which also showed that *Yr6yr6* segregated independently of the complementary genes that conferred the *YrA* resistance.

The recessive *Yr6* and the dominant *Yr7* were shown to be combined in cv.s Pavon 76 and Hermosillo 77. The presence of *Yr7* in parental stocks of Pavon 76 was also predicted from stem rust data indicating the presence of *Sr9g*. Inheritance studies using selected pathotypes of the stripe rust and stem rust pathogens confirmed the presence of *Yr7* and its close linkage with *Sr9g*. The *Sr9g-Yr7* 'complex' was linked in coupling with *Lr13* and showed recombination of 17.75 ± 5.20%. Hawthorn (1984) obtained an average linkage of 23.76 ± 0.73% for *Sr9* alleles and *Lr13*. As expected, *Yr6yr6* in Pavon 76 crosses segregated independently of both *Sr9g-Yr7* and *Lr13*. Kirmani *et al.* (1984) proposed that Pavon 76 carried *Yr8* , despite the fact that pathotype 6 E16, combining virulence for *Yr6* , *Yr7* and *Yr8* was virulent on this cultivar. The pedigree of Pavon 76 (Vicam 71//Ciano 67 sib / Siete Cerros 66 / 3 / Kalyansona / Bluebird) precludes the possibility of *Yr8* which was derived from *Aegilops comosa*. Perwaiz and Johnson (1985) confirmed that Pavon 76 was susceptible to pathotypes with virulence for *Yr6* and *Yr7* , although their stock of Pavon 76 was apparently genetically heterogeneous for *Yr7*.

The presence of *Yr6* and *Yr7* in Hermosillo 77, has not been reported previously. The presence of *Yr7* and the absence of *Sr9g* in Hermosillo 77 represents the first report of this genotype in bread wheats. McIntosh *et al.* (1981) reported that the durum cultivars, Acme and Kubanka, possessed *Sr9g* but not *Yr7* . The *Sr9g-Yr7* combination in bread wheats was derived from Iumillo durum (McIntosh *et al.*, 1981). The pedigrees of Pavon 76 and Hermosillo 77 are not clearly related, although Ciano sib and Bluebird are

both represented and these, together with wheats such as TZPP, are possible origins of these genes.

Inheritance of stripe rust resistances in triticales had not been investigated previously. Joshi *et al.* (1977) studied the responses of various Indian triticales to pathotypes of *P. striiformis* in the glasshouse and the field. Lines with pedigrees similar to Ningadhu and Coorong were noted to be resistant, whereas a line whose pedigree was similar to Dua, was susceptible. In the present studies, single genes were shown to confer seedling resistance to stripe rust in these triticales. It is possible that these resistances may be neutralised by single gene mutations for increased virulence in the pathogen, and thus stripe rust resistance in triticale may be as genetically vulnerable as that noted for stem rust resistance (McIntosh *et al.*, 1983).

Inheritance studies in all the wheats examined showed that materials resistant in seedling tests remained resistant as adult plants when tested with the same pathotype. On the other hand, plants susceptible in seedling tests often showed variable levels of adult-plant resistance in the field. The inheritance of adult-plant resistance was not studied in detail because the early studies had to be based on glasshouse investigations, due to voluntary restrictions aimed at preventing the release of the pathogen in the field. Later studies concentrated on the detection of possible adult-plant resistance in seedling susceptible segregates.

In certain crosses, low levels of host contamination were attributed to outcrossing or to contamination during machine threshing. In order to avoid such problems in future, it may be necessary to protect F1 spikes from outcrossing, and to use threshing machines that are less likely to permit contamination, or to thresh F1 plants by hand. Such low levels of contamination will be major problems only in instances where studies of allelism or close linkage are restricted to F2 populations. In these cases, contaminants can be recognised and discarded when F3 lines are tested.

7 GENERAL DISCUSSION

The occurrence of *P. striiformis f.sp. tritici* in Australasia afforded a spectacular and comparatively rare opportunity to study the adaption of an exotic pathogen in a large, geographically isolated continent. The introduction and establishment of barley stripe rust in South America in 1975 provided a similar example (Dubin and Stubbs, 1985). From the first reports in 1979, stripe rust spread rapidly within seasons, and is now widely distributed in all regions of the eastern Australian wheat belt with the exception of the central highlands of Queensland. The large distances involved in dispersal of this pathogen were exemplified by the appearance of stripe rust in New Zealand in 1980, presumably resulting from wind transport of inoculum from Australia. A notable feature, however, has been the failure of stripe rust to spread and establish in the wheat-growing areas of Western Australia where the main cultivars are known to be susceptible. This is believed to be due to the predominant west-to-east movement of weather patterns which generally results in frequent aerial dispersal of rust inoculum in an easterly direction (Watson and de Sousa, 1983). However, occasional dispersal of *P. graminis tritici* from eastern Australia to Western Australia has been suggested (Watson and Cass Smith, 1962).

Within the eastern Australian wheat belt, stripe rust has appeared in each season since 1979 and is now regarded as an endemic disease to the region. The means of survival of the pathogen during the frequently hot and dry summer months, when host material is at minimum availability, is unknown. Although a range of grasses of the sub-family Pooideae were shown to be susceptible to *P. striiformis tritici*, it was evident that susceptible wheats would assume a predominant role in the over-summer survival of the pathogen. Whereas specific sites of over-summer survival were not identified, it was apparent from the reported first sitings and the pathotypes identified in the initial phase of an epidemic, that the stripe rust pathogen survived at independent locations throughout the

wheat areas of eastern Australia.

Epidemic development was influenced by the time of first sighting of the disease in each season. From 1979 to 1982, stripe rust appeared in early spring and was prevalent in southern N.S.W. and Victoria. The early occurrences in 1983 and 1984 permitted inoculum increase throughout the winter months, resulting in widespread epidemics in spring throughout southern Queensland, N.S.W., Victoria and to a limited extent, South Australia. Data from pathotype surveys indicated that epidemic development was influenced by the initial inoculum occurring within local areas. However, frequencies and movements of particular pathotypes were related to the area and location of cultivars which permitted the development of those pathotypes.

In order to minimise epidemic development, it is clear that under Australian conditions, the levels of initial inoculum must be minimised. A number of options are available to achieve this. Firstly, over-summer survival of the pathogen could be reduced by reduction of wheat stubble regrowth and volunteer wheat plants, particularly in irrigated areas. However, the stripe rust pathogen is unlikely to be eliminated as shown by the apparent significant levels of over-summer survival which occurred during the widespread drought in eastern Australia in 1982. Thus, this strategy will not be completely effective when considering the large areas of wheat cultivation and the numerous opportunities for the pathogen to survive on suitable hosts in localised areas which have received adequate moisture for wheat growth, either as rainfall or irrigation.

Fungicide applications also present possibilities for reducing levels of initial inoculum. Aerial spraying was widely adopted in the major epidemic of 1983, resulting in expenditure on chemicals and application estimated at $12 million in N.S.W. alone. However, the relatively low yields under Australian dryland conditions and the variable responses of some cultivars means that economic returns from fungicide sprays will be marginal. Thus, this strategy will generally be restricted to small areas of susceptible

or moderately susceptible cultivars grown under irrigation, or under potentially high yielding conditions. Alternatively, the use of fungicides as seed dressings represents a relatively low cost alternative in providing protection during the early phases of crop establishement. The use of seed dressings could be particularly beneficial on late-sown cultivars, which are susceptible to stripe rust in the seedling stage, in areas where the disease is known to be present in earlier-sown crops.

The third option for minimising inoculum levels in crops is the development of resistant cultivars. A fundamental requirement of an effective breeding program aimed at the incorporation of diseases resistance is a knowledge of the pathogenic variation in the parasite. Pathogenicity surveys reported in these studies have confirmed that *P. striiformis tritici*, like other rust pathogens, has a capacity for producing new variants capable of neutralising the genetic protection of previously resistant cultivars. These relatively frequent variants were detected by assessing the response of a standard group of host materials to a large number of pathogen isolates. In order to distinguish the range of pathogenic variation present in Australia, it was proposed to add two testers (*i.e., T. spelta album, Yr5,* and Avocet R, *YrA*) to the system of pathotype nomenclature which was described by Johnson *et al.* (1972).

In the six years from 1979 to 1984, six distinct pathotypes have been identified. In the 1985 crop season, a further pathotype, virulent with respect to *Yr8*, was detected for the first time in Australia (Wellings, unpublished). However, among this group of pathogenic variants, two basic types have caused significant changes in the responses of commercial cultivars. Pathotypes virulent with respect to *YrA* (*i.e.,* 104 E137 A+ and 108 E141 A+) have caused estimated yield losses approaching 50% in cultivars carrying the corresponding *YrA* resistance (*e.g.* Avocet). Pathotypes virulent with respect to *Yr6* (*i.e.* 108 E141 A- and 108 E141 A+) have also caused

significantly higher responses in some cultivars with the matching host resistance gene (*e.g.* Millewa and Bindawarra). In contrast, pathotypes virulent with respect to *Yr5* or *Yr8* are not expected to cause significant changes in responses of Australian cultivars as these host resistance genes are not deployed. Although a range of pathogenic variability can be detected, it is of utmost importance that it be related to the host resistance genes in current commerical wheats and/or to resistance sources that are being used in breeding programs.

The occurrences of new pathotypes reported in the present studies is consistent with overseas experience. Bingham (1981a) reported that 10 previously unidentified pathotypes of the stripe rust pathogen had been found in England during the preceding 15 years. However, a limitation of Australian stripe rust pathogenicity surveys to date has been the failure to survey for variation in pathogenicity with respect to adult-plant resistance. It would appear desirable to develop techniques that would allow glasshouse investigations of adult-plant virulence, since field trials restrict the number of cultures for testing. However, the relationship between glasshouse and field responses require careful investigation, and, in a final analysis, the responses of cultivars to new pathotypes would have to be based on field data.

A feature of the pathotypes of *P. striiformis tritici* that were detected in Australasia was the variation which occurred in respect to single sources of host resistance. It appeared that mutation, rather than some other mechanism such as somatic hybridisation, was the probable cause of new variation. This feature was particularly useful in designing experiments to survey host stocks for resistance. Pathotype pairs could be selected and assumed to be identical except for the contrasting virulence and avirulence with respect to a particular host gene or resistance source. The interaction of these pathotypes with hosts provided data allowing firm conclusions to be drawn regarding the presence of the corresponding resistance

gene. In contrast, overseas laboratories with large collections of pathotypes isolated over many years cannot so readily select pairs of pathotypes where the background pathogenicity spectra can be assumed to be similar. Presumably difficulties of this type led Kirmani *et al.* (1983) to conclude that the susceptibility of Pavon 76 to pathotype 6 E16, which was virulent for *Yr6, Yr7* and *Yr8*, indicated the presence of *Yr8* in that cultivar.

