ASSESSMENT OF ADULT PLANT RESISTANCE TO STRIPE RUST IN WHEAT

By

Lizaan Pienaar

Dissertation submitted in partial fulfilment of the requirements for the degree Magister Scientiae Agriculturae in the Faculty Natural and Agricultural Sciences (Department of Plant Sciences - Plant Pathology) of the University of the Free State

Supervisor: Professor Z. A. Pretorius

MAY 2004

BLOEMFONTEIN

TABLE OF CONTENTS

ACKNOWLEDGEMENTS / DANKBETUIGINGS v

GENERAL INTRODUCTION 1

STRIPE RUST ON WHEAT: IMPORTANCE, BIOLOGY AND CONTROL

INTRODUCTION

2

ECONOMIC IMPORTANCE

3

PATHOGEN

Taxonomy, nomenclature and morphology 4

Life cycle 4

Symptoms 6

6

Pathogenic variability

7

HOST

10

HOST: PATHOGEN INTERACTIONS

11

Specific interactions

12

Nonspecific interactions

13

DISEASE CONTROL

13

Genetic resistance

13

Seedling resistance

16

Adult-plant resistance

17

Partial resistance

Symptom expression and assessment of resistance 18

Temperature and light intensity 18

Plant nutrition 20

Inter-plot interference 21

Earliness and observation date 21

Chemical control 21

22

Cultural methods

25

CONCLUSIONS

26

REFERENCES

26

TOWARDS IMPROVEMENT OF THE DETECTION OF ADULT-PLANT RESISTANCE TO STRIPE RUST IN WHEAT

INTRODUCTION

46

MATERIALS AND METHODS

Wheat and pathogen materials 47

Glasshouse experiments 47

Seedling tests 47

- Experimental population 47

- CIMMYT lines 47

Normal adult plant tests 48

Mini-adult plant tests 48

- Growth stage effects 49

- Experimental population 49

50
- Growth chamber and glasshouse comparison 50
- Pathotype effects

50
- Winter wheats

50
- CIMMYT lines

51
Field experiments

Experimental population 51

CIMMYT line evaluation 51

RESULTS 51

Glasshouse experiments 51

Seedling tests 51

- Experimental population 51

- CIMMYT lines 51

Normal adult plant tests 53

53

Mini-adult plant tests

54

- Growth stage effects

54

- Experimental population

54

- Growth chamber and glasshouse comparison 54

- Pathotype effects

55

- Winter wheats

- CIMMYT lines 55

Field experiments 56

Experimental population 56

CIMMYT line evaluation 56

DISCUSSION 57
REFERENCES 62

A GENETIC ANALYSIS OF STRIPE RUST RESISTANCE IN THE WHEAT CULTIVARS BAVIAANS AND SUNMIST

INTRODUCTION

104

MATERIALS AND METHODS

105

Crosses

Glasshouse experiment 105

Field experiment 106

RESULTS 106

107

Glasshouse experiment

107

Baviaans x Avocet S cross

Sunmist x Avocet S cross 107

Field experiment 107

Baviaans x Avocet S cross 107

Sunmist x Avocet S cross 107

DISCUSSION 108

REFERENCES 108

111

SUMMARY

132

OPSOMMING

134

APPENDICES

136

ACKNOWLEDGEMENTS

I would like to convey my sincere gratitude and appreciation to:

My supervisor, Prof. Z.A. Pretorius, for his continued support and assistance with the preparation of this dissertation. Also, for inspiring me to be a good scientist;

The NRF and Winter Cereal Trust, for financial support and the opportunity to conduct this research;

My parents, for all the opportunities granted to me through their hard work and sacrifices;

My fiancé Gustav Rademeyer, for his love, understanding and assistance;

Mrs. Annatjie Rademeyer, for her love and understanding;

My brother, sister, family and friends, for their love and understanding;

Mrs. Wilmarie Kriel, Mrs. Cornel Bender and Mrs. Zelda van der Linde, for their love and assistance, and

Finally, my Heavenly Father for His abundant grace.

DANKBETUIGINGS

Graag betuig ek my innige dank aan:

My studieleier, Prof. Z.A. Pretorius, vir sy volgehoue ondersteuning, insette en hulp met die voorbereiding van die verhandeling, asook vir sy

inspirasie om ‘n goeie wetenskaplike te wees;

Die NRF en Wintergraantrust vir finansiële steun en die geleentheid om hierdie studie te kon aanpak;

My ouers vir al die geleenthede wat hulle deur harde werk en baie opofferinge moontlik gemaak het;

Aan my verloofde Gustav Rademeyer vir sy liefde, begrip en bystand;

Mev. Annatjie Rademeyer vir haar liefde en begrip;

My broer, suster, familie en vriende vir hul liefde en begrip;

Mev. Wilmarie Kriel, Mev. Cornel Bender en Mev. Zelda van der Linde vir hul liefde en volgehoue ondersteuning, en

Laastens aan my Hemelse Vader vir Sy oorvloedige genade.

GENERAL INTRODUCTION

Stripe rust, caused by *Puccinia striiformis* Westend. f. sp. *tritici*, is a major disease of wheat (*Triticum aestivum* L.) in many cool and moist environments of the world. It was observed for the first time in South Africa in the Western Cape in August 1996 and within two years became established as an endemic disease in the major wheat producing areas. Since 1996, the control of stripe rust has cost South African wheat farmers millions of rands and although more efficient chemical control procedures have been developed, the use of resistant cultivars remains the best control method. This follows the approach in other countries where breeding for resistance against stripe rust has been a high priority.

The success of resistance breeding depends on relevant knowledge of pathogenicity and host genetics. Much of the earlier work on breeding for resistance to rust was based upon the exploitation of simply inherited major genes that were expressed in seedlings throughout the life cycle of the host plant. However, these major genes frequently interact in a gene-for-gene pattern with the pathogen and their efficacy has proven to be short-lived. Adult plant resistance cannot be identified in the seedling stage and has often been suggested as a possible source of durable resistance. Long-term resistance to rust diseases thus depends on the identification and use of durable resistance sources or on the continued use of new resistance sources and combinations of genes for specific resistance. Conventional resistance

breeding methods rely on time consuming field and/or greenhouse screening with *P. striiformis* f. sp. *tritici* races. Because of the large numbers of plants that must be screened in most wheat breeding programmes, a rapid yet reliable evaluation procedure is essential.

The objective of this study was firstly to summarise the available literature on stripe rust, including the economic importance, biology of the pathogen and disease control. Secondly, the possibility of detecting adult plant resistance to stripe rust in wheat seedlings was investigated, followed by optimisation of a system of accurate and reliable screening of adult wheat plants for stripe rust reaction in a controlled environment. Thirdly, the inheritance of adult plant resistance in the cultivars Baviaans and Sunmist was studied, in particular to determine if mini-adult wheat plants can be used in genetic studies of stripe rust resistance.

STRIPE RUST ON WHEAT: IMPORTANCE, BIOLOGY AND CONTROL

INTRODUCTION

Several thousand rust species attack a wide range of plants (Knott, 1989). These rusts often have narrow host ranges, being restricted to a single family, a single genus or even a single species (Kendrick, 1992). Even though rusts co-evolved with their hosts for millions of years, and do not usually kill them, rust fungi can severely reduce yields of domesticated plants, particularly cereals (Kendrick, 1992). Several rusts cause serious economic losses in crops, but none more than the three rusts that attack wheat (*Triticum aestivum* L.). The rusts are present everywhere wheat, the world's most important crop with respect to nutrition is grown (Afshari, 2000), and are among its most serious diseases (Knott, 1989). In general, rusts reduce plant vigour, limit grain filling and cause most damage when epidemics begin before or during flowering (Russell, Murray and Sutherst, 2000).

The wheat rusts are stem rust (*Puccinia graminis* Pers. f. sp. *tritici*), leaf rust (*Puccinia triticina* (Eriks.) = *P. recondita* Rob. ex Desm. f. sp. *tritici*) and stripe rust (*Puccinia striiformis* Westend. f. sp. *tritici*). Although these rusts belong to the same genus they differ in morphology, life cycle, and optimal environmental conditions for development (Knott, 1989). Stem rust is undoubtedly the most damaging of the rust diseases but the other two can cause losses in excess of

50% under optimum conditions (Afshari, 2000).

The continual extension of the geographic range of *P. striiformis* over the past 25 years has witnessed the progressive occurrence of the pathogen in new regions, e.g. eastern Australia (1979), New Zealand (1981) and South Africa (1996). The recent detection of *P. striiformis* in Western Australia represents the colonisation of the last major wheat-producing region of the world that had remained stripe rust free. This introduction occurred despite rigorous quarantine measures that have contributed to disease exclusion since the establishment of the grains industry in the 1800s (Wellings *et al.*, 2003).

The objective of this chapter is to summarise the literature available on stripe rust, including economic importance, biology and disease control.

ECONOMIC IMPORTANCE

Gadd described stripe rust for the first time in 1777 (Eriksson and Henning, 1896). Stripe rust, also known as yellow rust, was observed for the first time in South Africa in August 1996 in the Western Cape (Pretorius, Boshoff and Kema, 1997). Ensuing surveys during the 1996 season indicated that stripe rust occurred throughout most of the wheat-producing areas in the winter rainfall regions of the Northen, Western, and Eastern Cape provinces (Pretorius *et al.*, 1997). The disease was also observed on irrigated wheat in the summer rainfall region south of Kimberley. However, stripe rust was most severe in the

Western Cape, where prolonged cool and wet conditions favoured epidemic development. Due to spike infection and destruction of foliage, significant losses in yield and quality occurred in wheat fields (Pretorius *et al.*, 1997). In 1997 the disease was first observed in the southern and western regions of the Western Cape, followed by early detection in the western Free State from where it spread to other parts of the province, KwaZulu-Natal, Gauteng, North-West and Limpopo.

During 1998 a significant change in pathogenicity occurred (Boshoff and Pretorius, 1999). The stripe rust pathogen mutated and gained the ability to infect cultivars previously resistant to stripe rust. In view of the rapid dispersal of the pathogen since 1996, its ability to over summer in both the winter and summer rainfall areas, susceptibility of several high yielding cultivars, favourable climatic conditions, pathotype change, and strong economic impact due to yield losses and excessive chemical control costs, stripe rust is considered extremely damaging in South Africa (Pretorius, Bender and van der Linde, 2001).

Similar to many diseases caused by biotrophic plant pathogens, the amount of stripe rust may vary considerably from year to year, particularly where environmental factors during the year may be unfavourable for development of the pathogen (Hovmøller, 2001). Several factors affect the development of the disease such as inoculum pressure, meteorological conditions and cultivar susceptibility, causing incidence of individual diseases to

vary from year to year and from site to site (Cook, Hims and Vaughan, 1999). Yield losses varying from 40 to 84% have been reported throughout the world (Murray *et al.*, 1994; McIntosh, Wellings and Park, 1995). The control of foliar rusts on susceptible wheat cultivars has cost South African wheat farmers millions of rands. Wheat producers spent an estimated R28 million on fungicides to control this disease in the Western Cape in 1996 and despite the widespread application of chemicals, significant losses, varying from 5 to 50%, still occurred. Subsequent epidemic outbreaks in the eastern Free State in 1997 cost farmers R18 million in fungicide application (Boshoff, 2000). In 1998, the appearance of pathotype 6E22A- in the eastern Free State resulted in epidemic outbreaks of stripe rust on the previously resistant cultivars Hugenoot and Carina. The cost to control the disease on approximately 42 000 ha, excluding losses in yield and quality, was estimated at more than R6 million (Boshoff, 2000).

In a fungicide trial conducted in the Western Cape, mean yield in sprayed plots was increased by as much as 43% (Boshoff, Pretorius and Van Niekerk, 2003). Komen, Van Niekerk and Boshoff (2002) reported that stripe rust reduced grain yield of winter wheat by between 1.6 ton/ha (114%) and 2.5 ton/ha (177%) depending on the fungicide used and timing of the application. Considering yield components, stripe rust reduced hectolitre mass from 76.1 kg/hl to 64.8 kg/hl in the same trial.

PATHOGEN

Taxonomy, nomenclature and morphology

Rust fungi are all obligate biotrophs on vascular plants (Kendrick, 1992) and can only be cultured on living host material (Johnson, 1992a). Stripe rust belongs to the genus *Puccinia,* family Pucciniaceae, order Uredinales, and class Basidiomycetes (Littlefield, 1981). Stripe rust is caused by *Puccinia striiformis* f. sp. *tritici* (McIntosh *et al.*, 1995). In 1827 stripe rust was described as the third cereal rust under the name *Uredo glumarum* (Schmidt, 1827), and since then it was given several names e.g *Puccinia straminis* (Hassebrauk, 1965) and *Puccinia glumarum* (Eriksson and Henning, 1894). Cummins and Stevenson (1956), introduced the name *Puccinia striiformis* Westend (Manners, 1960).

Urediniospores, produced in uredinia, are defined as repeating vegetative spores produced on dikaryotic mycelium (Scott and Chakravorty, 1982). Savile (1984) described uredinia as small, often crowded, tardily naked, pale to bright yellow when fresh (paling as cytoplasmic pigment fades), occasionally with few thin-walled paraphyses, mainly adaxial, on narrow chlorotic streaks on older leaves but often scattered on young leaves. The urediniospores are 26-30(-33) × (16-)18-24.5(-26.5) μm, hyaline to subhyaline), often visibly bilaminate but usually have no pigment in the inner layer; echinulate (0.2)0.3-0.5(0.6) μm diam. and (0.8)1.0-2.3 μm between centers; germ spores

often obscure, scattered, apparently 7-13(-15), generally with very slight internal ring and no appreciable cap. The telia are mainly abaxial or on sheaths, covered by epidermis, plumbeous, elongate, with light to moderately heavy orange-brown stroma and are orange brown fused paraphyses ~50-70 μm long generally abundant and divide sorus into locules.

Teliospores are basidia-producing spores, and sori that produce teliospores are called telia (Scott and Chakravorty, 1982). Teliospores are occasionally one-celled (28-34 × 11-15.5 μm) or irregularly three- to four-celled, but typically two celled and 30-60(-65) × (13-)14-27(30)((-33)) μm. Usually they are slightly constricted, irregularly clavate or fusoid, rarely subcylindrical and often with faint longitudinal ridges. Teliospores are yellow-brown. The pedicels are pale to dark yellow, rarely to 16 μm long (Savile, 1984).

Stripe rust differs from the other two wheat rusts because it develops systemically in host tissue (Singh *et al.*, 2003). Leaf and stem rust only produce one new pustule at each infection site. *Puccinia striiformis* does not produce appressoria (De Vallavieille-Pope *et al.*, 1995) and has typical unrestricted growth of individual infections. Infections grow longitudinally in the leaf and scattered individual infections may coalesce. It is often impossible to distinguish individual infections on the leaf (Broers and López-Atilano, 1994). *Puccinia striiformis* germtubes do not seem to have any directional growth. Observations on germinating stripe rust spores on 0.8% agar as well as on

seedling leaves showed that germ tubes grew away from the surface, bent, and returned to the surface again. Germ tubes can cross four stomates without entering any of them (Broers and López-Atilano, 1994).

Life cycle

Stripe rust in the field may appear in foci ("hot spots") when spores land in a newly planted crop early in the season (Russell *et al.,* 2000). It has a microcyclic life cycle with no known alternate hosts (McIntosh *et al.*,1995; Russell *et al.,* 2000). Only three spore stages are known namely, urediniospores, teliospores and basidiospores (Knott, 1989). The life cycle of stripe rust involves a repetition of the asexual uredinial stage. The urediniospores germinate and infect at cooler temperatures with the optimum reported at 9-13°C (Roelfs, Singh and Saari, 1992) and a relative humidity of 100% (Rizvi, Schubert and Dixon, 2003). Stripe rust can survive periods of stress (cold winters and hot summers) as mycelium in tissue of living plants. Sporulating uredinia can survive at a temperature of -4°C (Hogg *et al.*, 1969).

However, if all above ground parts of the plant are killed, the rust will not survive (Knott, 1989). Only the uredinial stage is effective in the survival of *P. striiformis* and the teliospore stage - although vital for species identification -

has no known role in its survival (Scott and Chakravorty, 1982; Russell *et al.,* 2000).

De Vallavieille-Pope *et al.* (1995) found that an interruption of the wet period by a dry period did not affect ungerminated urediniospores, which were able to infect leaves during a subsequent dew period. The minimal continuous dew period necessary for infection increased from 4 to 6 h at optimal temperature (8°C) to at least 16 h at suboptimal temperatures. If the dry period occurs after the minimal dew duration for infection, percentage of infection is the same as with a continuous dew period (De Vallavieille-Pope *et al.*, 1995).

According to De Vallavieille-Pope *et al.* (1995) stripe rust is unable to infect seedlings if a dry period occurred between urediniospores germination and penetration. The narrower range of temperatures favourable to stripe rust is compensated for by the higher quantity of inoculum stored as ungerminated urediniospores, able to complete infection when suitable conditions returned. Also, *P. striiformis* compensates for a low infection efficiency with systemic growth within the leaf (De Vallavieille-Pope *et al.*, 1995).

Symptoms

Typical symptoms are long, yellow stripes on the leaves. All parts of the plant can be attacked, even kernels (Knott, 1989), but symptoms are more frequently

seen on the leaves (Kurt, 2001). The pustules are restricted by veins on older leaves but may grow several millimetres in length, whereas on seedling leaves lateral spread of the pustules is less restricted (Knott, 1989). Stripe rust is the only rust of wheat that consistently spreads beyond the initial infection point (Roelfs *et al.,* 1992). Individual pustules often give rise to chlorosis followed by necrosis on leaves (Kurt, 2001).

'Green islands' are regions of apparently healthy leaf tissue occurring at sites of individual infections when the remainder of the leaf is chlorotic and senescent (Al-Khesraji and Lose, 1980). Electron microscopical observation of 'green island' tissue revealed that most organelles such as chloroplasts, the nucleus and mitochondria showed remarkable changes in their structure as a result of the infection. It is well known that physiological changes, including those of chlorophyll content, are accompanied by ultrastructural changes, particularly in those cells directly associated with fungal mycelium in infection structures (Al-Khesraji and Lose, 1980). *Puccinia striiformis* may exert some degree of control over its host's physiology. The relationship between retention of chlorophyll in 'green islands' formed on detached wheat leaves and polyamines detected in such areas, particularly spermidine, has prompted the view that *P.striiformis* actively secretes polyamines (Al-Khesraji and Lose, 1980). Spermidine and spermine, are effective in retarding loss of chlorophyll in leaf tissue of radish (Altman, 1982). They also inhibit the degradation of protein and

the activity of ribonuclease (Altman, 1982).

Pathogenic variability

Pathogens that develop new races easily, and against which several to many race-specific resistance genes occur, are often specialized, biotrophic or hemibiotrophic, airborne or splashborne fungi (Parlevliet, 1993). As the sexual state has not been recorded for *P. striiformis*, the development of genetic variation must be due to other mechanisms, such as mutation and somatic recombination (Stubbs, 1985).

Within most species of the rust fungi, there are a number of *formae speciales*. These *forma speciales* are composed of many biotypes that differ in several characteristics but primarily in their virulence on host cultivars. A biotype is defined as a population of individuals of the same genotype; therefore, theoretically, the progeny of an aeciospore or urediniospore would constitute a pathogen biotype (Roelfs, 1984). The pathogen phenotype is described as avirulent, or having low pathogenicity, and virulent, or having high pathogenicity. The use of virulence in both generic and specific contexts is not acceptable, and no alternate term other than pathogenicity has been proposed (McIntosh *et al.*,1995).

The avirulence/ virulence pattern of an isolate is determined by inoculating a selected group of host plants of differing genotypes for rust

resistance (Roelfs, 1984). A group of biotypes with a similar pathogenicity pattern on a selected group of host plants is considered a physiologic race, also called a pathotype. The pathotype is a taxon below the *forma speciales* level, which is distinguished by physiological differences rather than morphological differences. These physiological differences are shown as differing pathogenicity patterns when the differential host series is independently inoculated with different cultures. Therefore, the pathotype could be a single biotype but is more likely to be a group of similar biotypes that can be distinguished from other phenotypes with a reasonable amount of certainty by differences in their virulence patterns on a selected differential series. The pathogenicity is determined from the disease infection types (Roelfs, 1984).

Different regions of the world use different environmental conditions, classification systems, and differential sets to identify pathotypes (De Vallavieille-Pope and Line, 1990). Most of the cereal rusts were grouped into pathotypes based on an internationally used set of differential hosts. Because of the local differences in pathogen virulence and host resistances, investigators gradually adopted local sets of differential hosts that better reflected their needs (Roelfs, 1984). Boshoff (2000) established a differential classifications system for South Africa by combining the World (1 to 9) and European (10 to 17) differentials supplemented with tester lines (18 to 42) to pathotypes 6E16A- and 6E22A- of *P. striiformis* (Table 1).

In stripe rust, the long latent period (the period during which the host plant is infected but not yet infectious; Zadoks and Schein, 1979), which results in a reduced number of multiplication cycles per cultural season, is compensated by the high monocyclic progeny/parent ratio and the semi-systemic lesion growth. According to Sache and de Vallavieille-Pope (1993), the strong sporulation capacity compensates for the low infection efficiency of stripe rust. Parlevliet (1993) indicated that the more inoculum of a pathogen present, the greater the chances that new variants can arise.

Since the initial detection of stripe rust in Australia in 1979, it has evolved into more than 20 pathotypes with assorted virulence characteristics in Australasia (Steele *et al.*, 2001). This evolution is believed to have occurred in a stepwise fashion from an original single pathotype, with no subsequent new introductions (Steele *et al.*, 2001). However, the recent introduction into Western Australia contradicts this statement (Wellings *et al.*, 2003). Newly introduced resistant cultivars lose their resistance within a few years owing to the appearance of new, often more complex races (Danial and Stubbs, 1992). *Puccinia striiformis* races with new pathogenicity combinations can spread across large areas so that similar cultivars in different countries are at risk when new pathotypes arise in one area. The risk is enhanced by the international use of similar cultivars across wide areas, and because breeders within a region often use the same source of resistance (Johnson, 1992a). The development of

new races as a response to the introduction of resistant cultivars is especially associated with breeding for major genes, developing high levels of resistance. When a new gene for virulence arises through mutation in one region, it can be spread to another region by wind dispersal of urediniospores or by unintentional human activity. Additionally, the same mutation can occur independently in more than one region. However, it may not be detected unless the corresponding host resistance gene is present as a selecting agent (Stubbs, 1985).

A study conducted by Steele *et al.* (2001) showed limited molecular variation within species. Different populations have different levels of molecular polymorphism. These differences could relate to the age of the population and to the relative number of migration events occurring between populations. The monomorphic nature of Australian isolates with respect to molecular markers is evidence that they are closely related to each other, suggesting that stepwise mutation of single virulence genes is the most probable means by which new pathotypes have originated (Steele *et al.*, 2001). In addition, Steele *et al.* (2001) stated that there was no evidence for a high mutation rate or chromosomal deletions in *P. striiformis* f. sp. *tritici*, and further analysis is required to determine whether the evolution of new pathotypes is caused by random mutations/deletion events or by more specific mechanisms at avirulence gene loci.

The possible number of different pathogen races that can be detected is 2^n where n is the number of genes. For seven genes, 2^7 = 128 races can be detected, indicating that the possible number of races is extensive, assuming that they all interact on a gene-for-gene basis with the pathogen. This calculation is based on the existence of each gene in a separate wheat line, so that the response of the pathogen for each resistance gene can be readily determined (Johnson, 1992b).

HOST

Puccinia striiformis is a pathogen of grasses and cereal crops, i.e. wheat, barley, triticale and rye (Roelfs *et al.,* 1992). Barley, rye and wheat are part of the tribe Triticeae Dumort (Hordeae Benth), a festucoid tribe of the family Poaceae (Gramineae), and have long been of great economic importance to humanity (Lupton, 1987).

The term wheat is normally used to refer to the cultivated species of the genus *Triticum*. This genus is complex and includes diploids ($2n = 2x = 14$), tertraploids ($2n = 4x = 28$) and hexaploids ($2n = 6x = 42$). Although a number of species have been cultivated over the years, cultivation is now restricted almost entirely to tetraploid durum wheat (*T. turgidum* L.) and hexaploid common or bread wheat (*T. aestivum* L.) (Knott, 1989). The hexaploid wheats are of two types: the major group with the formula AABBDD and a single hexaploid, *T.*

zhukovskyi AABBGG (Lupton, 1987). Isolates of *P.striiformis* taken from wheat are usually able to infect a wide range of wheat varieties but very few barley varieties, while the converse is true of isolates taken from barley. Although morphologically indistinguishable, these isolates from the two hosts clearly differ in their host range (Johnson and Lupton, 1987).

Puccinia striiformis f. sp. *tritici* is highly variable within and between geographical areas and is probably most closely related to the forma speciales *hordei,* although they can be readily distinguished in field nurseries involving variable germplasm of both host species (Zadoks, 1961). In South Africa, *Bromus catharticus* and *Hordeum murinum* have been identified as accessory hosts to *P. striiformis* f. sp. *tritici* (Boshoff, 2000).

HOST: PATHOGEN INTERACTIONS

All pathogens are variable with respect to host resistance but variation is, in itself, a variable quality. In pathogens that are biotrophic and grow in intimate contact with living host cells, such as rust, highly developed specificity occurs (Johnson, 1992a). Detailed genetic studies have been restricted to relatively few diseases of agricultural plants. In all instances more is known with regard to the genetics of reaction in the host than of the genetics of pathogenicity.

Genetic analysis of a disease is conducted on either the host or the pathogen while the genotype of the pathogen or host, respectively, is held

constant (Scott and Chakravorty, 1982). When several different pathogen isolates are used separately to infect an array of host cultivars they often show a variable ability to infect subsets of the array (Johnson, 1992a). According to Flor (1946) this variation is due to the operation of gene-for-gene interaction between the host and the pathogen. The four possible combinations of the resistance (R, r) and virulence (Av, v) alleles give resistance/avirulence when R in the host coincides with Av in the pathogen, and susceptibility for the combinations R/v, r/Av and r/v (Johnson, 1992a).

The minimum number of gene pairs to depict a gene-for-gene interaction is two, which was the original pattern Flor formulated (Day, 1974). The gene-for-gene system has the implication that resistance will not remain effective if the pathogen acquires the corresponding virulence by losing the avirulence allele that elicits resistance, either by deletion or by genetic change. It also shows that resistance genes can be combined and that the pathogen must evade the effect of each gene by change at a specific, corresponding locus. Thus, it must accumulate the necessary change to allow virulence (Johnson, 1992a).

An important consideration in understanding the genetic basis of host-pathogen interactions is that the simplest ideal model of the gene-for-gene interaction is based on the generality that each corresponding gene pair acts independently of the other corresponding gene pairs.

Dominance occurs for the resistance allele in the host and the avirulence allele in the pathogen. This consideration is based on Flor's original observations in flax and the flax rust pathogen which was consistent with these observations (Johnson, 1992a).

As more data accumulated from other host-pathogen systems, examples of recessive resistance that act in gene-for-gene systems were encountered (Singh and Johnson, 1988). Genes *Yr2* and *Yr6*, which give race-specific resistance to yellow rust, are recessive in at least some crosses and with some pathogen isolates (Singh and Johnson, 1988).

Host-pathogen interactions can be divided into two categories: specific and nonspecific. Specific interactions occur when a single pathogen isolate interacts with a single host genotype to produce a different disease response than another isolate with the same host in the same environment. Nonspecific interactions occur when all isolates result in a similar response on a given genotype (Roelfs *et al.,* 1992). Nonspecific resistance is theorized to be the better type of resistance to use in a breeding program (Roelfs *et al.*, 1992).

Specific interactions

Crop varieties which are immune to the pathogen do not become infected, so that the pathogen will die unless it has other hosts on which to survive. In such cases new races of the pathogens are not likely to arise, but unfortunately

immune varieties, which are also satisfactory in other aspects, are rarely achieved. Plants which are hypersensitive to the pathogen, developing only minute necrotic spots or flecks through which development of the pathogen is inhibited, have been referred to as immune (Tarr, 1972). The expression of incompatibility can occur early in the disease process and may result in an immune response, or incompatibility may be expressed slowly at the end of the process causing only a slight reduction in sporulation. The lower infection types are generally quite characteristic for a particular host-pathogen-environment-time interaction (Roelfs *et al.*, 1992).

If two specific resistance genes are present in the same host line, the infection type produced by an isolate, avirulent on both genes is generally that of the most effective gene (Roelfs *et al.*, 1992).

Nonspecific interactions

Resistance characterized by Johnson and Lupton (1987) as adult plant, horizontal, generalized, slow rusting, partial, minor gene, etc. has been placed in this group. This is not entirely correct as examples of race-specific genes for adult plant resistance to rust diseases exist (McIntosh *et al.*, 1995). As mentioned above, interactions visible as low and high infection types do not occur when varieties with nonspecific resistance are infected with different pathotypes.

DISEASE CONTROL

Genetic resistance

Plants employ a great variety of defence mechanisms to cope with the multitude of organisms that try to exploit them. These defence mechanisms can be classified as avoidance, resistance and tolerance (Parlevliet, 1993). Resistance mechanisms, used almost exclusively in breeding against pathogens, reduce the growth and/or development rate of the pathogen and are nearly always of a biochemical nature (Parlevliet, 1993).

According to Heath (1981; 1982), two mechanisms of resistance may be found in plants inoculated with inappropriate species or special forms of rust fungi: pre-haustorial and post-haustorial resistance. Pre-haustorial resistance is not associated with necrosis, and is very common in non-host interactions (Heath, 1981; 1982). Post-haustorial resistance is usually associated with necrosis of plant cells after initiation or formation of haustoria (Niks and Dekens, 1987). Pre-haustorial resistance is considered a general defence mechanism difficult to overcome by rust fungi (Heath, 1982; 1985). Rubiales and Niks (1992) described this type of resistance as more durable. Although resistance is readily achieved by wheat breeders, genetic variability in stripe rust often leads to reduced effectiveness of resistance, resulting in a continued effort to find, develop and deploy further resistance in order to maintain protection

(McIntosh, 1992).

The coevolution between *P. striiformis* and wheat has developed in a way which is different from other cereal rusts and has led to a different resistance mechanism in which reduction of the infection frequency is a very important component (Broers and López-Atilano, 1996). The best method of control is the use of resistant cultivars and breeding for resistance is a high priority (Johnson, 1992a; Hogenboom, 1993). Inherited resistance is a valuable attribute because it is easy for the grower to use and reduces the need for other methods of control. However, it is subject to some significant biological and financial constraints and breeding for resistance for this disease presents significant challenges (Johnson, 1992a).

Knowledge of the genetics of a quantitative character is descriptive and not in itself predictive. Its main use is in deciding upon a breeding strategy. Thus, the type of inheritance will determine which is the best generation in which to practice selection, the consequences of inbreeding, the genetic basis of heterosis and whether it is better to produce hybrids rather than homozygous varieties in a breeding programme (Snape, 1987).

Resistance to the three wheat rusts has been shown to be mostly race specific, and the pathogen populations have correspondingly been found to consist of different physiological races. There is also resistance that is not controlled by currently identified race-specific genes (Johnson and Lupton,

1987). Resistance of a cultivar to an isolate is a genetic character. Therefore, a cultivar never loses its resistance to that particular isolate (Roelfs *et al.*, 1992). At certain temperatures, inoculum densities, tissue types, host growth stages or host nutrition levels, the resistance may be ineffective or just not expressed, but the resistance gene remains (Roelfs *et al.*, 1992).

Stripe rust is best controlled by resistant wheat cultivars which have both major gene and polygenic resistance (Kurt, 2001). Investigations of wheat lines with varying levels of resistance indicated that several chromosomes were implicated in the control of resistance. A reduced dose of some of the chromosomes resulted in greater susceptibility to disease, whereas a reduced dose of others resulted in higher resistance expression (Johnson, 1992a).

The best prospect for breeding for durable resistance to stripe rust in wheat is to start with a cultivar for which there is reasonable evidence of durability, and ensure that the resistance selected is derived from this source (Johnson, 1978). In order to exploit durable resistance in breeding it would be beneficial to understand the genetic basis of the resistance. However, few cultivars with proven durable resistance to stripe rust have been investigated genetically (Johnson, 1992a). Wheat mutants present an interesting set of tools with which to study the genetics, physiology and biochemistry of developmentally regulated disease resistance.

Adult plant resistance (APR) has often been presented as a possible

source of durable resistance, and these mutants will allow the processes and genes involved in APR to be identified (Boyd and Minchin, 2001). A study indicated that many wild emmer wheat derivatives contained stripe rust resistance genes not existing in present-day bread wheat cultivars/lines (Sharma *et al.*, 1994). According to Danial, Kirigwi and Parlevliet (1995), it appears that the progression towards more complex races, especially for stripe rust, is inevitable for wheat-cereal rust pathosystems when the selection is for complete or near-complete resistance. Modern molecular techniques, which will improve the prospects of successful breeding for disease resistance, hold out the possibility of genetic linkage of important characters to easily assessable markers as well as for genetic transformation. However, they will not supplant the continuing need to apply well tried and established techniques of plant breeding for the foreseeable future, particularly with wide scale testing of materials, before they are released to farmers. The possible durability of disease resistance introduced by biotechnology will remain to be challenged by widespread use of cultivars possessing it (Johnson, 1992a).

Conventional resistance breeding methods rely on time consuming field and/or greenhouse screening with *P. striiformis* races. Molecular markers which are closely linked with target alleles, present a useful tool in plant breeding. These can help to detect the resistance genes of interest without the need of to carry out disease tests. PCR-based microsatellite markers are often

inherited in a co-dominant manner, and have excellent stability in comparison to RAPD markers. Furthermore, the PCR method used for microsatellites is easier, faster, cheaper and requires less DNA than the RFLP method (Ma *et al.*, 2001).

A range of designated and temporarily designated seedling genes, controlling stripe rust resistance, have been detected (Table 2) (Boshoff, 2000; http://www.umn.cdl.edu/ downloaded 20-05-2004). According to present records designated genes for stripe rust resistance genes have been numbered to *Yr35* (Z.A. Pretorius, personal communication).

Seedling resistance

Seedling resistance to wheat stripe rust is race-specific, and has proven to be short-lived (Johnson, 1992b). Much of the earlier work on breeding for resistance to rust was based upon the exploitation of simply inherited major genes that were expressed in seedlings and throughout the life cycle of the host plant. Most of these genes conferred high levels of resistance expressed as hypersensitive chlorotic flecks on plants exposed to infection. Plants carrying such genes could therefore be conveniently identified by tests carried out with precision on seedlings under glasshouse conditions. These tests are easy to conduct on a large scale, and are particularly convenient where it is desired to introduce resistance by back-crossing from an unadapted exotic source.

However, these major genes frequently interact in a gene-for-gene pattern with the pathogen. A series of otherwise promising varieties had to be withdrawn shortly before or after their release when their resistance to stripe rust failed. Others were still efficiently resistant, after increased pathogenicity occurred, to be used commercially, although sometimes only with the help of fungicides (Johnson and Lupton, 1987).

Seedling resistance is usually of a hypersensitive nature. Hypersensitivity is defined as the rapid death of the host cells surrounding the infected site and is accompanied by the restriction of the growth of the pathogen. Hypersensitivity of cereals to rust is commonly manifested as small necrotic flecks, in the centre of which some sporulation may occur. In contrast to the susceptible or high infection type, this is described as a resistant or low infection type (Parlevliet, 1985). The low infection types of the different genes expressed in seedlings include some which produce minute chlorotic flecks such as *Yr1*, *Yr8*, and *Yr10*, others that produce extensive necrosis with or without some sporulation such as *Yr7*, and others that give less consistent reactions sometimes ranging from a nonsporulating reaction to considerable sporulation and only slight chlorosis. *Yr2* and *Yr6* are included in this group, which may vary with the environment and also in response to the genetic background in which they occur. Some of the named genes are dominant but several are recessive at least in some crosses, including *Yr2*, *Yr6* and *Yr9* (Johnson, 1988).

Adult-plant resistance

APR cannot be identified in the seedling stage (Parlevliet, 1985), and is a potentially durable source of resistance (Johnson, 1992b). While the genetics of stripe rust APR has been studied, little is known of the physiology or biology of this type of resistance (Boyd and Minchin, 2001). Cool weather during the growing season can delay development of APR (Anonymous, 2003). The rate of stripe rust development after booting depends on the level of APR and the average temperature during the epidemic (Hovmøller, 2001).

