CONTROL OF FOLIAR RUSTS OF WHEAT IN SOUTH AFRICA WITH SPECIAL EMPHASIS ON *PUCCINIA STRIIFORMIS* F. SP. *TRITICI*

A thesis submitted in fulfilment of requirements for the degree of Doctor of Philosophy in the Faculty Natural and Agricultural Sciences, Department of Plant Pathology, University of the Free State

by

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GENERAL INTRODUCTION

During August 1996, stripe (yellow) rust, caused by *Puccinia striiformis* Westend. f. sp. *tritici* Eriks., was observed for the first time on bread wheat (*Triticum aestivum* L.) in the Western Cape. Professor Z.A. Pretorius and Dr. A.B. van Jaarsveld first identified the disease on the farm Grootvlei, near Moorreesburg, in the Swartland area. Ensuing surveys during the growing season indicated that stripe rust occurred throughout most of the wheat producing areas in the winter rainfall regions of the Northern, Western and Eastern Cape Provinces. The most severely infected wheat fields were near Darling, Hopefield, Moorreesburg and Malmesbury. Amongst factors contributing to the development of the epidemic were favourable weather conditions, the high level of susceptibility of most cultivars, prolonged weather conditions unsuitable for the application of fungicides, some producers postponing fungicide application and a shortage of fungicides and aerial crop sprayers at critical times. Producers spent an estimated R28 million on fungicides in the Western Cape during 1996. Spike infection and destruction of foliage contributed significantly towards losses in grain quantity and quality in seriously affected fields. In preliminary studies with susceptible cultivars, losses of 5 to 45% were measured.

The disease was again observed early in the 1997 season in the western Free State from where it spread to the rest of the province, KwaZulu-Natal, Gauteng, the North-West and Northern province. The rapid dispersal of the pathogen during the 1996 and 1997 wheat seasons, susceptibility of several high-yielding cultivars, and favourable climatic conditions in most wheat growing areas in South Africa, makes stripe rust a potentially damaging disease in local wheat production. A well-planned and co-ordinated research programme for stripe rust is, therefore, important.

The objectives of this study were firstly to summarise literature available on stripe rust, including the epidemiology, economic importance and disease control. Secondly, to monitor the occurrence and spread of the introduced stripe rust pathotype as well as the possible development of new variants. Thirdly, to determine the levels of resistance in South African wheat cultivars towards the prevailing stripe rust pathotype(s) and finally, to quantify the influence of foliar rusts on yield and quality of

wheat in South Africa as well as the economical justification of chemical control during different growth stages.

A REVIEW OF FOLIAR RUST DISEASES OF WHEAT WITH EMPHASIS ON *PUCCINIA STRIIFORMIS* F. SP. *TRITICI*

INTRODUCTION

Stripe rust, caused by *Puccinia striiformis* Westend. f. sp. *tritici* Eriks., is an important disease of wheat (*Triticum aestivum* L.) world-wide (Roelfs *et al.*, 1992). The disease was first described by Gadd in 1777 (Stubbs, 1985). Stripe rust is confined to areas with cool and wet environmental conditions and occurs prominently in North-Western Europe, the Mediterranean region, the Middle East, the North-West of the USA, Australia, the East African highlands, China, the Indian subcontinent, Central Asia, New Zealand, and the Andean region of South America (Danial, 1994). Stripe rust epidemics have been reported in Europe, the United Kingdom, Ethiopia, Turkey, and in the USA (Allan *et al.*, 1963; Bayles *et al.*, 1989; Chilosi & Corazza, 1990; Johnson, 1992b; Louwers *et al.*, 1992; Line, 1993). During severe epidemics, yield losses as high as 84% were recorded in Australia (Murray *et al.*, 1994). Depending on the area of wheat cultivation, factors that may influence the severity of stripe rust epidemics include rainfall in the preceding summer and autumn months, snowfall and low temperatures in the winter months, sowing date, onset of disease development, susceptibility of cultivars, yield potential, and spring temperatures (Coakley & Line, 1981; Ash & Brown, 1990; Ellison & Murray, 1992).

For the control of stripe rust producers largely rely on the cultivation of resistant cultivars and fungicide applications. Chemical applications, including foliar sprays and seed treatments, have been adopted effectively in controlling stripe rust epidemics, but genetic resistance is considered the most cost-effective and environmentally safe approach (Rakotondradona & Line, 1984; Jørgensen & Nielson, 1994; Ma *et al.*, 1995; Ma & Singh, 1996b; Cook *et al.*, 1999). Mixtures of cultivars, with different levels of resistance to stripe rust, present an economical alternative to reduce disease severity and increase yields relative to pure stands (Finckh & Mundt, 1992; Aslam & Fischbeck, 1993; Pradhanang & Sthapit, 1995; Mundt *et al.*, 1996; Akanda & Mundt, 1997).