The pathotypes of *P. striiformis tritici* described in the present studies were able to distinguish host resistances conferred by *Yr6, Yr7, YrA* and the combination *Yr6 + YrA*. It was shown that the variable expression of resistance in certain wheats carrying *Yr6* or *YrA* was related to variation in temperature or light intensity respectively. As a result of these studies, temperatures of 17C and light intensities greater than 1000 foot candles are recommended to enhance the expression of incompatible ITs produced by *Yr6* and *YrA*. Due to the restricted use of pathotype 106 E139 A-, which was virulent for *Yr7* and present only in New Zealand, the close linkage in coupling with *Sr9g* was exploited to confirm hypotheses regarding the presence of *Yr7*.

Inheritance studies confirmed reports that *Yr6* and *Yr7* respectively, behaved as recessive and dominant genes. *Yr7* showed linkage of 17.75 ± 5.20% with *Lr13* in chromosome 2B. This value was similar to that found for *Lr13* and *Sr9* alleles in several cultivars by Hawthorn (1984) and *Lr13* and *Sr9b* in Kenya Plume (Singh and McIntosh, 1986). On the other hand, *Yr6* showed the expected genetic independence from both *Yr7* and *Lr13*.

The *Yr7* and *Sr9g* genes were present in several wheats, including Sunkota, Gatcher R, Oroua, Chris, Norquay, Sinton, Thatcher, Manitou, South Africa 43, Corella and Pavon 76. In one segregating population of Pavon76/Line E, no recombinant between these genes was detected among 39 F3 lines. However, populations derived from a Hermosillo 77 cross, appeared to segregate for

Yr7yr7 but failed to detect *Sr9g*. If the gene in Hermosillo 77 is *Yr7*, as indicated from pathotype tests, then this is the first report of a breadwheat carrying *Yr7* without *Sr9g*. On the other hand, McIntosh *et al*. (1981) reported certain durum wheats carrying *Sr9g* but not *Yr7*. It seems likely that Hermosillo 77 represents a relatively rare recombinant between these resistance genes. Inheritance studies were useful in identifying the presence of *Yr6* and *Yr7* in Pavon 76 and Hermosillo 77. This identification could not have been achieved by using the pathotype array currently available in Australia.

The *YrA* resistance which was inherited as two complementary dominant genes, was present in certain Australian and exotic wheats. This resistance has not been previously identified. The Avocet R stock possessing *YrA* was sent to overseas laboratories for testing. R.W. Stubbs, Wageningen, The Netherlands (pers. comm.), was able to recognise variation in the response of this stock using a range of cultures of diverse origins. Apparently virulent and avirulent types were identified in growth cabinet tests conducted at high light intensities. However, R. Johnson, Plant Breeding Institute, Cambridge, England (pers. comm.) was unable to confirm evidence of variation. It is possible that under the prevailing conditions, light intensities were not adequate for clearly identifiable low reactions. A second alternative is that the Cambridge cultures were virulent for the *YrA* resistance. However, this appears unlikely because wheats known to possess this resistance were clearly resistant in field experiments conducted by J. Blackman at Plant Breeding Institute, Cambridge, in 1985 (pers. comm. to R.A. McIntosh). In contrast, the Avocet S line was clearly susceptible.

Johnson (pers. comm.) suggested that *Yr2* may be present in Avocet R. Perwaiz and Johnson (1985) concluded that *Yr2* was present in a range of CIMMYT-distributed wheats which have been shown in these studies to carry *YrA*. Wheats with the *Yr2* gene, present in Heines VII, are susceptible to Australasian pathotypes. However, *Yr2*, or a gene closely linked with *Yr2*, could be one of the

complementary genes determining the *YrA* resistance. This hypothesis could be tested by crossing cultivars possessing *Yr2* (*e.g.* Heines VII) with cultivars possessing *YrA* (*e.g.* Avocet R). If the hypothesis is correct, then F2 and F3 populations of such crosses should be HR in Europe when tested with *Yr2*-avirulent pathotypes, and should show single gene segregation in Australia using *YrA*-avirulent/*Yr2*-virulent pathotypes. If the hypothesis is not correct, then segregations in Australian tests should conform to 9 resistant : 7 susceptible, indicating that the parent carrying *Yr2* does not possess one of the two postulated complementary genes.

Pathotype tests and inheritance studies permitted conclusions regarding the stripe rust resistance genes present in a range of Australian and exotic wheats. These conclusions, presented in Table 7.1, represent the most extensive list that is currently available of stripe rust resistance genes in spring wheats.

The seedling resistances of current Australian cultivars is based on combinations of four genes, two of which constitute the *YrA* resistance (Table 7.1). Of these, *Yr7* remains effective in eastern Australia, although virulence was detected in New Zealand in 1982.

The same four genes are present in a wide range of exotic wheats. The resistances in United States and Canadian wheats were predominantly based on *Yr7*. This is undoubtedly due to the key role in breeding programs of cultivar Thatcher which occurs in the pedigrees of each of these wheats including Lee (McIntosh *et al.*, 1981). In contrast, the resistances in Brazilian wheats were based on *Yr6* which was presumably transferred from the cultivars Frontiera and Surpreza. The Kenyan cultivar, Romany, also possessed *Yr6*, as did several Mexican cultivars, and all were related to Frontiera. This cultivar, and its derivatives, have been widely exploited as a source of leaf rust resistance which was shown by Hawthorn (1984) to be conferred by *Lr13*.

The *YrA* resistance was common in Mexican wheats derived from the CIMMYT program. The Indian cultivar,

TABLE 7.1

Genes for resistance to *P. striiformis tritici* determined in spring wheats arranged according to country of origin

YrA	*Yr6*	*Yr7*	*Yr6 + YrA*	*Yr6 + Yr7*
Australian Wheats				
Avocet R[1]	Bindawarra	Eagle	CSP44	(Corella)[2]
Banks R	Jacup	Gatcher R		(Hartog)[3]
Cocamba	Miling	Sunkota		
Condor R	Millewa			
Egret R	Oxley			
Sunstar	Takari			
Vulcan	WW80			
WW15=Anza				
Exotic Wheats				
Mexico				
Inia 66	Atlas 66		Cajeme 71	Hermosillo 77
Nainari 60	Ciano 67			Pavon 76
Nuri 70	Penjamo 62R			
Tanori 71	Pitic 62			
	Tobari 66			
Brazil				
	Colotana			
	Frontana			
	Frontiera			
	Rio Negro			
	Supreza			
U.S.A. and Canada				
		Lee		
		Chris		
		Manitou		
		Norquay		
		Sinton		
		Thatcher		
Elsewhere				
Sonalika	Romany	Oroua		
	TZPP	South Africa 43		

[1]R implies a resistant selection of the particular cultivar.

[2]requires confirmation.

[3]assumed to have *Yr6 + Yr7* as this cultivar is a local selection of Pavon 'S'.

Sonalika, was selected from material distributed by CIMMYT. Again, *YrA* in Australian wheats can be traced to WW15, a selection from CIMMYT nurseries. It is apparent that the international distribution of the *YrA* resistance in spring wheats can be traced to materials produced by CIMMYT. A single ancestral source of this resistance is at present unknown but, since complementary genes are involved, the resistance may have emerged only after the appropriate cross of susceptible wheats possessing the complementary factors.

The *Yr6* gene was possibly introduced into the CIMMYT program with the initial use of South American wheats such as Frontana, Frontiera and their derivatives. This material was subsequently combined with germplasm carrying *YrA*. Mexican cultivars selected from this germplasm were shown in the present studies to possess either *Yr6* or *YrA*, while the combination of *Yr6 + YrA* was relatively rare. It was evident that the predominance of *Yr6* and *YrA* in Australian wheats was largely the consequence of the use of CIMMYT-generated germplasm, in particular, the wheats WW15 and WW80. Recent data (McIntosh pers. comm.) suggests that *YrA* occurs in several spring wheats from the Indian sub-continent and again, these were largely selected from CIMMYT materials or crosses involving such materials.

It is clear from these studies that the CIMMYT program had a large influence in the distribution of stripe rust resistant materials, especially in the spring wheat growing areas of the world. In addition to the resistances reported in the present studies, Perwaiz and Johnson (1985) suggested that *Yr2* was present in CIMMYT-generated cultivars, although this could not be confirmed in Australian studies, as all pathotypes were virulent for *Yr2*. Merker (1982) reported that the stripe rust resistance of Kavkaz wheat had been transferred to Veery and related cultivars produced by CIMMYT. The gene involved, designated *Yr9*, was present on a chromosome 1B/1R wheat/rye translocation which also possessed genes conferring resistance to stem rust (*Sr31*) and leaf rust (*Lr26*) (McIntosh, 1983). However, the presence of this translocated rye segment in

advanced lines from Australian wheat breeding programs) was apparently associated with flour quality defects (Martin and Stewart, 1984) and wheats with this resistance are unlikely to be released locally. Although Prescott (Anon., 1984) claimed that *Yr9* was effective in rust nurseries throughout Asia, Africa and the Middle East, pathotypes virulent with respect to *Yr9* have been detected in Europe (Bayles and Thomas, 1983; Johnson *et al.*, 1983). Kohli (Anon., 1984) reported that pathotypes with combined virulence for *Yr6, Yr7* and *Yr9* were prevalent during severe stripe rust epidemics on CIMMYT-derived wheats in Chile.

Thus, on the basis of results reported in these studies and the literature, it appears that the major genes for stripe rust resistance in CIMMYT-generated wheats include *Yr2, Yr6, Yr7, Yr9* and *YrA*. It was noted that "the level of resistance to leaf and stripe rust in CIMMYT materials does not . . . equal their stem rust resistance. Most released Mexican semidwarf wheats have lost their resistance to leaf and stripe rust within five years after widespread commercial production" (Anon., 1982).

However, despite the relatively few seedling-effective stripe rust resistance genes currently represented in Australian and exotic spring wheats, the results of the present studies indicated that effective adult-plant resistances, in addition to seedling resistances, are present in many cultivars. The inheritance and allelic relationships of stripe rust resistances effective only in adult-plants has not been thoroughly investigated, as many earlier field studies were confounded by the presence of seedling genes. However, several adult-plant resistances in Europe have been overcome by specific pathotypes (Stubbs, 1985) and thus these resistances may, in certain instances, be as vulnerable and empheral as many of the seedling resistances. A spectacular example was the ineffectiveness of the adult-plant resistance in Joss Cambier to a new pathotype identified in 1972 (Johnson and Taylor, 1972a).

Various breeding strategies aimed at the development

of stripe rust resistant wheats have been advocated. Dubin and Rajaram (1981) stated that the incorporation of rust resistance in the CIMMYT program was based on two strategies. The first involved the introduction of genetic diversity through pedigree breeding using double, or four-way, crosses. An assessment of the genetic basis of resistance was avoided as it was considered that "we must avoid danger of "paralysis by analysis"" (Dubin and Rajaram, 1981). However, the present studies suggest that spring wheats of CIMMYT origin have a relatively narrow genetic base for resistance to stripe rust, and this appears to be due to a fundamental, inadequate understanding of the lack of diversity of resistance in selected parental materials. The second strategy adopted by CIMMYT was the multilocational field testing of materials throughout the world. It was claimed that testing at a variety of sites provides disease response data for a wide range of pathogenic variants. However, there is no indication from published reports that the pathogenic variation at and between sites was systematically assessed. Torres and Rajaram (1983) identified four classes of materials based on examination of data from disease nurseries at diverse locations. They concluded that materials that were low or intermediate in disease response at all sites possessed "broad based resistance that is likely to be durable". Again, the present studies indicated that CIMMYT-derived wheats have relatively few genes for stripe rust resistance which can be and have been overcome by pathotypes with corresponding genes for virulence. In addition, McIntosh *et al.* (1983) described a situation of genetic vulnerability to stem rust in CIMMYT generated triticales which presumably had been developed on the basis of the above strategies. They concluded that stem rust resistance in these triticales was possibly based on as few as two genes.