One of the best described sources of APR to stripe rust is gene *Yr18.* This gene is completely linked with gene *Lr34*, known to confer durable leaf rust resistance. The level of resistance conferred by *Yr18* is usually not adequate when present alone (McIntosh, 1992). However, combinations of *Yr18* and two to four additional slow rusting genes result in adequate resistance levels in most environments (Singh and Rajaram, 1994). Genetic analyses indicated that the level of resistance improved with an increase in the number of these genes that individually have minor to intermediate but additive affects (Singh *et al.*, 2003). Testers for *Yr18* include the wheat lines RL6058, RL6077, Line 920, Condor and Jupateco 73R. The durable APR of Anza, which is related to Condor, is postulated to be attributed to *Yr18*, which is widespread in CIMMYT (International Maize and Wheat Improvement Center) germplasm and South

American wheats (Singh, 1992a). *Yr18* and *Lr34* are also known to be linked with gene *Ltn*, which confers a leaf tip necrosis in adult plants (Singh, 1992b). *Ltn* serves as a valuable marker for the linked genes (Singh, 1993).

Partial resistance. Partial resistance (PR) or quantitative resistance, also indicated as non-hypersensitive resistance, is a form of incomplete resistance whereby the individual lesions are characterized by a susceptible infection type (Parlevliet, 1985). It is often recessive and the result of the cumulative effects of several genes with small to intermediate effects.

The damage from virulent pathotypes on different cultivars depends on the general level of PR in such cultivars. For example, stripe rust on Hussar (*Yr9, Yr17*) and Ritmo (*Yr1*), both with additional PR expressed mainly at the adult plant stage, increased from zero to less than 20% of that of Anja (susceptible) in field trials. In contrast, the occurrence of virulence matching *Yr17* and *Yr9* resistance in cv. Brigadier, which does not have additional PR, led to an increase in disease severity from zero to about two times more than that observed on the susceptible control (Hovmøller, 2001).

The resistance in cv. Kraka (*Yr1*, CV) was increasingly effective in controlling stripe rust, because pathotypes with matching combination of virulence declined in the pathogen population. The pathotype frequency dynamics were thus influenced by selection forces within the country, and by

selection forces in areas where spores were spread to Denmark from outside. The importance of a sufficient level of partial resistance in the wheat germplasm to prevent too much damage by stripe rust epidemics, in the event that the resistance genes are overcome by the pathogen population, is emphasised (Hovmøller, 2001).

There are few host-pathogen systems where major, non-durable resistance genes are not present. This is also true for wheat and makes selection for partial resistance difficult (Parlevliet, 1993). A rightful PR breeding programme should therefore select against hypersensitive resistance and complete susceptibility, yet combine sources characterised by an increased latent period, reduced uredinium numbers and smaller uredinia in recurrent selection cycles (Parlevliet, 1993).

Symptom expression and assessment of resistance

An accurate assessment of resistance is essential in breeding for disease resistance, especially if quantitative resistance is applicable. Disease response for the cereal rust diseases can be assessed by either qualitative or quantitative means, or a combination of both (McIntosh *et al.*,1995).

For seedling scales, the infection type (IT) descriptions are based on the original scales proposed by Gassner and Straib (1932). Since then ITs have been described in slightly different ways (Roelfs, 1984). McNeal *et al.* (1971)

developed a 0 to 9 scale where 0 to 3 are classified as resistant, IT 4 to 6 as intermediate and IT 7 to 9 as susceptible. In the 0 to 4 IT scale of McIntosh *et al.* (1995), 0 reflects an immune host response, a fleck (;) reaction indicates a very resistant reaction and a ";n" reaction indicates a resistant reaction with accompanying necrosis. Roelfs (1985) mentioned that rust diseases could be assessed by enumerating numbers of uredinia (i.e. host receptivity), length of the latent period (i.e. time for pustule development) and duration of sporulation (McIntosh *et al.,* 1995).

For adult plant assessment the McNeal *et al.* (1971) scale has been used, whereas the Gassner and Straib (1932) scale is usually considered unsuitable for scoring adult plants. Peterson, Campbell and Hannah (1948) measured disease severity (DS) by estimating the percentage of tissue affected by the rust at a certain moment during the epidemic. The common approach under field conditions is to use the modified Cobb scale (Peterson *et al.*, 1948) as a quantitative measure of disease (McIntosh *et al.,* 1995) (Appendix1). A more labour intensive method of gaining a quantitative measure of disease is to use the area under disease progress curve (AUDPC). For cereal rusts, the major contribution made to the area value is the last one or two observations. Broers (1989) compared different methods to measure disease and concluded that DS and AUDPC were the most suitable parameters to measure the partial resistance to wheat leaf rust. Danial (1994) showed a high correlation between AUDPC

and DS for stripe rust. The benefit of multiple scores is the value of replication, and the likelihood of detecting early and potentially severe rusting genotypes (McIntosh *et al.,* 1995).

Several factors may interfere with an accurate assessment in the wheat/stripe rust pathosystem, e.g. interplot interference, nitrogen level, earliness, date of observation and leaf layer (Danial, 1994).

Temperature and light intensity

The expression of many genes is affected by environmental conditions such as light and temperature. Where this involves obligate pathogens it is often not possible to decide whether the observed affects is on the host, the pathogen or on the interaction between them (Johnson, 1992a). These effects from environmental conditions are more critical for stripe rust than for other cereal rusts (Stubbs, 1985).

Little is known about the effect of the environment on the epidemiological behaviour of quantitative resistance of spring wheat in different environments, but greenhouse studies showed that expression of quantitative resistance in winter wheat is sensitive to temperature (Qayoum and Line, 1985), resulting in a reduced expression at lower temperatures. According to Hovmøller (2001), the general level of stripe rust was strongly affected by weather conditions, which had an effect on winter survival of both the pathogen

and the host. In field observations at the Plant Breeding Institute, Cambridge, it was noted that most cultivars became less infected with stripe rust in warmer summers, even those which are too susceptible for commercial use (Johnson, 1992a).

High-temperature, adult-plant resistance (HTAP) to stripe rust has remained durable for at least 30 years in the USA (Schultz and Line, 1992). In contrast, cultivars that have only a moderate level of HTAP resistance, such as Nugaines, can be severely damaged by stripe rust in years with mild winters and cool, early spring temperatures. After booting, the apparent rates of infection on susceptible and moderately susceptible cultivars were positively correlated with the mean temperature during the period over which the rate was correlated, for the range 12.9-16.2°C (Hovmøller, 2001). Over this range, the apparent rate of infection of susceptible wheats increased at 0.045 per day per °C. From 16.2-20.3°C, the rate of susceptible wheats was negatively correlated with the mean temperature, and declined at 0.043 per day per °C. Murray *et al.* (1994) suggested that the final kernel mass is less influenced by the reduction in phytosynthetic area by stripe rust as temperature increases, because higher temperatures reduce the sink size. Another possibility is that the pathogen is less able to utilize host photosynthates at higher tempreatures.

Singh, Nelson and Sorrells (2000) identified and mapped a new gene from *Aegilops tauschii*, designated *Yr28* (located on wheat chromosome arm

4DS), that contributes to seedling and field resistance to the predominant race of stripe rust in the Mexican highlands and appears to increase in effectiveness at higher field temperatures. Stripe rust APR genes are commonly more effective at higher temperatures (Broers and López-Atilano, 1996). Temperature sensitivity is common in resistance to rust diseases of wheat and a particular form of temperature sensitivity, better expressed at high temperature, is not necessarily *per se* diagnostic for durable resistance (Johnson, 1992a).

Carstens V, Holzapfels Früh, and Chinese Spring were highly sensitive to changes in light intensity (Stubbs, 1985). Low light intensities gave susceptible reactions, whereas high light intensities yielded resistance reactions.

Plant nutrition

Fertility as an environmental factor may differ from soil to soil and year to year and might affect the assessment of resistance in breeding programs. Danial and Parlevliet (1995) found that stripe rust severity increased when wheat genotypes were exposed to higher N-levels, ranging from 0 to 90kg per ha.

Inter-plot interference

Screening for quantitative forms of resistance to airborne pathogens is generally carried out in small, adjacent plots (Danial, Broers, and Parlevliet, 1993). A fairly resistant entry may receive an abundance of inoculum if it has a highly

susceptible neighbour. The amount of pathogen on the fairly resistant entry can then be increased considerably, especially with airborne pathogens (Parlevliet, 1993). A representational error or inter-plot interference occurs then as an underestimation of the level of resistance and/or as an error in the ranking in the entries tested (Danial *et al.*, 1993).

Earliness and observation date

The difference in disease severity between similar host genotypes may be due to the difference in host growth stage. Susceptibility and resistance are often correlated with host growth stage even for race-specific resistance (Roelfs *et al.*, 1992). Both the rust development and the relationship to plant ontogeny, have an affect on the yield component.

According to Danial (1994), earliness tends to increase disease severity slightly, irrespective of the resistance level, but the confounding effect is small for stripe rust. Results indicate that early rust development negatively affected kernel number and that early infections (before jointing) can reduce the number of tillers per plant (Schultz and Line, 1992). Experiments done by Cook *et al.* (1999) provided indications that epidemics of foliar diseases initiated before flag leaf emergence had the greatest impact on yield. After this stage, yield loss averaged 27.2 $kg.ha^{-1}$ for each day that elapsed before fungicide was applied. From the end of heading to late milk, yield was significantly negatively

correlated with the proportion of leaf area affected by stripe rust. The correlation was greatest at the early milk stage of growth where the relationship was logarithmic with two factors significantly influencing this relationship. Yield loss increased as the length of the epidemic increased, and decreased as temperature increased during grain development (Murray *et al.*, 1994).

If the entries differ considerably in earliness the period of exposure to the pathogen varies as the assessment is usually done at the same moment for all entries. Resistance to head blight caused by *Fusarium* in wheat is considerably overestimated in late cultivars due to this aspect. The same is valid for *Septoria* leaf and glume blotch in wheat; the later the entry the lower the blotch scores (Parlevliet, 1993). However, the optimum plant growth stage for recording rust was different, depending on the population studied and the location (Schultz and Line, 1992).

Chemical control

Several fungicides are available that are effective, safe, and economical for use against rusts. In some cases, one spray may be sufficient, but depending on the compound, the weather and the length of the growing season, two or more sprays may be necessary (Knott, 1989).

Protectant fungicides safeguard plants against pathogen propagules establishing infections, but are ineffective against established infections, while

eradicant fungicides enter the plants, kill the established infections and also stop existing latent infections (Manners, 1993). The comparative efficacy of fungicides depends partly on the crop growth stage and disease level at the time of application, and may not accurately reflect their relative protectant or eradicant activity (Viljanen-Rollinson *et al.*, 2002).

Even though the resistance of the cultivar to the disease, as well as the type of fungicide used, are important factors to be considered (Viljanen-Rollinson *et al.*, 2002), the spray timing appeared to be more important than choice of fungicide (Cook *et al.*, 1999). Inappropriate timing can have important consequences: too soon and the protectant activity may have diminished before disease pressure is high; too late and the eradicant and reach-back activity will be insufficient to control the disease epidemic. Depending on disease pressure, fungicides on susceptible cultivars should be sprayed at or near flag leaf emergence (Viljanen-Rollinson *et al.*, 2002). The period from flag leaf emergence to ear emergence (G37-G59; Tottman, 1987) has been the recommended optimum growth stage for years (Anonymous, 1986). But, according to studies done by Cook *et al.* (1999), additional slightly earlier sprays are beneficial to ensure that the final three leaves remain disease-free from their emergence until natural senescence. These results show that yield loss owing to absence of treatment can occur from G32 (2nd node detectable), but is most consistent from about G37 (flag leaf just visible),

averaging 27 kg.ha^{-1} for each day by which treatment is delayed beyond that stage (Cook *et al.*, 1999). Since stripe rust generally occurs on the lower leaves first it may rapidly infect the upper leaves under favourable conditions. A heavy infection on a leaf will impede photosynthesis and grain fill, therefore it is important to protect the flag leaf, which provides about 50% of plant yield (Brown, 2002). It is also vital to ensure that wheat is well protected from disease, during and after stem extension and treatment should commence before the point at which yield starts to decline (Cook *et al.*, 1999).

Incorrect timing may also lead to poor apparent fungicide performance and excessive use of fungicides (Viljanen-Rollinson *et al.*, 2002). The potential exists for mutants to arise that are resistant to the fungicide used. These would have great selective advantage. However, with proper use of fungicides disease severities are kept low, thus reducing the numbers of urediniospores produced. Mutants produced will have little selective advantage on a susceptible host, and may be lost from the population by chance due to low disease levels (Stubbs, 1985).

A spring-sown field trial at Lincoln, New Zealand in the 2001/2002 growing season assessed the relative protectant and eradicant activity of the fungicides azoxystrobin and epoxiconazole for the control of wheat stripe rust. The AUDPC of flag leaves of the susceptible cultivar Tritea was reduced by 65% after application of azoxystrobin, and by 37% after application of epoxicanozole.

The AUDPC for the moderately susceptible cultivar Kamaru was reduced by 41-50% by both fungicides (Viljanen-Rollinson *et al.*, 2002). In South Africa, demethylation-inhibiting fungicides, including several triazole compounds, dominate fungicide use in the cultivation of both spring and winter wheat. One triazole seed treatment, six triazole fungicides and five triazole/ benzimidazole mixtures are registered for the control of stripe rust (Nel *et al.*, 1999). In a study done by Boshoff *et al.* (2003) combined seven and flag leaf treatments, over three susceptible cultivars, resulted in a 56% yield increase with the application of propiconazole (200 g a.i. ha^{-1}), followed by 49%, 44%, 39% and 25% for tebuconazole (187.5 g a.i. ha^{-1}), flutriafol (125 g a.i. ha^{-1}), bromuconazole (140 g a.i. ha^{-1}), and flusilazole (100 g a.i. ha^{-1}), respectively in the 1997 wheat season.

The economic importance of head infections in New Zealand were emphasized by Cromey (1989) who found that kernels from infected florets weighed up to 77% less than kernels from uninfected florets. Boshoff *et al.* (2003) found that head infection was reduced by only 8% when fungicides were applied at the seven leaf stage (GS 16 to 19). The best control of head infection was obtained with the application of triazole fungicides closely to, or just after head emergence (GS 49 to 59). Stripe rust head infection was reduced by 65% and 74% with a combined seven and flag leaf treatment and flag leaf treatments respectively (Boshoff *et al.* 2003).

Incidence of stripe rust on highly susceptible cultivars occurs despite the availability of effective fungicides because, due to weather or human factors, these cannot always be used optimally (Johnson, 1992a). According to Boshoff *et al.* (2003) the application of a triazole seed treatment to prevent the build-up of rust inoculum on susceptible cultivars during the early stages of growth, will reduce input costs in comparison to foliar sprays. Triazole seed treatment can either be used in combination with foliar treatments when the cultivars are highly susceptible to rust, or alone when yields are to low to justify foliar sprays (Line, 1993). Triticonazole (0.24 g a.i. kg^{-1} seed) can protect the crop up to the eight-leaf growth stage, but results from the study done by Boshoff *et al.* (2003), showed that triadimenol (0.23 g a.i. kg^{-1} seed) provided protection for a longer period. Triadimefon applied as a seed treatment, 0.25 g a.i. kg^{-1} seed, controlled foliar rusts through the tillering stage of plant growth (main shoot and nine or more tillers) (Rakotondradona and Line, 1984).

The best control of stripe rust was obtained with a combined seven and flag leaf treatment with propiconazole as well as triticonazole seed treatment combined with a seven and flag leaf treatment of propiconazole (Boshoff *et al.* 2003).

Cultural methods

Cultural practices provide an alternative measure for reducing risk of wheat rust epidemics. No single practice is effective under all conditions, but a series of

cultural practices greatly enhance the existing resistance (Roelfs *et al.*, 1992). The objective of cultural methods for rust control is to break the life cycle of the rust (Knott, 1989). Therefore, the removal of volunteer plants with strategic animal grazing, tillage or herbicides is an effective control measure for epidemics resulting from endogenous inoculum (Boshoff, 2000).

Resistance genes can be exploited through their guided distribution in space and time (gene deployment). Cultivars carrying the different resistance genes can be distributed within the same field, sown as a unit (the multiline or the cultivar mixture), or they can occur as different cultivars in different fields within the same farm (gene deployment at the farm level) (Parlevliet, 1993). Mixtures of different cultivars of a crop, without additional breeding for phenotypic uniformity have been shown to provide reductions of more than 50% of stripe rust in Oregon, USA (Finckh and Mundt, 1992). Five winter wheat cultivars, six two-component cultivar mixtures, and one four-way mixture were grown in the presence of stripe rust, eyespot, both diseases, and neither disease for three seasons (Mundt, Brophy and Schmitt, 1995). On average, mixtures reduced severity of stripe rust relative to their pure stands by 53%. Averaged over all years, the mixtures increased yield relative to the pure stands by 6.2% in the presence of stripe rust. The mixtures showed improved yield stability relative to the pure stands, with the four-component mixture being particularly stable (Mundt *et al.*, 1995). Such mixtures can greatly reduce the rate of epidemic development, as any given race of the pathogen will be

virulent on only part of the host population, and epidemic development will be greatly reduced in the secondary cycles (Browning and Frey, 1969; Mundt and Browning, 1985; Wolfe, 1985).

CONCLUSION

From reviewing the literature it is clear that a large body of information exists for wheat stripe rust. Despite the fact that stripe rust has only occurred in South Africa since 1996, researchers know which pathotypes occur, which *Yr* genes are effective, and which commercial cultivars have resistance to this disease. Furthermore, the host range has been determined and clear recommendations for chemical control formulated.

One area of research which has not been studied extensively is the genetics of resistance. As this knowledge is essential to keep abreast of the pathogen, more emphasis should be placed on genetic studies, whether traditional or molecular, to ultimately breed for durable stripe rust resistance. This disseration aims to optimise systems for phenotyping wheat accessions for adult plant resistance.

REFERENCES

Afshari, F. 2000. Studies on rust resistance in wheat with particular emphasis on stripe rust. Ph. D. thesis, The University of Sydney, Australia.

Al-Khesraji, T.O., and Lose, D.M. 1980. Intracellular structures of *Puccinia*

poarum on its alternate hosts. Transactions of the British Mycological Society 75: 397-411.

Altman, A. 1982. Retardation of radish leaf senescence by polyamines. Physiological Plant Pathology 54: 189-193.

Anonymous, 1986. Use of Fungicides and Insecticides on Cereals. MAFF Booklet 2257. Alnwick, UK: Ministry of Agriculture, Fisheries and Food.

Anonymous, 2003. Small Grain Wheat Diseases-Stripe Rust. Served from: http://pnw-ag.wsu.edu/smallgrains/Stripe%20Rust.html, 08-08-2003.

Boshoff, W.H.P. 2000. Control of foliar rusts of wheat in South Africa with special emphasis on *Puccinia striiformis* f. sp. *tritici.* Ph. D. thesis, University of the Free State, South Africa.

Boshoff, W.H.P., and Pretorius, Z.A. 1999. A new pathotype of *Puccinia striiformis* f. sp. *tritici* on wheat in South Africa. Plant Disease 83: 591.

Boshoff, W.H.P., Pretorius, Z.A., and Van Niekerk, B.D. 2003. Fungicide efficacy and the impact of stripe rust on spring and winter wheat in South Africa. South African Journal of Plant and Soil 20: 11-17.

Boyd, L.A., and Minchin, P.N. 2001. Wheat mutants showing altered adult plant disease resistance. Euphytica 122: 361-368.

Broers, L.H.M. 1989. Partial resistance to wheat leaf rust in 18 spring wheat cultivars. Euphytica 44: 247-258.

Broers, L.H.M., Cuesta Subías, X., and López Atilano, R.M. 1996. Field

assessment of quantitative resistance to yellow rust in ten spring bread wheat cultivars. Euphytica 90: 9-16.

Broers, L.H.M., and López-Atilano, R.M. 1994. A method for inoculating adult wheat plants with urediospores of *Puccinia striiformis* to measure components of resistance. Plant Disease 78: 353-357.

Broers, L.H.M., and López-Atilano, R.M. 1996. Effect of quantitative resistance in wheat on the development of *Puccinia striiformis* during early stages of infection. Plant Disease 80: 1265-1268.

Brown, D. 2002. Stripe Rust flourishes in Spring Wheat. This pdf was served from: http://cahenews.wsu.edu/RELEASES/2002/02045.htm, 08-08-2003.

Browning, J.A., and Frey, K.J. 1969. Multiline cultivars as a means of disease control. Annual Review of Phytopathology 14: 355-382.

Calonnec, A., Johnson, R., and De Vallavielle-Pope, C. 1997a. Genetic analysis of resistance to *Puccinia striiformis* in the wheat differential cultivars Heines VII, Heines Peko and Strubes Dickkopf. Plant Pathology 46: 373-386

Calonnec, A., Johnson, R., and De Vallavielle-Pope, C. 1997b. Identification and expression of the gene *Yr2* for resistance to *Puccinia striiformis* in the wheat differential cultivars Heines Kolben, Heines Peko and Heines VII. Plant Pathology 46: 387-396

Chen, X.M., Jones, S.S., and Line, R.F. 1995a. Chromosomal location of genes for stripe rust resistance in spring wheat cultivars Compair, Fielder, Lee,

and Lemhi and interactions of aneuploid wheats with races of *Puccinia striiformis*. Phytopathology 85: 375-381.

Chen, X.M., Line, R.F., and Leung, H. 1995b. Virulence and polymorphic DNA relationships of *Puccinia striiformis* f. sp. *hordei* to other rusts. Phytopathology 85: 1335-1342.

Chen, X., Jones, S.S., and Line R.F. 1996. Chromosomal location of genes for resistance to *Puccinia striiformis* in seven wheat cultivars with resistance genes at the *Yr3* and *Yr4* loci. Phytopathology 86: 1228-1233

Cook, R.J., Hims, M.J., and Vaughan, T.B. 1999. Effects of fungicide spray timing on winter wheat disease control. Plant Pathology 48: 33-50.

Cromey, M.G. 1989. Occurrence and effects of stripe rust in wheat spikes in New Zealand. New Zealand Journal of Horticultural Science 17: 155-158.

Cummins, G.B., and Stevenson, J.A. 1956. A check list of North American rust fungi (Uredinales). Plant Disease Reporter 240: 109-193.

Danial, D.L. 1994. Aspects of durable resistance in wheat to yellow rust. Ph. D. thesis, CIP-Data Koninklijke Bibliotheek, Den Haag.

Danial, D.L., Broers, L.H.M., and Parlevliet, J.E. 1993. Does interplot interference effect the screening of wheat for yellow rust resistance? Euphytica 70: 217-224.

Danial, D.L., and Parlevliet, J.E. 1995. Effects of nitrogen fertilization on disease severity and infection type of yellow rust on wheat genotypes varying in quantitative resistance. Journal of Phytopathology 143:

679-681.

Danial, D.L., Kirigwi, F.M., and Parlevliet, J.E. 1995. Lack of durability of resistance to cereal rusts in wheat when selection is for complete resistance. Plant Breeding 114: 539-541.

Danial, D.L., and Stubbs, R.W. 1992. Virulence of yellow rust races and types of resistance in wheat cultivars in Kenya. Pages165-175 in: The Seventh Regional Wheat Workshop For Eastern, Central and Southern Africa, 1991. D.G. Tanner and W. Mwangi (eds). CIMMYT, Mexico.

Day, P.R. 1974. Genetics of Host-Parasite Interaction. W.H. Freeman and Company, San Francisco.

De Vallavieille-Pope, C., and Line, R.F. 1990. Virulence in North American and European races of *Puccinia striiformis* on North American, world and European differential wheat cultivars. Plant Disease 74: 739-743.

De Vallavieille-Pope, C., Huber, L., Leconte, M., and Goyeau, H. 1995. Comparative effects of temperature and interrupted wet periods on germination, penetration, and infection of *Puccinia recondita* f. sp. *tritici* and *P. striiformis* on wheat seedlings. Phytopathology 85: 409-415.

Eriksson, J., and Henning, E. 1894. Die Hauptresultate einer neuen Untersuchung Uber die Getreiderostpilze. Zeitung für Pflanzenkrankheiten 4: 197-203.

Eriksson, J., and Henning, E. 1896. "Die Getreideroste." Norstedt and Söner, Stockholm.

Finckh, M.R., and Mundt, C.C. 1992. Stripe rust, yield and plant competition in wheat cultivar mixtures. Phytopathology 82: 905-913.

Flor, H.H. 1946. Genetics of pathogenicity in *Melampsora lini*. Journal of Agricultural Research 73: 335-357.

Gassner, G., and Straib, W. 1932. Die bestimmung der biologischen rassen des weizengelbrostes (*Puccinia glumarum* f. sp. *tritici* (Schmidt.) Eriks. und Henn.). Arbeiten der Biologischen Reichsansalt für Land und Forstwirtschaft, Berlin 20:141-163.

Hassebrauk, K. 1965. Nomenklatur, geographisshe Verbreitung und Wirtsberech des Gelbrostes, *Puccinia striiformis* West. Mitt. Biol. Bundesanst Land-Forstwirtsch., Berlin-Dahlem 116: 1-75.

Heath, M.C. 1981. Resistance of plants to rust infection. Phytopathology 71: 971-974.

Heath, M.C. 1982. Host defence mechanisms against infection by rust fungi. Pages 223-245 in: The Rust Fungi. K.J. Scott and A.K. Chakrovorty (eds). Academic Press, London.

Heath, M.C. 1985. Implications of non-host resistance for understanding host-parasite interactions. Pages 25-42 in: Genetic Basis of Biochemical Mechanisms of Plant Disease. J.V. Groth and W.R. Bushnell (eds). APS Press, St Paul, Minnesota.

Hogg, W.H., Hounam, C.E., Mallik, A.K., and Zadoks, J.C. 1969. Meteorological factors affecting the epidemiology of wheat rusts. WMO Tech Note 99.

Hogenboom, N.G. 1993. Economic importance of breeding for disease resistance. Pages 5-9 in: Durability of Disease Resistance. Th. Jacobs and J.E. Parlevliet (eds). Kluwer Academic Publishers, The Netherlands.

Hovmøller, M.S. 2001. Disease severity and pathotype dynamics of *Puccinia striiformis* f. sp. *tritici* in Denmark. Plant Pathology 50: 181-189.

Hyde, P.M., and Elahinia, S.A. 1990. The expression of partial resistance to *Puccinia striiformis* in wheat. Effects on colony growth and spore production. Journal of Phytopathology 129: 203-209.

Johnson, R. 1978. Practical breeding for durable resistance to rust diseases in self-pollinating cereals. Euphytica 27: 529-540.

Johnson, R. 1988. Durable resistance to yellow (stripe) rust in wheat and its implication in plant breeding. Pages 63-75 in: Breeding Strategies for Resistance to the Rusts of Wheat. N.W. Simmonds and S. Rajaram (eds). CIMMYT, Mexico.

Johnson, R. 1992a. Past, present and future opportunities in breeding for disease resistance, with examples from wheat. Euphytica 63: 3-22.

Johnson, R. 1992b. Reflections of a plant pathologist on breeding for disease resistance with emphasis on yellow rust and eyespot of wheat. Plant Pathology 41: 239-254.

Johnson, R., and Lupton, F.G.H. 1987. Breeding for disease resistance. Pages 369-425 in: Wheat Breeding. Its Scientific Basis. F.G.H. Lupton (ed). Chapman and Hall Ltd., New York.

Kendrick, B. 1992. The Fifth Kingdom. Second edition. Focus Information Group Inc., Newburyport, MA.

Knott, D.R. 1989. The Wheat Rusts - Breeding for Resistance. Springer-Verlag, Heidelberg, Germany.

Komen, J.S., Van Niekerk, B.D., and Boshoff, W.H.P. 2002. Chemiese beheer van streeproes in die Oos-Vrystaat (Chemical control of stripe rust in the Eastern Free State). Wheat Focus 20.4: 28-30.

Kurt, S. 2001. Screening of wheat cultivars for resistance to stripe rust and leaf blotch in Turkey. Crop Protection 21: 495 - 500.

Line, R.F. 1993. Integrated pest management for wheat: IPM in a wide-range system. Plant Disease 77: 303-307.

Littlefield, L.J. 1981. Biology of Plant Rusts. Iowa State University Press, Ames.

Lupton, F.G.H. 1987. Wheat Breeding. Its Scientific Basis. Chapman and Hall Ltd., New York.

Ma, J., Zhou, R., Dong, Y., Wang, L., Wang, X., and Jia, J. 2001. Molecular mapping and detection of yellow rust resistance gene *Yr26* in wheat transferred from *Triticum turgidum* L. using microsatellite markers. Euphytica 120: 219-226.

Manners, J.G. 1960. *Puccinia striiformis* Westend. var. *dactylidis* var.nov. Transcript of the British Mycological Society 43: 65-68.

Manners, J.G. 1993. Principles of Plant Pathology. Second edition.

Cambridge University Press, Cambridge, UK.

McIntosh, R.A. 1992. Close genetic linkage of genes conferring adult-plant resistance to leaf rust and stripe rust in wheat. Plant Pathology 41: 523-527.

McIntosh, R.A., Wellings, C.R., and Park, R.F. 1995. Wheat Rusts: An Atlas of Resistance Genes. CSIRO, East Melbourne, Australia.

McIntosh, R.A., Hart, G.E., Devos, K.M., Rogers, J., and Gale, M.D. 1998. Catalogue of gene symbols for wheat: 1997 supplement. Wheat Information Service 86: 54-91

McNeal, F.H., Conzak, C.F., Smith, E.P., Tade, W.S., and Russel, T.S. 1971. A uniform system for recording and processing cereal research data. USDA, ARS Bulletin.

Mundt, C.C., Brophy, L. S., and Schmitt, M. S. 1995. Disease severity and yield of pure-line wheat cultivars and mixtures in the presence of eyespot, yellow rust, and their combination. Plant Pathology 44: 173-182.

Mundt, C. C., and Browning, J. A. 1985. Genetic diversity and cereal rust management. Pages 527-560 in: The Cereal Rusts. Vol. 2. A.P. Roelfs and W.R. Bushnell (eds). Academic Press, Orlando.

Murray, G.M., Ellison, P.J., Watson, A., and Cullis, B.R. 1994. The relationship between wheat yield and stripe rust as effected by length of epidemic and temperature at the grain development stage of crop growth. Plant Pathology 43: 397-405.

Nel, A., Krause, M., Ramautar, N., and Van Zyl, K. 1999. A Guide for the Control of Plant Disease. National Department of Agriculture, Pretoria.

Niks, R.E., and Dekens, R.G. 1987. Histological studies on the infection of triticale, wheat and rye by *Puccinia recondita* f.sp. *tritici* and *P. recondita* f.sp.*recondita*. Euphytica 36: 275-285.

Parlevliet, J.E. 1985. Resistance of the non-race-specific type. Pages 501-525 in: The Cereal Rusts. Vol. 2. A.P. Roelfs and W.R. Bushnell (eds). Academic Press, Orlando.

Parlevliet, J.E. 1993. What is durable resistance: a general outline. Pages 23-39 in: Durability of Disease Resistance. Th. Jacobs and J.E. Parlevliet (eds). Kluwer Academic Publishers, The Netherlands.

Peterson, R.F., Campbell, A.B., and Hannah, A.E. 1948. A diagrammatic scale for estimating rust intensity on leaves and stems of cereals. Canadian Journal of Research Section C 26: 496-500.

Pretorius, Z.A., Boshoff, W.H.P, and Kema, G.H.J. 1997. First report of *Puccinia striiformis* f. sp. *tritici* on wheat in South Africa. Plant Disease 81: 424.

Pretorius, Z.A., Bender, C.M., and Van der Linde, Z. 2001. Analysis and improvement of resistance in bread wheat to stripe rust caused by *Puccinia striiformis* f. sp. *tritici*. Final Report to Winter Cereal Research Trust, South Africa.

Qayoum, A., and Line, R.F. 1985. High-temperature, adult-plant resistance to stripe rust of wheat. Phytopathology 75: 1121-1125.

Rakotondradona, R., and Line, R.F. 1984. Control of stripe rust and leaf rust of wheat seed treatments and effects of treatments on the host. Plant Disease 68: 112-117.

Rizvi, S.A., Schubert, T.S., and Dixon, W.N. 2003. Pest Alert - Stripe Rust of Wheat in Florida. Served from: http://www.doacs.state.fl.us/-pi/enpp/pathology/striperust.html, 11-12-2003.

Roelfs, A.P. 1984. Race specificty and methods of study. Pages 131-161 in: The Cereal Rusts. Vol. 1. A.P. Roelfs and W.R. Bushnell (eds). Academic Press, Orlando.

Roelfs, A.P. 1985. Wheat and rye stem rust. Pages 3-37 in: The Cereal Rusts. Vol. 2. A.P. Roelfs and W.R. Bushnell (eds). Academic Press, Orlando.

Roelfs, A.P., Singh, R.P., and Saari, E.E. 1992. Rust Diseases of Wheat: Concepts and Methods of Disease Management. Mexico, D.F.: CIMMYT.

Rubiales, D., and Niks, R.E. 1992. Histological responses in *Hordeum chilense* to brown and yellow rust fungi. Phytopathology 41: 611-617.

Russell, B., Murray, G., and Sutherst, B. 2000. Wheat Stripe Rust-Reports. Served from:http://www.ento.csiro.au/research/pestmgmt/IPM ModelingNetwork/wheat_rust_repo,11-12-2003

Sache, I., and De Vallavieille-Pope, C. 1993. Comparison of the wheat brown and yellow rusts for monocyclic sporulation and infection processes, and their polycyclic consequences. Journal of Phytopathology 138: 55-65.

Savile, D.B.O. 1984. Taxonomy of the cereal rust fungi. Pages 79-112 in: The Cereal Rusts. Vol. 1. A.P. Roelfs and W.R. Bushnell (eds). Academic Press, Orlando.

Schmidt, J.K. 1827. "Allgemeine okonisch-technische Flora oder Abbildungen und Beschreibungen aller in bezug auf Okonomie und Technologie, merkwurdigen Gewachse". Vol. 1. Jena, Germany.

Schultz T.R., and Line, R.F. 1992. High-temperature, adult plant resistance to wheat stripe rust and effects on yield components. Agronomy Journal 84: 170-175.

Scott, K.J., and Chakravorty, A.K. 1982. The Rust Fungi. Academic Press, London.

Sharma, S., Louwers, J.M., Karki, C. B., and Snijders, C.H.A. 1994. Postulation of resistance genes to yellow rust in wild emmer wheat derivatives and advanced wheat lines from Nepal. Euphytica 81: 271-277.

Singh, R.P. 1992a. Genetic association of leaf rust resistance gene *Lr34* with adult-plant resistance to stripe rust in bread wheat. Phytopathology 82: 835-838.

Singh, R.P. 1992b. Association between gene *Lr34* for leaf rust resistance and leaf tip necrosis in wheat. Crop Science 32: 874-878.

Singh, R.P. 1993. Genetic association of gene *Bdv1* for tolerance to barley yellow dwarf virus with genes *Lr34* and *Yr18* for adult-plant resistance to rust in bread wheat. Plant Disease 77: 1103-1106.

Singh, H., and Johnson, R. 1988. Genetics of resistance to yellow rust in Heines VII, Soissonais and Kalyansona. Pages 885-890 in: Proceedings of the Seventh International Wheat Genetics Symposium. T.E. Miller and R.M.D. Koebner (eds). IPSR, Cambridge.

Singh, R.P., Nelson, J.C., and Sorrells, M. E. 2000. Mapping *Yr28* and other genes for resistance to stripe rust in wheat. Crop Science 40: 1148-1155.

Singh, R.P., and Rajaram, S. 1994. Genetics of adult plant resistance to stripe rust in ten spring bread wheats. Euphytica 72:1-7.

Singh, R.P., Rajaram, S., Huerta-Espino, J., and William, M. 2003. Durable Resistance to Yellow (Stripe) Rust in Wheat. Served from: http://www.cimyt.org/Research/Wheat/map/research_results/ResHighlights/pdfs/resHigh_DurabResist.pdf, 06-11-2003.

Snape, J.W. 1987. Conventional methods of genetic analysis in wheat. Pages 109-128 in: Wheat Breeding. Its Scientific Basis. F.G.H. Lupton (ed). Chapman and Hall Ltd., New York.