Nevertheless, it is recognised that the strategy of multilocation testing has resulted in the development of wheats that are high yielding and widely adapted. The danger, however, is that the adoption of this germplasm may lead to a situation of global genetic vulnerability to the

cereal rust diseases.

Sharp (1983) concluded that "minor" genes conferred durable resistance to stripe rust in wheats grown in northwestern U.S.A. These genes were noted to be temperature-sensitive and additive in effect. Moreover, transgressive segregation to increased resistance occurred in some crosses. It was proposed that intercrosses between agronomically adapted wheats that were susceptible to stripe rust could result in commercial cultivars with potentially durable resistance. However, the value of such resistances in practical wheat breeding is still to be demonstrated.

Resistance to stripe rust is a major objective in the pedigree breeding system used at Plant Breeding Institute, Cambridge, England (Bingham, 1981a). The aim of this program was to select for adult-plant resistance which may require not more than a single application of fungicide in order to achieve maximum yield. Due to experiences with seedling resistances which, when present in single or multiple combinations, had failed to provide long term protection, seedling susceptible breeding lines were being selected (Bingham, 1981b). In addition, parental materials were chosen from among cultivars which had apparent durable resistance. Combinations of these parents with other materials which may carry specific seedling or specific adult-plant resistance genes, were tested in subsequent generations using pathotypes with appropriate virulence combinations. This method, which was originally described by Johnson (1978), increases the probability of recovering durable resistances. A stated feature of the Cambridge program was its reliance on fundamental investigations which, it was anticipated, would allow the future development of durable resistances without the need for fungicide support.

From the present studies, it was evident that stripe rust resistance under Australian conditions will need to be effective during most of the growing season. This should be a particular requirement of early sown

wheats as these cultivars, if susceptible, can become infected early, and then allow inoculum increase during winter, leading to severe spring epidemics. Mid-season and late sown wheats ideally should be resistant from the seedling stage, as they may be sown in areas where the disease is already established. Seedling protection could be achieved with fungicidal seed dressings, or by the use of seedling resistances. Although seedling resistances to stripe rust have not been durable, some authors caution against the abandonment of such genes (Person *et al.*, 1976; Macer, 1972; Riley, 1973). Johnson (1984) identified a number of instances in which durable resistance to disease was conferred by a single gene. Bingham (1981a) stated "the ideal strategy to control yellow rust might be to superimpose genetic factors for seedling resistance on a background of durable adult plant resistance." As there are only 16 catalogued genes for stripe rust resistance, the search for further genes, including seedling types, may be a worthwhile venture.

The use of recognised sources of durable resistance in Australian breeding programs may be a long-term objective as agronomic type, seed colour, grain quality and resistance to other diseases among these wheats are not suitable for local conditions. Nevertheless, some progress has been made in transferring adult-plant resistances from the North American wheat Brevor, and the French wheat Flinor, to a susceptible Australian genotype (Wellings, unpublished). The attempted exploitation of transgressive segregates with improved resistance from intercrosses of Australian wheats is also in progress (Smithson and McIntosh, unpublished). High levels of adult-plant resistance in cultivars Oxley and Cook are also being recommended for use in eastern Australian breeding programs.

The present studies have provided data on the epidemiology and pathogenic variation within *P. striiformis tritici* in Australia, and on the genetic basis of seedling resistances in Australian and exotic wheats. These results contribute towards a basis for the future development of wheats resistant to stripe rust under Australian conditions.

BIBLIOGRAPHY

ALLISON, C.C. and K. ISENBECK. 1930. Biologische Specialisierung von *Puccinia glumarum tritici* Eriksson and Henning. Phytopathologische Zeitschrift 2, 87-98.

ALLAN, R.E. and L.H. PURDY. 1967. Single gene control of stripe rust resistance in wheat. Plant Disease Reporter 51, 1041-1043.

ALLAN, R.E. and L.H. PURDY. 1970. Reaction of F2 seedlings of several crosses of susceptible and resistant wheat selections to *Puccinia striiformis* Phytopathology 60, 1368-1372.

ALLAN, R.E. and O.A. VOGEL. 1961. Stripe rust resistance of Suwon 92 and its relationship to several morphological characters in wheat. Plant Disease Reporter 45, 778-779.

ALLAN, R.E., O.A. VOGEL and L.H. PURDY. 1963. Influence of stripe rust upon yields and test weights of closely related lines of wheat. Crop Science 3, 564-565.

ALLAN, R.E., L.H. PURDY and O.A. VOGEL. 1966. Inheritance of seedling and adult reaction of wheat to stripe rust. Crop Science 6, 242-245.

ALLARD, R.W. 1956. Formulas and tables to facilitate the calculation of recombination values in heredity. Hilgardia 24, 235-277.

AHMAD, S.T., S. SINGH and D.P. MISRA. 1970. Additions to the wheat rust races in India. Races 14 and 38 of *P. striiformis* identified during 1965. Indian Phytopathology 22, 522-524.

AMITAI, Z.K., C.H. van SILFHOUT and A. GRAMA. 1985. An undescribed gene for resistance to *Puccinia striiformis* in *Triticum dicoccoides* sel. G-25. (unpublished manuscript).

ANIKSTER, Y. 1984. The *Formae Speciales*. In "The Cereal Rusts, Volume I". Eds. W.R. Bushnell and A.P. Roelfs. Academic Press. p.115-129.

ANONYMOUS. 1974. Maris Huntsman and yellow rust. Cereal Rusts Bulletin 2,46.

ANONYMOUS. 1975. Wheat Research. Australian Wheat Board 1,2.

ANONYMOUS. 1982. CIMMYT Review 1982. El Batan, Mexico, pp.130.

ANONYMOUS. 1984. CIMMYT Report on Wheat Improvement, 1981. pp.135.

ANONYMOUS. 1985. The Wheat Industry. In "Australian Agricultural Year Book, 1985", Strand Publishing. p.249-258.

ARTHUR, J.C. 1962. Manual of the Rusts in United States and Canada. Haffner Publishing Company. pp.186.

BAKER, E.P. 1966. Isolation of complementary genes conditioning crown rust resistance in the oat variety Bond. Euphytica 15,313-318.

BAKER, H.G. 1970. Plants and Civilization. MacMillan and Co. Ltd., London .pp.194.

BAKSHI, J.S. and R.N. SAWHNEY. 1965. Inheritance of resistance of La Prevision to three races of yellow rust. Indian Journal of Genetics and Plant Breeding 25, 227-230.

BARTOS, P., J. VALKOUN, J. KOSNER and V. SLOVENCIKOVA. 1973. On the genetics of rust resistance on the wheat cultivar Kavkaz. Cereal Rusts Bulletin 1, 27.

BAWDEN, M., J.G. MANNERS and R.H. PRIESTLEY. 1978. Occurrence of physiologic races of *Puccinia striiformis* in England and Wales, 1931-1933. Transactions of the British Mycological Society 70, 1-5.

BAYLES, R.A. and J.E. THOMAS. 1983. Yellow rust of barley. U.K. Cereal Pathogen Virulence Survey, 1983 Annual Report. p.53-56.

BEAVER, R.G. and R.L. POWELSON. 1969. A new race of stripe rust pathogenic on the wheat variety Moro, C.I.13740. Plant Disease Reporter 53, 91-93.

BERESFORD, R.M. 1982. Stripe rust (*Puccinia striiformis*), a new disease of wheat in New Zealand. Cereal Rusts Bulletin 10,35-41.

BEVER, W.M. 1934a. Effect of light on the development of the uredial stage of *Puccinia glumarum*. Phytopathology 24, 507-516.

BEVER, W.M. 1934b. Physiologic specialization in *Puccinia glumarum* in the United States. Phytopathology 24, 686-688.

BEVER, W.M. 1937. Influence of stripe rust on growth, water economy, and yield of wheat and barley. Journal of Agricultural Research 54, 375-385.

BIFFEN, R.H. 1905. Mendel's laws of inheritance and wheat breeding. Journal of Agricultural Sciences 1, 4-48.

BINGHAM, J. 1981b. The achievements of conventional plant breeding. Philosophical Transactions of the Royal Society of London B292, 441-455.

BINGHAM, J. 1981a. Breeding for disease resistance. p.13-14 in 'Strategies for the Control of Cereal Diseases.' J.F. Jenkyn and R.T. Plumb (eds.), Blackwell. pp.219.

BRENT, K. 1983. Protecting wheat against pests and diseases. In "Better British Wheat". Agricultural Research Council, June 1983. p.24-25.

BRIGGLE, L.W. and O.A. VOGEL. 1968. Breeding short-stature, disease resistant wheats in the United States. Euphytica Supplement No. 1, 107-130.

BRITTON, M.P. and G.B. CUMMINS. 1956. The reaction of species of *Poa* and other grasses to *Puccinia striiformis*. Plant Disease Reporter 40, 643-645.

BRÖNNIMANN, A. 1964. Yellow rust problems in Switzerland. Proceedings Cereal Rust Conference, Cambridge. p.71-81.

BROWN, J.F. and E.L. SHARP. 1969. Interactions of minor host genes for resistance to *Puccinia striiformis* with changing temperature regimes. Phytopathology 59, 999-1001.

BURDON, J.J., D.R. MARSHALL, N.H. LUIG and D.J.S. GOW. 1982. Isozyme studies on the origin and evolution of *Puccinia graminis f.sp. tritici* in Australia. Australian Journal of Biological Sciences 35, 231-238.

BURLEIGH, J.R. and J.W. HENDRIX. 1970. The winter biology of *Puccinia striiformis* West. in the Pacific Northwest. Washington Agricultural Experiment Station Technical Bulletin 65. pp.20.

CHAMBERLAIN, N.H., J.K. DOODSON and R. JOHNSON. 1971. The occurrence of two new physiologic races of *Puccinia striiformis* Westend. in Britain. Plant Pathology 20, 92-95.

CHESTER, K.S. 1946. "The Nature and Prevention of the Cereal Rusts as Exemplified in the Leaf Rust of Wheat". Chronica Botanica. pp.269.

COAKLEY, S.M. 1978. The effect of climate variability on stripe rust of wheat in the Pacific Northwest. Phytopathology 68, 207-212.

CUMMINS, G.B. 1971. "The Rust Fungi of Cereals, Grasses and Bamboos". Springer-Verlag. pp.570.

DAY, P.R. 1974. "Genetics of Host-Parasite Interaction". W.H. Freeman and Company, San Francisco. pp.238.

DOLING, D.A. 1967. Yellow rust of wheat. M.A.F.F. Advisory Leaflet 527. pp.4

DOLING, D.A. and J.K. DOODSON. 1968. The effect of yellow rust on the yield of spring and winter wheat. Transactions of the British Mycological Society 51, 427-434.

DOODSON, J.K., J.G. MANNERS and A. MYERS. 1964. Some effects of yellow rust (*Puccinia striiformis*) on the growth and yield of a spring wheat. Annals of Botany N.S.28, 459-472.

DOUSSINAULT, G., F. DOSBA and J. JAHIER. 1983. New results on the improvement of the level of resistance to eyespot in wheat. Proceedings 6th International Wheat Genetics Symposium . pp.193-198.

DUBIN, H.J. and S. RAJARAM. 1981. The Strategy of the International Maize and Wheat Improvement Centre (CIMMYT) for breeding disease resistant wheat: an international approach. p. 27-35 in "Strategies for the Control of Cereal Diseases". J.F. Jenkyn and R.T. Plumb (eds.), Blackwell. pp.219.

DUBIN, H.J. and R.W. STUBBS. 1985. Epidemic spread of barley stripe rust in South America. Plant Disease (in press).

DUTLU, C. 1984. The inheritance of resistance to stripe rust in some bread wheat cultivars. Proceedings of Fifth European and Mediterranean Cereal Rusts Conference. p.47-53.

EL-BEDEWY R. and G. RÖBBELEN. 1982. Chromosomal location and change of dominance of a gene for resistance against yellow rust, *Puccinia striiformis* West., in wheat, *Triticum aestivum* L. Zeitschrift für Pflanzenzüchtung 89, 145-157.

ELLINGBOE, A.H. 1975. Horizontal resistance: an artifact of experimental procedure? Australian Plant Pathology Newsletter 4, 44-46.

ERIKSSON, J. 1894. Ueber die Specialisierung des Parasitismus bei den Getreiderostpilzen. Ber. Devt. Bot. Ges. 12, 292-331.

ERIKSSON, J. and E. HENNING. 1894. Die Hauptresultate einer neuen Untersuchung über die Getreideroste. Zeitschrift Pflanzenzüchtung 4, 66-73,140-142, 197-203.257-262.

FANG, C.T. 1944. Physiologic specialization of *Puccinia glumarum* Erikss. and Henn. in China. Phytopathology 34, 1020-1024.

FARRER, W. 1889. The making and improvement of wheats for Australian conditions. Agricultural Gazette of New South Wales 9, 131-168,242-260.

FAVRET, E.A. and J. VALLEGA. 1953. Inheritance of resistance to Argentine races of *Puccinia glumarum* in the wheat "Chino 166". Phytopathology 43, 471 (abstract).

FLOR, H.H. 1942. Inheritance of pathogenicity in *Melampsora linii*. Phytopathology 32, 653-659.

FLOR, H.H. 1956. The complementary gene systems in flax and flax rust. Advances in Genetics 8, 29-54.

FLOR, H.H. 1971. Current status of the gene-for-gene concept. Annual Review of Phytopathology 9, 275-296.

FUCHS, E. 1960. Physiologic races in yellow rust (*Puccinia glumarum* (Schm.) Erikss. et Henn.) of wheat. Proceedings of the Second European Yellow Rust Conference, Wageningen, The Netherlands. p.9-21.

GANDHI, S.M. 1971. Inheritance of adult-plant resistance to stripe rust in wheat. Indian Journal of Genetics and Plant Breeding 31, 256-274.

GASSNER, G. and W. STRAIB. 1932. Die Bestimmung der biologischen Rassen des Weizengelbrostes (*Puccinia glumarum f.sp. tritici* (Schmidt.) Erikss. und Henn.). Arbeiten der biologischen Reichsanstalt für Land und Forstwirtschaft, Berlin 20, 141-163.

GILL, K.S., S.S. AUJILA, Y.R. SHARMA, G.S. NANDA and C. SINGH. 1978. Studies on multilines in wheat (*T. aestivum*) 7. Seedling and adult plant reaction of components of Kalyansona and PV18 multilines against yellow rust. Cereal Research Communications 6, 167-173.

GODDARD, M.V. 1976. The production of a new race 105 E137 of *Puccinia striiformis* in glasshouse experiments. Transactions of the Britich Mycological Society 67, 395-398.

GRAMA, A. and Z.K. GERECHTER-AMITAI. 1974. Inheritance of resistances to stripe rust (*Puccinia striiformis*) in crosses between wild emmer (*Triticum dicoccoides*) and cultivated tetraploid and hexaploid wheats II *Triticum aesitivum*. Euphytica 23, 393-398.

GRAMA, A., Z.K. GERECHTER-AMITAI and A. BLUM. 1983. Wild emmer as a donor of genes for resistance to stripe rust and for high protein content. Proceedings Sixth International Wheat Genetics Symposium, Japan. p.187-192.

GRAMA, A., Z.K. GERECHTER-AMITAI and C.H. van SILFHOUT. 1984. Additive gene action for resistance to *Puccinia striiformis f.sp. tritici* in *Triticum dicoccoides*. Euphytica 33, 281-287.

GERECHTER-AMITAI, Z.K. and A. GRAMA. 1974. Inheritance of resistance to stripe rust (*Puccinia striiformis*) in crosses between wild emmer (*Triticum dicoccoides*) and cultivated tetraploid and hexaploid wheats I *Triticum durum*. Euphytica 23, 387-392.

GERECHTER-AMITAI, Z.K. and R.W. STUBBS. 1970. A valuable source of yellow rust resistance in Israeli populations of wild emmer, *Triticum disoccoides* Koern. Euphytica 19, 12-21.

GERECHTER-AMITAI, Z.K., E.L. SHARP and M. REINHOLD. 1984. Temperature-sensitive genes for resistance to *Puccinia striiformis* in *Triticum dicoccoides*. Euphytica 33, 665-672.

GHOSH, S., S.M. SIKKA and M.Y. RAO. 1958. Inheritance studies in wheat.IV Inheritance of rust resistance and other characters. Indian Journal of Genetics and Plant Breeding 18, 142-162.

GREEN, G.J. 1965. Stem rust of wheat, rye and barley in Canada in 1964. Canadian Plant Disease Survey 45, 23-29.

GREEN, G.J., and A.B. CAMPBELL. 1979. Wheat cultivars resistant to *Puccinia graminis tritici* in western Canada: their development, performance and economic value. Canadian Journal Plant Pathology 1,3-11.

GREWAL, A.S., A.S. RANDHAWA and A.S. MINHAS. 1976. Inheritance of stripe rust resistance in wheat (*Triticum aestivum* L.). Punjab Agricultural University, Journal of Research 13, 124-126.

HANSON, W.D. 1959. Minimum family sizes for the planning of genetic experiments. Agronomy Journal 51, 711-715.

HASSAN, S.F., M. HUSSAIN and S.A. RIZVI. 1979. Wheat disease situation in Pakistan. Proceedings National Seminar on Wheat Research and Production, Islamabad. pp.231-234.

HASSEBRAUK, K. 1965. Nomenklatur, geograpgische Verbreitung und Wirstbereich des Gelbrostes, *Puccinia striiformis* West. Mitteilungen aus der Biologischen Bundesanstalt für Land-und Forstwirtschaft, Berlin-Dahlem 116, pp.75.

HAWTHORN, W.M. 1984. "Genetic Analyses of Leaf Rust Resistance in Wheat". Ph.D. Thesis, University of Sydney pp.299.

HELM, J.H. and R.E. ALLAN. 1970. A genetic study of reactions to stripe rust and flag smut in hexaploid wheat. Agronomy Abstracts 11-12.

HENDRIX, J.W., J.R. BURLEIGH and J.-C. TU. 1965. Oversummering of stripe rust at high elevations in the Pacific Northwest-1963. Plant Disease Reporter 49, 275-278.

HENRIKSEN, G.B. and W.K. POPE. 1971. Additive resistance to stripe rust in wheat. Crop Science 11, 825-827.

HERMANSEN, J.E. and CHR. STAPEL. 1973. Notes on the yellow rust epiphytotic in Denmark in 1972. Cereal Rusts Bulletin 1, 5-8.

HUMPHREY, H.B. and R.O. CROMWELL. 1930. Stripe rust, *Puccinia glumarum*, on wheat in Argentina. Phytopathology 20, 981-986.

HUMPHREY, H.B. and C.W. HUNGERFORD. 1924. Stripe rust (*Puccinia glumarum*) of cereals and grasses in the United States. Journal of Agricultural Research, 29, 209-227.

HUMPHREY, H.B., C.W. HUNGERFORD and A.G. JOHNSON. 1924. Stripe rust (*Puccinia glumarum*) of cereals and grasses in the United States. Journal of Agricultural Research 29, 209-227.

HUNGERFORD, C.W. and C.E. OWENS. 1923. Specialized varieties of *Puccinia glumarum* and hosts for variety *tritici*. Journal of Agricultural Research 25, 363-413.

HUSSAIN, M., S.F. HASSAN and M.A.S. KIRMANI. 1980. Virulences in *Puccinia recondita* Rob. ex. Desm. f.sp. *tritici* in Pakistan during 1978 and 1979. Fifth European & Mediterranean Cereal Rusts Conference, Bari-Rome, Italy. p.179-184.

HYLANDER, N., I. JORSTAD and J.A. NANNFELDT. 1953. Enumeratio uredinearum Scandinavicarum Op. bot. (Bot. Notiser. Suppl.) 1,1-102.

JOHNSON, R. 1978. Practical breeding for durable resistance to rust diseases in self-pollinating cereals. Euphytica 27, 529-540.

JOHNSON, R. 1981a. Durable resistance: definition of, genetic control and attainment in plant breeding. Phytopathology 71, 567-568.

JOHNSON, R. 1981b. Durable disease resistance. In "Strategies for the Control of Cereal Diseases". Eds. J.F. Jenkyn and R.T. Plumb. Blackwell. p.55-63.

JOHNSON, R. 1983a. Breeding for durable resistance to yellow rust in wheat. In "Better British Wheat". Agricultural Research Council June, 1983, p.14-15.

JOHNSON, R. 1983b. Genetic background of durable resistance. In "Durable Resistance in Crops". Eds. F. Lamberti, J.M. Waller and N.A. Van der Graaf. Life Sciences 55, Plenum Press. p.5-26.

JOHNSON, R. 1983c. Pathogenic variability: strain survey methods, purpose and effectiveness - United Kingdom. In "The Current Status of Stripe Rust Research". Proceedings of a National Workshop, Horsham, Australia. ed. L. O'Brien. p.36-40.

JOHNSON, R. 1984. A critical analysis of durable resistance. Annual Review of Phytopathology 22, 309-330.

JOHNSON, V.A. and H.L. BEEMER. 1977. Wheat in the people's republic of China. National Academy of Sciences, Washington, pp.190.