Steele, K.A., Humphreys, E., Wellings, C.R., and Dickinson, M. J. 2001. Support for a stepwise mutation model for pathogen evolution in Australasian

Puccinia striiformis f. sp. *tritici* by use of molecular markers. Plant Pathology 50: 174-180.

Stubbs, R.W. 1985. Stripe rust. Pages 61-91 in: The Cereal Rusts. Vol. 2. A.P. Roelfs and W.R. Bushnell (eds). Academic Press, Orlando.

Tarr, S.A.J. 1972. Principles of Plant Pathology. The Macmillan Press, London.

Tottman, D.R. 1987. Decimal codes for the growth stages of cereals with illustrations. Annals Applied Biology 110: 441-451.

Viljanen-Rollinson, S.L.H., Parkes, R.A., Armour, T., and Cromey, M.G. 2002. Fungicide control of stripe rust in wheat: protection or eradication? Pages 336-340 in: 55th Conference Proceedings (2002) of the New Zealand Plant Protection Society Incorporated. Served from: http://www.hortnet.co.nz, 08-08-2003.

Wellings, C.R., Wright, D.G., Keiper, F., and Loughman, R. 2003. First detection of wheat stripe rust in Western Australia: evidence for a foreign incursion. Australian Plant Pathology 32: 321-322.

Wolfe, M.S. 1985. The current status and prospects of multiline cultivars and variety mixtures for disease resistance. Annual Review of Phytopathology 23: 251-273.

Zadoks, J.C. 1961. Yellow rust on wheat. Studies in epidemiology and physiologic specialization. *Tijdschrift Over Plantenziekten* 67: 69-256.

Zadoks, J.C., and Schein, R.D. 1979. Epidemiology and plant disease

management. Oxford University Press, Oxford.

Table 1. Seedling infection types produced by the World (1 to 9) and European (10 to 17) differentials, and supplemental tester lines (18 to 42) to pathotypes 6E16A- and 6E22A- of *Puccinia striiformis* f. sp. *tritici*[a]

Cultivar	*Yr* gene(s)	Low infection type[b]	Seedling response	
			pt. 6E16A-	pt. 6E22A-
Differentials				
1. Chinese 166	*1*	0;	0;	;
2. Lee	*7*	;n, 1n	4	4
3. Heines Kolben	*2*[c], *6*	;, n1	4	4
4. Vilmorin 23	*3a, 4a*[d]	;	;n	;n, 1c
5. Moro	*10, Mor*[e]	0;	;	;
6. Strubes Dickkopf	*Sd, 25*[f]	-	;c, 1cn	;c, 1cn
7. Suwon 92/Omar	*4, Su*	;	0;	0;, 1cn
8. Clement	*2, 9, 25*[f], *Cle*[e]	0;	0;	;
9. *Triticum spelta album*	*5*	0;, ;	0;	;
10. Hybrid 46	*4b*[h]	;	;	;
11. Reicherberg 42	*7, 25*[f]	;n, 1n	;1cn	4
12. Heines Peko	*2, 6, 25*[f]	;n, n1	;n	4
13. Nord Deprez	*3a, 4*[b]	;	;	;, ;c
14. Compare	*8, 19*[i]	0;, ;	4	4
15. Carstens V	*Cv*		;	;c
16. Spaldings Prolific	*Sp*		0;	0;
17. Heines VII	*2, 25*[f], *HVII*[e]	0;, 2	;c	;c, ;1c
Supplemental set				
18. *Yr1/6**AvS	*1*	0;	;	;
19. Kalyansona	*2*	0;, 2	4	4
20. *Yr5/6**AvS	*5*	0;, ;	0	0;
21. *Yr6/6**AvS	*6*	;, ;n1	3	3
22. *Yr7/6**AvS	*7*	;n, 1n	3	3
23. *Yr8/6**AvS	*8*	0;, ;	3	3

Table 1 (cont.). Seedling infection types produced by the World (1 to 9) and European (10 to 17) differentials, and supplemental tester lines (18 to 42) to pathotypes 6E16A- and 6E22A- of *Puccinia striiformis* f. sp. *tritici*[a]

Cultivar	*Yr* gene(s)	Low infection type[b]	Seedling response	
			pt. 6E16A-	pt. 6E22A-
Supplemental set (cont.)				
24. Federation/4*Kavkaz	*9*	0;	0;	0;
25. *Yr9/6* *AvS	*9*	0;	0	0;
26. *Yr10/6* *AvS	*10*	0;	;	0;
27. *Yr11/3* *AvS	*11*		3	3
28. Wembley	*14*[h]		3	3
29. *Yr15/6* *AvS	*15*	0;	0	;
30. Trident	*17*	;c, ;1	4	4
31. *Yr17/3* *AvS	*17*	;c, ;1	3	3
32. Jupateco R	*18*		4	4
33. *Yr18/3* *AvS	*18*		4	4
34. *Yr24/3* *AvS	*24*		;	;
35. *Yr26/3* *AvS	*26*		;	;
36. Selkirk	*27*[d]		1cn, 3	1cn, 3
37. Yr27/3*AvS	*27*		;, 1p=4	;
38. Avocet R	*A*	;cn1, 2+	;c, 1c	;c, 1c
39. YrSp/3*AvS	*Sp*		;	;
40. Avocet S			4	4
41. Federation 1221			4	4
42. Japateco S			4	4

[a]Boshoff (2000).
[b]McIntosh *et al*. (1995); [c]Calonnec, Johnson and De Vallavielle-Pope (1997b).
[d]McIntosh *et al*. (1998); [e]Chen, Jones and Line (1995b).
[f]Calonnec, Johnson and De Vallavielle-Pope (1997a); [g]not available.
[h]Chen, Jones and Line (1996). [i]Chen, Line and Leung (1995a).
[j]Hyde and Elahinia (1990).

Table 2. Designated and temporarily designated resistance genes for *Puccinia striiformis* f. sp. *tritici*, genome location, source and tester lines[a]

Yr gene	Genome location	Original source	Tester line	Other genes in tester	Remarks
Designated					
1	2A	Chinese 166	Chinese 166		
2	7B	Heines VII	Heines VII	*HVII*	
3a	1B	Vilmorin 23	Vilmorin 23	*V23*	
3b	1B	Hybrid 46	Hybrid 46	*4b, H46*	
3c	1B	Minister	Minister	*Min*	
4a	6B	Capelle-Deprez	Capelle-Deprez	*3a, 16*	
4b	6B	Hybrid 46	Hybrid 46	*3b, H46*	
5	2BL	*Triticum spelta album*	*T. spelta album*		
6	7BS	Heines Kolben	Heines Kolben	*2, HK*	
7	2BL	Lumillo durum	Lee	*Le1, Le2*	
8	2D	*T. comosum*	Compare	*Com*	
9	1BL	Imperial rye	Riebesel 47/51		
10	1BS	Moro	Moro	*Mor*	
11		Joss Cambier	Joss Cambier		APR
12		Caribo	Mega		APR
13		Ibis	Maris Huntsman		APR

Table 2 (cont.). Designated and temporarily designated resistance genes for *Puccinia striiformis* f. sp. *tritici,* genome location, source and tester lines[a]

Yr gene	Genome location	Original source	Tester line	Other genes in tester	Remarks
Designated					
14		Falco	Maris Bilo		APR
15	1BL	Dippes Triumph	*T. dicoccoides* G-25		
16	2DS	Capelle-Deprez	Capelle-Deprez	*3a, 4a*	APR
17	2AS	*T. ventricosa*	VPM1		test at 10ºC with high light
18	7DL	Frontana	Jupateco R 73R		APR
19	5B	Compair		*8*	see *YrCom*
20	6D	Fielder			see *YrFie*
21	1B	Lemhi			see *YrLem*
22	4D	Lee	Lee	*7, 23*	see *YrLe1*
23	6D	Lee	Lee	*7, 22*	see *YrLe2*
24	1B	K733 (durum)	K733		
25	1D	TP1295	Strubes Dickopf		
26	6AS	Haynaldia villosa	Yangmai-5		
27	2BS	Selkirk			
28	4DS	*T. tauschii W-219*			
29	1BL	Lalbahadur	Lalbahadur		APR

Table 2 (cont.). Designated and temporarily designated resistance genes for *Puccinia striiformis* f. sp. *tritici,* genome location, source and tester lines[a]

Yr gene	Genome location	Original source	Tester line	Other genes in tester	Remarks
Designated					
30	3BS		Opata 85		APR
Temporary designated					
A		Anza			
A		Avocet	Avocet		
A1		Gaines			APR
A2		NuGaines		*A1*	APR
A3		Luke		*A4*	APR
A4		Luke		*A3*	APR
A5		Durch		*A6a*	APR
A6		Durch		*A5*	APR
A7		Stephens		*A8*	APR
A8		Stephens		*A7*	APR
Ab		Alba	Alba	*Alb*	APR
Alb		Alba	Alba	*Ab*	
B-a	R	Bersee	Bersee	*14, B-b, B-c*	APR
B-b		Bersee	Bersee	*14, B-a, B-c*	APR

Table 2 (cont.). Designated and temporarily designated resistance genes for *Puccinia striiformis* f. sp. *tritici,* genome location, source and tester lines[a]

Yr gene	Genome location	Original source	Tester line	Other genes in tester	Remarks
Temporary designated					
B-c		Bersee	Bersee	*14, B-a, B-b*	APR
C		Yecora Rojo			APR
CaV		Zdar	Zdar	*4b, 5*	
Ck		Cook, Oxley	Cook		
Cle	4B	Clement	Clement	*9*	
Com	5B	Compair	Compair	*8*	see *Yr19*
CV1		Carstens V	Carstens V	*12, CV2, CV3*	
CV2		Carstens V	Carstens V	*12, CV1, CV3*	
CV3		Carstens V	Carstens V	*12, CV1, CV2*	
D	6A	Druch	Druch	*3a, Dru*	
D		Yecora Rojo			
Da1	1A	Daws	Daws	*Da2*	
Da2	5D	Daws	Daws	*Da1*	
Dru	5B	Druch	Druch	*3a, D*	
DT		DippesTriumph	DippesTriumph		APR
E	3E	*Elytrigia* (*Agropyron elongatum*)	Cns sub line	*Yr18*	

Table 2 (cont.). Designated and temporarily designated resistance genes for *Puccinia striiformis* f. sp. *tritici,* genome location, source and tester lines[a]

Yr gene	Genome location	Original source	Tester line	Other genes in tester	Remarks
Temporary designated					
EDC		Etoile de Choisy	Etoile de Choisy		APR
Falco		Falco	Falco		APR
Fie	6D	Fielder	Fielder	*6*	see *Yr20*
Fmg		Flamingo	Flamingo		APR
G		Gaby	Gaby		
H		Anza			APR, recessive
Hr		Harrier	Harrier		APR
H Peko		Heines Peko	Heines Peko		APR
H4		Heines IV	Heines IV		
H46	6A	Hybrid 46	Hybrid 46	*3b, 4b*	
H52	1BL	*T. turgidum dicoccoides*			
HVII	4A	Heines VII	Heines VII	*2*	
J		Glennson 81			
K733	1B	K733 durum	K733		
KK-1		Kenya Kubangu			
KK-2		Kenya Kubangu			

Table 2 (cont.). Designated and temporarily designated resistance genes for *Puccinia striiformis* f. sp. *tritici,* genome location, source and tester lines[a]

Yr gene	Genome location	Original source	Tester line	Other genes in tester	Remarks
Temporary designated					
Kg1		King	Flinders		APR
Kg2		King	King	*Kg1*	APR
L		Ollanta			
LD		Langs Dfoerfler 5111-41-173	Langs Dfoerfler 5111-41-173		
Le1	4D	Lee	Lee	*7, Le2*	see *Yr22*
Le2	6D	Lee	Lee	*7, Le1*	see *Yr23*
Lely		Lely	Lely		APR
Lem	1B	Lemhi	Lemhi		
Luq	2B	Luqiyu	Luqiyu		
Min	4A	Minster	Minster	*3c*	
Mor	4B	Moro	Moro	*10*	
ND	4A	Nord Deprez	Nord Deprez	*3a*	
Opal		Opal	Opal		APR
P1		Pavon 76	Pavon 76	*P2*	partial additive APR
P2		Pavon 76	Pavon 76	*P1*	partial additive APR
Pa1		Paha	Paha	*Pa2, Pa3*	

Table 2 (cont.). Designated and temporarily designated resistance genes for *Puccinia striiformis* f. sp. *tritici,* genome location, source and tester lines[a]

Yr gene	Genome location	Original source	Tester line	Other genes in tester	Remarks
Temporary designated					
Pa1		Paha	Paha	*Pa1, Pa3*	
Pa1		Paha	Paha	*Pa2, Pa3*	
Pr1		Produra	Produra	*Pr1, Pr2*	
Pr2		Produra	Produra	*Pr1, Pr3*	
Pr3		Produra	Produra	*Pr1, Pr2*	
S	3B	Stephens	Stephens	*3a, Ste*	
SD	-	Strubes Dickkopf	Strubes Dickkopf		
Slk	2B	Selkirk	Selkirk		see *Yr27*
Sp	-	Spaldings Prolific	Spaldings Prolific		
Ste	2B	Stephens	Stephens	*3a, S*	
Su92	2B	Suwon 92/ Omar	Suwon 92/ Omar		
T1		Tonichi 81	Tonichi 81	*18, T2*	additive APR
T2		Tonichi 81	Tonichi 81	*18, T1*	additive APR
Tr1	6D	Tres	Tres	*Tr2*	
Tr2	3A	Tres	Tres	*Tr1*	
Tye	6D	Tyee	Tyee		

Table 2 (cont.). Designated and temporarily designated resistance genes for *Puccinia striiformis* f. sp. *tritici,* genome location, source and tester lines[a]

Yr gene	Genome location	Original source	Tester line	Other genes in tester	Remarks
Temporary designated					
V23	6A	Vilmorin 23	Vilmorin 23	*3a*	
Yam	4B	Yamhill	Yamhill	*2, 4a*	
Z		Zaragoza 75	Zaragoza 75		APR[b]

[a] http://www.cdl.umn.edu/res_gene/wstr.html downloaded 20-05-2004.

[b] APR - Adult-plant resistance.

TOWARDS IMPROVEMENT OF THE DETECTION OF ADULT-PLANT RESISTANCE TO STRIPE RUST IN WHEAT

INTRODUCTION

Stripe rust (caused by *Puccinia striiformis* Westend. f. sp. *tritici*) is a serious disease of wheat (*Triticum aestivum* L.) in many parts of the world (Viljanen-Rollinson and Cromey, 1998) and has become endemic in South Africa after being observed for the first time in the Western Cape during August 1996 (Pretorius, Boshoff and Kema, 1997). Despite the widespread application of chemicals during the seasons following this introduction (R28 million's worth in the Western Cape in 1996, R18 million and R6 million in the Free State in 1997 and 1998), significant crop losses occurred, varying from 5 to 50% (Boshoff, 2000). A substantial amount of these losses could be ascribed to the reluctance of many farmers to invest in expensive fungicide application against a new and unknown disease. Although more efficient chemical control procedures have been developed (Boshoff, 2000), the use of resistant cultivars remains the best control method. This substantiates the approach in other countries where breeding for resistance against stripe rust has been a high priority (Johnson, 1992).

Inherited resistance is a valuable attribute because it is easy for the grower to use and reduces the need for other methods of control. Seedling resistance to stripe rust is race-specific and has proven to be short-lived (Johnson, 1992). Effective and durable stripe rust resistance is mainly of the post-seedling type (McIntosh, 1992), also referred to as adult plant resistance

(APR). APR develops progressively at later growth stages, is often quantitative (i.e. partial), and is based on single to many genes (Johnson, 1992). It has frequently been presented as a possible source of durable resistance (Boyd and Minchin, 2001), resistance that remains effective in cultivars grown over wide areas in environments favourable to disease over long periods of time (Johnson, 1992).

Breeding for resistance to this disease presents significant challenges which are unique considering the other wheat rusts (Johnson, 1992). Conventional resistance breeding relies on time consuming field and/or glasshouse screening with *P. striiformis* (Ma *et al.*, 2001). Seedling resistance can easily be detected in glasshouse studies, but APR is difficult to detect and better expressed in the field (Boshoff, 2000). The objectives of this study were to investigate the possibility of detecting adult plant resistance to stripe rust in wheat seedlings, and to optimise a system of accurate and reliable screening of APR for stripe rust reaction in a controlled environment.

MATERIALS AND METHODS

Wheat and pathogen materials

Thirteen South African and 21 Australian wheat entries were selected as an experimental population based on their resistance (adult plant or seedling) or susceptibility to stripe rust**.** The following conditions apply to all experiments conducted, unless otherwise stated: plants were grown in a steam-sterilized, soil-peat mixture in a disease-free glasshouse cubicle at 24.6±0.5°C/19.5±0.5°C with a 14/10 h day/night cycle. Natural daylight was supplemented with 120 $\mu molm^{-2}s^{-1}$ photosynthetically active radiation (PAR) emitted by cool-white fluorescent tubes, arranged directly above the plants. Seven days after planting, followed by weekly applications, all wheat treatments received 50 ml of 3 g/l hydroponic nutrient solution (6.5-2.7-13 N-P-K plus micro-elements) per pot as a soil drench. Fertilisation continued until

experiments were terminated. All experiments were conducted with fresh spores of *P. striiformis* f. sp. *tritici.* Urediniospores of the different pathotypes were multiplied, in advance, in isolation on seedlings of selective wheat hosts (Fig. 1). Inoculated wheat plants were incubated at 11±1°C and >96 % relative humidity in a dew chamber for 48 h. Initial experiments were conducted with pathotype 6E16A-, whereas most comparisons between glasshouse and field studies were done with 6E22A-. Since 6E16A- is more avirulent than 6E22A-, it was argued that the former pathotype would be more appropriate in detecting resistance in seedlings.

Glasshouse experiments

Seedling tests

***Experimental population*.** Primary leaf reactions of the 34 cultivars were tested to determine whether APR could be detected in seedlings. Five entries were sown in clumps (5-10 seeds/entry) per 10-cm-diameter pot . When the primary leaves had fully expanded and before inoculation, all treatments were subjected to a specific pre-conditioning period (Table 1) for 24 h. Seedlings were sprayed with a mineral oil suspension of 42 $\times 10^4$ spores/ml (pathotype 6E16A-) and incubated as mentioned above. Upon completion of the incubation period, treatments were conditioned at specific temperature and light conditions (Table 1). Thereafter, the seedlings were moved back to a glasshouse cubicle where a 14/10 h day/night cycle of 17.3±0.7°C/15.8±0.1°C was maintained. Infection types were recorded 14 days after inoculation, using a 0 to 4 scale (Appendix 1) (McIntosh, Wellings and Park, 1995). All seedling infection types were confirmed in independent experiments.

***CIMMYT lines*.** As a reference for adult plant responses, the seedling reactions of the 23rd and 24th CIMMYT Elite Spring Wheat Yield Trial (ESWYT's, 49 pure lines) were tested to pathotype 6E22A- of *P. striiformis*. f. sp. *tritici*. Two control cultivars (Steenbras and Morocco) were included. Prior to inoculation (42 $\times 10^4$ spores/ml oil) all seedlings were conditioned at 18°C for 24 h (200 $\mu molm^{-2}s^{-1}$ PAR) in a growth chamber. Upon completion of the

incubation period, the seedlings were dried for 3 h in a growth chamber at 18°C and moved to a glasshouse cubicle (17.3±0.7°C/15.8±0.1°C with a 14/10 h day/night cycle). Infection types were recorded as explained above and confirmed in a repeat experiment.

Normal adult plant tests

Experience has shown that flag leaves of mature wheat plants do not respond well to prolonged periods of leaf wetness at ~10°C (Z.A. Pretorius, personal communication). Five sets of the wheat entries Avocet S (susceptible check), Kariega, Leichhardt, SST88 and Sunlyn were planted in 1-liter-capacity pots (two seeds per pot). Plants were grown in a disease free cubicle at 22.0±0.2°C/16.4±0.5°C in a 14/10 h day/night cycle. Twelve weeks after planting foliage was inoculated with an urediniospore suspension (12 $\times 10^4$ spores/ml sterile, distilled water) of pathotype 6E22A- containing Tween 20® as surfactant. Each set was incubated at 11±1°C and >96 % relative humidity in a dew chamber for 6 h, 12 h, 24 h, 36 h and 48 h, respectively. After incubation the plants were dried for 3 h in an air-conditioned room before being placed in a glasshouse cubicle where a 14/10 h day/night cycle was maintained at 19.2±1.3°C/15.4±0°C. Twenty-five days later the flag leaf infection types (0 - 4 scale) and whole plant reaction to stripe rust (HR, R, RMR, MR, MRMS, MS, MSS and S [Appendix 1]) were rated (McIntosh *et al.*, 1995).

Mini-adult plant tests

Mini-adult wheat plants refer to those raised in a manner that will accelerate plant growth. The objective of this approach is to reduce the time needed to obtain adult plant data. By manipulating plant density, pot size, light and temperature, wheat plants will reach maturity quicker than those grown as described in the previous section (Pretorius, Park and Wellings, 2000). When subjected to these conditions wheat plants are considerably smaller in stature, thus the term “mini-adults”.

***Growth stage effects*.** To determine the optimum growth stage for

inoculation, three sets of 12 selected entries were grown at 25°C under continuous light (200 $\mu molm^{-2}s^{-1}$ PAR emitted by fluorescent tubes and incandescent bulbs arranged 30 cm above the plants) in a growth chamber. Six to eight seeds were sown in tapered plastic cones (4 x 4 x 10 cm**,** 95 cm^3) filled with sterilized soil. Sets were planted 10 days apart. Fertilisation started 20 days after planting, followed by weekly applications. Inoculation took place when the first set was four weeks old (Zadoks growth stage 39, Stubbs *et al.,* 1986), the second set five weeks old (Zadoks growth stage 57) and the third set six weeks (Zadoks growth stage 64) old. The plants were inoculated with a water suspension (25 $\times 10^4$ spores/ml) of pathotype 6E16A- containing Tween 20®, and incubated as previously described. After incubation the plants were dried for 3 h in an air-conditioned room before being placed in a glasshouse cubicle where a 14/10 h day/night cycle was maintained at 19.1±0.5°C/15.4±0°C. Daylight was supplemented with 120 $\mu molm^{-2}s^{-1}$ PAR. Fifteen days later the flag leaves (0 - 4 scale) as well as the whole plant reaction to stripe rust (HR, R, RMR, MR, MRMS, MS, MSS and S) were rated. The same rating procedure was applied to all the mini-adult experiments.

Experimental population**.** A set of the 34 entries was grown in a growth chamber to compare their responses with field results, thereby determining the feasibility of using this procedure.

Growth chamber and glasshouse comparison**.** Twelve entries were grown at 25°C under continuous light in a growth chamber. A duplicate set was grown at 24.6±0.5°C/19.5±0°C in a glasshouse. Five weeks after sowing the plants (Fig. 2) were inoculated with a water suspension (25 $\times 10^4$ spores/ml) of pathotype 6E16A- containing Tween 20®, and incubated as described above. Post-incubation and assessment procedures as mentioned above were used. Flag leaf and stem dimensions were measured to quantify plant development in both environments at the time of inoculation.

Pathotype effects**.** Four isolates, viz. 6E16A-, 6E22A-, 6E22A-(SG102) and

7E22A- of *P. striiformis* f. sp. *tritici* were tested separately on 12 entries in the glasshouse. The plants were grown at 24.6±0.5°C/19.5±0°C and inoculated six weeks after sowing with the four different isolates in water suspensions of 25 $\times 10^4$ spores/ml and Tween 20®, respectively. The incubation, post-incubation and assessment procedures were as mentioned above.

***Winter wheats*.** To test the feasibility of evaluating wheat genotypes that require vernalisation as mini-adults, seed of eight South African winter wheat cultivars were surface-sterilised and germinated in seed trays (95 cm^3 cones) filled with vermiculite (Fig. 3 A) at 11±1°C. After six weeks, germlings were transplanted to seedtrays (95 cm^3 cones) filled with sterilized soil (Fig. 3 B) and kept at 24.6±0.5°C/19.5±0°C with a 14/10 h day/night cycle. Two spring wheats, Baviaans (resistant) and Morocco (susceptible), were included as controls. Six weeks later the plants were inoculated with a water suspension (25 $\times 10^4$ spores/ml) of pathotype 6E22A- containing Tween 20®. The incubation and post-incubation, and assessment procedures as mentioned above were used.

***CIMMYT lines*.** Flag leaf reactions to pathotype 6E22A- of entries in the 23rd and 24th CIMMYT ESWYT grown as mini-adults in seedling cones in a glasshouse as described, were determined. The experiments were repeated in independent inoculation tests.

Field experiments

Experimental population

To serve as control for the different pre- and post-inoculation treatments of seedlings, the response of the 34 test wheats was determined in a field plot at Greytown, KwaZulu-Natal in 2002. A stripe rust epidemic was initiated by inoculating spreader rows, consisting of a mixture of susceptible cultivars, with pathotype 6E22A-. Three spreader rows surrounded the entire plot area whereas single spreader rows were planted perpendicular (in pathways) to all trial entries. Furthermore, Morocco was included after every 10 th entry to

facilitate stripe rust development among plots. Trial entries were planted in 2-m-rows spaced 90 cm apart (ca. 60 seeds per row). Disease severity for each cultivar was assessed from visual scores according to the modified Cobb Scale (0-100%) (Peterson, Campbell and Hannah, 1948) combined with a field reaction type (Appendix 1).

CIMMYT line evaluation

To validate the accelerated screening system (mini-adults), the flag leaf response of entries in the 23rd and 24th CIMMYT ESWYT's was determined in field plots at Greytown in 2002 and 2003, respectively. Baviaans (resistant), Kariega (resistant), Morocco (susceptible) and Steenbras (resistant) were included as controls. The trials were conducted as described in the preceding section.

RESULTS

Glasshouse experiments

Seedlings tests

***Experimental population*.** Seedling infection types obtained after exposing the test population to different pre- and post-infection environments are shown in Tables 2 and 3. In the set grown in the glasshouse and subjected to 24 h light immediately prior to inoculation (treatment 2), 13 entries were more susceptible, and only two cultivars, Sunlyn and Wyuna, had a more resistant infection type than those detected in the control set (treatment 1) (Table 2, Fig. 4). The remaining cultivars generally responded similar to the control.

Treatment 3 was subjected to 24 h of darkness in the growth chamber at 18°C prior to inoculation. Twenty eight of the entries appeared more resistant than the control set. Characteristic of this treatment was the occurrence of infected leaf areas in bands, indicated by the suffix "b" (Table 2, Fig. 4). Two cultivars, Biedou and Cook, exhibited marginally more susceptible reactions than their unconditioned glasshouse controls. Corrigan C and Swift had similar infection types to that of the control, but the formation of the infection was in bands. Olifants and Sunmist were the only two cultivars displaying the same infection type as the control.

Treatment 4, which consisted of 12 h light and 12 h darkness preconditioning at 18°C, was quite similar to treatment 3. The infection mostly occurred in bands (Fig. 4) and 29 of the cultivars had a more resistant reaction than the control. Corrigan C and Goroke were similar to the control, with the exception of banding. Compared to the control, infection types on Olifants and Wyuna were consistent. Tasman showed some necrosis at the leaf tips.

Treatment 5 was subjected to a 24 h light period at 25°C in the growth chamber before inoculation. No obvious effect was noted, either between this treatment and its counterpart preconditioned at 18°C (treatment 2), or the glasshouse control (Table 2, Fig. 5). In treatment 6 (24 h darkness at 25°C) infection was suppressed (Table 2) in most entries, including Avocet S, appearing more resistant than the control (Fig. 5). In pre-inoculation treatment 7 (12 h light and 12 h darkness at 25°C) 16 cultivars were more resistant and eight more susceptible than the glasshouse control. In some cases sporulation occurred in bands.

The remaining treatments (8 to 13, Table 1) were all subjected to a pre-inoculation conditioning of 24 h light in the growth chamber at 18°C, followed by different post-inoculation treatments (Table 3). When the dew period was succeeded by 12 h light and 12 h darkness at 18°C (treatment 8), 18 cultivars were more resistant and three more susceptible than the control (treatment 1)(Fig. 6). In treatment 9 (6 h light and 18 h darkness period at 18°C), reduced sporulation (indicated by minus and double minus) was observed for most entries (Table 3). None of the cultivars had the same reaction as the control. Fig. 7 illustrates differences in sporulation on Olifants in treatments 1, 8, 9 and 10.

Stripe rust development in treatment 10 (18 h light and 6 h darkness period at 18°C) was generally comparable to the glasshouse control. In treatments 11 (12 h light at 18°C and 12 h darkness at 25°C) and 12 (12 h light and 12 h darkness at 25°C) disease development was suppressed and pustules were generally smaller than those in the control (Figs. 6 and 8). With the exception of minor variation, treatment 13 (18 h light and 6 h darkness at 10°C) was comparable to the control. A resistant ;1c infection type was

observed for Sunstate as compared to the moderately susceptible 3++c in the control environment. Similarly, more resistant variants were detected within Biedou, Kariega (Fig. 9), Leichhardt and Sunbrook in treatment 13 than in the control.

CIMMYT lines. Forty nine advanced lines of both the 23rd and 24th CIMMYT ESWYT's were tested as seedlings in the glasshouse to determine their primary leaf infection types to pathotype 6E22A-. Seedling infection types from entries in the 23rd ESWYT ranged from highly resistant to susceptible. ESWYT entries 44, 48 and 50 were moderately susceptible to susceptible as seedlings (Table 4). In the 24th ESWYT primary leaf infection types varied from a highly resistant fleck (;) (Fig. 10 A) to susceptibility (4) (Table 5). Entries 2, 3, 5, 18, 21, 28, 37 and 39-50 (Figs. 10 B, 11 A and B) displayed moderate to high infection types. Several lines from both trials appeared heterogeneous for seedling reaction to stripe rust.

Normal adult plant tests

The results from testing normal glasshouse-grown adult plants are shown in Table 6. Extensive leaf damage occurred in most treatments and precluded meaningful rust phenotyping. The flag leaf infection types determined for Avocet S were accurate for all incubation periods, but except for 12 h (Fig. 12), no sporulation occurred on the rest of the plant precluding reaction type ratings.

Some flecking was visible on Kariega, SST88 and Sunlyn for different incubation periods, but infection types could not consistently be recorded. Leichhardt appeared more resistant to leaf damage and showed a ;cn infection type on all plants (Fig. 13). For cultivars allowing a rating of reaction type, 48 h of incubation were required.

Mini-adult plant tests

Growth stage effects. Plants from the first growth stage were clearly more susceptible to stripe rust (Table 7, Fig. 14 A-C). With the exception of Arnhem and SST876, all entries infection types from the first group were

susceptible. SST88, for example, showed a susceptible infection type (3++) on the terminal inoculated leaf, even though the rest of the plant reaction was moderately resistant. Likewise, the response of Kariega, which was at stage 55 (half of ear emerged) in the first group, was quite different from a later growth stage.

Considering overall plant growth and quality of infection types, the second growth stage was the most suitable. It was apparent that flag leaves of mini-adult plants, beyond flowering at inoculation, were not appropriate for reliable infection studies. Exceptions occurred but extensive chlorosis/necrosis complicated assessments.

SST876, which has hypersensitive seedling resistance to stripe rust, was the only cultivar with a consistent response at all three growth stages.

Experimental population. Reactions determined in the growth chamber are shown in Table 8 and presented under Field experiments.

Growth chamber and glasshouse comparison. Of the selection of 12 lines compared for accelerated growth and reliable disease scores in the growth chamber and the glasshouse, infection types could be determined on only Morocco and SST88 raised in the growth chamber (Table 9). Flag leaves of the remaining cultivars were nearly or totally necrotic (Fig. 15 A, B, and C). Whole plant reaction type was more reliable for rust assessment in the growth chamber. Based on reaction type Leichhardt and SST88 were more resistant in the growth chamber, most likely from a growth stage effect. Growth chamber-grown Kariega, Sunelg and Sunlyn were moderately resistant, compared to being resistant in the glasshouse. At the time of inoculation, glasshouse-grown plants were considerably slower in development rate than those in the growth chamber (Table 9).

The glasshouse plants were more vigorous than those in the growth chamber (Table 10). The glasshouse grown plants had larger (97 x 4.4 mm on average) flag leaves than the growth chamber grown plants (53.6 x 2.6 mm).

Stem thickness and plant height were fairly similar in both environments (Table 10, Figs. 16 - 19).

Pathotype effects. According to flag leaf infection type and whole plant reaction 6E16A- proved to be less virulent than the other three pathotypes, which were similar (Table 11). Pathotype 6E22A-(SGI02), collected by the Small Grain Institute at Bultfontein, Free State with assumed increased virulence on wheat with APR to stripe rust (J.S. Komen, personal communication), did not differ from 6E22A-.

Winter wheats. Eight winter wheat cultivars were tested as mini-adults to determine whole plant reactions and flag leaf infection types (Table 12, Figs. 20 and 21). Two control cultivars, Baviaans (resistant) and Morocco (susceptible), were included. From the beginning of vernalisation to rust assessment, the experiment took 98 days to complete. Reactions from both the control cultivars correlated with their reactions in the field. When compared to the data of Boshoff (2000), the glasshouse reactions of Elands, Gariep, Hugenoot, PAN3377, SST333 and Tugela were similar to their reaction types observed over a range of field environments. SST363 showed an MR reaction in the glasshouse, compared to the predominant susceptible responses reported by Boshoff (2000). However, resistant and moderately resistant to susceptible reactions have been noted for SST363 at certain locations (Boshoff, 2000). SST399 produced an MS in the glasshouse whereas this cultivar is known to possess high levels of APR in the field (Z.A. Pretorius, personal communication).

CIMMYT lines. Forty three entries in both the 23rd and 24th ESWYT's (Tables 4 and 5) were highly resistant (Figs. 10 A, B and 11B) to pathotype 6E22A- when tested as mini-adults in the glasshouse. Entry 10 from the 23rd ESWYT was heterogeneous for stripe rust reaction whereas entries 44 to 48 (Figs. 22 and 23) displayed MR-MS reaction types. In the 24th ESWYT entries 40, 41, 42 and 46 all had moderate resistant reactions, while ESWYT43 and

ESWYT44 (Fig. 11 A) both had moderately resistant to moderately susceptible reactions.

Field experiments

Experimental population

Field reactions types of the 13 South African and 21 Australian spring type wheats were determined on three consecutive occasions (Table 8). Starting on 10 September, ratings were done 13 and 22 days apart. The first rating was done relatively early in the rust season when the flag leaf sheaths were just opening (Zadoks growth stage 47). Except for Avocet S and Morocco, all the cultivars had a resistant reaction at this stage. At the second rating entries had higher severity scores, but generally remained in the resistant category. Sporulation was more often encountered at the last rating, with Datatine C and Osprey showing high MS reactions.

When the mini-adult reactions of these entries are compared to the field reactions (Table 8), Corrigin C, Datatine C, Leichhardt, Osprey, Sunlyn and Sunmist were rated marginally more susceptible in the growth chamber. Wyuna appeared completely susceptible in the growth chamber whereas in the field it was rated as 70MR-R. Osprey, Sunmist and Wyuna were late maturing and did not all have flag leaves (Zadoks growth stage 36) at the time of inoculation. Cunningham, Goroke, Meering, Olifants, Oxley and SST88 were slightly more susceptible in the field.

CIMMYT line evaluation

The majority of entries in the 23rd (Fig. 24) and 24th ESWYT's were resistant to pathotype 6E22A- of *P. striiformis* f. sp. *tritici* in the field (Tables 4 and 5). Almost no sporulation occurred on any of the lines. In the 23rd ESWYT 32 entries had no visible symptoms (0) whereas 11 showed only a trace resistant (tR) reaction, indicated by a few isolated flecks. ESWYT44 and ESWYT50 had the most susceptible reactions showing a 10MR-MS reaction type.

More susceptible lines occurred in the 24th ESWYT. Twenty three lines had no visible symptoms (0) and eight had a tR. Eighteen entries displayed

moderate to high reaction types, with entries 42 and 50 being most susceptible with 70MR-MS and 80MR-MS, respectively.

When the mini-adult plants were compared with their corresponding field entries, only six lines from both the 23rd and 24th ESWYT's sets differed for stripe rust reaction. In the 23rd set entry 10 had a heterogeneous reaction in the glasshouse (S,R), compared to the tR reaction in the field. Entries 45, 46, 47 and 48 all had a more susceptible reaction in the glasshouse than in the field. ESWYT50 was the only line that had a more resistant reaction in the glasshouse than in the field.