JOHNSON, R. and D.E. BOWYER. 1974. A rapid method of measuring production of yellow rust spores on single seedlings to assess differential interactions of wheat cultivars with *Puccinia striiformis*. Annals of Applied Biology, 77, 251-258.

JOHNSON, R. and P.L. DYCK. 1984. Resistance to yellow rust in *Triticum spelta* var. *album* and bread cultivars Thatcher and Lee. Proceeding of Sixth European and Mediterranean Cereal Rusts Conference. p.71-74.

JOHNSON, R. and C.N. LAW. 1973. Cytogenetic studies on the resistance of the wheat variety Bersée to *Puccinia striiformis*. Cereal Rusts Bulletin 1, 38-43.

JOHNSON, R. and C.N. LAW. 1975. Genetic control of durable resistance to yellow rust (*Puccinia striiformis*) in the wheat cultivar Hybride de Bersée. Annals of Applied Biology 81, 385-391.

JOHNSON, T. and M. NEWTON. 1946. Specialization, hybridization, and mutation in the cereal rusts. The Botanical Review, 12, 337-392.

JOHNSON, R. and A.J. TAYLOR. 1972a. Isolates of *Puccinia striiformis* collected in England from the wheat varieties Maris Beacon and Joss Cambier. Nature 238, 105-106.

JOHNSON, R. and A.J. TAYLOR. 1972b. Yellow rust of wheat. Plant Breeding Institute, Cambridge, Annual Report 1972. p.136-139.

JOHNSON, R. and A.J. TAYLOR. 1975. Yellow rust of wheat. Plant Breeding Institute, Cambridge, Annual Report. p.126-128.

JOHNSON, R. and A.J. TAYLOR. 1976a. Spore yield of pathogens in investigations of the race-specificity of host resistance. Annual Review Phytopathology 14, 91-119.

JOHNSON, R. and A.J. TAYLOR. 1976b. Yellow rust of wheat. Plant Breeding Institute, Cambridge, Annual Report. p.106-109.

JOHNSON, R. and A.J. TAYLOR. 1977. Yellow rust of wheat. Plant Breeding Institute, Cambridge, Annual Report. p.119-122.

JOHNSON, R. and A.J. TAYLOR. 1980. Pathogenic variation in *Puccinia striiformis* in relation to the durability of yellow rust resistance in wheat. Symposium, "Variation in Plant Pathogens". Annals of Applied Biology 94, 283-286.

JOHNSON, T., G.J. GREEN AND D.J. SAMBORSKI. 1967. The world situation of the cereal rusts. Annual Review of Phytopathology 5, 183-200.

JOHNSON, R., R.W. STUBBS, E. FUCHS and N.H. CHAMBERLAIN. 1972. Nomenclature for physiologic races of *Puccinia striiformis* infecting wheat. Transactions of the British Mycological Society 58, 475-480.

JOHNSON, R., A.J. TAYLOR and G.M.B. SMITH. 1975. Samples of *Puccinia striiformis* from yellow rust infections on Maris Huntsman in 1974. Cereal Rusts Bulletin 3, 4-6.

JOHNSON, R., A.J. TAYLOR and G.M.B. SMITH. 1983. Yellow rust of wheat. Plant Breeding Institute, Cambridge, Annual Report 1983. pp.82-85.

JOHNSON, R., A.J. TAYLOR and G.M.B. SMITH. 1984. Yellow rust of wheat. Plant Breeding Institute, Cambridge. Annual Report 1984. p.85-88.

JÖNSSON, J.O. 1978. Race non-specific resistance to *Puccinia striiformis f.sp. tritici*. Agri. Hortique Genetica 36,13-22.

JOSHI, L.M., L.B. GOEL and V.C. SINHA. 1976. Role of the Western Himalayas in the annual recurrence of yellow rust in northern India. Cereal Rusts Bulletin 4,27-30.

JOSHI, L.M., S. NAGARAJAN and K.D. SRIVASTAVA. 1977. Epidemiology of brown and yellow rust of wheat in north India I. Place and time of appearance and spread. Phytopathologische Zeitschrift 90, 116-122.

JOSHI, L.M., D.V. SINGH and K.D. SRIVASTAVA. 1985. Status of rusts and smuts in India. Rachis 4, 10-16.

KAIDASH, A.S. and V.T. BESSMEL'TSEV. 1976. Potential losses of grain yield of winter wheat caused by rust, *Puccinia striiformis*. Mikologiya i Fitopatalogiya 10, 509-510.

KEMA, G.H.J., C.H. van SILFHOUT and R.W. STUBBS. 1984. Analysis of resistance to yellow rust (*Puccinia striiformis* West. f.sp. *tritici*) in Dutch wheats. VI European and Mediterranean Cereal Rusts Conference. p.75-79.

KIRMANI, M.A.S., S.S.A. RIZVI and R.W. STUBBS. 1984. Postulated genotypes for stripe rust resistance in wheat cultivars of Pakistan. VI European and Mediterranean Cereal Rusts Conference. p.95-98.

KOCHHAR, S., K.S. GILL and G.S. NANDI. 1983. Genetic analysis of resistance in some varieties of bread wheats (*Triticum aestivum*) to three races of yellow rust (*Puccinia striiformis*). Wheat Information Service 56, 237-240.

KRUPINSKY, J.M. and E.L. SHARP. 1978. Additive resistance in wheat to *Puccinia striiformis*. Phytopathology 68, 1795-1799.

KRUPINSKY, J.M. and E.L. SHARP. 1979. Reselection for improved resistance of wheat to stripe rust. Phytopathology 69, 400-404.

LABRUM, K.E. 1980. The location of *Yr2* and *Yr6*, genes conferring resistance to yellow rust. Proceedings of Fifth European and Mediterranean Cereal Rusts Conference, Bari, Italy. p.41-45.

LARGE, E.C. 1940. "The Advance of the Fungi". Jonathan Cape, London. pp.488.

LATCH, G.C.M. 1976. Stripe rust, *Puccinia striiformis f.sp. dactylidis* , on *Dactylis glomerata* in New Zealand. New Zealand Journal of Agricultural Research 19, 535-536.

LEONARD, W.H. and J.H. MARTIN. 1967. "Cereal Crops". Macmillan Company, New York. pp.824.

LEWELLEN, R.T. and E.L. SHARP. 1968. Inheritance of minor gene combinations in wheat to *Puccinia striiformis* at two temperature profiles. Canadian Journal of Botany 46, 21-26.

LEWELLEN, R.T., E.L. SHARP and E.R. HEHN. 1967. Major and minor genes in wheat for resistance to *Puccinia striiformis* and their responses to temperature changes. Canadian Journal of Botany 45, 2155-2172.

LINE, R.F. 1976. Factors contributing to an epidemic of stripe rust on wheat in the Sacramento Valley of California in 1974. Plant Disease Reporter 60, 312-316.

LINE, R.F. 1980a. Specific and non-specific resistance to *Puccinia striiformis* and *Puccinia recondita* in western United States. Proceedings Fifth Meeting European and Mediterranean Cereal Rusts Foundation, Italy. p.91-92.

LINE, R.F. 1980b. Specific and non-specific resistance to stripe rust and leaf rust of wheat. Third International Wheat Conference Proceedings, Madrid. p.495-499.

LINE, R.F. 1983a. Chemical control of stripe rust and assessment of losses caused by rust in the United States of America. In "The Current Status of Stripe Rust Research, Proceedings of a National Workshop", Horsham, Australia. Ed. L. O'Brien. p.24.

LINE, R.F. 1983b. Pathogenicity and evolution of *Puccinia striiformis* in the United States of America. In "The Current Status of Stripe Rust Research", Proceedings of a National Workshop, Horsham, Australia. Ed. L. O'Brien. p.31-35.

LINE, R.F. 1983c. Resistance to *Puccinia striiformis* in United States of America. In "The Current Status of Stripe Rust Research", Proceedings of a National Workshop, Horsham, Australia. Ed. L. O'Brien. p.48-49.

LINE, R.F., E.L. SHARP AND R.L. POWELSON. 1970. A system for differentiating races of *Puccinia striiformis* in the United States. Plant Disease Reporter 54, 992-994.

LINE, R.F., C.F. KONZAK and R.E. ALLAN. 1974. Evaluating resistance to *Puccinia striiformis* in wheat. In "Induced Mutations for Resistance in Crop Plants", International Atomic Energy Agency. p.125-132.

LINE, R.F., R.E. ALLAN and C.F. KONZAK. 1976. Identifying and utilizing resistance to *Puccinia striiformis* in wheat. "Induced Mutations for Disease Resistance in Crops", International Atomic Energy Agency. p.151-158.

LINE, R.F., A. QAYOUM and G. MILUS. 1983. Durable resistance to stripe rust of wheat. Abstract 812, Fourth International Congress of Plant Pathology, Melbourne, Australia.

LITTLE, R. and J.G. MANNERS. 1969a. Somatic recombination in yellow rust (*Puccinia striiformis*) I. The production and possible origin of two new physiological races. Transactions of the British Mycological Society 53, 251-258.

LITTLE, R. and J.G. MANNERS. 1969b. Somatic recombination in yellow rust (*Puccinia striiformis*) II. Germ tube fusions, nuclear number and nuclear size. Transactions of the British Mycological Society 53, 259-267.

LOEGERING, W.Q. 1966. The relationship between host and pathogen in stem rust of wheat. Proceedings Second International Wheat Genetics Symposium. Hereditas Supplement 2, 167-177.

LUIG, N.H. 1977. The establishment and success of exotic strains of *Puccinia graminis tritici* in Australia. Proceedings of Ecological Society of Australia 10, 89-96.

LUIG, N.H. 1985. Epidemiology in Australia and New Zealand. In "The Cereal Rusts Volume II". Eds. A.P. Roelfs and W.R. Bushnell. Academic Press. p.301-328.

LUIG, N.H. and I.A. WATSON. 1970. The effect of complex genetic resistance in wheat on the variability of *Puccinia graminis f.sp. tritici*. Proceedings of the Linnean Society, N.S.W. 95, 22-45.

LUIG, N.H., and I.A. WATSON. 1972. The role of wild and cultivated grasses in the hybridization of formae speciales of *Puccinia graminis*. Australian Journal of Biological Sciences 25, 235-242.

LUIG, N.H. and I.A. WATSON. 1977. The role of barley, rye and grasses in the 1973-74 wheat stem rust epiphytotic in southern and eastern Australia. Proceedings of the Linnean Society of N.S.W. 101, 65-76.

LUIG, N.H., J.J. BURDON and W.M. HAWTHORN. 1985. An exotic strain of *Puccinia recondita tritici* in New Zealand . Canadian Journal of Plant Pathology 7, 173-176.

LUPTON, F.G.H. 1982. Recent advances in cereal breeding. Netherlands Journal of Agricultural Science 30, 11-24.

LUPTON, F.G.H. and R.C.F. MACER. 1962. Inheritance of resistance to yellow rust (*Puccinia glumarum* Erikss. and Henn.) in seven varieties of wheat. Transactions British Mycological Society 45, 21-45.