DISCUSSION

Of the three wheat rusts, stripe rust appears to be the most sensitive to environmental effects (Stubbs, 1988). Environmental conditions during the pre- and post-inoculation phases have often been shown to affect the outcome of interactions between host plant and pathogen (Park, Ash and Rees, 1992), and seedling infection types obtained during this study confirmed this.

Environmental conditions experienced immediately before inoculation influenced the quality of infection types. An increase in sporulation was obtained with the treatments subjected to 24 h light prior inoculation. Several cultivars had more susceptible reactions than the control. In previous studies all cultivars for which disease response was found to be modified by post-inoculation temperature, displayed a shift toward resistance at higher temperatures (Park *et al*., 1992). In the present study temperature did not have such a significant influence as did day length. All treatments subjected to less than 14 h day length (treatments 3, 4, 6 - 9, 11 and 12) showed a reduction in infection levels. It is therefore suggested that all wheat seedlings be subjected to a pre-inoculation conditioning period at sufficient light intensity to ensure consistent and accurate assessment of stripe rust reactions.

De Vallavieille-Pope *et al*. (2002), however, proved that not only the point in time of light application, but also the quantity (intensity x duration) of natural

or artificial light improved stripe rust infection. In their study the infection efficiency of wheat seedlings was enhanced from 0.4 to 36% depending on the light quantity received prior to inoculation. Furthermore, dark periods experienced prior to inoculation were not conducive to infection type development. Clear banding of infection zones and reduced sporulation occurred in treatments 3, 4 and 7. In the tests done by De Vallavieille-Pope *et al.* (2002) a 16 h darkness period before inoculation was also detrimental to infection by *P. striiformis*.

The reactions of adult plants and seedlings to pathogen infection are sometimes quite different. Such phenological differences have yet to be fully explained (Mackenzie, 1991). The development of stripe rust in the leaves of the wheat cultivar Maris Widgeon showing seedling susceptibility and APR, was examined using light and electron microscopy. Mid-sections of fully expanded fourth seedling leaves to APR of wheat infected by *P. striiformis* indicated that the infection was qualitatively similar to that of leaves on mature plants (Mares, 1979). Cultivars showing APR to physiological race 104 E137 were seedling susceptible, but thereafter resistance increased progressively in successive leaves (Mares, 1979). With further microscopic studies done by Cartwright and Russel (1981) on the development of *P. striiformis* in the susceptible winter wheat, Nord Deprez, a different perspective was formed. Leaves of seedlings and adult plants were studied using a fluorescent staining technique showing that there are important differences between the development of *P. striiformis* on seedlings and on adult wheat plants. In adult plants, the growth of the fungus is highly polarized, with the formation of stripes of uredinial pustules between the vascular bundles.

In previous studies, Park and Rees (1989) detected resistance to stripe rust in primary and secondary leaves of some Australian wheat cultivars with APR when plants were grown under controlled conditions (21/6°C, 12 h light/dark cycle, approx. 600 µE m^{-2} s^{-1}). Under field conditions, these cultivars were not susceptible until later growth stages, suggesting that in these cases the expression of the resistance may be sensitive to environmental

conditions (Park *et al*., 1992).

Although seedlings can be used to screen for partial resistance to wheat leaf rust (*Puccinia recondita* f. sp. *tritici* [syn. *P. triticina]*) if temperatures are maintained at about 8-13°C (night-day) (Broers and Wallenburg, 1989; McIntosh *et al*., 1995), no environment was conducive to expression of APR to stripe rust in seedlings evaluated in this study. This conclusion was based on the lack of correlation between low seedling infection types and adult responses observed in the field. According to Stubbs (1988), in describing the methodology of stripe rust in the mature plant stage, cultivars hardly show their race-specific mature plant resistance in the seedling stage or, at least, do not do so at the temperature used for identification of races (18/15°C and 18/6 h day night cycles). According to results presented here it is not possible to detect adult plant stripe rust resistance analogous to the seedling chlorosis gene linked to *Sr2* (Brown, 1997), or the low temperature resistance of *Lr34* (Drijepondt, Pretorius and Rijkenberg, 1991). Apparently the gene-for-gene-for-environment interaction is not the same for all wheat rusts. The changes in infection types when certain Thatcher *Lr* lines were inoculated with specific isolates of *P. recondita* f. sp. *tritici*, especially under variable temperatures, indicated a complex interaction for temperature sensitivity in wheat leaf rust (Statler and Christianson, 1993).

The lack of an efficient seedling screening system implies that adult plant testing needs to be improved to find a more rapid and cost-effective evaluation procedure. If resistance is based on genes effective only at the adult plant stages, then adult plant tests should be conducted on a routine basis (McIntosh, 1992). In the present study this proved to be difficult, since several attempts to investigate the reaction of normally grown adult plants in the glasshouse failed. In the system used at the University of the Free State, normal adult plants cannot be accurately rated due to leaf damage. If the dew period is reduced, the level of infection is not enough. In previous experiments only about half of the experimental set could be evaluated due to leaf necrosis

of the normally raised adult plants (data not shown). Apparently the thicker flag leaves cannot reverse the effects of water-soaking experienced at lower incubation temperatures.

In contrast the smaller and thinner leaves of mini-adults are not damaged when they are incubated at 11°C for 48 h. When the stature of normal adult plants is compared to that of the mini-adult plants, the latter are notably smaller. The average flag leaf of the mini-adults was 57 x 2.7 mm compared to the 226 x 10.5 mm of the normal adult plants. Also, the mean height of normal adult plants was 561.1 mm compared to the 314.4 mm of the mini-adult plants. In terms of leaf damage the mini-adults were thus better suited for adult plant evaluation. Provided that the system is optimised for growth stage, growth habit and true reflection of a particular wheat genotype, the mini-adult plants appear more reliable and user-friendly.

Variation in expression of resistance due to plant age, age of leaf tissue or to difference in growth stage has been demonstrated in many host-pathogen associations in cereals (Qayoum and Line, 1985; Pretorius, Rijkenberg and Wilcoxson, 1988; Ellison and Murray, 1992; Warren and Keane, 2000). From the present data it can be concluded that the mini-adult plants should be inoculated between heading and flowering. Although an accelerated growth period is desirable in a quick turn-over of adult plant data, one shortcoming of mini-adults is the relatively quick maturation of flag leaves. If these small flag leaves are inoculated after flowering they loose their chlorophyll too rapidly for accurate rust ratings. In previous studies Qayoum and Line (1985) found that when wheat cultivars were infected with stripe rust, flag leaves produced higher infection types than the lower leaves. Differences were ascribed to flag leaves being greener and not as hardened as the older leaves. The flag leaf is an important photosynthate source for developing heads, and due to translocation a flag leaf may not have sufficient reserves to express maximum levels of resistance (Patterson and Moss, 1979). In the present study the level of flag leaf resistance ranged from moderate to almost immune, indicating that

genotype, and not leaf age only, also play a role in stripe rust phenotype.

Glasshouse grown mini-adults proved to be more successful than those raised in a growth chamber. The average flag leaf size of the glasshouse grown mini-adults was 97 x 4.4 mm compared to the 53.6 x 2.6 of the growth chamber grown plants. Stem thickness and plant height were fairly similar between the environments. Due to larger leaves the glasshouse plants were more vigorous and healthier looking and therefore better material to work with. At this point it is not known which physiological processes are affected by the expedited growth in a growth chamber isolated from natural light and how these influence rust response. The glasshouse environment is thus recommended as a better alternative.

Results of surveys done by Boshoff (2000) revealed the presence of two stripe rust pathotypes in South Africa. Pathotype 6E16A- caused major yield losses in the summer rainfall areas in 1997 resulting in a major shift to the cultivation of the resistant cultivars Hugenoot and Carina the next year. High selection pressure evidently resulted in the development of pathotype 6E22A- (Boshoff, 2000). According to flag leaf infection type and whole plant reactions observed in the present study, pathotype 6E16A- proved to be less virulent than the other three pathotypes, which were similar. The highly susceptible cultivar, Avocet S, was not completely susceptible to the three pathotypes tested. Pathotype 6E22A-(SGI02) with assumed virulence on wheat with APR to stripe rust (J.S. Komen, personal communication), did not differ from 6E22A- and suggests that the variation observed in the field was caused by environment. Pathotype 7E22A- is a single-step mutation from 6E22A-, adding virulence for *Yr1*. It can be concluded that APR in the wheats tested is effective against South African pathotypes, and that the mini-adults provided an acceptable system for comparing adult plants to different isolates.

This system accurately detected APR in all winter wheats except SST399. Likewise, APR in the CIMMYT entries was accurately described. With both sets only six of the 49 CIMMYT lines differed in their reactions between the two environments, which gives an accuracy of 88%. A 77% accuracy was obtained when the experimental (34 entries) set's mini-adults

were compared to the field reactions. The better correlation for the CIMMYT germ plasm can also be attributed to the occurrence of many seedling resistances in this material. Variation in expression of this type of hypersensitive resistance has been observed to be much less than in adult plant or partial resistance.

In conclusion, the accelerated system used in this study to identify APR to stripe rust gives results comparable to those in the field. It is clear that some underestimations occurred in the glasshouse, but this system is useful if the cut-off for resistance is less conservative. Although the method is labour intensive at present (requires more regular watering), it opens possibilities for pre-screening large numbers of breeding lines, and for more detailed studies, e.g. microscopic studies of the development of stripe rust in wheat flag leaves. Likewise, it provides a screening method for APR which is not hampered by the severe leaf damage encountered with previous methods.

REFERENCES

Boshoff, W.H.P., 2000. Control of foliar rusts of wheat in South Africa with special emphasis on *Puccinia striiformis* f. sp. *tritici.* Ph. D. thesis, University of the Free State, South Africa.

Boyd, L.A., and Minchin, P.N. 2001. Wheat mutants showing altered adult plant disease resistance. Euphytica 122: 361-368.

Broers, L.H.M., and Wallenburg, S.C. 1989. Influence of post-infection temperature on three components of partial resistance in wheat to wheat leaf rust. Euphytica 44: 215-224.

Brown, G. 1997. The inheritance and expression of leaf chlorosis associated with gene *Sr2* for adult plant resistance to wheat stem rust. Euphytica 95: 67-71.

Cartwright, D.W., and Russel, G.E., 1981. Development of *Puccinia striiformis* in a susceptible winter wheat variety. Transactions of the British Mycological Society 76: 197-204.

De Vallavieille-Pope, C., Huber, L., Leconte, M., and Bethenod, O. 2002. Pre-inoculation effects of light quantity on infection efficiency of *Puccinia striiformis* and *P. triticina* on wheat seedlings. Phytopathology 92: 1308-1314.

Drijepondt, S.C., Pretorius, Z.A., and Rijkenberg, F.H.J. 1991. Expression of two wheat leaf rust resistance gene combinations involving *Lr34*. Plant Disease 75: 526-528.

Ellison, P.J., and Murray, G.M. 1992. Epidemiology of *Puccinia striiformis* f. sp. *tritici* on wheat in southern New South Wales. Australian Journal of Agricultural Research 43: 29-41.

Johnson, R. 1992. Past, present and future opportunities in breeding for disease resistance, with examples from wheat. Euphytica 63: 3-22.

Ma, J., Zhou, R., Dong, Y., Wang, L., Wang, X., and Jia, J. 2001. Molecular mapping and detection of yellow rust resistance gene *Yr26* in wheat transferred from *Triticum turgidum* L. using microsatellite markers. Euphytica 120: 219-226.

Mackenzie, D.R. 1991. Progress in plant disease resistance research. FAO Plant Protection Bulletin 39: 147-154.

Mares, D.J. 1979. Microscopic study of the development of yellow rust (*Puccinia striiformis*) in a wheat cultivar showing adult plant resistance. Physiological Plant Pathology 15: 289-296.

McIntosh, R.A. 1992. Pre-emptive breeding to control wheat rusts. Euphytica 63: 103-113.

McIntosh, R.A., Wellings, C.R., and Park, R.F. 1995. Wheat Rusts: An Atlas of Resistance Genes. CSIRO, East Melbourne, Australia.

Park, R.F., and Rees, R.G. 1989. Expression of adult-plant resistance and its effect on the development of *Puccinia striiformis* f.sp *tritici* in some Australian wheats. Plant Pathology 38: 200-208.

Park, R.F., Ash, G.J., and Rees, R.G. 1992. Effects of temperature on the response of some Australian wheat cultivars to *Puccinia striiformis* f. sp. *tritici*. Mycological Research 96: 166-170.

Patterson, T.G., and Moss, D.N. 1979. Senescence in field-grown wheat. Crop Science 19: 635-640.

Peterson, R.F., Campbell, A.B., and Hannah, A.E. 1948. A diagrammatic scale for estimating rust intensity of leaves and stems of cereals. Canadian Journal of Research Section C 26: 496-500.

Pretorius, Z.A., Boshoff, W.H.P., and Kema, G.H.J. 1997. First report of *Puccinia striiformis* f. sp. *tritici* on wheat in South Africa. Plant Disease 81:424.

Pretorius, Z.A. Park, R.F., and Wellings, C.R. 2000. An accelerated method for

evaluating adult-plant resistance to leaf and stripe rust in spring wheat. Acta Phytopathologica et Entomologica Hungarica 35: 359-364.

Pretorius Z.A., Rijkenberg F.H.J., and Wilcoxson, R.D. 1988. Effects of growth stage, leaf position and temperature on adult-plant resistance of wheat infected by *Puccinia recondita* f. sp. *tritici*. Plant Pathology 37: 36-44.

Qayoum, A., and Line, R.F. 1985. High-temperature adult plant resistance to stripe rust in wheat. Phytopathology 75: 1121-1125.

Statler, G.D., and Christianson, T. 1993. Temperature studies with wheat leaf rust. Canadian Journal of Research Section 15: 97-101.

Stubbs, R.W. 1988. Pathogenicity analysis of yellow (stripe) rust of wheat and its significance in a global context. Pages 23-39 in: Breeding Strategies for Resistance to the Rusts of Wheat. N.W. Simmonds and S. Rajaram (eds). Mexico, D. F., CIMMYT.

Stubbs, R.W., Prescott, J.M., Saari, E.E., and Dubin, H.J. 1986. Cereal disease methodology manual. International Maize and Wheat Improvement Centre in Cooperation with the Research Institute for Plant Protection, Wageningen, The Netherlands.

Viljanen-Rollinson, S.L.H., and Cromey, M.G. 1998. The effectiveness of stripe rust resistance gene *Yr18* in wheat cultivars. Pages 336-340 in: 51st Conference Proceedings (1998) 0f the New Zealand Plant Protection Society Incorporated. Downloaded from: http://www.hortnet.co.nz, 06-11-2003

Warren, M.A., and Keane, P.J. 2000. The nature of adult plant resistance to stripe rust (*Puccinia striiformis*) in the wheat cultivar, Meering. Proceedings of the 12th Biennial Congress of the Australasian Plant Pathology Society, Canberra, Australia.

Table 1. Pre- and post-inoculation treatments of wheat seedlings inoculated with pathotype 6E16A- of *Puccinia striiformis* f. sp. *tritici*

Treatment	Pre-inoculation	Special conditioning[a]	Post-inoculation
1	GH (25/15°C, 14/10 h day/night)	-	GH (18/15°C, 14/10 h day/night)
2	GH (25/15°C, 14/10 h day/night)	GC (18°C, 24 h light)	GH (18/15°C, 14/10 h day/night)
3	GH (25/15°C, 14/10 h day/night)	GC (18°C, 24 h darkness)	GH (18/15°C, 14/10 h day/night)
4	GH (25/15°C, 14/10 h day/night)	GC (18°C, 12 h light/ 12 h darkness)	GH (18/15°C, 14/10 h day/night)
5	GH (25/15°C, 14/10 h day/night)	GC (25°C, 24 h light)	GH (18/15°C, 14/10 h day/night)
6	GH (25/15°C, 14/10 h day/night)	GC (25°C, 24 h darkness)	GH (18/15°C, 14/10 h day/night)
7	GH (25/15°C, 14/10 h day/night)	GC (25°C, 12 h light/ 12 h darkness)	GH (18/15°C, 14/10 h day/night)
8	GH (25/15°C, 14/10 h day/night)	GC (18°C, 24 h light)	GC (18°C, 12 h light/12 h darkness)
9	GH (25/15°C, 14/10 h day/night)	GC (18°C, 24 h light)	GC (18°C, 6 h light/18 h darkness)
10	GH (25/15°C, 14/10 h day/night)	GC (18°C, 24 h light)	GC (18°C, 18 h light/6 h darkness)
11	GH (25/15°C, 14/10 h day/night)	GC (18°C, 24 h light)	GC (25°C 12 h light/18°C 12 h darkness)
12	GH (25/15°C, 14/10 h day/night)	GC (18°C, 24 h light)	GC (25°C, 12 h light/12 h darkness)
13	GH (25/15°C, 14/10 h day/night)	GC (18°C, 24 h light)	GC (10°C, 18 h light/6 h darkness)

[a] Following the special conditioning period, inoculated wheat was incubated at 11±1°C and >96 % relative humidity in a dew chamber for 48 h.

Table 2. The effect of different pre-inoculation temperature and light cycles on wheat seedling infection types to pathotype 6E16A- of *Puccinia striiformis* f. sp. *tritici*[a]

Wheat cultivar/ line	18°C (Treatment)				25°C (Treatment)		
	GH (control) (1)	24 h light (2)	24 h darkness (3)	12 h light/ 12 h darkness (4)	24 h light (5)	24 h darkness (6)	12 h light/ 12 h darkness (7)
Arnhem[b]	3++	3++	3-b	3+b	3++	1++b LT	3++b
Avocet S[e]	3++	3++	3b	3+b	3++	3=b	3+
AvS-*Yr18*[e]	;cn,3++	5p;,1p4	;	;	4p;c,3p3c	3p;,1p3=b	4p;c,2p3c
Baviaans[d]	3+c	3++	;1c,3b	3b	3++c	3=b	2cn
Biedou[d]	;c,2c	3++	;c,3	3b	3++	2+cb	3b
Cook[b]	2c	2++c	2c,3c	;1b	3cn	;cn1=	2+cn
Corrigan C[b]	3++	3++	3++b	3++b	3++4	3+	3++
Cunningham[b]	3c	3++	3=c	1p;,3p2+cb	3c	1++ LT	3-c
Datatine C[b]	4	4	3-b	3++b	4	2b	3++
Dollarbrid[b]	3++	3++	3b	3+b	3++	2+b	3+
Goroke[b]	3+	3++	3b	3+b	3+	3+	3++
Janz[b]	3++	3++	3b	3+b	3+	2	3cb
Kariega[d]	3++	3++	3-b	3-b	3++	2b	3++

Table 2 (cont.). The effect of different pre-inoculation temperature and light cycles on wheat seedling infection types to pathotype 6E16A- of *Puccinia striiformis* f. sp. *tritici*[a]

Wheat cultivar/ line	18°C				25°C		
	GH (control) (1)	24 h light (2)	24 h darkness (3)	12 h light/ 12 h darkness (4)	24 h light (5)	24 h darkness (6)	12 h light/ 12 h darkness (7)
Leichhardt[b]	4	4	3b	3b	3++	3=b	3+
Meering[b]	3c	3c	3-b	3b	3c	1+cb	3c
Olifants[d]	;1cn	2+cn	;1cn	;1cn	1cn	;1=cn	1cn
Osprey[b]	3+	3c,4	3b	3b	4	3=cb	3c
Ouyen[b]	2c	3c	5p0,1p1c	;1,2b	2+cn	0c	2cn
Oxley[b]	3+	3+	2p0 LT,5p2+c	3-b	3+cn	3cb LT,3=	3=c
SST57[c]	2c	2c,3c	;1-	;,;1c	1cn,3c	2-c	2cn
SST88[c]	3++	3++	3b	3=b	3++	3+b	3++
SST806[c]	2+c	3c	;1c	3p;c,1p2c	2cn	1cn	2cn
SST825[c]	3c	1p;,6p3c	2=	6p0;,2p;1c	3++c	2c	3+c
SST876[c]	0;	0;	0	0	0	0;	0;
Steenbras[d]	3+	3+	3= LT	;1b	3++	2-b	3+
Sunbrook[b]	3++	3++	3p;1,2p3c	3-b	3=c	2+c	3c

Table 2 (cont.). The effect of different pre-inoculation temperature and light cycles on wheat seedling infection types to pathotype 6E16A- of *Puccinia striiformis* f. sp. *tritici*

Wheat cultivar/ line	18°C				25°C		
	GH (control) (1)	24 h light (2)	24 h darkness (3)	12 h light/ 12 h darkness (4)	24 h light (5)	24 h darkness (6)	12 h light/ 12 h darkness (7)
Suneca[b]	3++	3++	3-b LT	3-b	3++	3-3b	3+b
Sunelg[b]	3++	3++	3-b LT	3=b	3++	3+b	3+b
Sunlyn[b]	3c	2c	0	6p0;,1p3- LT	3+	1++b	3+cb
Sunmist[b]	3++	4	3++	3+b	3++	3+b	3=b
Sunstate[b]	;1c	2c	;cn	0;	2++c	0cn	1cn
Swift[b]	3++	3++	3++b	3p0,2p3 LT	3++	3 LT	3++b
Tasman[b]	3c	3c	2c,1p3b	3c LT	3++	3= LT	3++ LT
Wyuna[b]	2p;,5p3++	3p;,2p3	4p;,2p3=b	1p;,1p;1,4p3++	3++	3b	3++b

[a] Following the pre-inoculation treatments, all infection types were determined at 17.3±0.7°C/15.8±0.1°C in a glasshouse. Infection types (0 to 4 scale) followed by "p", "b", "c", "LT" and "n" indicate total plants, bands, chlorosis, leaf tip and necrosis, respectively.

[b] Australian cultivars.

[c] Monsanto cultivars.

[d] Small Grain Institute cultivars.

[e] Near-isogenic *Yr*-lines developed by PBI, The University of Sydney, Australia.

Table 2. The effect of different pre-inoculation temperature and light cycles on wheat seedling infection types to pathotype 6E16A- of *Puccinia striiformis* f. sp. *tritici*[a]

Wheat cultivar/ line	18°C (Treatment)				25°C (Treatment)		
	GH (control) (1)	24 h light (2)	24 h darkness (3)	12 h light/ 12 h darkness (4)	24 h light (5)	24 h darkness (6)	12 h light/ 12 h darkness (7)
Arnhem[b]	3++	3++	3-b	3+b	3++	1++b LT	3++b
Avocet S[e]	3++	3++	3b	3+b	3++	3=b	3+
AvS-*Yr18*[e]	;cn,3++	5p;,1p4	;	;	4p;c,3p3c	3p;,1p3=b	4p;c,2p3c
Baviaans[d]	3+c	3++	;1c,3b	3b	3++c	3=b	2cn
Biedou[d]	;c,2c	3++	;c,3	3b	3++	2+cb	3b
Cook[b]	2c	2++c	2c,3c	;1b	3cn	;cn1=	2+cn
Corrigan C[b]	3++	3++	3++b	3++b	3++4	3+	3++
Cunningham[b]	3c	3++	3=c	1p;,3p2+cb	3c	1++ LT	3-c
Datatine C[b]	4	4	3-b	3++b	4	2b	3++
Dollarbrid[b]	3++	3++	3b	3+b	3++	2+b	3+
Goroke[b]	3+	3++	3b	3+b	3+	3+	3++
Janz[b]	3++	3++	3b	3+b	3+	2	3cb
Kariega[d]	3++	3++	3-b	3-b	3++	2b	3++

Table 2 (cont.). The effect of different pre-inoculation temperature and light cycles on wheat seedling infection types to pathotype 6E16A- of *Puccinia striiformis* f. sp. *tritici*[a]

Wheat cultivar/ line	18°C				25°C		
	GH (control) (1)	24 h light (2)	24 h darkness (3)	12 h light/ 12 h darkness (4)	24 h light (5)	24 h darkness (6)	12 h light/ 12 h darkness (7)
Leichhardt[b]	4	4	3b	3b	3++	3=b	3+
Meering[b]	3c	3c	3-b	3b	3c	1+cb	3c
Olifants[d]	;1cn	2+cn	;1cn	;1cn	1cn	;1=cn	1cn
Osprey[b]	3+	3c,4	3b	3b	4	3=cb	3c
Ouyen[b]	2c	3c	5p0,1p1c	;1,2b	2+cn	0c	2cn
Oxley[b]	3+	3+	2p0 LT,5p2+c	3-b	3+cn	3cb LT,3=	3=c
SST57[c]	2c	2c,3c	;1-	;,;1c	1cn,3c	2-c	2cn
SST88[c]	3++	3++	3b	3=b	3++	3+b	3++
SST806[c]	2+c	3c	;1c	3p;c,1p2c	2cn	1cn	2cn
SST825[c]	3c	1p;,6p3c	2=	6p0;,2p;1c	3++c	2c	3+c
SST876[c]	0;	0;	0	0	0	0;	0;
Steenbras[d]	3+	3+	3= LT	;1b	3++	2-b	3+
Sunbrook[b]	3++	3++	3p;1,2p3c	3-b	3=c	2+c	3c

Table 2 (cont.). The effect of different pre-inoculation temperature and light cycles on wheat seedling infection types to pathotype 6E16A- of *Puccinia striiformis* f. sp. *tritici*

Wheat cultivar/ line	18°C				25°C		
	GH (control) (1)	24 h light (2)	24 h darkness (3)	12 h light/ 12 h darkness (4)	24 h light (5)	24 h darkness (6)	12 h light/ 12 h darkness (7)
Suneca[b]	3++	3++	3-b LT	3-b	3++	3-3b	3+b
Sunelg[b]	3++	3++	3-b LT	3=b	3++	3+b	3+b
Sunlyn[b]	3c	2c	0	6p0;,1p3- LT	3+	1++b	3+cb
Sunmist[b]	3++	4	3++	3+b	3++	3+b	3=b
Sunstate[b]	;1c	2c	;cn	0;	2++c	0cn	1cn
Swift[b]	3++	3++	3++b	3p0,2p3 LT	3++	3 LT	3++b
Tasman[b]	3c	3c	2c,1p3b	3c LT	3++	3= LT	3++ LT
Wyuna[b]	2p;,5p3++	3p;,2p3	4p;,2p3=b	1p;,1p;1,4p3++	3++	3b	3++b

[a] Following the pre-inoculation treatments, all infection types were determined at 17.3±0.7°C/15.8±0.1°C in a glasshouse. Infection types (0 to 4 scale) followed by "p", "b", "c", "LT" and "n" indicate total plants, bands, chlorosis, leaf tip and necrosis, respectively.

[b] Australian cultivars.

[c] Monsanto cultivars.

[d] Small Grain Institute cultivars.

[e] Near-isogenic *Yr*-lines developed by PBI, The University of Sydney, Australia.

Table 4. Seedling, mini-adult and field[a] response of the 23rd CIMMYT Elite Spring Wheat Yield Trial to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici* in 2002

Wheat cultivar/ entry	Pedigree[b]	Glasshouse			Field
		Seedling response	Mini-adult response		Normal adult response
		Infection type[c]	Infection type	Reaction type	Reaction type
Baviaans[d]	Queen Fan(A50)/4/Jup/Emu"S"//Gjo"S"/3/Kvz/K4500L-6-A-4	–	;	R	tR
Kariega[d]	SST44//K4500.2/Sapsucker"S"	–	;	R	tR
Morocco[e]	P.I. 377890	3++	3++	S	100S
Steenbras[d]	SST44/SST66/4/Hoopv/CI 297001/3/T.aest/Bon//Cno/7C	3+c	;	R	–
ESWYT2	Attila	;cn	;	R	tR
ESWYT3	BL 1724	7p;cn,1p2+	;	R	5-10R
ESWYT4	Inqalab 91	;2cn	0	R	tR
ESWYT5	Kariega	;2	0	R	tR
ESWYT6	PBW343	;	;	R	tR
ESWYT7	Turaco/2*Borl95	;,cn LT	;	R	tR
ESWYT8	Skauz/2*Star	1cn	;	R	tR
ESWYT9	Skauz/2*Star	1cn,2cn	;	R	5R-20R
ESWYT10	F60314.76/Mrl//Cno79/3/Ka/Nac/4/Star	1cn,2cn	;,3++	S,R	tR

Table 4 (cont.). Seedling, mini-adult and field[a] response of the 23rd CIMMYT Elite Spring Wheat Yield Trial to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici* in 2002

		Glasshouse			Field
		Seedling response	Mini-adult response		Normal adult response
Wheat cultivar/ entry	Pedigree[b]	Infection type[c]	Infection type	Reaction type	Reaction type
ESWYT11	He1/3*Cno79//2*Seri/3/Borl95/4/yaco	;,cn LT	;	R	tR
ESWYT12	Kea/Tan/4/Tsh/3/Kal/Bb//Tqfn/5/pavon/9/...	2cn	0	R	tR
ESWYT13	Rabe/2*Mo88	;,cn LT	;	R	0
ESWYT14	Kauz//Bow/Nkt	;	;	R	0
ESWYT15	Buc/Prl//Weaver	;,cn LT	;	R	0
ESWYT16	Prinia/Star	;, cn LT	;	R	0
ESWYT17	Cndo/R143//Ente/Mexi_2/3/...	;,cn LT	;	R	0
ESWYT18	Chen/Aegilops squarrosa (Taus)//Bcn/3/Kauz	;,cn LT	;	R	0
ESWYT19	Chen/Aegilops squarrosa (Taus)//Bcn/3/Kauz	;,cn LT	;	R	0
ESWYT20	Chen/Aegilops squarrosa (Taus)//Bcn/3/Kauz	;, cn LT	;	R	0
ESWYT21	Croc_1/Ae. squarrosa (205)//Kauz/3/Sasia	0;	;	R	0
ESWYT22	Croc_1/Ae.squarrosa (205)//Kauz/3/Attila	;	;	R	tR
ESWYT23	Croc_1/Ae.squarrosa (205)//Kauz/3/Attila	;	;	R	0
ESWYT24	Croc_1/Ae.squarrosa (205)//Kauz/3/Attila	;	;	R	5R

Table 4 (cont.). Seedling, mini-adult and field[a] response of the 23rd CIMMYT Elite Spring Wheat Yield Trial to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici* in 2002

		Glasshouse			Field
		Seedling response	Mini-adult response		Normal adult response
Wheat cultivar/ entry	Pedigree[b]	Infection type[c]	Infection type	Reaction type	Reaction type
ESWYT25	Choix/Star/3/He1/3*Cno79//2*Seri	2p;,2p2cn	;cn	R	0
ESWYT26	Choix/Star/3/He1/3*Cno79//2*Seri	5p;,3p1cn	;cn	R	0
ESWYT27	Jup/Zp//Coc/3/Pvn/4/Gen/5/Bow//Buc/Bul/6/...	;,cn LT	;cn	R	0
ESWYT28	Kauz/Pastor	;,cn LT	;	R	0
ESWYT29	Kauz/Pastor	;	;	R	0
ESWYT30	Weaver/4/Nac/Th.Ac//3*Pvn/3/Mirlo/Buc	;2	0	R	0
ESWYT31	Weaver/3/Lira/Buc/Pvn	5p;cn LT,1p2cn	;	R	0
ESWYT32	Galvez/Weaver/3/Vorona/Cno79//Kauz	;,cn LT	;	R	tR
ESWYT33	Pastor/Kauz	;,cn LT	;	R	0
ESWYT34	Pastor/3/Kauz*2/Opata//Kauz	;	;	R	0
ESWYT35	Site/Mo/Cettia	;,cn LT	;cn	R	0
ESWYT36	Site/Mo/3/Vorona/Bau//Bau	;[c]	;	R	0
ESWYT37	Site/Mo/4/Nac/Th.Ac//3*Pvn/3/Mirlo/Buc	1cn,2cn	;	R	0
ESWYT38	Kauz/Site	;	;	R	0

Table 4 (cont.). Seedling, mini-adult and field[a] response of the 23rd CIMMYT Elite Spring Wheat Yield Trial to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici* in 2002

		Glasshouse			Field
		Seedling response	Mini-adult response		Normal adult response
Wheat cultivar/ entry	Pedigree[b]	Infection type[c]	Infection type	Reaction type	Reaction type
ESWYT39	Cndo/R143//Ente/Mexi_2/3/...	;,cn LT	;	R	0
ESWYT40	Cndo/R143//Ente/Mexi_2/3/...	;	;	R	0
ESWYT41	Picus/3/Kauz*2/Bow//Kauz	;,cn LT	0	R	0
ESWYT42	Kauz/3/Myna/Vul//Buc/Flk/4/Milan	;	;	R	0
ESWYT43	Munia/Chto/3/Pfau/Bow//Vee#9/4/Chen/...	0;	;	R	0
ESWYT44	Bow/Prl*3/6/Wrm/4/Fn/3*Th//K58/2*N/3/...	3c	;1	MR-MS	10MR-MS
ESWYT45	Pyn/Bau//Milan	2cn	;2	MR-MS	0
ESWYT46	Pyn/Bau//Milan	;,cn LT	;1	MR-MS	0
ESWYT47	Pyn/Bau//Milan	3p;cn LT,2p1cn	;1	MR-MS	0
ESWYT48	Weebill1	3++c	;1,;2	MR-MS	5R
ESWYT49	Weaver/Tsc//Weaver/3/Weaver	2+	;	R	0
ESWYT50	Fret2	3++	;	R	0-10MR-MS

[a] Field trials done at Greytown, KZN, [b] CIMMYT field book.

[c] Infection types (0 to 4 scale) followed by "p", "c", "LT" and "n" indicate plants, chlorosis, leaf tip and necrosis , respectively.

[d] Susceptible seedling control and resistant adult control, [e] susceptible control, ¯ not included in field trial.

Table 5. Seedling, mini-adult and field[a] response of the 24th CIMMYT Elite Spring Wheat Yield Trial to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici* in 2003

		Glasshouse			Field
		Seedling response	Mini-adult response		Normal adult response
Wheat cultivar/ entry	Pedigree[b]	Infection type[c]	Infection type	Reaction type	Reaction type
Baviaans[d]	Queen Fan(A50)/4/Jup/Emu"S"//Gjo"S"/3/Kvz/K4500L-6-A-4	–	–	–	tR
Kariega[d]	SST44//K4500.2/Sapsucker"S"	–	0;	R	tR-70MR-MS
Morocco[e]	P.I. 377890	3++	4	S	100S
Steenbras[d]	SST44/SST66/4/Hoopv/CI 297001/3/T.aest/Bon//Cno/7C	2+c	;cn	R	–
ESWYT2	Rayon F 89	3	;	R	0
ESWYT3	Seri/Rayon	3++	;	R	5R
ESWYT4	Bl 1724	;	;	R	0
ESWYT5	Inqalab 91	2cn	0;	R	tR
ESWYT6	Up 2338	;,;c,1++	0;	R	0
ESWYT7	Opata/Rayon//Kauz	;	;	R	0
ESWYT8	Opata/Rayon//Kauz	;	;	R	0
ESWYT9	Skauz*2.Fct	;	;	R	0
ESWYT10	W462//Vee/Koel/3/Peg//Mrl/Buc	;c	;c	R	0

Table 5 (cont.). Seedling, mini-adult and field[a] response of the 24th CIMMYT Elite Spring Wheat Yield Trial to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici* in 2003

		Glasshouse			Field
		Seedling response	Mini-adult response		Normal adult response
Wheat cultivar/ entry	Pedigree[b]	Infection type[c]	Infection type	Reaction type	Reaction type
ESWYT11	Buc/Prl//Weaver	;	;	R	0
ESWYT12	Weaver/Prinia	;	0;	R	0
ESWYT13	Snb//Cmh79A.955/3*Cno79/3/Attila	;1-	;	R	tR
ESWYT14	Bow/Prl//Buc/3/Wh576	;1cn	;	R	0
ESWYT15	Chen/Aegilops squarrosa (Taus)//Bcn/3/Kauz	;cn	0;	R	10R
ESWYT16	Croc_1/Ae. squarossa (205)//Kauz/3/Attila	;	;	R	0
ESWYT17	Croc_1/Ae. squarossa (205)//Kauz/3/Attila	;	;	R	0
ESWYT18	Prl/Sara/Tsi/Vee#5/3/Ducula	2+3	;	R	5R
ESWYT19	Choix/Star/3/He1/3*Cno79//2*Seri	;,1,2+3	;	R	10R
ESWYT20	Kauz/Pastor	;	;	R	0
ESWYT21	Weaver/4/Nac/Th.Ac//3*Pvn/3/Mirlo/Buc	2cn	0;	R	5R
ESWYT22	Weaver/4/Nac/Th.Ac//3*Pvn/3/Mirlo/Buc	;1cn	0;	R	5R
ESWYT23	Pastor/Kauz	;c	;	R	0
ESWYT24	Site/Mo/3/Vorona/Bau//Bau	;cn	;	R	0

Table 5 (cont.). Seedling, mini-adult and field[a] response of the 24th CIMMYT Elite Spring Wheat Yield Trial to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici* in 2003

		Glasshouse			Field
		Seedling response	Mini-adult response		Normal adult response
Wheat cultivar/ entry	Pedigree[b]	Infection type[c]	Infection type	Reaction type	Reaction type
ESWYT25	Site/Mo/4/Nac/Th.Ac//3*Pvn/3/Mirlo/Buc	;cn,2,3+	;	R	10-20R
ESWYT26	Sw89.5181/Kauz	;	;	R	tR
ESWYT27	Cndo/R143//Ente/Mexi_2/3/...	;	;	R	0
ESWYT28	Chen/Aegilops squarrosa (Taus)//Bcn/3/Vee#7/…	3+c	;	R	0
ESWYT29	Chen/Aegilops squarrosa (Taus)//Bcn/3/2*Kauz	;	;	R	0
ESWYT30	Chen/Aegilops squarrosa (Taus)//Bcn/3/2*Kauz	;	;	R	0
ESWYT31	Para2//Jup/Bjy/3/Vee/Jun/4/2*Kauz/5/Bow/…	;c	;	R	0
ESWYT32	Para2//Jup/Bjy/3/Vee/Jun/4/2*Kauz/5/Bow/…	;	;	R	tR
ESWYT33	Bow/Ures//2*Weaver/3/Bow/Prl//Buc	;c	;c	R	20R
ESWYT34	Chen/Aegilops squarrosa (Taus)//Bcn/3/…	;c	;	R	5R
ESWYT35	Chen/Aegilops squarrosa (Taus)//Bcn/3/…	;	;	R	0
ESWYT36	Chen/Aegilops squarrosa (Taus)//Turaco/4/…	0;	0;	R	0
ESWYT37	Vee/Pjn//Kauz/3/Pastor	3c	;	R	tR
ESWYT38	Hpo/Tan//Vee/3/2*Pgo/4/Chen/…	;	;	R	0

Table 5 (cont.). Seedling, mini-adult and field[a] response of the 24th CIMMYT Elite Spring Wheat Yield Trial to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici* in 2003

		Glasshouse			Field
		Seedling response	Mini-adult response		Normal adult response
Wheat cultivar/ entry	Pedigree[b]	Infection type[c]	Infection type	Reaction type	Reaction type
ESWYT39	Falke*2/Bisu/3/Chen/…	2+3c	0;	R	tR
ESWYT40	Skauz/Ks94U215//Skauz	0;,;c,3++	;,2	R,MR	20-40R
ESWYT41	Babax/Lr39//Babax	3++	1,2cn	MR	20MR-MS
ESWYT42	Babax/Lr42//Babax	3++	;c,;1cn	R,MR	70MR-MS
ESWYT43	Babax/Lr43//Babax	3++	1cn,2	MR,MS	20MR-MS
ESWYT44	Babax/Lr43//Babax	4	2cn	MR,MS	30MR-MS
ESWYT45	Pbw65/2*Seri.1B	3++	;c	R	5-10R
ESWYT46	Pfau/Seri.1B//Amad	;c,3++	;1cn	R,MR	40MR-MS
ESWYT47	Prl/2*Pastor	3++	;	R	tR
ESWYT48	Attila*2/Pastor	3++	;	R	tR
ESWYT49	Huw234+Lr34*2//Prl/Vee#10	2+	;	R	35MR
ESWYT50	Huw234+Lr34*2/Pastor	3++	;	R	80MR-MS

[a] Field trials done at Greytown, KZN, [b] CIMMYT field book.

[c] Infection types (0 to 4 scale) followed by "p", "c", "LT" and "n" indicate plants, chlorosis, leaf tip and necrosis , respectively.

[d] Susceptible seedling control and resistant adult control, [e] susceptible control, ¯ not included in field trial.

Table 6. The effect of incubation period on normally grown adult wheat plants in the glasshouse to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici*

Wheat cultivar/ line	Growth stage	6 h		12 h		24 h		36 h		48 h	
		Infection type	Reaction type	Infection type	Reaction type	Infection type	Reaction type	Infection type	Reaction type	Infection type	Reaction type
Avocet S	57	3	–	3	S	3	–	3	–	3	–
Kariega	59	–	–	–	–	–	–	;	–	–	–
Leichhardt	51-53	;cn	–	;cn	–	;cn	–	;cn	–	;cn	MR-MS
SST88	<41	–	–	;	–	;	–	;	–	;	5-10R
Sunlyn	43	–	–	;	–	;	–	;	–	;	R

– not possible to determine infection types due to extensive leaf chlorosis.

Table 7. The effect of growth stage on stripe rust reaction of a collection of resistant wheat cultivars and lines tested as mini-adults in a growth chamber to pathotype 6E16A- of *Puccinia striiformis* f. sp. *tritici*

Wheat	First growth stage			Second growth stage			Third growth stage		
cultivar/ line	Growth[a] stage	Infection type	Reaction type	Growth stage	Infection type	Reaction type	Growth stage	Infection type	Reaction type
Arnhem	36	2cn	MR	43	;cn	R	69	;cn[b]	R
Avocet S	41	4	S	61	;3	MS-S	69	;2	MS
Dollarbird	36	3	MS-S	59	;cn	MR-R	61	;cn[b]	MR-R
Kariega	55	3cn	MR-MS	57	;,;cn	MR-R	61	¯	MR-R
Leichhardt	36	3++	MR-MS	61	;cn	MR-R	69	;cn[b]	MR-R
Meering	39	3++	MS-S	59	;cn	MR-R	69	;1cn	MR-R
Morocco	36	4	S	61	4	S	61	3	S
SST88	36	3++	MR-R	43	1cn	MR-MS	57	;	R
SST876	36	0;	R	59	0;	R	61	;cn[b]	R
Sunelg	41	3++	MS-S	61	;cn	MR-MS	61	;cn[b]	MR-MS
Sunlyn	36	3++	MR-MS	59	;cn	MR-R	65	;cn[b]	MR-MS
Tasman	41	3++	MS-S	59	;cn	MR-R	65	;cn[b]	MR-R

[a] Zadoks scale of cereal growth stages (Stubbs *et al,* 1986).

[b] Flag leaves have much more necrosis than growth stage 2.

¯ not possible to determine infection types due to extensive leaf chlorosis.

Table 8. Growth chamber mini-adult reponse and field reaction of 12 South African and 22 Australian spring wheat cultivars to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici* at Greytown, KZN, South Africa during 2002

Wheat cultivar/ line	Growth chamber response		Field response			
			First reading (10/09/02)		Second reading (23/09/02)	Third reading (15/10/02)
	Growth stage	Reaction type	Growth stage	Reaction type	Reaction type[a]	Reaction type
Arnhem	59	R	57 - 59	30R	40R ltn	–
Avocet S	69	MS-S	47	80S	100S	–
AvS-*Yr18*	69	R	47	5R	tR-40MR ltn	5MS-MR
Baviaans	59	R	59	5R	tR ltn	tR
Biedou	65-69	R	49	15R	10R ltn	10R
Cook	65	R	47	15R	15R ltn	SR
Corrigan C	59	MS	47	10R	20R ltn	20R ltn
Cunningham	51	R	47	15R	30R-MR ltn	30R-MR
Datatine C	57	S	47	20R	40R ltn	40MR- MS
Dollarbrid	61	R	47	30R	40R ltn	40-50R
Goroke	69-75	R	47	30R	40R ltn	30-40MR-R
Janz	65-69	R	47	20R	30R ltn	20-30R
Kariega	59	R	55 - 59	5R	5R ltn	tR
Leichhardt	59	MR	47	20R	30R ltn	50R

Table 8 (cont.). Growth chamber mini-adult reponse and field reaction of 12 South African and 22 Australian spring wheat cultivars to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici* at Greytown, KZN, South Africa during 2002

Wheat cultivar/ line	Growth chamber response		Field response			
			First reading (10/09/02)		Second reading (23/09/02)	Third reading (15/10/02)
	Growth stage	Reaction type	Growth stage	Reaction type	Reaction type	Reaction type
Meering	69	R	47	15R	40R ltn	40MR-R
Morocco	61	S	47	100S	-	-
Olifants	41	R	47	tR	tR ltn	5MR
Osprey	36	MS[b]	47	20R	30R-MR	40MR-MS
Ouyen	61	R	47	20R	20R	-
Oxley	39	R	47	10R	10R ltn	15-20MS-MR
SST57	45-47	R	47	5R	10R	15R
SST88	49-55	R	47	5R	tR	20MR-R
SST806	57	R	59	tR	10R ltn	tR
SST825	51	R	55 - 59	tR	5R	5R
SST876	59	R	55 - 59	5R	10R ltn	0
Steenbras	49-53	MR	59	15R	20R-MR	-
Sunbrook	41	R	47	20R	20R[b]	0
Suneca	45-47	R	47	20R	30R	30R

Table 8 (cont.). Growth chamber mini-adult reponse and field reaction of 12 South African and 22 Australian spring wheat cultivars to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici* at Greytown, KZN, South Africa during 2002

Wheat cultivar/ line	Growth chamber response		Field response			
			First reading (10/09/02)		Second reading (23/09/02)	Third reading (15/10/02)
	Growth stage	Reaction type	Growth stage	Reaction type	Reaction type	Reaction type
Sunelg	69	MR	47	20R	20R ltn	15-20MR-R
Sunlyn	65-69	MR	47	tR	tR ltn	tR
Sunmist	39-41	MS	37	20R	30R-MR[b]	50-60MR-R
Sunstate	61	R	53 - 59	10R	5R ltn	30R
Swift	45-49	MR	47	15R	30R ltn	40MR-R
Tasman	59	MR	49	15R	20R ltn	30MR-R
Wyuna	41	S	37	20R	30R-MR	70MR-R

[a] ltn = leaf tip necrosis.

[b] late maturing.

¯ not possible to determine infection types due to extensive leaf chlorosis.

Table 9. Mini-adult response in the growth chamber and the glasshouse to pathotype 6E16A- of *Puccinia striifomis* f. sp. *tritici*

Wheat cultivar/ line	Growth chamber			Glasshouse		
	Growth[a] stage	Infection[b] type	Reaction[c] type	Growth stage	Infection type	Reaction type
Arnhem	59	¯	R	41	;cn	R
Avocet S	61	¯	MS-S	43	3c	MS-S
Dollarbird	59	¯	R	47-49	;cn	R
Kariega	61	¯	MR-R	59	;cn	R
Leichhardt	59	¯	R	41-43	;cn	MR-R
Meering	61-65	¯	MR-R	43	;cn	MR-R
Morocco	61	4	S	43-45	4	S
SST88	59-61	;cn	R	36-37	;cn	MR-R
SST876	61	¯	R	43	;cn	R
Sunelg	59-61	¯	MR-R	57-59	;cn	R
Sunlyn	59-61	¯	MR-R	43	;cn	R
Tasman	59-61	¯	MR-R	45-47	;cn	MR-MS

[a] Zadoks scale of cereal growth stages (Stubbs *et al,* 1986).

[b] Glasshouse response was measured on a 0 to 4 infection type scale.

[c] Field response was measured according to R (resistant), MR (moderately resistant), MS (moderately susceptible) and S (susceptible) reaction type classes.

¯ not possible to determine infection types due to extensive leaf chlorosis.

Table 10. A comparison of the flag leaf length, flag leaf width and total plant height in mm of growth chamber grown mini-adult plants and glasshouse grown mini-adult plants

Wheat	Mini-adult plants in growth chamber (mm)												Mini-adult plants in glasshouse (mm)											
cultivar/	Flag leaf length			Flag leaf width			Stem width			Total plant height			Flag leaf length			Flag leaf width			Stem width			Total plant height		
line	Min	Max	Aver.	Min	Max	Aver.	Min	Max	Aver.	Min	Max	Aver.	Min	Max	Aver.	Min	Max	Aver.	Min	Max	Aver.	Min	Max	Aver.
Arnhem	75,9	85,9	80,9	3,2	3,8	3,5	1,1	1,4	1,3	350,0	450,0	400,0	92,1	96,4	94,3	3,8	4,3	4,1	1,2	1,7	1,5	310,0	375,0	342,5
Avocet S	59,6	63,2	61,4	2,2	2,4	2,3	0,9	1,1	1,0	380,0	390,0	385,0	91,8	92,4	92,1	3,1	3,8	3,5	1,3	1,3	1,3	350,0	375,0	362,5
Dollarbrid	65,3	75,6	70,5	2,9	2,9	2,9	1,3	1,3	1,3	335,0	380,0	357,5	113,8	114,3	114,1	4,1	4,6	4,4	1,1	1,6	1,4	365,0	370,0	367,5
Kariega	28,5	33,3	30,9	1,6	1,7	1,7	1,1	1,1	1,1	310,0	320,0	315,0	63,0	65,7	64,4	3,6	3,7	3,7	1,1	1,1	1,1	370,0	420,0	395,0
Leichhardt	52,1	52,6	52,4	2,4	2,6	2,5	1,1	1,4	1,3	385,0	400,0	392,5	97,0	111,3	104,2	4,1	4,4	4,3	1,1	1,4	1,3	390,0	405,0	397,5
Meering	46,0	48,1	47,1	2,8	2,9	2,9	1,1	1,1	1,1	370,0	385,0	377,5	93,9	103,6	98,8	3,5	3,9	3,7	1,1	1,2	1,2	310,0	315,0	312,5
Morocco	55,5	60,0	57,8	3,0	3,2	3,1	1,0	1,2	1,1	250,0	330,0	290,0	93,7	103,9	98,8	5,3	6,3	5,8	1,1	1,4	1,3	370,0	390,0	380,0
SST88	56,5	68,8	62,7	2,1	2,8	2,5	1,0	1,2	1,1	370,0	380,0	375,0	-	-	-	-	-	-	1,0	1,3	1,2	290,0	310,0	300,0
SST876	57,8	71,6	64,7	2,4	3,4	2,9	1,0	1,5	1,3	330,0	370,0	350,0	96,6	98,3	97,5	4,7	5,3	5,0	1,3	1,4	1,4	310,0	340,0	325,0
Sunelg	46,4	50,1	48,3	2,6	2,8	2,7	0,7	0,9	0,8	280,0	300,0	290,0	83,8	90,3	87,1	3,4	3,8	3,6	1,4	1,5	1,5	280,0	320,0	300,0
Sunlyn	47,9	53,1	50,5	2,1	3,9	3,0	1,2	1,3	1,3	270,0	280,0	275,0	115,5	118,2	116,9	3,7	3,9	3,8	1,4	1,5	1,5	300,0	315,0	307,5
Tasman	43,5	64,7	43,5	2,2	3,3	2,8	1,1	1,2	1,2	320,0	340,0	330,0	91,2	91,7	91,5	4,8	5,6	5,2	1,1	1,4	1,3	345,0	360,0	352,5
Total aver.	53,6			2,6			1,1			339,8			97,0			4,4			1,3			343,8		

- No flag leaves available, plants too young.

Table 11. The effect of pathotypes of *Puccinia striiformis* f. sp. *tritici* on stripe rust reaction of a collection of resistant wheat cultivars and lines tested as mini-adults in a glasshouse

		pt. 6E16A-		pt. 6E22A-		pt. 7E22A-		6E22A-(SGI02)	
Entry	Growth stage	Infection type	Reaction type	Infection type[b]	Reaction type	Infection type	Reaction type	Infection type	Reaction type
Arnhem	45-47	;	R	Z2	R	Z2	R	Z2	R
Avocet S	43	3	MS	3c	MS-S	3c	MS-S	3c	MS-S
Dollarbird	49-53	;	R	;1+c	R-MR	;1+c	R-MR	;1+c	R-MR
Kariega	61	;	R	;c	R	;c	R	;c	R
Leichhardt	49-51	;	R	Z2,3	R-MR	Z2,3	R-MR	Z2,3	R-MR
Meering	49-51	;	R	;1cn	MR	;1cn	MR	;1cn	MR
Morocco	45	4	S	4	S	4	S	4	S
SST88	43	;	R	;	R	;	R	;	R
SST876	55-57	;	R	;	R-MR	;	R	;	R
Sunelg	53-57	;1c	MR	;c	MR	;c	MR	;c	MR
Sunlyn	41-43	;	R	;1c	R-MR	;1c	R-MR	;1c	R-MR
Tasman	49-51	;	R	;1c	R	;1c	R-MR	;1c	R-MR

[a] Collection by SGI at Bultfontein, Free State with assumed increase virulence on wheat with adult-plant resistance to stripe rust.

[b]Z = variable size with larger uredia towards the leaf base.

Table 12. The response of South African winter wheat cultivars tested as mini-adults in the glasshouse to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici*

Wheat Cultivar/ line	Growth stage	Infection type	Reaction type
Baviaans[a]	69	;	R
Elands	45	;2	MR-MS
Gariep	59	;3	MS
Hugenoot	45	;3	MR-MS
Morocco[b]	45	4	S
PAN3377	41	;	R
SST333	61	;1	MR
SST363	69	;3	MR
SST399	37	¯	MS
Tugela	39	;cn	R

[a] Resistant control.

[b] Susceptible control.

¯ No flag leaves, plants too young.

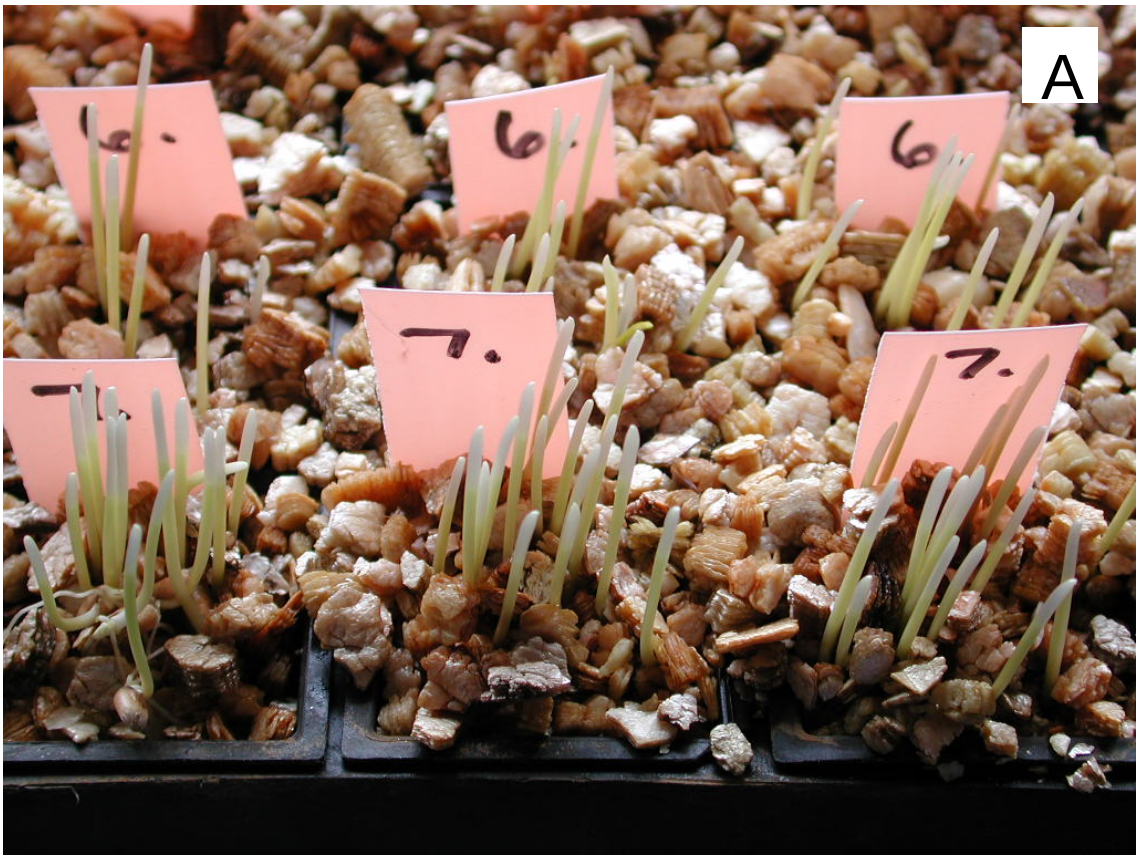
Fig. 1. Urediniospore multiplication of *Puccinia striiformis* f. sp. *tritici* in the glasshouse.

Fig. 2. Seedtrays with accelerated mini-adult plants in the glasshouse.

1

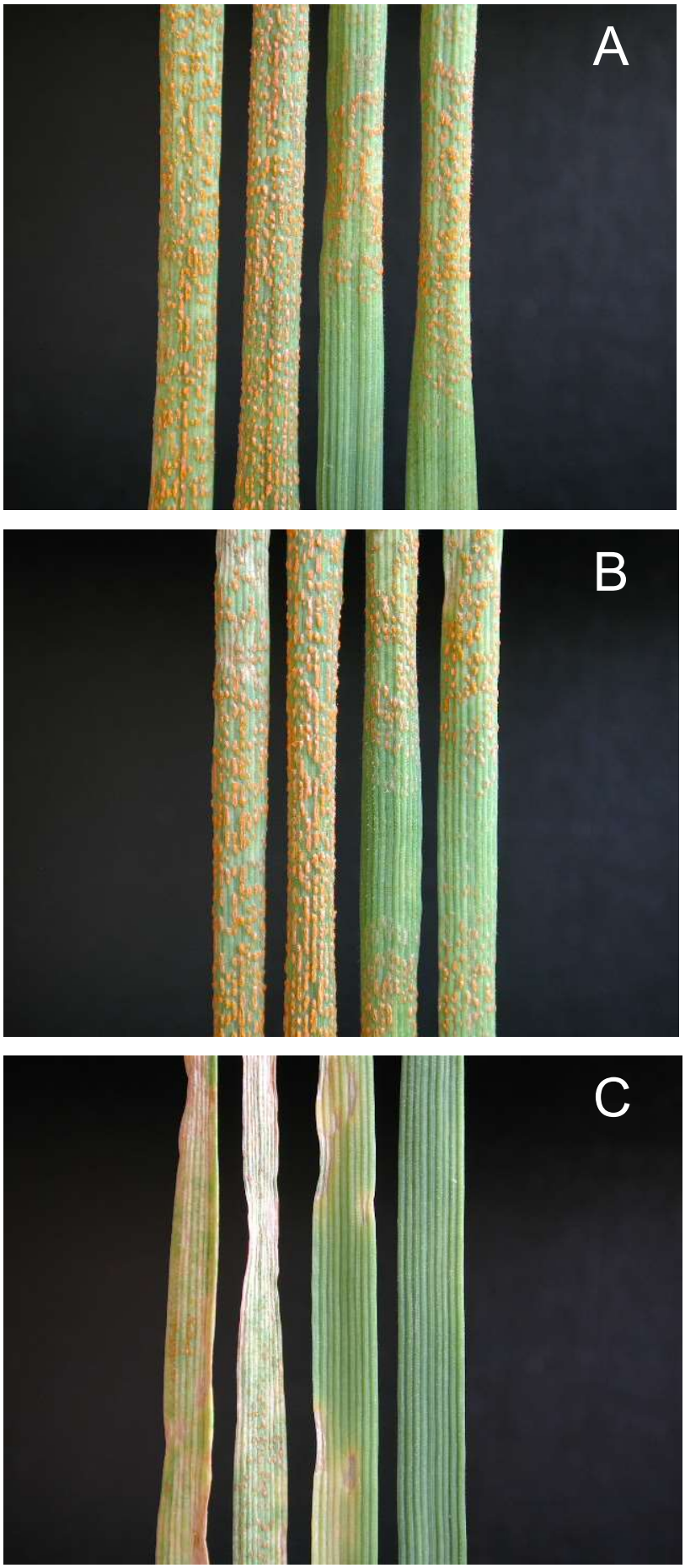
2

Fig. 3. Winter wheat seedlings in a seedtray with vermiculite in the coldroom, after six weeks of vernalization (A), and after transplantion from vermiculite to soil in a seedtray in the glasshouse (B).


A

B
8.

Fig. 4. Primary leaf reactions of Avocet S (A), Baviaans (B) and Sunstate (C), exposed to different pre-inoculation temperature and light cycles, to pathotype 6E16A- of *Puccinia striiformis* f. sp. *tritici.* Treatments from left to right: glasshouse (control); 18°C, 24 h light; 18°C, 24 h darkness and 18°C, 12 h


A
B
C

light/ 12 h darkness.

Fig. 5. Primary leaf reactions of Avocet S (A), Baviaans (B) and Sunstate (C), exposed to different pre-inoculation temperature and light cycles, to pathotype 6E16A- of *Puccinia striiformis* f. sp. *tritici.* Treatments from left to right: glasshouse (control); 25°C, 24 h light; 25°C, 24 h darkness and 25°C, 12 h light/ 12 h darkness.

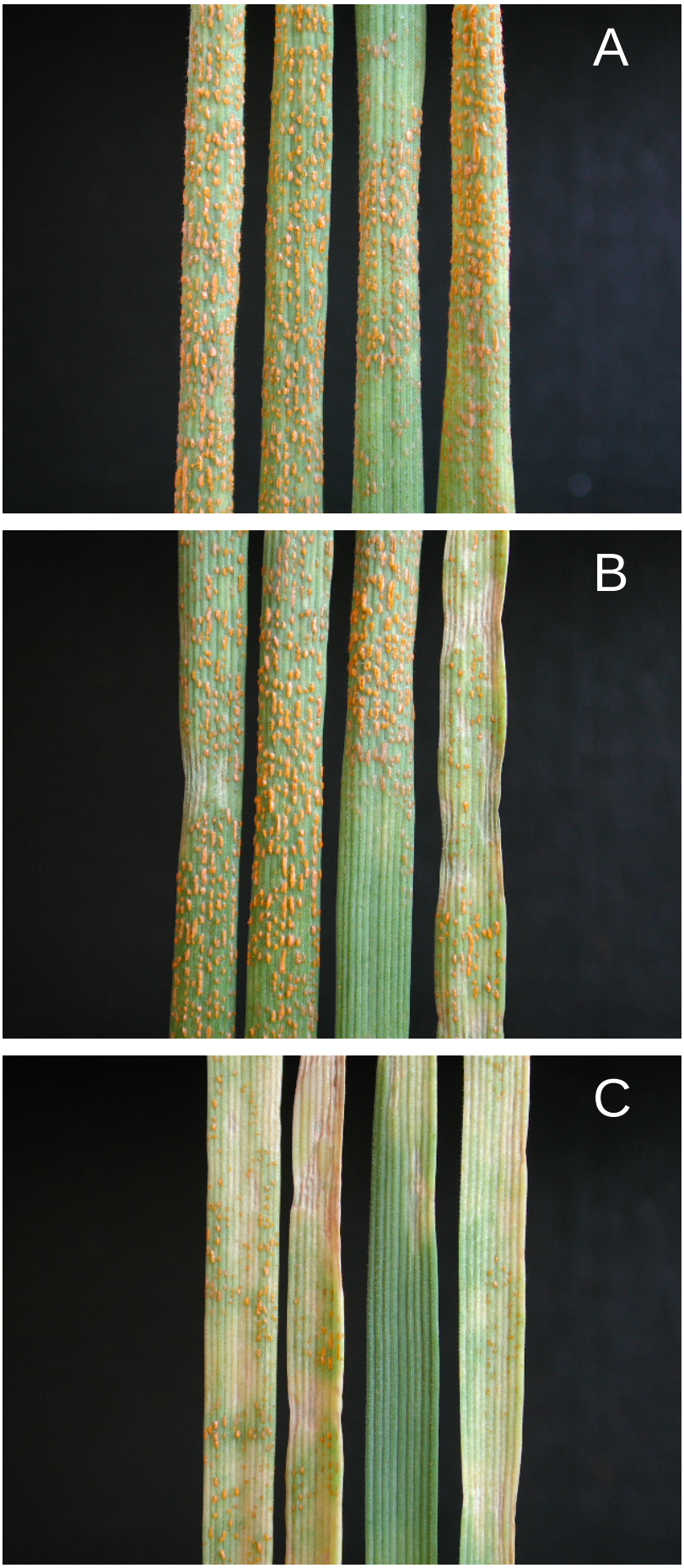

A
B
C

Fig. 6. Primary leaf reactions of Avocet S (A), Cunningham (B) and SST806 (C),

exposed to different pre-inoculation temperature and light cycles, to pathotype 6E16A- of *Puccinia striiformis* f. sp. *tritici.* Treatments from left to right: glasshouse (control); 18°C, 12 h light / 12 h darkness; 18°C 12 h light/ 25°C 12 h darkness and 25°C, 12 h light/ 12 h darkness.

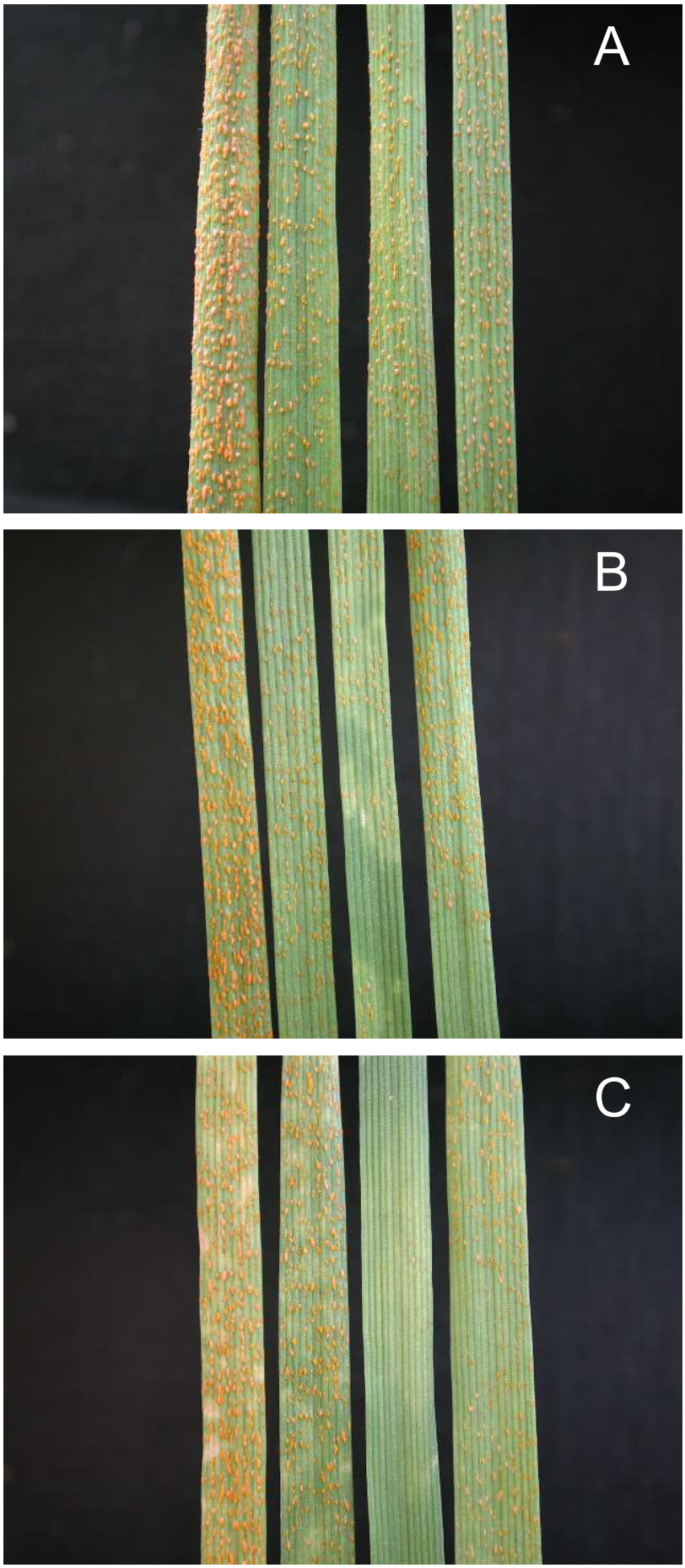

A
B
C

Fig. 7. Primary leaf reactions of Olifants, exposed to different post-inoculation temperature and light cycles, to pathotype 6E16A- of *Puccinia striiformis* f. sp. *tritici.* Treatments from left to right: glasshouse (control); 18°C, 12 h light/ 12 h darkness; 18°C, 6 h light/ 18 h darkness and 18°C, 18 h light/ 6 h darkness.

Fig. 8. Primary leaf reactions of Tasman, exposed to different post-inoculation temperature and light cycles, to pathotype 6E16A- of *Puccinia striiformis* f. sp. *tritici.* Treatments from left to right: glasshouse (control); 18°C, 12 h light / 12 h darkness; 18°C 12 h light/ 25°C 12 h darkness and 25°C, 12 h light/ 12 h darkness.

Fig. 9. Primary leaf reactions of Kariega exposed to a post-inoculation temperature of 10°C with 18 h light and 6 h darkness to pathotype 6E16A- of *Puccinia striiformis* f. sp. *tritici.* Infection types from left to right: two leaves 0; and two leaves 3=.

7

8

9

Fig. 10 A. Primary and flag leaf reactions of the 24th CIMMYT Elite Spring Wheat Yield Trial, entry 34. From left to right: primary leaf infection type, ;;c (three leaves), followed by mini-adult flag leaf infection type ; (three leaves).

Fig. 10 B. Primary and flag leaf reactions of the 24th CIMMYT Elite Spring Wheat Yield Trial, entry 37. From left to right: primary leaf infection type 3c (three leaves), followed by mini-adult flag leaf reaction ; (three leaves).