MACER, R.C.F. 1966. The formal and monosomic genetic analysis of stripe rust (*Puccinia striiformis*) resistance in wheat. Proceedings Second International Wheat Genetics Symposium, Sweden 1963. Heriditas Supplement 2, 127-143.

MACER, R.C.F. 1972. The resistance of cereals to yellow rust and its exploitation by plant breeding. Proceedings of Royal Society of London B.181, 281-301.

MACER, R.C.F. 1975. Plant Pathology in a changing world. Transactions of the British Mycological Society 65, 351-374.

MAINS, E.B. 1933. Studies concerning heteroecious rusts. Mycologia 25, 407-417.

MANNERS, J.G. 1950. Studies on the physiologic specialization of yellow rust (*Puccinia glumarum* (Schm.) Erikss. & Henn.) in Great Britain. The Annals of Applied Biology 37, 187-214.

MANNERS, J.G. 1960. *Puccinia striiformis* Westend. var *dactylidis* var. nov. Transactions of British Mycological Society 43, 65-68.

MARES, D.J. and S. COUSEN. 1977. The interaction of yellow rust (*Puccinia striiformis*) with winter wheat cultivars showing adult plant resistance: macroscopic and microscopic events associated with the resistant reaction. Physiological Plant Pathology 10, 257-274.

MARSHALL, D.R. 1984. The future of disease control. Australian Institute of Agricultural Science Occasional Publication 15, 21-27.

MARTIN, N.M. and J.W. HENDRIX. 1967. Comparison of root systems produced by healthy and stripe rust-inoculated wheat in mist-water and sand-culture. Plant Disease Reporter 51, 1074-1076.

MARTIN, D.J. and B.G. STEWART. 1984. Dough Stickiness and mixing properties of wheat cultivars derived from rye. Proceedings of Fourth Assembly of the Wheat Breeding Society of Australia, Toowoomba. p.139.

McINTOSH, R.A. 1976. Genetics of wheat and wheat rusts since Farrer. Journal of Australian Institute of Agricultural Science 42, 203-216.

McINTOSH, R.A. 1980. Report to Wheat Industry Research Council. pp.9.

McINTOSH, R.A. 1983. A catalogue of gene symbols for wheat (1983 edition). Proceedings Sixth International Wheat Genetics Symposium, Kyoto, Japan. p.1197-1254.

McINTOSH, R.A. and I.A. WATSON. 1982. Genetics of host-pathogen interactions in rusts. In "The Rust Fungi". Eds. K.J. Scott and A.K. Chakravorty, Academic Press. p.121-149.

McINTOSH, R.A. and C.R. WELLINGS. 1986. Wheat rust resistance - the continuing challenge. The Daniel McAlpine Memorial Lecture. Australasian Plant Pathology 15 (in press).

McINTOSH, R.A., N.H. LUIG, R. JOHNSON and R.A. HARE. 1981. Cytogenetical studies in wheat. XI. *Sr9g* for reaction to *Puccinia graminis tritici*. Zeitschrift für Pflanzenzüchtung 87, 274-289.

McINTOSH, R.A., T.E. MILLER and V. CHAPMAN. 1982. Cytogenetical studies in wheat XII. *Lr28* for resistance to *Puccinia recondita* and *Sr34* for resistance to *P. graminis tritici*. Zeitschrift für Pflanzenzüchtung 89, 295-306.

McINTOSH, R.A., N.H. LUIG, D.L. MILNE and J. CUSICK. 1983. Vulnerability of triticales to wheat stem rust. Canadian Journal of Plant Pathology 5, 61-69.

MERKER, A. 1982. "Veery" - a CIMMYT spring wheat with the 1B/1R chromosome translocation. Cereal Research Communications 10, 105-106.

METZGER, R.J. and B.A. SILBAUGH. 1970. Inheritance of resistance to stripe rust and its association with brown glume colour in *Triticum aestivum* L., 'PI 178383'. Crop Science 10, 567-568.

MILUS, G. and R.F. LINE. 1984a. Heritability of durable, adult-plant, temperature sensitive resistance to *Puccinia striiformis* in Pacific Northwest Wheats. (Abstract) Phytopathology 74, 794.

MILUS, G. and R.E. LINE. 1984b. Number of genes controlling resistance to stripe rust in three Pacific Northwest wheats. (Abstract) Phytopathology 74, 1140.

MINHAS, A.S. and S. SINGH. 1973. Inheritance of stripe rust resistance in intervarietal crosses of *Triticum aestivum*. Madras Agricultural Journal 60, 45-48.

MULDER, J.L. and C. BOOTH. 1971. C.M.I. Descriptions of Pathogenic Fungi and Bacteria, No. 291 " *Puccinia striiformis* ". Commonwealth Mycological Institute, Kew, England.

MUNDY, E.J. 1973. The effect of yellow rust and its control on the yield of Joss Cambier winter wheat. Plant Pathology 22, 171-176.

MURRAY, G.M., P. DUNSTAN and M. MAY. 1984. Effect of stripe rust on yields of wheat cultivars in southern New South Wales in 1983. New South Wales Department of Agriculture, Biology Branch Plant Disease Survey (1983-84). p.16-17.

NAGARAJAN, S. 1983. Yellow rust (*P. striiformis*) programme. Annual Report, Indian Agricultural Research Institute Regional Station, Flowerdale, Simla. p.36-51.

NAGARAJAN, S., P. BAHADUR and S.K. NAYAR. 1984. Occurrence of a new virulence, 47S102 of *Puccinia striiformis* West., in India during crop year, 1982. Cereal Rusts Bulletin 12, 28-29.

NAMBISAN, P.N.N. and S.P. KHOLI. 1961. Inheritance of seedling resistance to races 13 and H of *Puccinia glumarum* (Schm.) Erikss. and Henn. in crosses of *Triticum aestivum*. Indian Journal of Genetics and Plant Breeding 21, 15-22.

O'BRIEN, L., J.S. BROWN, R.M. YOUNG and I. PASCOE. 1980. Occurrence and distribution of wheat stripe rust in Victoria and susceptibility of commercial wheat cultivars. Australasian Plant Pathology 9,14.

OMAR, A.M., A.K.A. SELIM, S.H. HASSANEIN and O.H.S. KHALIL. 1970. Inheritance of mature plant reaction to stripe rust of wheat in U.A.R. United Arab Republic Journal of Botany 13, 131-142.

PAICHADZE, L.V. 1974. Recovery, development and survival of wheat yellow rust in Georgia. Soobshchenyia Akademii Nauk Gruzinskoi S.S.R. 76, 173-176.

PAL, B.P., S.M. SIKKA and M.V. RAO. 1956. Inheritance studies in wheat. Indian Journal of Genetics and Plant Breeding 16, 32-46.

PARLEVLIET, J.E. 1979. Components of resistance that reduce the rate of epidemic development. Annual Review of Phytopathology 17, 203-222.

PERSON, C., J.V. GROTH and O.M. MYLYK. 1976. Genetic change in host-parasite populations. Annual Review Phytopathology 14, 177-188.

PERWAIZ, M.S. and R. JOHNSON. 1985. Genes for resistance to yellow rust in seedlings of wheat cultivars from Pakistan tested with British isolates of *Puccinia striiformis*. Unpublished manuscript, pp.10.

PETERSON, R.F., A.B. CAMPBELL and A.E. HANNAH. 1948. A diagramatic scale for estimating rust intensity on leaves and stems of cereals. Canadian Journal of Research C. 26, 496-500.

POPE, W.K. 1968. Interaction of minor genes for resistance to stripe rust in wheat. Proceedings Third International Wheat Genetics Symposium, Canberra, Australia. p.251-257.

PRIESTLEY, R.H. 1978. Detection of increased virulence in populations of wheat yellow rust. In "Plant Disease Epidemiology". Eds. P.R. Scott and A. Bainbridge, Oxford, U.K. p.63-70.

PRIESTLY, R.H. and D.A. DOLING. 1974. Aggressiveness of *Puccinia striiformis* isolates on wheat cultivars. Transactions of the British Mycological Society 63, 549-557.

PRIESTLEY, R.H. and D.A. DOLING. 1976. A seedling method for measuring the susceptibility of wheat cultivars to *Puccinia striiformis*. Annals of Applied Biology 83, 199-206.

PRIESTLEY, R.H., J. SMITH, J.K. DOODSON and N.H. CHAMBERLAIN. 1974. Changes in the frequency of occurrence of physiologic races of *Puccinia striiformis* on wheat in the U.K. Cereal Rusts Bulletin 2,12-15.

PRIESTLEY, R.H., J. SMITH and J.K. DOODSON. 1975. Identification of eight isolates of *Puccinia striiformis* collected from Maris Huntsman in the U.K., 1974. Cereal Rusts Bulletin 3,1-3.

PRIESTLEY, R.H., R.A. BAYLES and J.E. THOMAS. 1984a. Identification of specific resistances against *Puccinia striiformis* (yellow rust) in winter wheat varieties I. Establishment of a set of type varieties for adult plant tests. Journal of National Institute of Agricultural Botany 16, 469-476.

PRIESTLEY, R.H., R.A. BAYLES and J. RYALL. 1984b. Identification of specific resistances against *Puccinia striiformis* (yellow rust) in winter wheat varieties II. Use of cluster analysis. Journal of National Institute of Agricultural Botany 16, 477-485.

PURDY, L.H. and R.E. ALLAN. 1963. Seedling and mature plant reaction of wheat to stripe rust. Plant Disease Reporter 47, 797-799.

PURDY, L.H. and R.E. ALLAN. 1966. A stripe rust race pathogenic to 'Suwon 92' wheat. Plant Disease Reporter 50, 205-207.

PURDY, L.H., W.Q. LOEGERING, C.F. KONZAK, C.S. PETERSON and R.E. ALLEN. 1968. A proposed standard method for illustrating pedigrees of small grain varieties. Crop Science 8, 405 - 406.

QUALSET, C.O., H.E. VOGT and N.E. BORLAUG. 1984. Registration of Anza wheat. Crop Science 24, 827-828.

RAO, M.V., V.S. MATHUR, S.G. AHIRE and P.N. NARULA. 1963. Inheritance studies in wheat.XVIII Inheritance of field reaction to yellow rust and other characters of an Ethiopian durum wheat. Indian Journal of Genetics and Plant Breeding 23, 19-24.

RAPILLY, F. 1979. Yellow rust epidemiology. Annual Review of Phytopathology 17, 59-73.

REES, R.G. 1972. *Agropyron scabrum* and its role in the epidemiology of *Puccinia graminis* in north-eastern Australia. Australian Journal of Agricultural Research 23, 789-798.

REINHOLD, M., E.L. SHARP and Z.K. GERECHTER-AMITAI. 1983. Transfer of additive "minor-effect" genes for resistance to *Puccinia striiformis* from *Triticum dicoccoides* into *Triticum durum* and *Triticum aestivum*. Canadian Journal of Botany 61, 2702-2708.

RILEY, R. 1973. Genetic changes in hosts and the significance of disease. Annals of Applied Biology 75, 128-132.

RILEY, R., V. CHAPMAN and R.C. MACER. 1966. The homoeology of an *Aegilops* chromosome causing stripe rust resistance. Canadian Journal of Genetics and Cytology 8, 616-630.