A

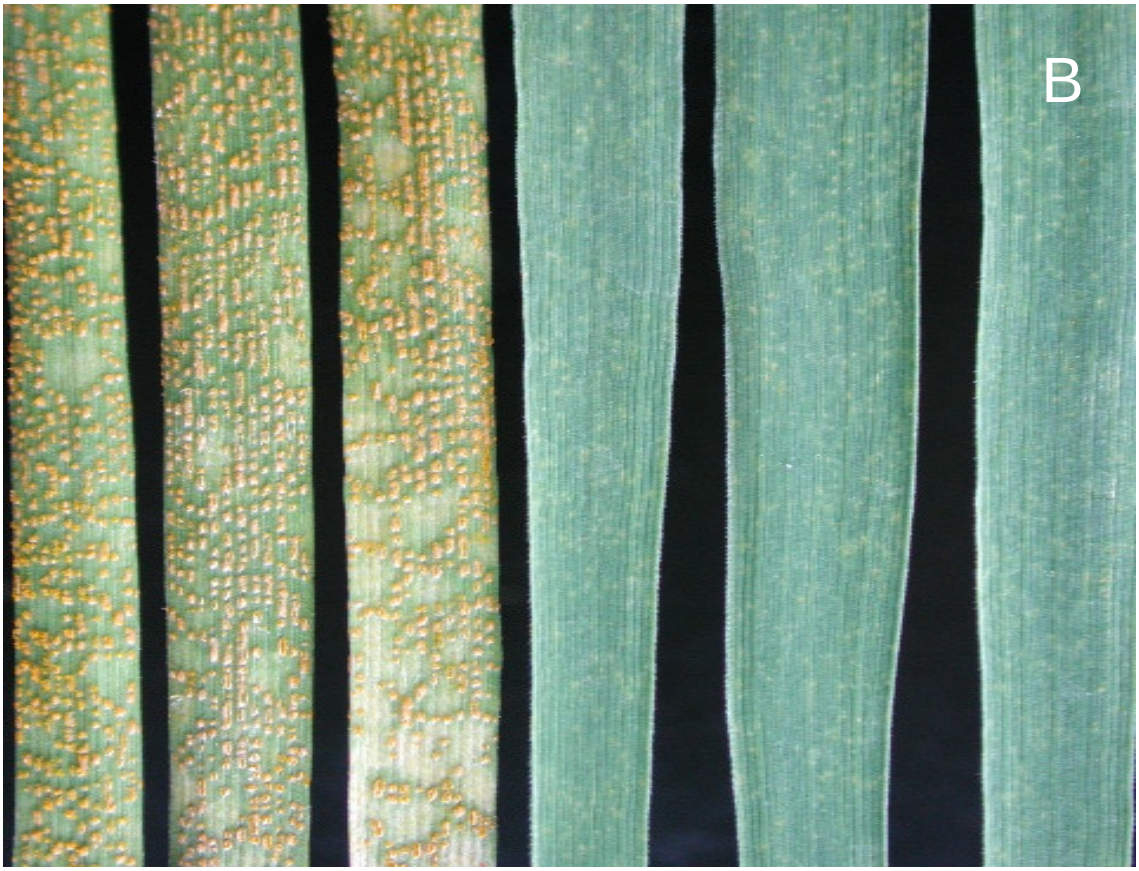
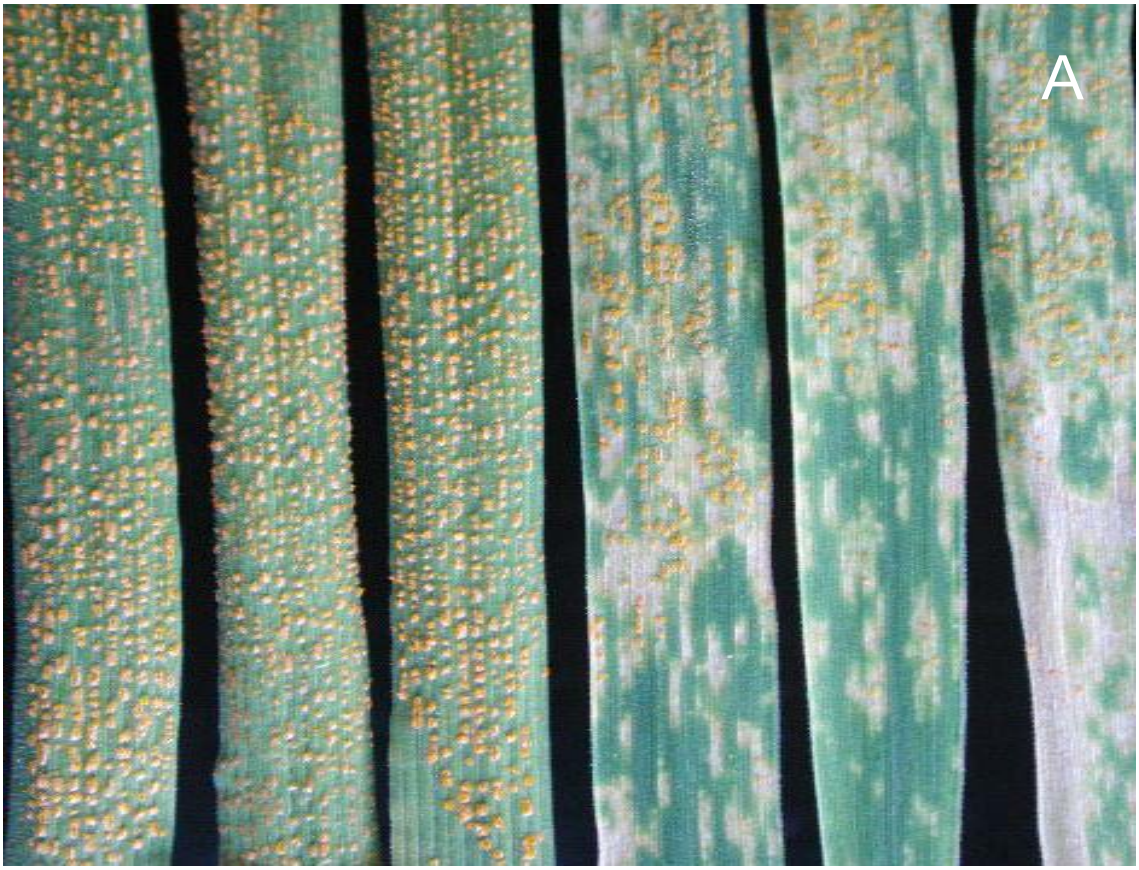

B

Fig. 11 A. Primary and flag leaf reactions of the 24th CIMMYT Elite Spring Wheat Yield Trial, entry 44. From left to right: primary leaf reaction 3++ (three leaves), followed by mini-adult flag leaf reaction 2cn (three leaves).

Fig. 11 B. Primary and flag leaf reactions of the 24th CIMMYT Elite Spring Wheat Yield Trial, entry 48. From left to right: primary leaf reaction 3++ (three leaves), followed by mini-adult flag leaf reaction ; (three leaves).


A

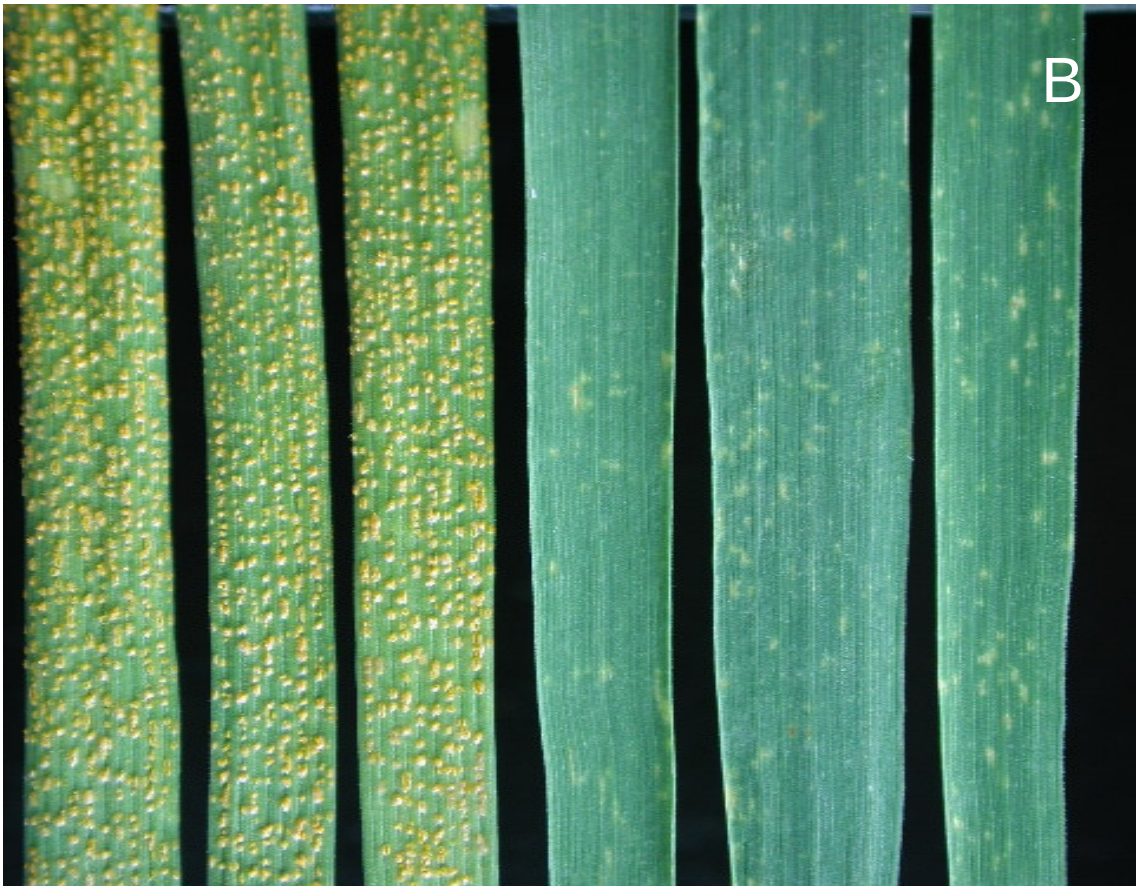

B

Fig. 12. Flag leaf reactions of normally grown Avocet S plants in the glasshouse to different incubation periods to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici.* From left to right: two leaves from the 12 h incubation period and two leaves from the 36 h incubation period.

Fig. 13. Flag leaf reactions of normally grown Leichhardt plants in the glasshouse, following different incubation periods, to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici.* From left to right incubation periods of 6 h, 12 h, 24 h, 36 h and 48 h.

12

13

Fig. 14. Flag leaf reactions of accelerated mini-adult plants of Leichhardt (A), SST88 (B) and Sunelg (C), inoculated at different growth stages with pathotype 6E16A- of *Puccinia striiformis* f. sp. *tritici*. From left to right: growth stage 1, growth stage 2 and growth stage 3.

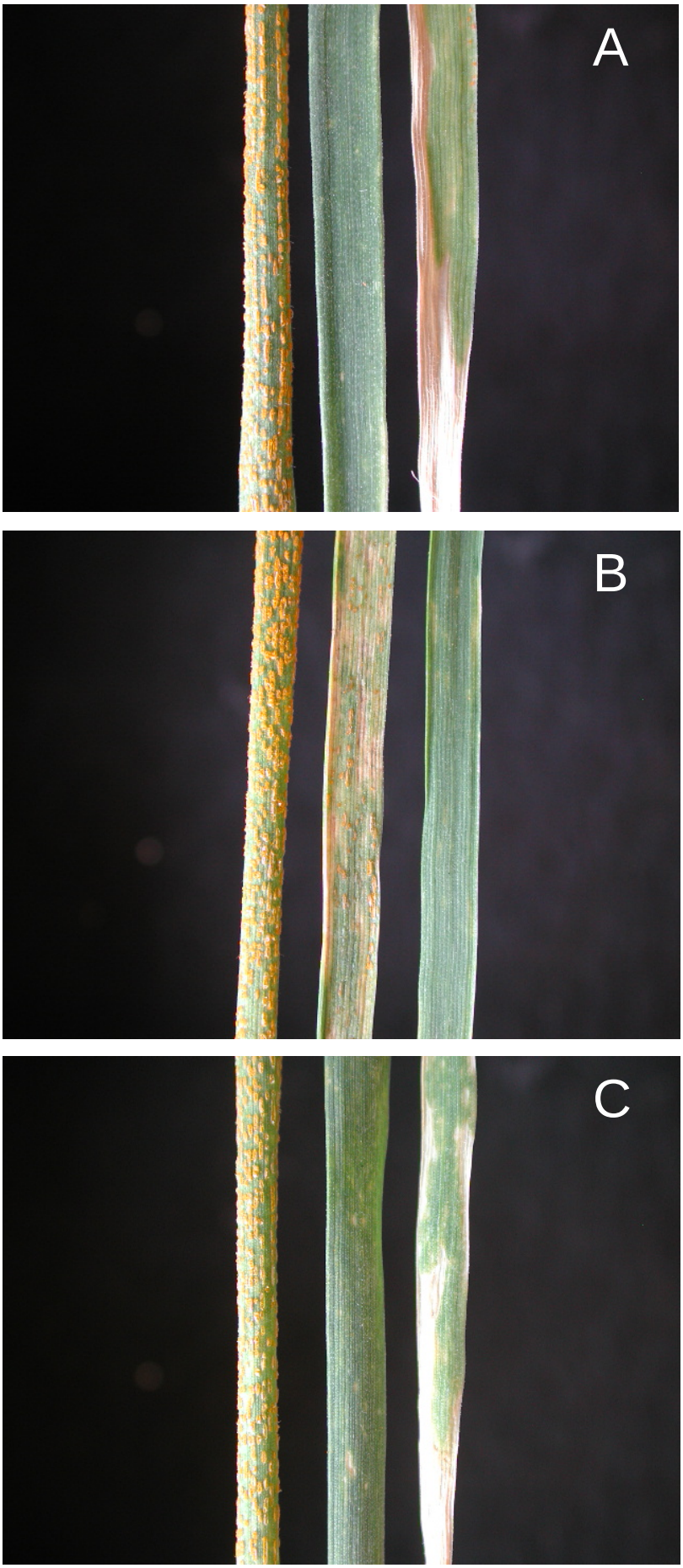
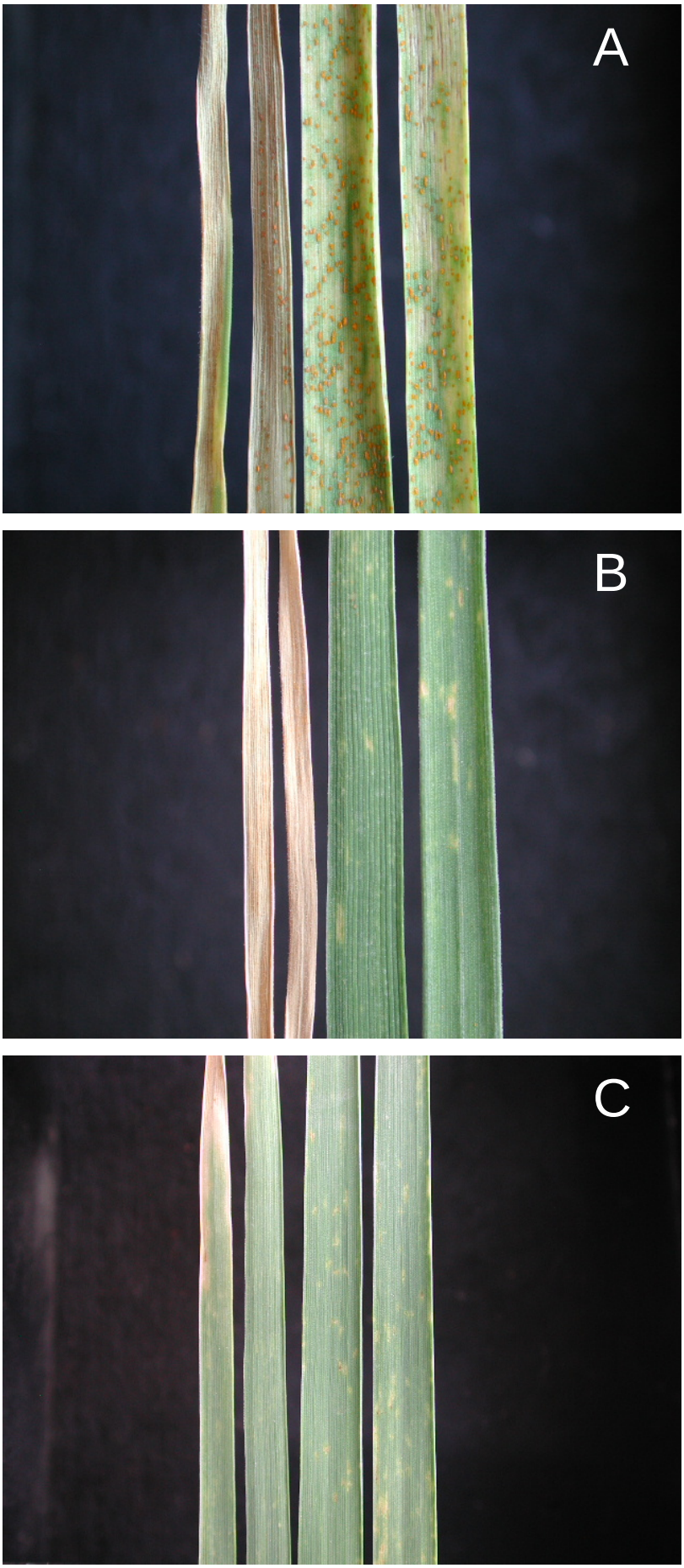

A
B
C

Fig. 15. Flag leaf reactions of accelerated mini-adult plants of Avocet S (A), Leichhardt (B) and SST88 (C) grown in the growth chamber (two leaves on the left) and glasshouse (two leaves on the right) to pathotype 6E16A- of *Puccinia striiformis* f. sp. *tritici.*


A
B
C

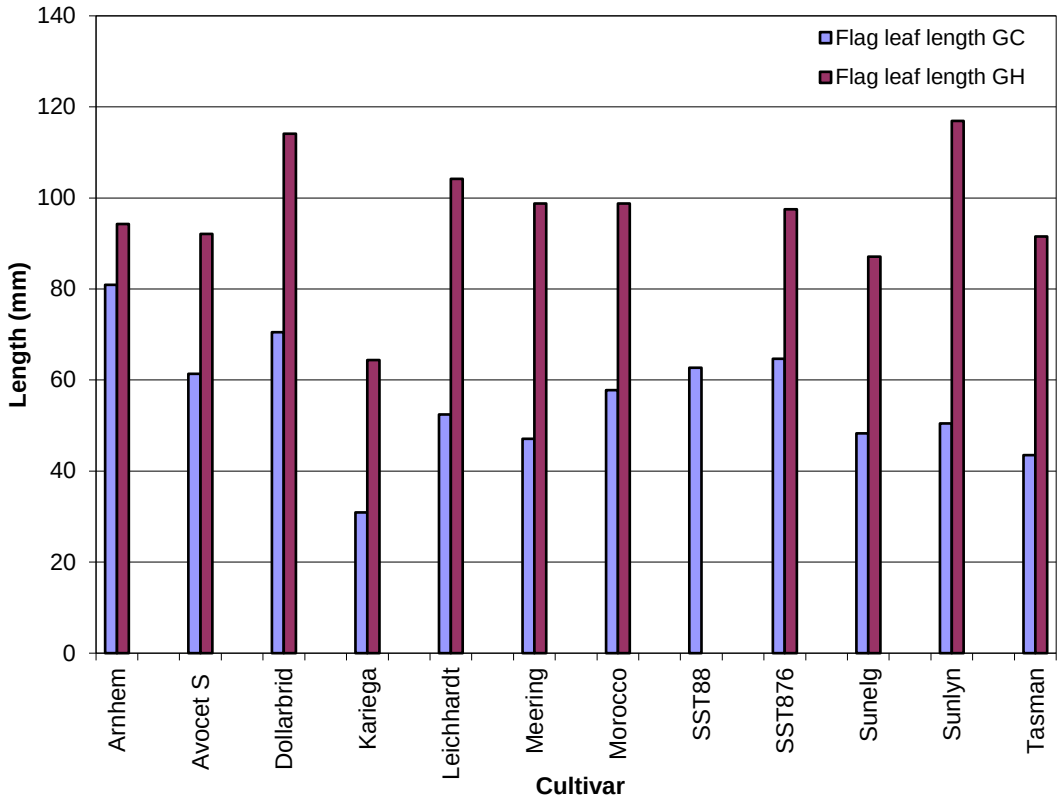


Fig. 16. A comparison between the flag leaf length of growth chamber (GC) and glasshouse (GH) grown mini-adult plants.

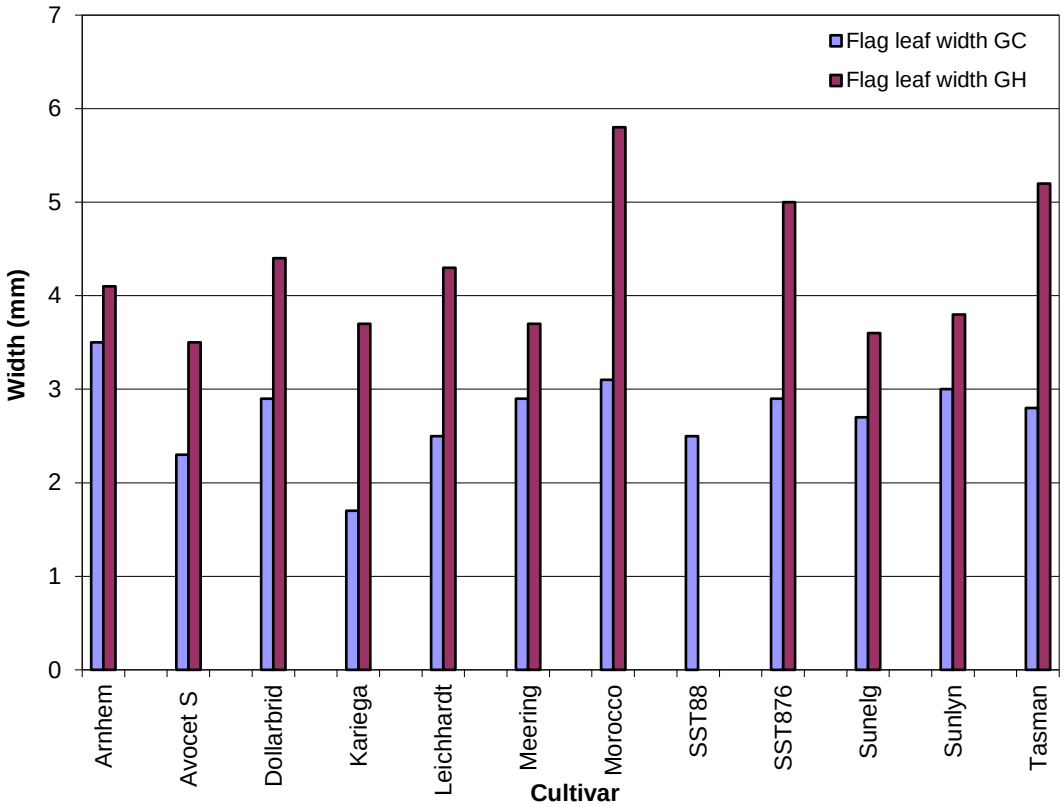


Fig. 17. A comparison between the flag leaf width of growth chamber (GC) and glasshouse (GH) grown mini-adult plants.

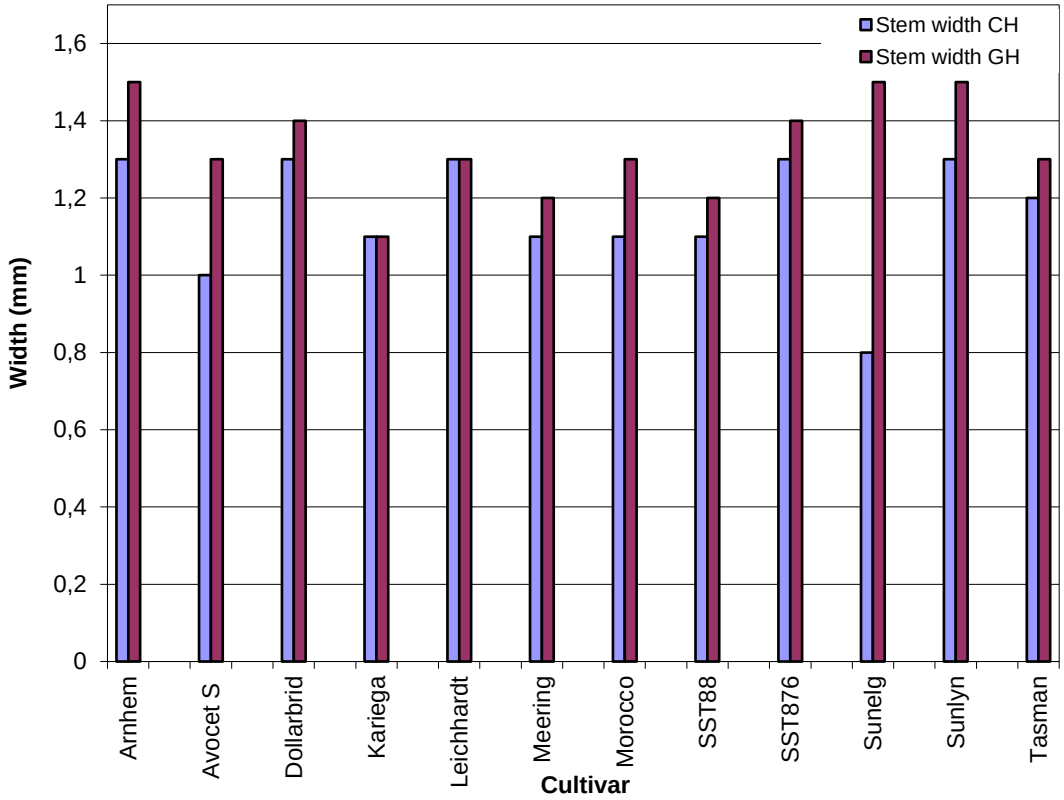


Fig. 18. A comparison between the stem width of growth chamber (GC) and glasshouse (GH) grown mini-adult plants.

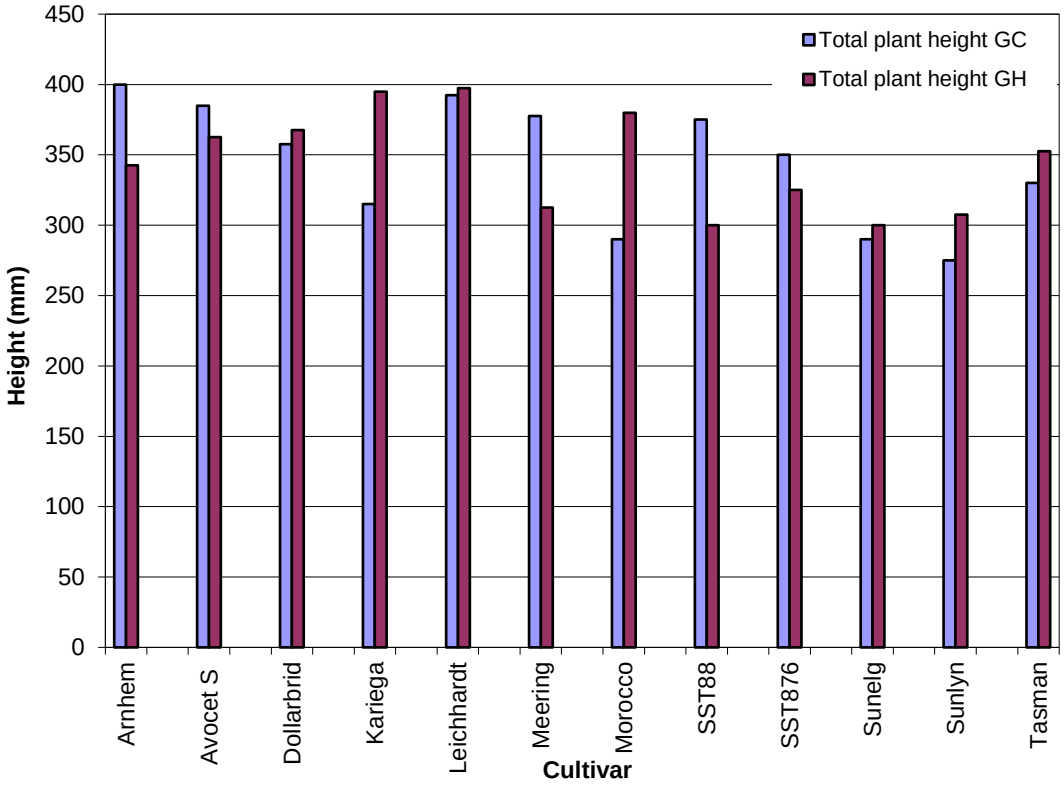
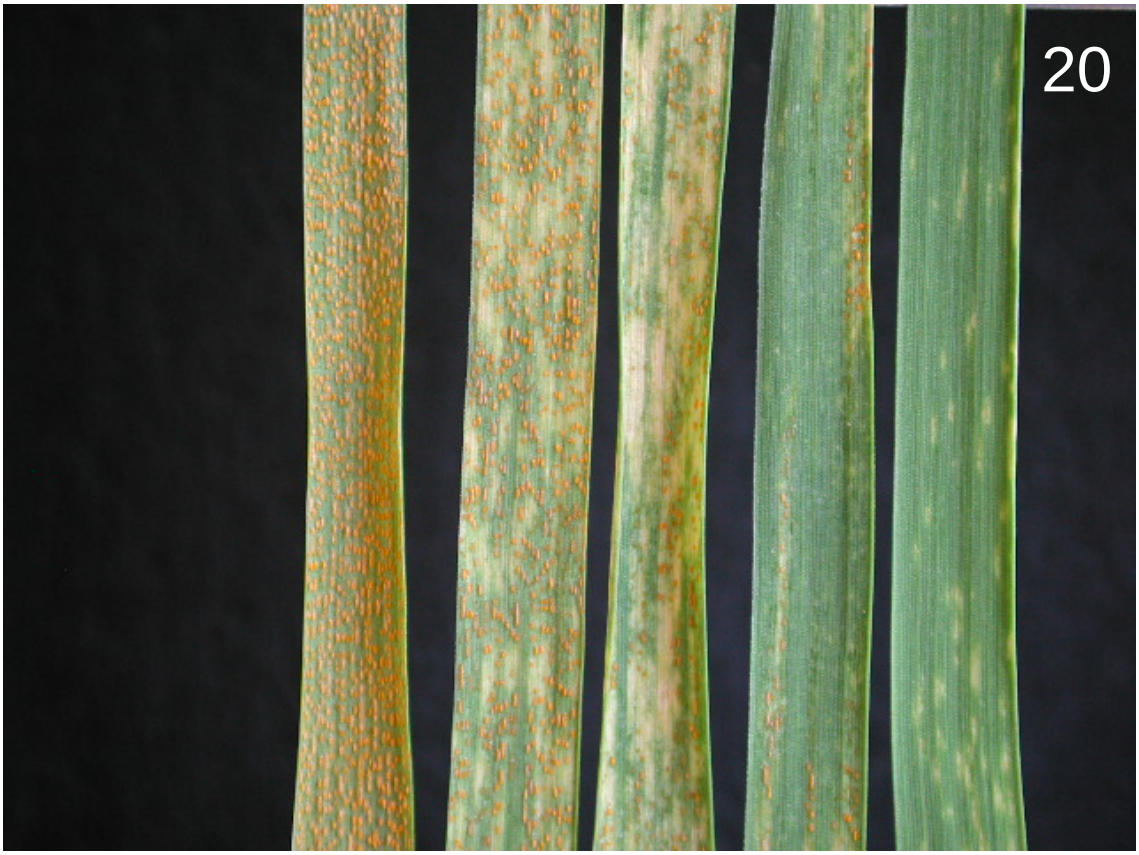


Fig. 19. A comparison between plant height of growth chamber (GC) and glasshouse (GH) grown mini-adult plants.

Fig. 20. Flag leaf reactions of intermediate and true-winter wheat cultivars tested as accelerated mini-adult plants in the glasshouse to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici.* From left to right: Morocco (control), Gariep, SST363, SST333 and PAN3377.


20

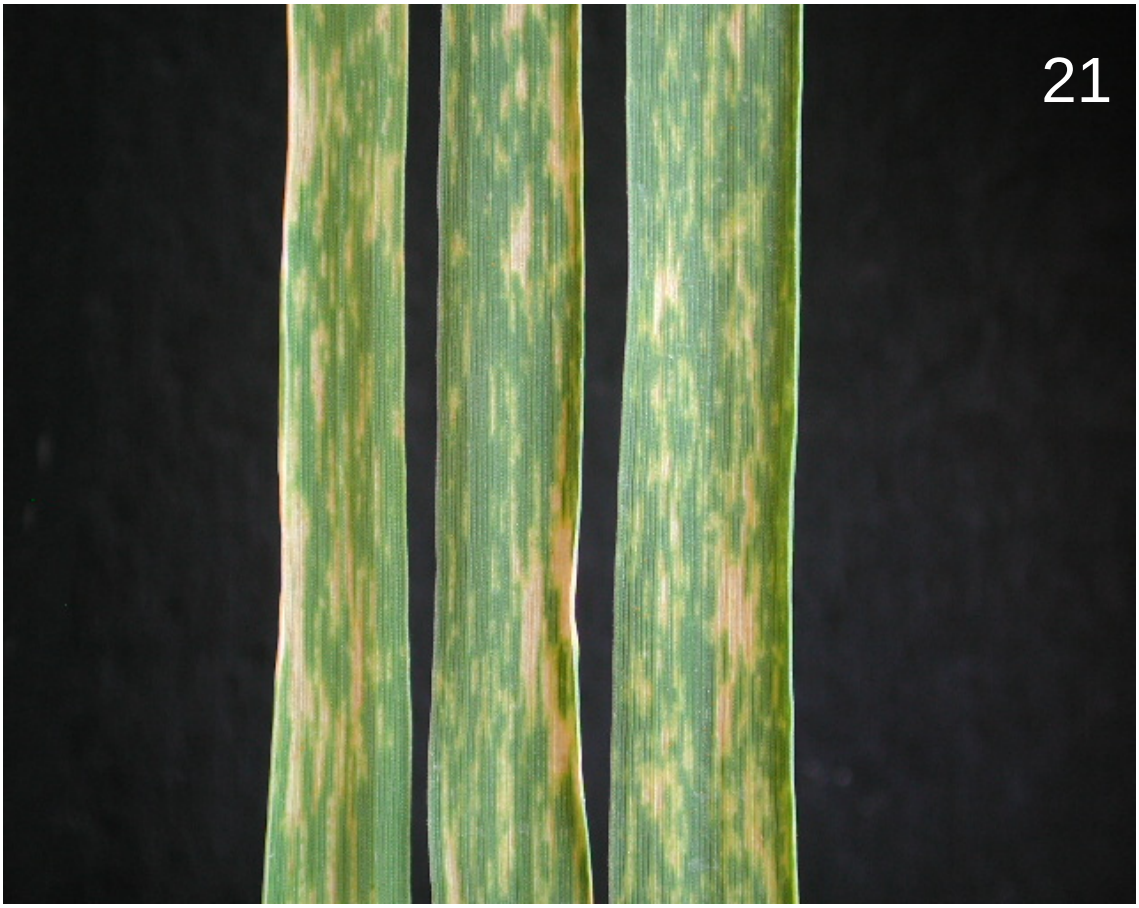
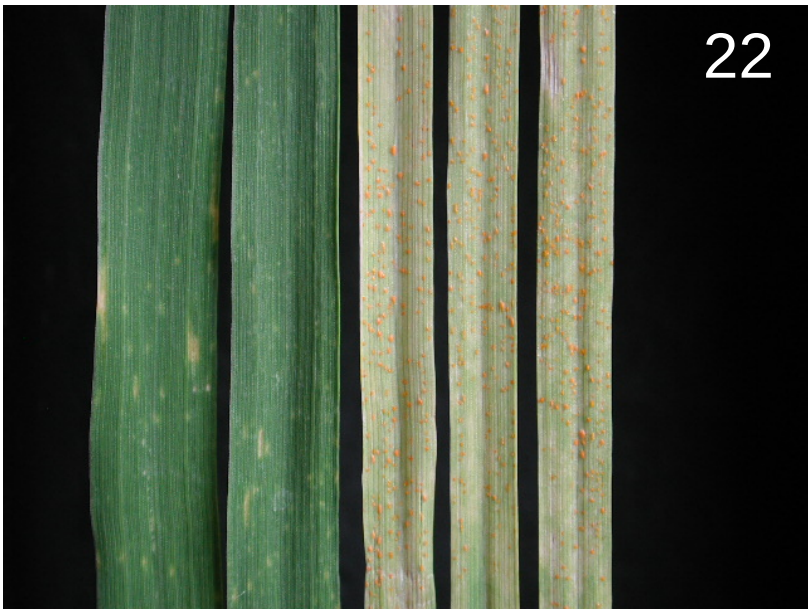

21

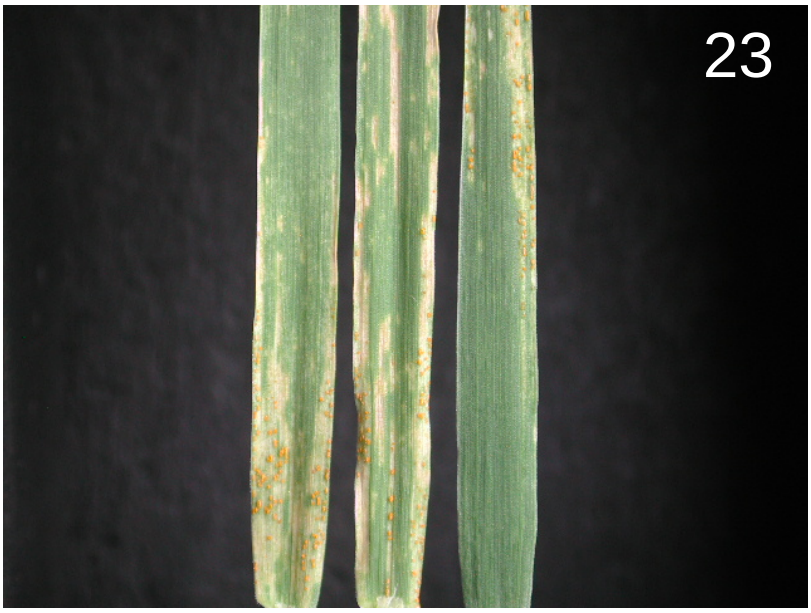
Fig. 21. Flag leaf reaction of the resistant winter wheat Tugela, tested as accelerated mini-adult plants in the glasshouse, to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici.*

Fig. 22. Flag- and lower leaf reactions of accelerated mini-adult line 44 of the 23rd CIMMYT Elite Spring Wheat Yield Trial to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici* in the glasshouse.

Fig. 23. Flag leaf reactions of accelerated mini-adult line 48 of the 23rd CIMMYT Elite Spring Wheat Yield Trial to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici* in the glasshouse.

Fig. 24. Field reaction of the 23rd CIMMYT Elite Spring Wheat Yield Trial lines to *Puccinia striiformis* f. sp. *tritici* in field plots at Greytown, KZN.


22


23

24

A GENETIC ANALYSIS OF STRIPE RUST RESISTANCE IN THE WHEAT CULTIVARS BAVIAANS AND SUNMIST

INTRODUCTION

Durable resistance in a crop variety remains effective despite widespread cultivation of the crop for a long period of time in an environment favourable to disease (Johnson, 1981). Breeding for durable resistance to stripe rust (caused by *Puccinia striiformis* f. sp. *tritici*) of wheat (*Triticum aestivum* L.) depends to a large extent on knowledge of variation in the pathogen and knowledge of the genetics controlling resistance (Worland, Gale and Law, 1987; Pretorius, 1998). By analysing wheat genotypes for the number and identity of resistance genes, and their mode of inheritance (Zwer and Qualset, 1991), information is gained by which similar resistances can be constructed in other breeding lines. Limitations in our understanding of the genetic basis of durable resistance often restrict the breeding of disease-resistant varieties that are satisfactory in all characters for commercial use (Worland *et al*., 1987).

The genetics of hexaploid bread wheat is complicated because the duplication and triplication of genes resulting from polyploidy lead to complex segregation patterns and epistatic effects, which can be difficult to analyse and resolve into the effects of component genes (Worland *et al*., 1987). Also, there are only a few reports on the gene action (whether genes are expressed in an additive, dominant, and/or epistatic fashion) for inheritance of adult-plant

resistance of wheat to stripe rust (Lupton and Johnson,1970; Zwer and Qualset, 1991; Chen and Line, 1993). Furthermore, epistasis of resistance genes varies depending on the pathogen race and genetic background of the parent in the cross (Chen and Line, 1992). Lupton and Johnson (1970) reported that durable, adult-plant stripe rust resistance in the wheat cultivar Little Joss was recessive and that its genetic control was complex. In genetic studies done by Calonnec, De Vallavieille-Pope and Johnson (2002) on the wheat differential cultivars Carstens V and Spaldings Prolific, resistance in both cultivars appeared complicated. According to Chen, Line and Jones (1995) more studies are needed to understand the complexity of the gene interactions between wheat cultivars and stripe rust races.

Because of the large numbers of plants that must be screened in most wheat breeding programmes, it is not possible for breeders to analyse components of resistance, such as rate of infection or spore production on individual plants or progenies. More detailed assessments of disease resistance can be applied to parents or to small numbers of selected lines in the later generations of breeding programmes, but a rapid method is essential for application to large numbers of segregating progeny in early generations (Worland *et al*., 1987). In the previous chapter a system was described to more efficiently obtain adult-plant reactions to *Puccinia striiformis* f. sp. *tritici*. Whereas this procedure was found to be reasonably reliable for screening pure-breeding wheat lines, information on its accuracy in phenotyping resistance in segregating populations is lacking.

The objective of this project was to study the inheritance of adult-plant resistance in the cultivars Baviaans and Sunmist, in particular to determine if

mini-adult wheat plants can be used in genetic studies of stripe rust resistance.

MATERIALS AND METHODS

Crosses

To obtain segregating populations allowing the above objectives to be met, lines with known stripe rust resistance were crossed with a susceptible cultivar. Crosses were made between Baviaans, Cook, SST57, Steenbras, Suneca, Sunlyn, Sunmist and Swift and the susceptible cultivar Avocet S (AvS), respectively. Considering infrastructure and logistics required to study inheritance in eight populations, the crosses Baviaans x AvS and Sunmist x AvS were selected for this study. Following hybridisation in April 2002, hand-harvested F_1 seeds of each cross (derived from a single pollinated spike) were surface-sterilised for 1 min. with a 1:6 v/v diluted commercial Jik (containing 3.5% sodium hypochlorite). Seeds were then washed with distilled water and germinated on filter paper moistened with 1% H_2O_2 in petri dishes. Seven days later germinated seedlings were transplanted to soil in plastic pots and grown in the glasshouse. During November 2002 the F_2 seed was harvested. In January 2003, 50 F_2 seeds of each of the Baviaans x AvS and Sunmist x AvS crosses were planted in the glasshouse. F_3 seed was harvested per plant in May 2003 and families numbered from 1 to 50.

Glasshouse experiment

To validate the accelerated screening system (mini-adults), the flag leaf

response of F_2 and F_3 plants of the two crosses was determined. For F_2 evaluations six to eight seeds were sown in 4 x 4 x 10 cm (95 cm^3) cones. F_3 families were sown in a similar way to obtain *ca*. 30 plants per family. Three replications of each parent and Morocco (susceptible control) were included. Plants were grown at ±25°C/16°C in a glasshouse in sterilized soil. Standard procedures for growing mini-adults were followed (previous chapter). Six weeks after sowing the plants were inoculated with a water suspension (25 $\times 10^4$ spores/ml) of pathotype 6E22A-containing Tween 20® as surfactant, and incubated as described in chapter 2. Fifteen days later the flag leaves were rated according to a 0 to 4 scale (Appendix 1) to group the F_2 plants as either susceptible, intermediate or resistant, and the F_3 families as susceptible, segregating or resistant.

Field experiment

The response of the F_2 and F_3 progenies of both the Baviaans x AvS and Sunmist x AvS crosses to *P. striiformis* f. sp. *tritici* was determined in field plots at Greytown, KwaZulu-Natal (KZN) in 2003. To allow rating of individual plants, 10 F_2 seeds were space-planted per 2-m row. For F_3 families 60 seeds were sown per 2-m row. Rows were spaced 90 cm apart. Three rows of each parent bordered the beginning and end of each population. Parents were furthermore replicated five times within the experimental plot. A stripe rust epidemic was initiated by inoculating spreader rows with pathotype 6E22A-. Three spreader rows, consisting of a mixture of Morocco, Hugenoot, Palmiet and McNair, surrounded the entire plot area whereas single spreader rows were planted perpendicular (in pathways) to all trial entries. Furthermore, Morocco was included after every tenth entry to facilitate stripe rust development among plots. Disease assessments were carried out on 18 September, 30 September

and 6 October 2003. Disease severity, combined with a field reaction type (Appendix 1) for each entry, was allocated according to the modified Cobb Scale (0-100%) (Peterson, Campbell and Hannah, 1948). Similar to the glasshouse screening procedure, F_2 plants were rated as either susceptible, intermediate or resistant and the F_3 families as susceptible, segregating or resistant. In Chi-square analyses of observed and expected ratios obtained for F_2 populations, data from the intermediate and resistant groups were pooled.

RESULTS

Glasshouse experiment

Baviaans x Avocet S cross

F_2 data from 149 plants are presented in Table 1. Of these, 114 plants had a resistant reaction to pathotype 6E22A-, 16 were intermediate and 19 had a susceptible reaction. Chi-square analyses indicated that the cross segregated for one dominant and one recessive gene for resistance ($\chi^2_{13:3}$ = 3.519), or alternatively one dominant gene and two independent recessive genes ($\chi^2_{55:9}$ = 0.2218) (Table 1).

In the F_3, 21 families were resistant, 28 showed within-family segregation for resistance and susceptibility (Figs. 1 - 4), and one family was homozygous susceptible (Tables 1 and 2). This suggests that Baviaans could have two dominant resistance genes ($\chi^2_{7:8:1}$ = 1.84) or that there are three resistance genes present in different combinations of dominance and recessivity ($\chi^2_{37:26:1}$ = 5.1331) (Table 1).

Sunmist x Avocet S cross

F_2 data were obtained for 82 plants in the glasshouse (Table 3). Of these, 27 plants were resistant, 21 were intermediate and 34 were susceptible to stripe rust. According to the Chi-square analyses, the best segregation fit was for three independent recessive genes for resistance ($\chi^2_{37:27}$ = 0.0179) (Table 3).

F_3 family data are presented in Tables 3 and 4. Four families were resistant, 45 showed within-family segregation for resistance and susceptibility (Figs. 5 - 8), and one family was homozygous susceptible. None of the ratios fitted adequately explained segregation of resistance genes in Sunmist (Table 3).

Field experiment

Baviaans x Avocet S cross

In the F_2 generation, nine plants were resistant, 54 were intermediate and 19 were susceptible to the pathotype 6E22A- (Table 1). Chi-square analyses indicated that the cross segregated for one dominant gene for resistance ($\chi^2_{3:1}$ = 0.5455).

Table 2 shows the data obtained for the 50 F_3 families to pathotype 6E22A-. Twenty three families were resistant, 24 showed within-family segregation, and three families were susceptible. Chi-square analysis indicated

that two dominant genes for resistance ($\chi^2_{7:8:1}$ = 0.1029) could be present in Baviaans (Table1).

Sunmist x Avocet S cross

Of the 95 F_2 plants, were 16 resistant, 32 intermediate and 47 susceptible to stripe rust (Table 3). Chi-square analyses suggested either two independent recessive genes for resistance ($\chi^2_{7:9}$ = 1.7726), or three independent recessive genes for resistance ($\chi^2_{37:27}$ = 2.0678), present in the cross (Table 3).

F_3 family data are presented in Table 4. Eight families were resistant, 38 showed within-family segregation for resistance and susceptibility, and four had a susceptible reaction to the pathotype. Chi-square analysis could not determine the number of resistance genes present in Sunmist (Table 3).

DISCUSSION

Genetic studies of rust resistance proceed in several stages and usually begin with the identification of resistant germ plasm (McIntosh, Wellings and Park, 1995). Presumed resistant genotypes are then crossed with a susceptible genotype to permit determination of the number of genes for resistance to a particular pathotype (McIntosh *et al.,* 1995). Because of the likely application of genetic studies to resistance breeding, it is essential to correlate glasshouse tests with field assessments. Parallel tests are best based on F_2 families from backcrosses or F_3 lines from direct crosses (McIntosh *et al.,* 1995).

With regard to adult-plant resistance the principle of confirming

glasshouse data in the field is even more applicable, as it is unlikely that the full complement of resistance will be recognised in seedling evaluations done in a controlled environment (Zwer and Qualset, 1991). Johnson (1981) mentioned the example of where seedling resistance transferred from Capelle Desprez was not durable in other varieties when Capelle Desprez itself remained resistant under similar conditions. It has been more difficult to identify resistance genes and chromosomal location for genes expressed only in adult-plants. In many crosses there may be several genes affecting resistance to stripe rust, and environment may alter the expression of resistance, often making it difficult to obtain segregation into discreet classes in the progeny (Worland *et al*., 1987).

The approach followed in the present study conformed to the guidelines in terms of selecting cultivars with high levels of adult-plant resistance not previously analysed, as well as comparing glasshouse and field data. For the Baviaans x AvS cross more than one ratio was significant. Segregation ratios indicated that there could be one to three genes present in Baviaans. Based on the fact that Baviaans illustrated a high level of resistance to the pathogen in both environments (; to tR reaction), it can be concluded that dominant resistance is present. The best fit was obtained with the 7:8:1 ratio ($\chi^2_{7:8:1}$ = 0.1029) in the field, suggesting the presence of two dominant genes. The hypothesis was further substantiated by the clear expression of two phenotypes (Fig. 9). The two genes are expressed as a resistant fleck and necrotic stripe reaction, respectively, and resemble the phenotypes observed in a Kariega x AvS population analysed by Ramburan *et al*. (2004). In the Sunmist x AvS cross only two ratios suggested meaningful Mendelian ratios, *i.e*. either two or three independent recessive genes for resistance. The best fit observed in the glasshouse ($\chi^2_{37:27}$ = 0.0176) was also confirmed in the field ($\chi^2_{37:27}$ = 2.0678). However, these data were obtained in the F_2 generation where ratings are based on single plants without the confirmation of replication. Due to the occurrence of chlorosis in the glasshouse, most probably derived from AvS

(Ramburan *et al*., 2004), distinctions between the intermediate and susceptible class were difficult and resistance data for Sunmist can thus not be regarded as conclusive.

A comparison between glasshouse and field data is summarised in Table 5 and Fig. 10 for Baviaans x AvS and in Table 6 and Fig. 11 for the Sunmist x AvS cross. The Chi-square test was used to compare the homogeneity of the samples. The actual calculations are given in Appendices 4 and 5. For the Baviaans x AvS cross the two environments differed significantly for the F_2 generation (χ^2 = 9.7498), but did not differ significantly for the F_3 (χ^2 =1.3986). The Sunmist x AvS cross responded similarly in both environments for the F_2 (χ^2 = 1.4645) and F_3 generations (χ^2 =3.7238). The system proved to be 75% accurate in a direct comparison of disease reactions of families from both crosses (differences are indicated with asterisks in Tables 2 and 4). The resemblance of the parental reactions between the two environments is illustrated in Figs. 1, 2, 5 and 6. However, when the segregating lines are compared (Figs. 3, 4, 7 and 8) a more resistant reaction (more chlorosis) was observed in the glasshouse. Different reactions between environments are not unexpected in the wheat stripe rust pathosystem. It has been documented that factors such as light and temperature could alter the expression of resistance (Zwer and Qualset, 1991; Chen and Line, 1993). Where this involves obligate pathogens it is often not possible to decide whether the observed effects are primarily due to the host, the pathogen or the interaction between them. Because of these variations in gene action it is not always possible to interpret segregation data or to make a full inventory of gene-for-gene interactions (Johnson, 1992).

In addition to a shorter evaluation cycle, the mini-adult screening system is more economic in terms of space. When the accelerated screening system

is compared to the normal adult-plant tests done in the glasshouse, more replicates could be tested in the same glasshouse space. One bench in the glasshouse used measures approximately 1.7 m^2 and can carry 100 1-liter-capacity pots (108 kg potting mix). On the same bench 245 95-cm^3-cones can be accommodated for mini-adults, using only 17 kg potting mix.

In conclusion, there appear to be two resistance genes present in Baviaans. Genetic studies of several sources indicate that adult-plant resistance to stripe rust is conferred by combinations of two to several genes (McIntosh, 1992; Singh, 1992; McIntosh and Brown, 1997; Ramburan *et al.*, 2004). Results from the Sunmist x AvS cross were not conclusive. This can be due to inaccurate interpretation of the intermediate class for this cross, due to the chlorosis obtained. According to Luig and Rajaram (1972) even in favourable environments, the expression of resistance genes would be determined by the genetic background of the parents used in the cross. Chen and Line (1992) mentioned that more than one ratio was significant for some crosses between stripe rust resistant and susceptible lines. Best fits occurred when the intermediate infection types were analysed separately or when they were combined with resistant infection types, but not when combined with the susceptible category. Combining the intermediate with the resistant infection types did not change the number of genes but did change the gene interaction. The present study suggested that inheritance of resistance to stripe rust should not be studied in F_2 or F_3 generations, but rather in replicated plots of a doubled-haploid population, where environmental and genetic variance can be separated. Ramburan *et al*. (2004) used this approach successfully to characterize stripe rust resistance in the South African wheat cultivar Kariega.

In segregating populations the accelerated screening system did not prove to be as successful as in screening pure lines. Based on the fact that the reaction of Avocet S was not completely susceptible (Fig. 12) in the glasshouse, and most likely influenced ratios, the lower success rate appears to be a function of the parental genotypes rather than the system in general.

REFERENCES

Calonnec, A., de Vallavieille-Pope, C., and Johnson, R. 2002. Genetic analysis of resistance of the differential cultivars Carstens V and Spaldings Prolific to two races of *Puccinia striiformis*. Plant Pathology 51: 777-786.

Chen, X., and Line, R.F. 1992. Identification of stripe rust resistance genes in wheat genotypes used to differentiate North American races to *Puccinia striiformis.* Phytopathology 82: 1428-1434.

Chen, X., and Line, R.F. 1993. Inheritance of stripe rust resistance in wheat cultivars postulated to have resistance genes at *Yr3* and *Yr4* loci. Phytopathology 83: 382-388.

Chen, X., Line, R.F., and Jones, S.S. 1995. Chromosomal location of genes for resistance to *Puccinia striiformis* in winter wheat cultivars Heines VII, Clement, Moro, Tyee, Tres, and Daws. Phytopathology 85: 1362-1367.

Johnson, R. 1981. Durable resistance: definition of, genetic control, and attainment in plant breeding. Phytopathology 71: 567-568.

Johnson, R. 1992. Past, present and future opportunities in breeding for disease resistance, with examples from wheat. Euphytica 63:3-22.

Luig, N.H., and Rajaram, S. 1972. The effect of temperature and genetic background on host gene expression and interaction to *Puccinia graminis tritici*. Phytopathology 62: 1171-1174.

Lupton, F.G.H., and Johnson, R. 1970. Breeding for adult-plant resistance to yellow rust in wheat. Annals of Applied Biology 66: 137-143.

McIntosh, R.A. 1992. Pre-emptive breeding to control wheat rusts.

Euphytica 63: 103-113.

McIntosh, R.A., and Brown, G.N. 1997. Anticipatory breeding for resistance to rust diseases in wheat. Annual Review of Phytopathology 35: 311-326.

McIntosh, R.A., Wellings, C.R., and Park, R.F. 1995. Wheat Rusts: An Altas of Resistance Genes. CSIRO, East Melbourne, Australia.

Peterson, R.F., Campbell, A.B., and Hannah, A.E. 1948. A diagrammatic scale for estimating rust intensity of leaves and stems of cereals. Canadian Journal of Research Section C 26: 496-500.

Pretorius, Z.A. 1998. Report of a study visit to the Plant Breeding Institute-Cobbitty, University of Sydney, Australia.

Ramburan, V.P., Pretorius, Z.A., Louw, J.H., Boyd, L.A., Smith, P.H., Boshoff, W.H.P., and Prins, R. 2004. A genetic analysis of adult plant resistance to stripe rust wheat cultivar Kariega. Theoretical Applied Genetics 108: 1426-1433.

Singh, R.P. 1992. Genetic association of leaf rust resistance gene *Lr34* with adult-plant resistance to stripe rust in bread wheat. Phytopathology 82: 835-838.

Worland, A.J., Gale, M.D., and Law, C.N. 1987. Wheat genetics. Pages 129-171 in Wheat Breeding. Its Scientific Basis. F,G.H. Lupton, eds. Chapman and Hall, London.

Zwer, P.K., and Qualset, C.O. 1991. Genes for resistance to stripe rust in four spring wheat varieties. Euphytica 58: 171-181.

Table 1. Analysis[a] of segregating ratios expected for a one, two or three gene hypothesis in glasshouse-grown mini-adult plants and field plots of the Baviaans x Avocet S F_2 and F_3 populations to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici*

Cross	Environment	Number of plants			Gene hypothesis	Expected ratio	χ^2
		Resistant	Intermediate	Susceptible			
Baviaans x Avocet S F_2	Glasshouse	114	16	19	1 (one dominant gene)	3:1	11.9217*
			130				
	Field	9	54	25	1 (one dominant gene)	3:1	0,5455
			63				
	Glasshouse				2 (two dominant genes)	15:1	10.7495*
	Field					15:1	73.7455*
	Glasshouse				2 (one dominant, one	13:3	3,5190
	Field				recessive gene)	13:3	5.3893*
	Glasshouse				2 (two independent	7:9	114.5594*
	Field				recessive genes)	7:9	27.7173*
	Glasshouse				3 (one dominant, two	55:9	0,2118
	Field				independent recessive genes)	55:9	14.9877*
		Number of families					
		Resistant	Segregating	Susceptible			
Baviaans x Avocet S F_3	Glasshouse	21	28	1	1 (one dominant or	1:2:1	16.7200*
	Field	23	24	3	one recessive gene)	1:2:1	16.0800*
	Glasshouse				2 (two dominant genes)	7:8:1	1,8400
	Field					7:8:1	0,1029
	Glasshouse				3 (different combinations of	37:26:1	5,1331
	Field				dominant or recessive genes)	37:26:1	19.0385*

[a] The Chi-square test was used to compare homogeneity of samples; * differed significantly from expected ratio.

Table 2. Glasshouse mini-adult plant response and field reaction of the Baviaans x Avocet S F_3 population to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici*

Family	Glasshouse response[a]			Response	Field response[c]			Response
	Resistant	Intermediate	Susceptible	category[b]	Resistant	Intermediate	Susceptible	category
Avocet S[d]			2++c	Susceptible			60-70S	Susceptible
Baviaans[e]	;			Resistant	tR			Resistant
Morocco[f]			3++c,4	Susceptible			100S	Susceptible
1	;	1c	2++c	Segregating*	5R			Resistant
2	;		2++c	Segregating	30MR-R		90-100S	Segregating
3	;	1c		Segregating*	5R-30R			Resistant
4	;	1c	2++c	Segregating	5R		90S	Segregating
5	;		2++c	Segregating*	5R-30R			Resistant
6	;			Resistant*	5R		90S	Segregating
7	;			Resistant	5R-30R			Resistant
8	;	1c	2++c	Segregating*	5R			Resistant
9	;			Resistant	5R-30R			Resistant
10	;		2++c	Segregating	30R		90S	Segregating
11	;			Resistant	tR			Resistant
12	;			Resistant	0R			Resistant

Table 2 (cont.). Glasshouse mini-adult plant response and field reaction of the Baviaans x Avocet S F_3 population to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici*

Family	Glasshouse response[a]			Response category[b]	Field response[c]			Response category
	Resistant	Intermediate	Susceptible		Resistant	Intermediate	Susceptible	
13	;			Resistant*	5R-30R		90S	Segregating
14	;	1c	2++c	Segregating	30R		90S	Segregating
15	;			Resistant	5R-30R			Resistant
16	;			Resistant*	30R		90S	Segregating
17	;	1c		Segregating*	5R-30R			Resistant
18	;	1cn	2++c	Segregating	5R	40MR-MS	90S	Segregating
19	;	1c		Segregating	5R	40MR-MS	90S	Segregating
20	;	1c	2++c	Segregating	10R		90S	Segregating
21	;	1c	2++c	Segregating*			90S	Susceptible
22	;			Resistant	20MR			Resistant
23	;	1c		Segregating	5R-30R		90S	Segregating
24	;	1c		Segregating*	5R-30R			Resistant
25	;	1c	2++c	Segregating	5R	40MR-MS	90S	Segregating
26	;			Resistant	5R-30R			Resistant
27	;	1c	2++c	Segregating	30R		90S	Segregating
28	;			Resistant	5R-30R			Resistant

Table 2 (cont.). Glasshouse mini-adult plant response and field reaction of the Baviaans x Avocet S F_3 population to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici*

Family	Glasshouse response[a]			Response category[b]	Field response[c]			Response category
	Resistant	Intermediate	Susceptible		Resistant	Intermediate	Susceptible	
29	;			Resistant	5R-30R			Resistant
30	;			Resistant	tR-30R			Resistant
31	;	1c	2++c	Segregating	5R	40MR-MS	90S	Segregating
32	;			Resistant	tR-30R			Resistant
33	;		2++c	Segregating	10R		90S	Segregating
34	;	1cn		Segregating	10R		90S	Segregating
35	;	1c	2++c	Segregating	10R	40MR-MS	90S	Segregating
36	;	1cn		Segregating		40MR-MS	90S	Segregating
37	;			Resistant	5R-30R			Resistant
38	;	1+c		Segregating*			80-90MS-S	Susceptible
39	;	1c	2++c	Segregating	5R	40MR-MS	90S	Segregating
40	;			Resistant	5R-30R			Resistant
41	;			Resistant*	5R	40MR-MS	90S	Segregating
42	;			Resistant	Tr			Resistant
43	;	1c	2c	Segregating		30MR-MS	90S	Segregating
44	;			Resistant	5R-30R			Resistant

Table 2 (cont.). Glasshouse mini-adult plant response and field reaction of the Baviaans x Avocet S F_3 population to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici*

Family	Glasshouse response[a]			Response category[b]	Field response[c]			Response category
	Resistant	Intermediate	Susceptible		Resistant	Intermediate	Susceptible	
45	;			Resistant	5R-30R			Resistant
46	;			Resistant*	5R		90S	Segregating
47			2++c	Susceptible			90S	Susceptible
48	;		2c	Segregating*	40MR			Resistant
49	;	1c	2++c	Segregating	5R	40MR-MS	90S	Segregating
50	;	1c	2++c	Segregating	5R	40MR-MS	90S	Segregating

[a] Glasshouse response was measured on a 0 to 4 infection type scale.

[b] Glasshouse reponse different (*) from field reaction.

[c] Field response was measured according to R (resistant), MR (moderately resistant), MS (moderately susceptible) and S (susceptible) reaction type classes.

[d] Male parent.

[e] Female parent.

[f] Susceptible control.

Table 3. Analysis[a] of segregating ratios expected for a one, two or three gene hypothesis in glasshouse-grown mini-adult plants and field plots of the Sunmist x Avocet S F_2 and F_3 populations to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici*

Cross	Environment	Number of plants			Gene hypothesis	Expected ratio	χ^2
		Resistant	Intermediate	Susceptible			
Sunmist x Avocet S F_2	Glasshouse	27	21	34	1 (one dominant gene)	3:1	11.8537*
			48				
	Field	16	32	47	1 (one dominant gene)	3:1	30.3474*
			48				
	Glasshouse				2 (two dominant genes)	15:1	173.5317*
	Field					15:1	302.9116*
	Glasshouse				2 (one dominant, one recessive gene)	13:3	27.7686*
	Field					13:3	58.8634*
	Glasshouse				2 (two independent recessive genes)	7:9	7.2853*
	Field					7:9	1,7726
	Glasshouse				3 (three independent, recessive genes)	37:27	0,0176
	Field					37:27	2,0678
		Number of families					
		Resistant	Segregating	Susceptible			
Sunmist x Avocet S F_3	Glasshouse	4	45	1	1 (one dominant or one recessive gene)	1:2:1	32.3600*
	Field	8	38	4		1:2:1	14.1600*
	Glasshouse				2 (two dominant genes)	7:8:1	32.0510*
	Field					7:8:1	15.8057*
	Glasshouse				3 (different combinations of dominant or recessive genes)	37:26:1	51.5258*
	Field					37:26:1	43.7833*

[a] The Chi-square test was used to compare homogeneity of samples; * differed significantly from expected ratio.

Table 4. Glasshouse mini-adult plant response and field reaction of the Sunmist x Avocet S F_3 population to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici*

Family	Glasshouse response[a]			Response category[b]	Field response[c]			Response category
	Resistant	Intermediate	Susceptible		Resistant	Intermediate	Susceptible	
Avocet S[d]			2++c	Susceptible			60-70S	Susceptible
Morocco[e]			3++,4	Susceptible			100S	Susceptible
Sunmist[f]	;			Resistant	30R-MR			Resistant
1	;	1c	2++c	Segregating		60MR	100S	Segregating
2		1c	2++cn	Segregating		60MR	80S	Segregating
3	;	1c	2++c	Segregating	20MR	50MR	80S	Segregating
4	;	1c	2++c	Segregating	40MR		90MS-S	Segregating
5	;	1c	2++c	Segregating*			70S	Susceptible
6	;	1c	2++c	Segregating	40MR		80S	Segregating
7	;	1c	2++c	Segregating	40MR		90S	Segregating
8	;	1c		Segregating	30R-MR		100S	Segregating
9	;	1c	2++c	Segregating	30R-MR		100S	Segregating
10	;	1c	2++c	Segregating	30 - 40R-MR		90 - 100S	Segregating
11			2++c	Susceptible			100S	Susceptible
12	;	1c	2++c	Segregating	30MR-MS		100S	Segregating

Table 4 (cont.). Glasshouse mini-adult plant response and field reaction of the Sunmist x Avocet S F_3 population to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici*

Family	Glasshouse response[a]			Response category[b]	Field response[c]			Response category
	Resistant	Intermediate	Susceptible		Resistant	Intermediate	Susceptible	
13	;	1c	2++c	Segregating	20-50MR		90MS-S	Segregating
14	;	1c	2cn,2++c	Segregating		70MR-MS	90MS-S	Segregating
15	;	1c	2++c	Segregating*			100S	Susceptible
16	;	1c		Segregating		70MR-MS	90-100S	Segregating
17	;	1c	2++c	Segregating		70MR-MS	90-100S	Segregating
18	;	1cn	2++c	Segregating*			100S	Susceptible
19	;	1c	2++c	Segregating	20MR-R	70MR-R	90MS-S	Segregating
20		1c	2++c	Segregating	20MR	60MR-MS	90MS-S	Segregating
21	;			Resistant*		60MR-R	90MS-S	Segregating
22	;	1c	2++c	Segregating	20MR-R	70MR-R	90MS-S	Segregating
23	;	1c	2++c	Segregating		80-90MR-MS	100S	Segregating
24	;	1c	2++c	Segregating		60MS	100S	Segregating
25	;	1c		Segregating	10R		100S	Segregating
26	;	1c	2++c	Segregating		60MR-R	90-100S	Segregating
27	;	1c	2++c	Segregating	20MR-R	70MR-R	90MS-S	Segregating
28	;	1c		Segregating	10R-MR	40MR-MS	90MS-S	Segregating

Table 4 (cont.). Glasshouse mini-adult plant response and field reaction of the Sunmist x Avocet S F_3 population to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici*

Family	Glasshouse response[a]			Response category[b]	Field response[c]			Response category
	Resistant	Intermediate	Susceptible		Resistant	Intermediate	Susceptible	
29	;	1c	2++c	Segregating	Tr		100S	Segregating
30	;	1c		Segregating*	30R-MR			Resistant
31	;	1c		Segregating	20MR-R	70MR-R	90MS-S	Segregating
32	;			Resistant*	20MR-R	70MR-R	90MS-S	Segregating
33	;	1c	2++c	Segregating*	30R			Resistant
34	;	1c		Segregating		70MR-MS	90-100S	Segregating
35	;	1c		Segregating*	30MR-R			Resistant
36		1c	2++c	Segregating		80MR-MS	90S	Segregating
37	;	1c	2++c	Segregating		60MS	100S	Segregating
38	;			Resistant	30R-MR			Resistant
39	;			Resistant	30R-MR			Resistant
40	;	1c	2++c	Segregating	20MR-R	70MR-R	90MS-S	Segregating
41	;	1c	2++c	Segregating	20R		90MR-MS	Segregating
42	;	1c	2++c	Segregating	30MR-R		90MS-S	Segregating
43	;	1c		Segregating	20MR-R	70MR-R		Segregating
44	;	1c		Segregating		50MR-R	90MS-S	Segregating

Table 4 (cont.). Glasshouse mini-adult plant response and field reaction of the Sunmist x Avocet S F_3 population to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici*

Family	Glasshouse response[a]			Response category[b]	Field response[c]			Response category
	Resistant	Intermediate	Susceptible		Resistant	Intermediate	Susceptible	
45	;	1c		Segregating*	30MR-MS			Resistant
46	;	1c	2++c	Segregating	20MR-R	70MR-R	90MS-S	Segregating
47	;	1c	2++c	Segregating	20MR-R	70MR-R	90MS-S	Segregating
48	;	1c	2++c	Segregating*		70R-MR		Resistant
49	;	1c	2++c	Segregating		70MR-MS	90MS-S	Segregating
50	;	1c		Segregating*	30MR-R			Resistant

[a] Glasshouse response was measured on a 0 to 4 infection type scale.

[b] Glasshouse reponse different (*) from field reaction.

[c] Field response was measured according to R (resistant), MR (moderately resistant), MS (moderately susceptible) and S (susceptible) reaction type classes.

[d] Male parent.

[e] Susceptible control.

[f] Female parent.

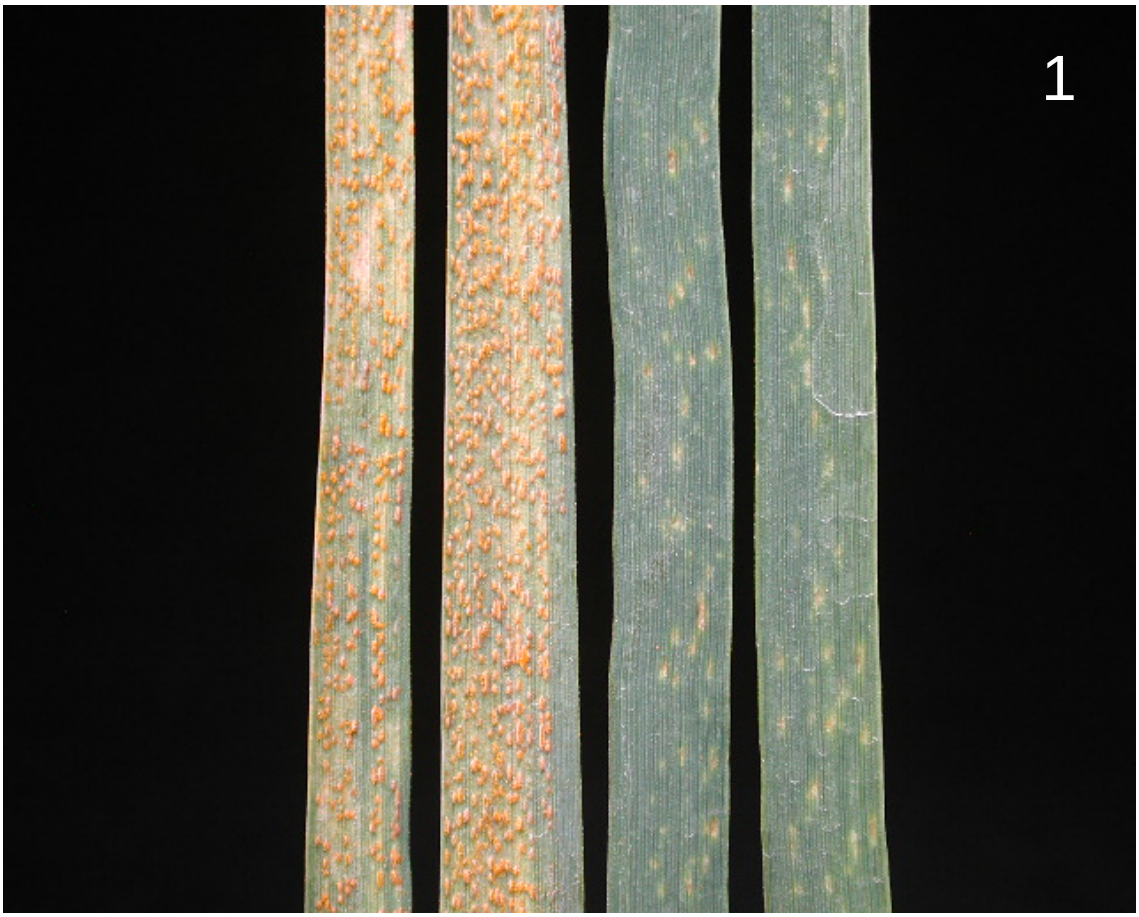
Table 5. A comparison[a] of the effect of environment on segregation ratios of Baviaans x Avocet S progenies tested as mini-adults in a glasshouse and in the field to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici*

Cross		Number of plants		χ^2		$\Sigma\chi^2$	$\chi^2_{0.050}$
		Glasshouse	Field	Glasshouse	Field		
Observed							
Baviaans x Avocet S F_2	Resistant	130	63				
	Susceptible	19	25	0,6694	1,1250		
Expected						9.7498*	3.8400[b]
Baviaans x Avocet S F_2	Resistant	121	72	2,8929	5,0625		
	Susceptible	28	16				
		Number of families					
Observed							
Baviaans x Avocet S F_3	Resistant	21	23				
	Segregating	28	24				
	Susceptible	1	3	0,0455	0,0455		
Expected				0,1538	0,1538	1,3986	5.9000[c]
Baviaans x Avocet S F_3	Resistant	22	22	0,5000	0,5000		
	Segregating	26	26				
	Susceptible	2	2				

[a] Standard Chi-square test was used to compare homogeneity of samples (Appendix 4), [b]1 *df*; [c] 2 *df.*
* Differed significantly from expected ratio.

Fig. 1. Flag leaf reactions of accelerated mini-adult plants of Avocet S (two leaves on the left) and Baviaans (two leaves on the right) to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici* in the glasshouse.

Fig. 2. Flag leaf reactions of Avocet S (left) and Baviaans (right) to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici* in field plots at Greytown, KZN.