RILEY, R., V. CHAPMAN and R. JOHNSON. 1968a. Introduction of yellow rust resistance of *Aeigilops comosa* into wheat by genetically induced homoeologous recombination. Nature 217, 383-384.

RILEY, R., V. CHAPMAN and R. JOHNSON. 1968b. The incorporation of alien disease resistance in wheat by genetic interference with the regulation of meiotic chromosome synapsis. Genetic Research, Cambridge 12, 199-219.

RÖBBELEN, G. and E.L. SHARP. 1978. Mode of inheritance, interaction and application of genes conditioning resistance to yellow rust. Advances in Plant Breeding 9, Verlag Paul Parey. pp.88.

ROBINSON, R.A. 1969. Disease resistance terminology. Review of Applied Mycology 58, 593-606.

ROELFS, A.P. 1978. Estimated losses caused by rust in small grain cereals in the United States - 1918-76. U.S. Department of Agriculture, Miscellaneous Publication Number 1363, pp.85.

RUDORF, W. 1929. Beiträge zur Immunitätszüchtung gegen *Puccinia glumarum tritici* (Streifenrost des Weizens). Phytopathologische Zeitschrift 1, 465-526.

RUSSELL, G.E. 1978. Plant Breeding for Pest and Disease Resistance. Butterworths pp.485.

SANDHU, T.S. and G. SINGH. 1970. Inheritance of field reaction to yellow and brown rusts in crosses of common wheat. Punjab Agricultural University, Journal of Research 7, 5-9.

SANDHU, B.S., A.S. KHEHRA and J.S. CHOHAN. 1977. Rust resistance in Indian wheat varieties. Indian Journal Agricultural Science 47, 542-545.

SAVILLE, D.B.O. 1979. Fungi as aids in higher plant classification. The Botanical Review 45, 377-503.

SAVILLE, D.B.O. 1984. Taxonomy of the cereal rust fungi. In "The Cereal Rusts Volume I". Ed. W.R. Bushnell and A.P. Roelfs, Academic Press. p.79-112.

SAWHNEY, R.N. and J.K. LUTHRA. 1970. Identification of new genes for resistance and the isolation of differential hosts for Indian races of stripe rust. Indian Journal of Genetics and Plant Breeding 30, 491-496.

SCHEINPFLUG, H., V. PAUL and P. KRAUS. 1978. Studies on the mode of action of Bayleton ® against cereal disease. Pflanzenschutz-Nachrichten Bayer 31, 101-115.

SHANER, G. and R.L. POWELSON. 1973. The oversummering and dispersal of inoculum of *Puccinia striiformis* in Oregon. Phytopathology 63,13-17.

SHARMA, S.K., S. SINGH and L.B. GOEL. 1973. Note on a new record of 'Sonalika'-infecting race of yellow-rust in India and sources of its resistance. Indian Journal of Agricultural Science 43, 964-965.

SHARP, E.L. 1965. Prepenetration and postpenetration environment and development of *Puccinia striiformis* on wheat. Phytopathology 55, 198-203.

SHARP, E.L. 1983. Experience of using durable resistance in the U.S.A. p.385-396 in "Durable Resistance in Crops". F. Lamberti, J.M. Waller and N.A. van der Graff (eds.), Plenum Press, pp.454.

SHARP, E.L. and R.B. VOLIN. 1970. Additive genes in wheat conditioning resistance to stripe rust. Phytopathology 60, 1146-1147.

SIKKA, S.M., M.V. RAO and M. AHLUWALIA. 1960. Inheritance in wheat X Inheritance of field reaction to rusts and other characters. Indian Journal of Agricultural Science 30, 223-232.

SINGH, R.P. 1983. Genetics of rust resistance in wheat. Ph.D. thesis, University of Sydney. pp.224.

SINGH, R.P. and R.A. McINTOSH. 1984a. Complementary genes for reaction to *Puccinia recondita tritici* in *triticum aestivum* I Genetic and linkage studies. Canadian Journal of Genetics and Cytology 26, 723-735.

SINGH, R.P., and R.A. McINTOSH. 1984b. Complementary genes for reaction to *Puccinia recondita tritici* in *Triticum aesitivum* II Cytogenetic studies. Canadian Journal of Genetics and Cytology 26, 736-742.

SINGH, R.P. and R.A. McINTOSH. 1986. Genetics of resistance to *Puccinia graminis tritici* and *Puccinia recondita tritici* in Kenya Plume Wheat. Euphytica 35 (in press).

SINGH, H.B. and G.S. SETHI. 1978. Field reaction of wheats to yellow and brown rusts. Indian Journal of Genetics and Plant Breeding 38, 163-167.

SINGH, S., L.B. GOEL, S.K. SHARMA, S.K. NAYAR, S.C. CHATTERJEE, V.C. SINHA and P. BAHADUR. 1978. Prevalence and distribution of physiologic races of wheat rusts in India during 1969-74. Indian Journal of Agricultural Science 48, 1-7.

SKOVMAND, B. and S. RAJARAM. 1978. Semi dwarf bread wheats. Names, parentage, pedigrees, origin. Information Bulletin 34, CIMMYT, Mexico. pp.16.

SLOVENCIKOVA, V. 1974. The variable stripe rust reaction of the wheat cultivar Chambord. Cereal Rusts Bulletin 2,25-27.

SLOVENCIKOVA, V. 1980. Genetics of field resistance of the winter wheats Benno, Zorba, Orlando and Saladin to race 232 E137 ("Clement" race) of *Puccinia striiformis*. Cereal Rusts Bulletin 8, 12-16.

STAKMAN, E.C. and J.G. HARRAR. 1957. Principles of Plant Pathology. Ronald Press. pp.581.

STAKMAN, E.C. and M.N. LEVINE. 1922. The determination of biologic forms of *Puccinia graminis* on *Triticum spp.* Technical Bulletin of the Minnesota Agricultural Experimental Station 8, 1-10.

STAKMAN, E.C. and F.J. PIEMEISEL. 1917. A new strain of *Puccinia graminis*. Phytopathology 7, 73-80.

STAKMAN, E.C., D.M. STEWART and W.Q. LOEGERING. 1962. Identification of physiologic races of *Puccinia graminis var. tritici*. U.S. Department of Agriculture, Agricultural Research Service. pp.53.

STEEL, R.G. and J.H. TORRIE. 1980. Principles and Procedures of statistics. A Biometrical Approach. McGraw-Hill Kogakusha. pp.633.

STUBBS, R.W. 1966. Recent aspects of the physiological specialisation of yellow rust in The Netherlands. Proceedings 3rd European Yellow Rust Conference, Cambridge 1964. pp.47-54.

STUBBS, R.W. 1967. Influence of light intensity on the reactions of wheat and barley seedlings to *Puccinia striiformis*. Phytopathology 57, 615-617.

STUBBS, R.W. 1977. Observations on horizontal resistance to yellow rust (*Puccinia striiformis f.sp. tritici*) . Cereal Rusts Bulletin 5, 27-32.

STUBBS, R.W. 1980. Yellow rust in Europe. Annual Report, Research Institute for Plant Protection, Wageningen, The Netherlands. pp.27-28.

STUBBS, R.W. 1985. Stripe rust. In "The Cereal Rusts Volume II". Eds. A.P. Roelfs and W.R. Bushnell. Academic Press. p.61-101.

STUBBS, R.W., E. FUCHS, H. VECHT and E.J.W. BASSET. 1974. The international survey of factors of virulence of *Puccinia striiformis* Westend. in 1969, 1970 and 1971. Nederlands Graan-Centrum, Technisch Bericht 21, pp.88.

STUBBS, R.W. V. SLOVENCIKOVA and P. BARTOS. 1977. Yellow rust resistance of some European wheat cultivars derived from rye. Cereal Rusts Bulletin 5, 44-47.

STUBBS, R.W., M. SANDERS and A.C. ZEVEN. 1984. A recessive resistance gene for yellow rust (*Puccinia striiformis* West.) in bread wheat (*Triticum aestivum* L.). Euphytica 33, 561-562.

STROBEL, G.A. and E.L. SHARP. 1965. Proteins of wheat associated with infection type of *Puccinia striiformis*. Phytopathology 55, 413-417.

SYME, J.R. 1983. Working with the world for plant improvement. Journal of Australian Institute of Agricultural Science 49, 181-190.

TAYLOR, A.J., G.M.B. SMITH and R. JOHNSON. 1981. Race-specific genetic factors for resistance to *Puccinia striiformis* in wheat cultivars from the Plant Breeding Institute. Cereal Rusts Bulletin 9, 33-45.

THE, T.T. 1976. Variability and inheritance studies in *Triticum monococcum* for raction to *Puccinia graminis f.sp. tritici* and *P. recondita*. Zeitschrift für Pflanzenzüchtung 76, 287-298.

TOLLENAAR, H. 1967. A comparison of *Puccinia striiformis f.sp. poae* on bluegrass with *P. striiformis f.sp. tritici* and *f.sp. dactylidis*. Phytopathology 57, 418-420.

TOLLENAAR, H. and B.H. HOUSTON. 1967. A study on the epidemiology of stripe rust, *Puccinia striiformis* West., in California. Canadian Journal of Botany 45, 291-307.

TORRES, E. and S. RAJARAM. 1983. International testing as a means to implement durable resistance: The experience of CIMMYT with wheat rusts. p.363-367 in "Durable Resistance in Crops." F. Lamberti, J.M. Waller and N.A. Van der Graaff (eds.), Plenum Press, pp.454.

TOTTMAN, D.R. and R.J. MAKEPEACE. 1979. An explanation of the decimal code for the growth stages of cereals, with illustrations. Annals of Applied Biology 93, 221-234.

UBELS, E., R.W. STUBBS and J.C.S. JACOB. 1965. Some new races of *Puccinia striiformis*. Netherlands Journal of Plant Pathology 71, 14-19.

VALLEGA, V. and G. ZITELLI. 1979. Epidemics of yellow rust in Italy. Cereal Rusts Bulletin 6, 17-22.

VAN DER PLANK, J.E. 1968. "Disease Resistance in Plants". Academic Press, New York. pp.206.

VOLIN, R.B. and E.L. SHARP. 1973. Physiologic specialization and pathogen aggressiveness in stripe rust. Phytopathology 63, 699-703.

VOSS, E.G. (Ed.). 1983. International Code of Botanical Nomenclature. International Association for Plant Taxonomy, Utrecht. pp.472.

WALTHER, U. and H. HERDAM. 1984. On the inheritance of field resistance to stripe rust (*Puccinia striiformis* West.) in some wheat cultivars. Proceedings of Fifth European and Mediterranean Cereal Rusts Conference, pp.107-110.

WALLWORK, H. and R. JOHNSON. 1984. Transgressive segregation for resistance to yellow rust in wheat. Euphytica 33, 123-132.

WATERHOUSE, W.L. 1929. Australian rust studies I. Proceedings of the Linnean Society of N.S.W. 54, 616-680.

WATERHOUSE, W.L. 1936. Some observations on cereal rust problems in Australia. Proceedings of Linnean Society of N.S.W. 61,5-38.

WATSON, I.A. and F.C. BUTLER. 1984. Wheat Rust Control in Australia. National Conferences and other Initiatives and Developments. The University of Sydney. pp.79.

WATSON, I.A. and W.P. CASS SMITH. 1962. Movement of wheat rusts in Australia. Journal of the Australian Institute of Agricultural Science 28, 279-287.

WATSON, I.A. and C.N.A. DE SOUSA. 1983. Long distance transport of spores of *Puccinia graminis tritici* in the southern hemisphere. Proceedings of the Linnean Society of N.S.W. 106, 311-321.

WATSON, I.A. and N.H. LUIG. 1961. Leaf rust on wheat in Australia: A systematic scheme for the classification of strains. Proceedings of Linnean Society of N.S.W. 86, 235-258.

WATSON, I.A. and H.H. LUIG. 1963. The classification of *Puccinia graminis* var. *tritici* in relation to breeding resistant varieties. Proceedings of Linnean Society of N.S.W. 88, 235-258.

WATSON, I.A. and N.H. LUIG. 1968. The ecology and genetics of host-pathogen relationships in wheat rusts in Australia. Proceedings Third International Wheat Genetics Symposium, Canberra. p.227-238.

WELLINGS, C.R. and N.H. LUIG. 1984. Wheat rusts: the old and the new. Australian Institute of Agricultural Science, Occasional Publication 15, 5-15.

WELLINGS, C.R. and R.A. McINTOSH. 1982. Stripe rust - A new challenge for the wheat industry. Agricultural Gazette of N.S.W. 92, 2-4.

WHEELER, D.J.B., S.W.L. JACOBS and B.E. NORTON. 1982. "Grasses of New South Wales". University of New England Monographs, 3. The University of New England, Armidale, Australia. pp.295.

WIESE, M.V. 1977. Compendium of Wheat Diseases. American Phytopathology Society, pp.106.

WOLFE, M.S. 1971. Fungicides and the fungus population problem. Proceedings Sixth British Insecticide and Fungicide Conference, Brighton, England 3, 724-734.

WOLFE, M.S. and R.C.F. MACER. 1964. The use of kinetin in the detached leaf culture of *Puccinia striiformis*. Proceedings Third Cereal Rusts Conference. p.58-63.

WORLAND, A.J. and C.N. LAW. 1985. Genetic analysis of chromosome 2D of wheat I. The location of genes affecting height, day-length insensitivity, hybrid dwarfism and yellow rust resistance (unpublished manuscript).

WRIGHT, R.G. and J.H. LENNARD. 1978. Mitosis in *Puccinia striiformis* I. Light microscopy. Transactions of the British Mycological Society. 70, 91-98.

WRIGHT, R.G. and J.H. LENNARD. 1980. Origin of a new race of *Puccinia striiformis*. Transactions of the British Mycological Society 74, 283-287.

WRIGHT, R.G., J.H. LENNARD and D. DENHAM. 1978. Mitosis in *Puccinia striiformis* II. Electron microscopy. Transactions of the British Mycological Society 70, 229-237.

ZADOKS, J.C. 1961. Yellow rust on wheat. Studies in epidemiology and physiologic specialization. Tijdschrift over Plantenziekten 67, 69-256.

ZADOKS, J.C. 1967. International dispersal of fungi. Netherlands Journal of Plant Pathology 73, Supplement 1, 61-80.

ZADOKS, J.C. 1979. Strategies in combating cereal diseases in Europe, with special reference to yellow rust of wheat. Proceedings of Rice Blast Workshop, I.R.R.I., Philippines, p.183-198.

ZELLER, F.J. 1973. 1B/1R wheat-rye chromosome substitutions and translocations. Proceedings of the Fourth International Wheat Genetics Symposium, Columbia, U.S.A. p.209-221.

ZELLER, F.J. and E. FUCHS. 1983. Cytologie und Krankheitsresistenz einer 1A/1R-und mehrerer 1B/1R-weizen-roggen-translokationss orten. Zeitschrift für Pflanzenzüchtung 90, 285-296.

ZEVEN, A.C. and N.C. ZEVEN-HISSINK. 1976. "Genealogies of 1400 Wheat Varieties". Netherlands Cereal Centre, Wageningen, and CIMMYT, Mexico. pp.121.

APPENDIX TABLE 1.

Species of family Poaceae inoculated with *Puccinia striiformis f.sp. tritici* under glasshouse conditions and found to be symptomless

SUB-FAMILY PANICOIDEAE

Andropogoneae

Bothriochloa macra (Stued.) S.T. Blake
Cymbopogon refractus (R.Br.) A. Camus
Dicanthium sericeum (R.Br.) A. Camus
Hemarthria uncinata R.Br.
Heteropogon contortus (L.) Beauv. ex Roem. & Schult.
Imperata cyclindrica Beauv.
Sorghum halepense (L.) Pers.
Themeda australis (R.Br.) Stapf
Themeda avenacea (F. Muell) Maiden & Betche

Paniceae

Cenchrus ciliarus L.cv. Molopo, Runibank
Digitaria brownii (Roem. & Schult.) Hughes
Digitaria ternata (Hochst.) Stapf.
Eriochloa pseudoacrotricha (Stapf. ex Thell) J.M. Black
Panicum coloratum L.
Panicum effusum R.Br.
Panicum maximum Jacq.
Panicum prolutum F. Muell.
Paspalum dilatatum Poir.
Paspalum wetsteinii Hack.
Pennisetum alopecuroides (L.) Spreng.
Pennisetum clandestinum Hochst. ex Chiov. cv. Whittet

SUB-FAMILY ERAGROSTOIDEAE

Aristideae

Aristida calycina R.Br.
Aristida leptopoda Benth.
Aristida ramosa R.Bt.

Eragrosteae

Astrebla lappacea (Lindl.) Domin
Dactyloctenium radulans (R.Br.) Beauv.
Eragrostis elongata Jacq.
Eragrostis leptostachya Stevd.
Eragrostis molybdea Vickery

Pappophoreae

Enneapogon avenaceus (Lindl.) C.E. Hubbard

Chlorideae

Chloris acicularis Lindl.
Chloris truncata R.Br.
Chloris ventricosa B.Br.
Chloris virgata Sw.
Cynodon dactylon (L.) Pers.

Sporoboleae

Sporobolus africanus (Poir.) Robyns & Tournay
Sporobolus elongatus R.Br.

Zoysieae

Tragus australianus Blake

SUB-FAMILY POOIDEAE

Agrosteae

Agrostis avenacea Gmel.
Dichelachne crinita (L.) Hook.
Dichelachne micrantha (Cav.) Domin.

Aveneae

Aira cupaniana Guss.
Avena sativa L.
Holcus lanatus L.

Triticeae

Agropyron cristatum (L.) Gaertn

Phalarideae

Phalaris angusta Nees ex Trin.
Phalaris aquatica L.
Phalaris canariensis L.
Phalaris minor Retz.

Poeae

Briza maxima L.
Briza minor L.
Briza subarista Lamk.
Bromus brevis Stevd.
Bromus gussonii Parl.
Bromus hordeaceus L.
Bromus molliformis F.E. Lloyd
Dactylis glomerata L.
Lolium perenne L.
Lolium rigidum Gaudin
Lolium multiflorum Lamk.
Poa annua L.
Poa sieberana Spreng.
Vulpia bromoides (L.) Gray
Vulpia megalura (Nutt.) Rydb.
Vulpia myuros (L.) Gmel.

SUB-FAMILY BAMBUSOIDEAE

Stipeae

Stipa aristiglumis F. Muell.
Stipa verticillata Nees ex Spreng

Ehrharteae

Microlaena stipoides (Labill.) R.Br.

SUB-FAMILY ARUNDINOIDEAE

Arundineae

Danthonia induta Vickery
Danthonia laevis Vickery
Danthonia linkii Kunth.
Danthonia racemosa R.Br.
Danthonia richardsonii Cashmore
Monachather paradoxa Stevd.

APPENDIX TABLE 2

Response (per cent leaf area affected) of cultivars, arranged in groups according to postulated resistance genes, after inoculation with three pathotypes of *P. striiformis tritici* in the field

Cultivar	104 E137 A+			108 E141 A-			108 E141 A+		
	Replicate 1	Replicate 2	Replicate 3	Replicate 1	Replicate 2	Replicate 3	Replicate 1	Replicate 2	Replicate 3
Group A (*Yr4*)									
Avocet R	10	10	10	20	20	5	10	5	30
Avocet S	20		15	50	10	30	50	20	10
Banks R	5	5		15	5		20	20	20
Banks S		20	20	10	10	15	10	10	20
Egret R	5	10	10	10	10	10	10	20	15
Egret S		20	20	10	15	20	50	60	10
Condor R	10	5	10	10	10	10	15	20	20
Condor S	5	10		10	15	15	15	10	
Sunstar	5	10	10	10	10	15	10	10	10
Vulcan	5	5	5	5	20	5	5		10
WW15	5	10		10	20	20		20	15
Sonalika	30	30	20	50	50	40	40	50	50
Inia 66	40			10	10	10	30	30	10
Nainari 60	15	20	10	15	15	5		5	20
Group B (*Yr7*)									
Sunkota	5	20	20	10	20	5	20	10	10
Gatcher R	30	30	30	30	20	20	20	20	20
Gatcher S	60	40	50	50	40	60	40	40	50
Norquay	5	10	10	10	10	15	5	10	10
Group C (*Yr6*)									
Oxley	10	20	5	5	10	15	5	5	5
Jacup		60	10	50	50	20	50	40	50
Miling	10	30	20	50	30	30	10	50	40
Millewa	80	30	10	80	50	30	60	50	70
Takari	10	5	30	20	10	15	30	20	10
Bindawarra	5	50	20	60	50	50	40	60	10
CSP44 (*Yr6*)	5	5		15	10	10	5	10	15
WW 80	5	20	5	10	10		10	10	5
Heines Peko	10	10	5	5	20	10	20	10	10
Pitic 62	5	30	30	40	50	30	10	10	20
Penjamo 62R	20	20	20	10	30	20	10	20	10
Penjamo 62S	80	40	50	10	60	60	15		70
Frontana	5	10	10		10	5	5	10	5
Tobari 66	20	30		5	20	20		10	10
Atlas 66	10	5	5	5	10		60	5	10
Shoshi	5	15	5	10	10	5		10	10
Romany	10	10	20	30	20	20	10	10	5
Frontiera	50	10		40	40		50	5	
Group D (*Yr6 + A*)									
Cajeme 71	15	15	5	10	5	10	10	10	5
CSP44	5	5	15	10	10	15	5	20	10
Group E (*Yr6 + 7*)									
Pavon 76	5	10	5		10	20	5	10	5
Hermosillo 77	10	5	10	5	10	10	5	5	10
Group F									
Corella	5	5	5		5	5	5	5	5
Group G									
Nacozari 76	20	10	5	5	10	10	10	5	5

[1]Missing data due to failure of some plots.