1

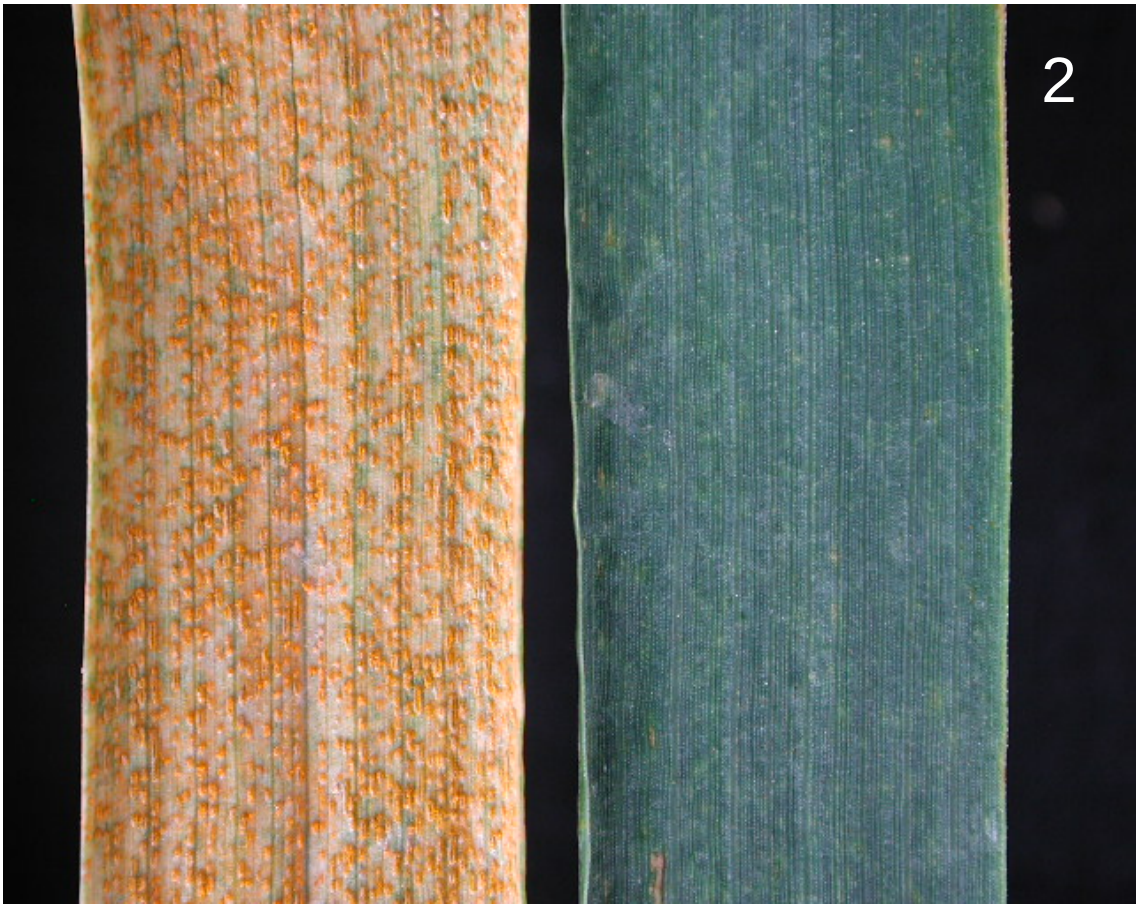

2

Fig. 3. Flag leaf reactions of accelerated mini-adult plants of the Baviaans x Avocet S cross in the glasshouse to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici*. From left to right: two leaves Avocet S (parent); two leaves Baviaans (parent); three leaves F_3 family 18 (infection types ;, 1cn, 2++c).

Fig. 4. Flag leaf reactions of the Baviaans x Avocet S F_3 family 18 to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici* in field plots at Greytown, KZN. From left to right: 5R, 40MR-MS and 90S.

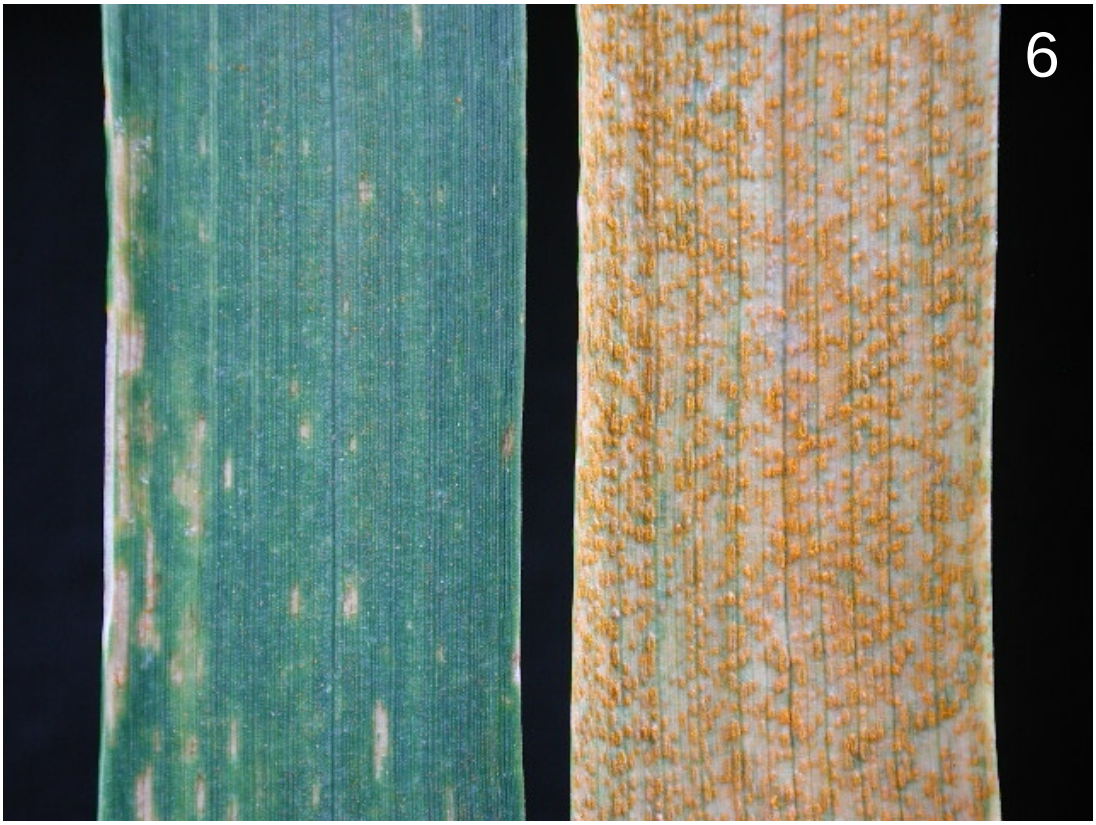
3

4

Fig. 5. Flag leaf reactions of accelerated mini-adult plants of Sunmist (two leaves on the left) and Avocet S (two leaves on the right) to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici* in the glasshouse.

Fig. 6. Flag leaf reactions of Sunmist (left) and Avocet S (right) to pathotype

5


6

6E22A- of *Puccinia striiformis* f. sp. *tritici* in field plots at Greytown, KZN.

Fig. 7. Flag leaf reactions of accelerated mini-adult plants of the Sunmist x Avocet S cross in the glasshouse to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici*. From left to right: two leaves Sunmist (parent); two leaves Avocet S (parent); three leaves Sunmist x Avocet S F_3 family 13 (infection types ;, 1c, 2++c).

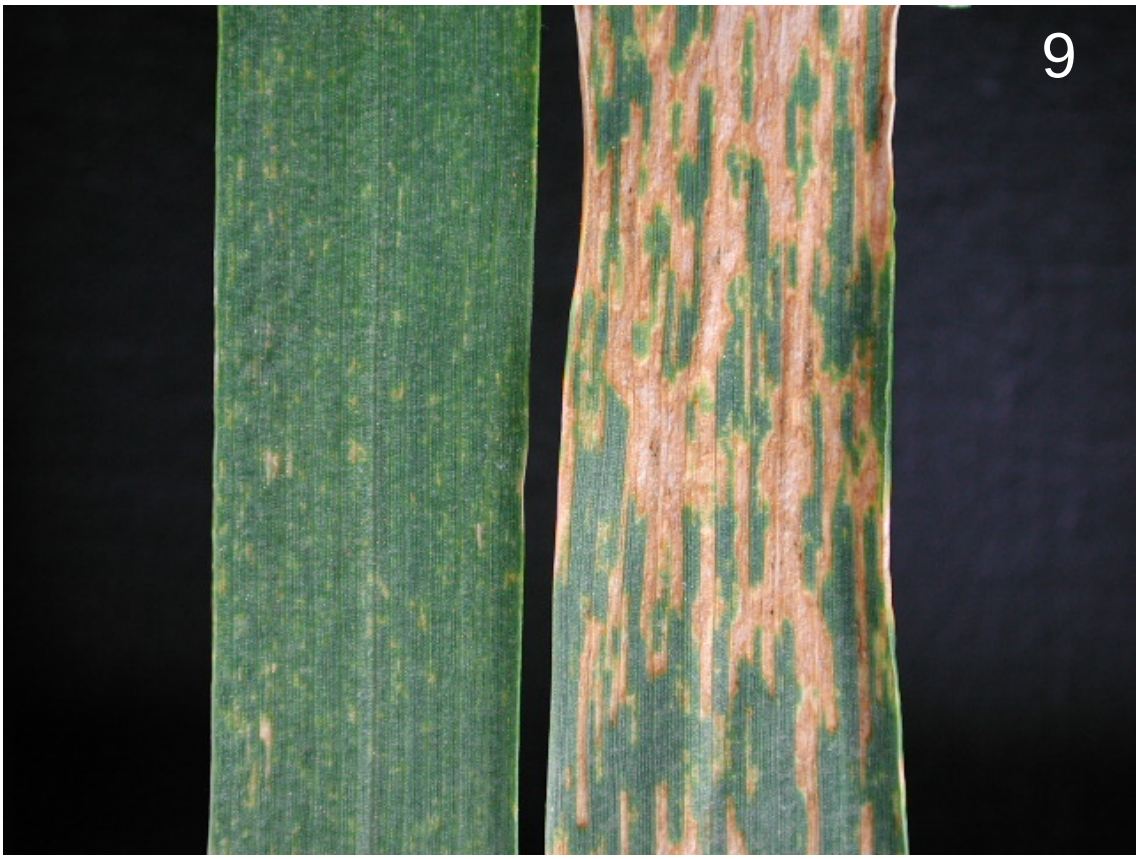
Fig. 8. Flag leaf reactions of the Sunmist x Avocet S cross F_3 family 13 to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici* in field plots at Greytown, KZN.

7

8

Fig. 9. The expression of the two proposed resistance genes in Baviaans to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici* in field plots at Greytown,

KZN as observed in the F_3 generation of a Baviaans x Avocet S cross.


9

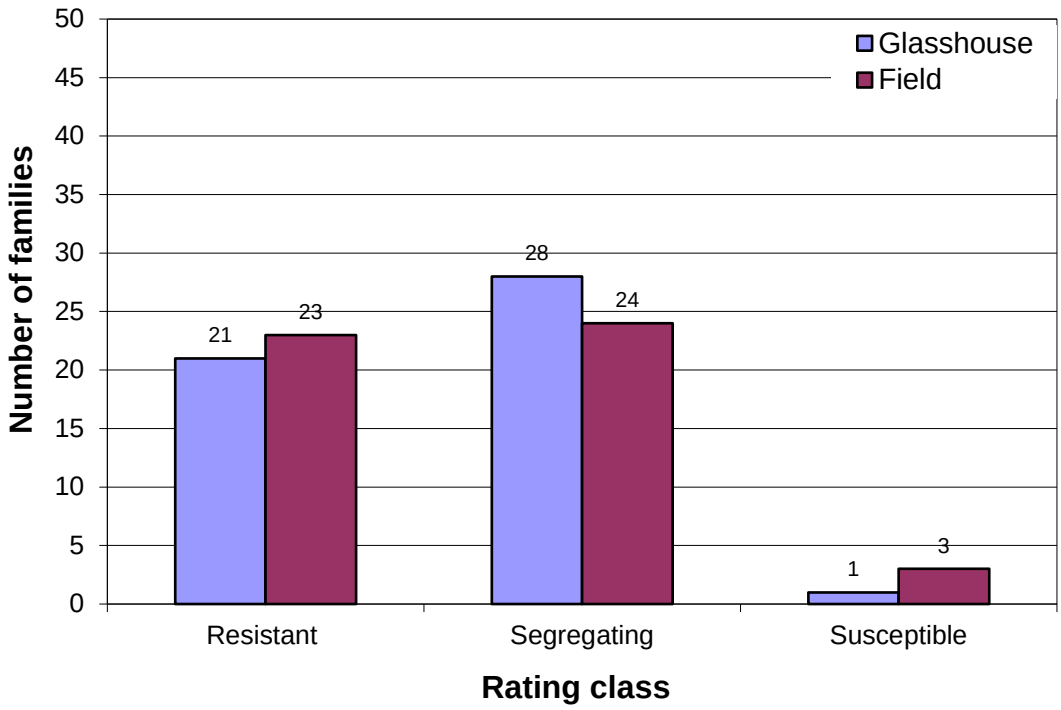


Fig. 10. A glasshouse and field comparison of the mini-adult plant response of the Baviaans x Avocet S F_3 population to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici*. Actual values are given above each bar.

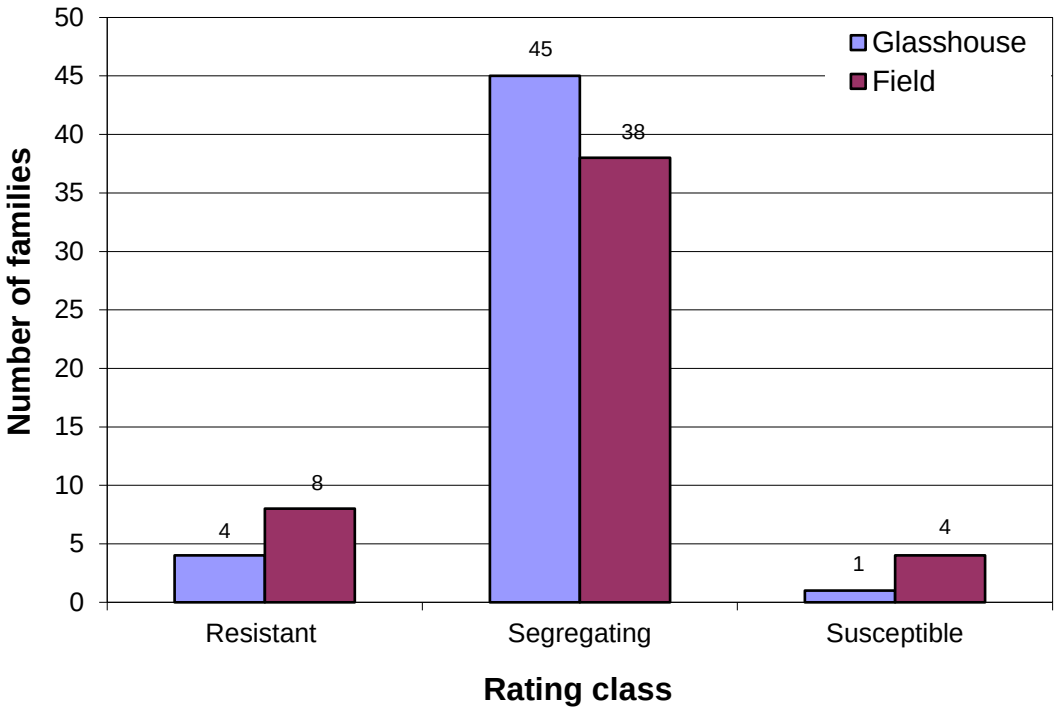
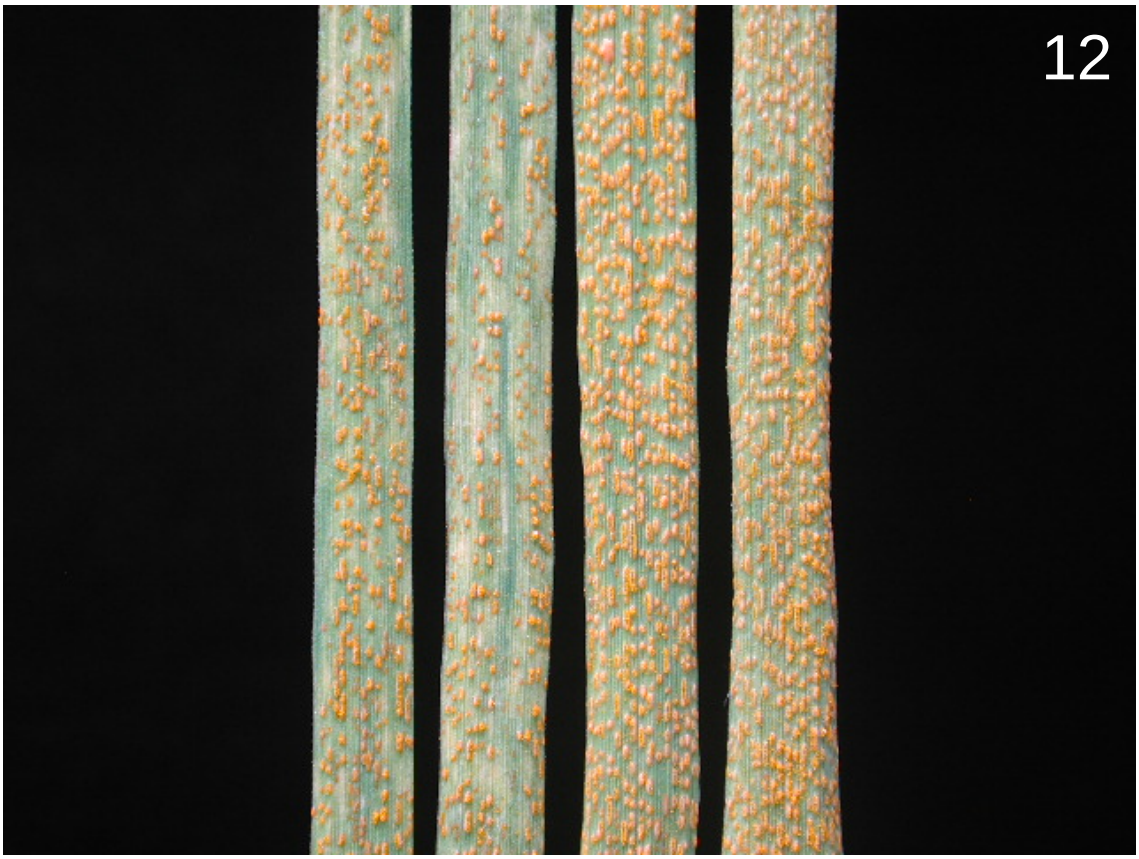


Fig. 11. A glasshouse and field comparison of the mini-adult plant response of the Sunmist x Avocet S F_3 population to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici*. Actual values are given above each bar.

Fig. 12. A comparison between mini-adult flag leaf reactions of Avocet S (two leaves on the left) and Morocco (two leaves on the right) to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici* in the glasshouse. Avocet S consistently showed varying degrees of flag leaf chlorosis in the glasshouse.


12

SUMMARY

The objective of this study was to develop and optimise methods to detect adult-plant resistance (APR) in wheat to stripe rust caused by *Puccinia striiformis* f.sp. *tritici*. A collection of spring wheats was tested at the seedling stage with different temperature and light regimes to test the hypothesis that environmental variation can induce APR expression in primary leaves. Treatments included pre-inoculation conditioning of seedlings at either 18° or 25°C, followed by post-inoculation temperature and light treatments. In some treatments intermittent low and high temperatures (18° or 25°C) were used as well as a treatment with continuous low temperature (10°C). The highest infection types were encountered with a pre-inoculation treatment of 18°C combined with a 24 h light cycle, and a post-inoculation treatment of 18°C combined with a 18 h light and 6h darkness cycle. Day length influenced infection types more than temperature. All treatments subjected to less than 14 h light per day showed a reduction in infection levels. Dark periods experienced prior to inoculation were not conducive to infection type development. Clear banding of infection zones and reduced sporulation occurred. Based on the lack of correlation between seedling infection types and adult responses observed in the field, no environment was conducive to expression of APR to stripe rust in seedlings.

Rating of adult plants for stripe rust resistance has traditionally been problematic in terms of infection levels and repeatability. Furthermore, several attempts to investigate the reaction of normally grown adult plants in the

glasshouse failed due to leaf damage. To improve these methods, an accelerated system of producing adult wheat plants in a controlled environment was tested. By manipulating plant density, pot size, light and temperature, wheat plants reached maturity quicker than normally grown adult plants and were referred to as mini-adult plants. In terms of leaf damage the mini-adults were better suited for flag (terminal) leaf evaluation. For optimum results, the mini-adult plants should be grown in a glasshouse rather than growth chamber and inoculated between heading and flowering.

According to flag leaf infection type and whole plant reactions observed, the mini-adults provided an acceptable system for comparing adult plants to different isolates of *P. striiformis*. This system accurately detected APR in most winter wheats tested and was reliable for a collection of CIMMYT spring type wheats. Disease ratings of 98 CIMMYT lines tested with this accelerated system showed a correlation of 88% with field ratings.

The mini-adult plant system was also tested with populations segregating for APR. Because of the likely application of genetic studies to resistance breeding, it is essential to correlate glasshouse tests with field assessments. The approach followed in the present study conformed to guidelines in terms of selecting cultivars with high levels of APR not previously analysed, as well as comparing glasshouse and field data. Segregation ratios indicated the presence of two resistance genes in the F_3 of a Baviaans x Avocet S cross ($\chi^2_{7:8:1}$ = 0.1029), but were inconclusive for a Sunmist x Avocet S cross where Mendelian ratios could not be confirmed. The accelerated screening system was less successful than the screening of pure lines. Based on the fact that the reaction of Avocet S was not completely susceptible in the glasshouse, and most likely influenced ratios, the lower success rate appeared to be a function of the parental genotypes rather than in the system in general.

Compared to the normal glasshouse procedure, the improved APR

system took significantly less time and physical resources to achieve ratings.

OPSOMMING

Die doel van hierdie studie was om ‘n metode te ontwikkel en te optimiseer waarmee volwasse-plantweerstand (VPW) in koring teen streeproes (*Puccinia striiformis* f.sp. *tritici*) geïdentifiseer kan word. ‘n Versameling lentekorings is op saailingstadium aan verskillende temperatuur- en ligbehandelings blootgestel om die hipotese te toets dat omgewingstoestande VPW in saailinge tot uiting kan bring. Behandelings het bestaan uit voor-inokulasie blootstelling van saailinge aan 18° of 25°C, gevolg deur na-inokulasie temperatuur- en ligbehandelings. In sommige behandelings is lae en hoë temperature (18° of 25°C) afwisselend getoets, en in ander volgehoue lae temperatuur (10°C). Die hoogste infeksietipes is verkry met ‘n voor-inokulasie temperatuur van 18°C gekombineer met 24 h lig behandeling, en ‘n na-inokulasie temperatuur van 18°C, met ‘n kombinasie van 18 h lig en 6 h donker periode. Alle behandelings wat minder as 14 h ligblootstelling gehad het, het ‘n afname in infeksietipe getoon. Donker periodes voor inokulasie het nie goeie infeksie tot gevolg gehad nie. Infeksie het in duidelike bande voorgekom, met verminderde sporulasie. As gevolg van die swak korrelasie tussen die saailing infeksietipes en volwasse-plantreaksies in die veld, is tot die gevolgtrekking gekom dat omgewing nie VPW akkuraat in saailinge uitdruk nie.

Lesings van volwasse-plantreaksies is tradisioneel moeilik in terme van

infeksie vlakke en herhaalbaarheid. Verskeie pogings om die reaksie van normaal gekweekte volwasse plante in die glashuis te toets het misluk weens blaarskade. Om hierdie metode te verbeter, is ‘n versnelde sisteem ontwikkel wat volwasse plante in ‘n beheerde omgewing toets. Deur plantdigtheid, potgrootte, lig en temperatuur te manipuleer is die volwasseplantstadium vinniger bereik as met normale plante, en word na hulle verwys as mini-volwasse plante. In terme van blaarbeskadiging, het die mini-volwasse plante beter vlagblaarreaksies getoon. Vir optimale resultate moet die mini-volwasse plante eerder in die glashuis as in groeikabinette gekweek word, en geïnokuleer word tussen aarvorming en blomstadium. Na aanleiding van vlagblaarinfeksietipe en totale plantreaksie, bied mini-volwasse plante ‘n aanvaarbare sisteem om verskillende streeproes-isolate met mekaar te vergelyk. Die sisteem het VPW akkuraat in meeste winterkorings asook in ‘n versameling CIMMYT lentekorings beskryf. Siektelesings van 98 CIMMYT lyne is getoets met hierdie versnelde sisteem en het 88% gekorrelleer met die veldreaksies.

Die mini-volwasse sisteem is ook getoets met populasies wat segregeer vir VPW. As gevolg van die toepassing van genetiese studies in weerstandsteling, is dit noodsaaklik om glashuistoetse met veldreaksies te vergelyk. Die benadering gevolg in hierdie studie het gehou by riglyne in terme van seleksie van kultivars met hoë weerstandsvlakke, wat nie voorheen getoets is nie, asook om glashuisdata met velddata te vergelyk. Segregasie-verhoudings het gedui op die teenwoordigheid van twee weerstandsgene in die F_3 van ‘n Baviaans x Avocet S kruising ($\chi^2_{7:8:1}$ = 0.1029),

maar was onbeduidend vir die Sunmist x Avocet S kruising ten opsigte van Mendeliese segregasie-verhoudings. Die versnelde sisteem was minder suksesvol vir genetiese studies as vir die evaluasie van suiwertelende lyne. As gevolg van die feit dat Avocet S nie volkome vatbaar was in die glashuis nie, wat ook vermoedelik die segregasieverhoudings beïnvloed het, kan die verlaagde sukses eerder toegeskryf word aan die ouergenotipe as aan die sisteem in die algemeen.

Die versnelde sisteem benodig betekenisvol minder tyd en fisiese hulpbronne om akkurate lesings te produseer.

Appendix 1. Seedling infection type, field response, and severity classes used in the evaluation of wheat lines and cultivars to infection by *Puccinia striiformis* f. sp. *tritici*[a]

Seedling infection type[b]	Description
0	no visible uredia
;	necrotic flecks
;n	necrotic areas without sporulation
1	necrotic and chlorotic lesions with restricted sporulation
2	moderate sporulation with necrosis and chlorosis
3	sporulation with chlorosis
4	abundant sporulation without chlorosis
Z	variable sixe with larger uredia towards the leaf base

[b] Variations are indicated by the use of - (less than average for the class) and + (more), as well as c and n to indicate more than usual degrees of chlorosis and necrosis. A comma is used to indicate heterogeneity between plants in a single test.

Field response	
0	no visisble symptoms
R	resistant, visible chlorosis or necrosis, no uredia are present
MR	moderately resistant, small uredia are present and surrounded by either chlorotic or necrotic areas
MS	moderately susceptible, medium sized uredia are present and possibly surrounded by chlorotic areas
S	susceptible, large uredia are present, generaly with little or no chlorosis and no necrosis

Severity (Field response)	Severity is recorded as the percetage (0-100%) leaf area infected and is usually combined with field response
tR	trace severity with a resistant field response
5MR	5% severity with a moderately resistant field response
100S	100% severity with a susceptible field response

[a] McIntosh, R.A., Wellings, C.R., and Park, R.F. 1995. Wheat Rusts: An Atlas of Resistance Genes. CSIRO, East Melbourne, Australia.

Appendix 2. Names, pedigrees, synonyms, year and state of release, and genes for resistance to stripe rust in Australian wheat cultivars (AUSVAR set)

Cultivar/ line	Synonym	Year	State	Pedigree	*Yr* Genes
Arnhem				Pitic/2*Hartog[a]	*Yr6,7+*
Avocet S				Thatcher-*Ag. elongatum* /3*Pinnacle//WW15/3/Egret[b]	
AvS-*Yr18*				Jupateco R/3*Avocet S[c]	
Cook	UQ7401,UQ7401A	1977	Qld.	Timgalen/Condor sib//Condor[a]	
Corrigan C	77Z:832 77Z379-100-9*2	1989	W.A.	Tincurrin*//Gamenya/Iassul[a]	
Cunningham	Qt3286	1990	Qld.	3Ag/4 Condor//Cook[a]	
Datatine C	84W:1147	1994	W.A.	3Ag3/3*Halberd//4*Tincurrin[a]	
Dollarbrid	K2012	1987	N.S.W.	Wren/Gabata//Kalyansona/BB[a]	*Yr7*
Goroke	ED089	1993	Qld.	TM56*2/AUSEN4-21//3Ag3/4*Condor[a]	
Janz				3Ag3/4*Condor//Cook[d]	
Leichhardt				Genaro/4*Hartog[d]	
Meering				Condor selection[c]	
Osprey	M2386	1983	N.S.W.	Condor*2/WW33B[a]	
Ouyen	EL254	1993	Vic.	Takari/TM56//Cocamba[a]	
Oxley	UQ139	1974	Qld.	WW80/2*WW15[a]	

Appendix 2 (cont.). Names, pedigrees, synonyms, year and state of release, and genes for resistance to stripe rust in Australian wheat cultivars (AUSVAR set)

Cultivar/ line	Synonym	Year	State	Pedigree	*Yr* Genes
Sunbrook	SUN224A	1995	N.S.W	Suneca/2*Hartog[a]	*Yr6*
Suneca	SUN44E	1982	N.S.W	II69.66.b.M.b.MGW.86.2.b.b.b.=Ciano/2/Spica/Amber Mutant Sonora 64[a]	
Sunelg	SUN69A	1984	N.S.W	Darf*4/3AG14[a]	
Sunlyn				? (has Suneca in pedigree)[a]	*Yr17*
Sunmist	SUN61A	1992	N.S.W	Miskle 'S'[a]	
Sunstate	SUN148L	1992	N.S.W	VPM1/5 Cook//4 Hartog[a]	*Yr17*
Swift	WW879	1993	N.S.W	Condor/3Ag14//Romany/1489[a]	
Tasman	QT4546	1993	Qld.	Torres///Gaboto/Siete Cerros//Bluebird/Ciano[a]	*Yr6*
Wyuna	SD34	1984	Vic.	DX3-134/Olympic[a]	

[a] Pretorius, Z.A. 1998. Report of a study visit to the Plant Breeding Institute-Cobbitty, Univesity of Sydney, Australia.

[b] Pretorius, Z.A (personal communication).

[c] Wellings C.R. (personal communication).

[d] Sivapalan, S., O'Brien, L., Ortiz-Ferrara, G., Hollamby, G.J., Barclay, I., Martin, P.J. 2001. Yield performance and adaptation of some Australian and CIMMYT/ICARDA developed wheat genotypes in the West Asia North Africa (WANA) region. Australian Journal of Agricultural Research 52: 661-670.

Appendix 3. Names and pedigrees of the South African cultivars

Cultivar/ line	Pedigree
Baviaans	Queen Fan(A50)/4/Jup/Emu"S"//Gjo"S"/3/Kvz/K4500L-6-A-4[a]
Biedou	Kariega*2/4/SST3//Scout*5/Ag/3/Kasteel/Py487(W82-1)[a]
Elands	Betta//Monon/Arthur.Oh130/3/*3Gaudam 1/Fisai[b]
Gariep	Betta//Monon/Arthur.Oh130/3/*3Gaudam 1/Fisai[b]
Hugenoot	Betta//Flamink/Amigo[b]
Kariega	SST44//K4500.2/Sapsucker"S"[b]
PAN3377	Confidential[b]
Olifants	Jup"S"/Bow"S"//Vee#5/Buc"S"/3/Tui"S"[a]
SST57	SST16*3//5*T4/S67-336/3/A2398[b]
SST88	Confidential
SST333	SST124*4/ENT1[b]
SST363	SST124*3/RWA-R[b]
SST399	Confidential
SST806	Confidential
SST825	Kavkaz/Buho//Kalyansona/Bluebird/3/Hermosilo 77/Sapsucker (Tui "S")[b]
SST876	Palmiet/A2398//Adam Tas/3/SST 825[b]
Steenbras	SST44/SST66/4/Hoopv/CI 297001/3/T.aest/Bon//Cno/7C[a]
Tugela	Kavkaz/Jaral[b]

[a] Komen, J.S. (personal communication).
[b] Boshoff, W.H.P. 2000. Control of foliar rusts of wheat in South Africa with special emphasis on *Puccinia striiformis* f. sp. *tritici.* Ph. D. thesis, University of the Free State, South Africa.

Appendix 4. Contingency table for stripe rust response groups as observed for Baviaans x Avocet S F_2 and F_3 populations tested as mini-adults in a glasshouse and as field plots at Greytown

F_2 population

Observed

	Resistant	Susceptible	Total
Glasshouse	130	19	149
Field	63	25	88
Total	193	44	237

Expected resistant glasshouse value: (193 x 149)/237 = 121
Expected susceptible glasshouse value: 149 - 121 = 28
Expected resistant field value: (193 x 88)/237 = 72
Expected susceptible field value: 88 - 72 = 16

Expected

	Resistant	Susceptible	Total
Glasshouse	121	28	149
Field	72	16	88
Total	193	44	237

χ^2 (Resistant, glasshouse) = 0.6694 ($\chi^2 = \Sigma(O - E)^2/E$)
χ^2 (Susceptible, glasshouse) = 2.8929
χ^2 (Resistant, field) = 1.1250
χ^2 (Susceptible, field) = 5.0625
$\Sigma\chi^2$ = 9.7498 $\chi^2_{0.050}$ = 3.8400 (1*df*)

F_3 population

Observed

	Resistant	Intermediate	Susceptible	Total
Glasshouse	21	28	1	50
Field	23	24	3	50
Total	44	52	4	100

Expected

	Resistant	Intermediate	Susceptible	Total
Glasshouse	22	26	2	50
Field	22	26	2	50
Total	44	52	4	100

$\chi 2$ (Resistant, glasshouse) = 0.0455
χ^2 (Intermediate, glasshouse) = 0.1538
χ^2 (Susceptible, glasshouse) = 0.5000
χ^2 (Resistant, field) = 0.0455
χ^2 (Intermediate, field) = 0.1538
χ^2 (Susceptible, field) = 0.5000
$\Sigma\chi^2$ = 1.3986 $\chi^2_{0.050}$ = 5.9900 (2 *df*)

Appendix 5. Contingency table for stripe rust response groups as observed for Sunmist x Avocet S F_2 and F_3 populations tested as mini-adults in a glasshouse and as field plots at Greytown

F_2 population

Observed

	Resistant	Susceptible	Total
Glasshouse	48	34	82
Field	48	47	95
Total	96	81	177

Expected resistant glasshouse value: (96 x 82)/177 = 44
Expected susceptible glasshouse value: 82 - 44 = 38
Expected resistant field value: (96 x 95)/177 = 52
Expected susceptible field value: 95 - 52 = 43

Expected

	Resistant	Susceptible	Total
Glasshouse	44	38	82
Field	52	43	95
Total	96	81	177

χ^2 (Resistant, glasshouse) = 0.3636 ($\chi^2 = \Sigma(O - E)^2/E$)
χ^2 (Susceptible, glasshouse) = 0.4211
χ^2 (Resistant, field) = 0.3077
χ^2 (Susceptible, field) = 0.3721
$\Sigma\chi^2$ = 1.4645 $\chi^2_{0.050} = 3.8400$ (1 *df*)

F_3 population

Observed

	Resistant	Intermediate	Susceptible	Total
Glasshouse	4	45	1	50
Field	8	38	4	50
Total	12	83	5	100

Expected

	Resistant	Intermediate	Susceptible	Total
Glasshouse	6	41,5	2,5	50
Field	6	41,5	2,5	50
Total	12	83	5	100

χ^2 (Resistant, glasshouse) = 0.6667
χ^2 (Intermediate, glasshouse) = 0.2952
χ^2 (Susceptible, glasshouse) = 0.9000
χ^2 (Resistant, field) = 0.6667
χ^2 (Intermediate, field) = 0.2952
χ^2 (Susceptible, field) = 0.9000
$\Sigma\chi^2$ = 3.7238 $\chi^2_{0.050} = 5.9900$ (2 *df*)