Resistance to stripe rust has been divided into pathotype-non-specific (quantitative) and pathotype-specific (qualitative) resistance (Johnson, 1988). Several cultural and environmental factors may influence the expression of resistance to stripe rust including plant nutrition, temperature and light intensity (Sharp, 1965; Sharp *et al.*, 1976; Wellings *et al.*, 1988; Daamen *et al.*, 1989; Ash & Brown, 1991). According to Roelfs *et al.* (1992) stripe rust requires more specialised environmental control in the glasshouse due to its sensitivity towards environmental influences and the production of less discrete infection types on host plants. The latter may be attributed to the presence of numerous resistance genes, additive effects of some resistance genes, the presence of temperature-sensitive genes, and many genes functioning only in the adult-plant stages.

With the appearance of stripe rust in South Africa (Pretorius *et al.*, 1997), *P. striiformis* has now spread to all the major wheat production areas in the world. The global dissemination of *Puccinia striiformis* has, however, been slower than for other rust pathogens. For example, *Puccinia striiformis* f. sp. *tritici* did not occur in Australia until 1979 (O'Brien *et al.*, 1980), and was not found in New Zealand until 1980 (Beresford, 1982). Physical isolation and effective quarantine barriers are factors that may have contributed to this.

Leaf or brown rust of wheat caused by *P. triticina* (Eriks.) (=*P. recondita* f. sp. *tritici*) is an important disease in most wheat producing regions of the world (Roelfs *et al.*, 1992; McIntosh *et al.*, 1995; Ortelli *et al.*, 1996; Sayre *et al.*, 1998; Brahma *et al.*, 1999; Moschini & Pérez, 1999; Manisterski *et al.*, 2000). Disease spread can be rapid under favourable conditions with severe epidemics and losses occurring when the flag leaf is infected before heading (Chester, 1946). Similar to stripe rust, producers mainly rely on genetic resistance and fungicide applications to control this disease (Broers, 1989; Cook *et al.*, 1999; Sundin *et al.*, 1999).

The purpose of this review is to summarise the present literature on stripe rust placing special emphasis on the causal organism, environmental conditions conducive to epidemic development, economic importance, and disease control. The latter include chemical measures such as foliar application of fungicides, seed treatment and resistance breeding. Furthermore, available literature on losses caused by *P. triticina*, and chemical control of this disease, is reviewed.

WHEAT STRIPE RUST

PATHOGEN

Puccinia striiformis f. sp. *tritici* is an obligate parasite completely dependent on living host tissue (Wiese, 1987). This particular *forma speciales* can infect numerous wheat cultivars but also a few barleys, triticale, rye and certain grasses (Wiese, 1987; Roelfs *et al.*, 1992). The pathogen has a hemiform life cycle comprising of uredial and telial stages. The sexual stage of the fungus has not been encountered and so far no alternate hosts have been found (Stubbs, 1985). Numerous, host specific pathotypes of *P. striiformis* occur (Johnson, 1992a), and are probably formed by mutation and somatic recombination (Stubbs, 1985). Shan *et al.* (1999) used DNA fingerprinting to examine genetic variation among 160 stripe rust isolates. They found 97 phenotypes with phenotypic diversity varying among different regions in China. Previously Chen *et al.* (1993) found a low association between virulence and RAPD patterns of *P. striiformis* f. sp. *tritici* isolates which suggested that DNA polymorphisms are independent of virulence.

Host specialisation

Based on the host genus, eight *formae speciales* of *P. striiformis* have been reported: *Puccinia striiformis* f. sp. *tritici* on wheat (*T. aestivum*), *P. striiformis* f. sp. *hordei* on barley (*Hordeum vulgare* L.), *P. striiformis* f. sp. *secalis* on rye (*Secale cereale* L.), *P. striiformis* f. sp. *elymi* on *Elymus* spp., *P. striiformis* f. sp. *agropyri* on *Agropyron* spp., *P. striiformis* f. sp. *dactylidis* on orchard grass (*Dactylis glomerata*), *P. striiformis* f. sp. *poa* on Kentucky bluegrass (*Poa pratensis*), and *P. striiformis* f. sp. *leymi* on *Leymus secalinus* (Chen *et al.*, 1995b).

Pathogenicity studies and random amplified polymorphic DNA (RAPD) analyses by Chen *et al.* (1995b) supported the separation of *P. striiformis* f. sp. *hordei*, *P. striiformis* f. sp. *tritici* and *P. striiformis* f. sp. *poa* in North America. Although *P. striiformis* f. sp. *hordei* and *P. striiformis* f. sp. *tritici* primarily attack barley and wheat, respectively, *P. striiformis* f. sp. *hordei* is virulent on some wheat varieties, and *P. striiformis* f. sp. *tritici* is virulent on some cultivars of barley. Isolates

of *P. striiformis* f. sp. *hordei* and *P. striiformis* f. sp. *tritici* did not infect bluegrass, and isolates of *P. striiformis* f. sp. *poa* did not infect wheat and barley cultivars. Based on field and glasshouse data, supported by isozyme and double-stranded RNA analyses, *P. striiformis* f. sp. *hordei* and *P. striiformis* f. sp. *tritici* were also proved to be distinctly different (Newton *et al.*, 1985; Stubbs, 1985; Line & Chen, 1996). Johnson & Lovell (1994) concluded that some genes for resistance could interact with more than one *forma speciales* of a rust pathogen. Part of the separation between *formae speciales* in their host preference may thus be due to pathotype-specific genes, of which some may be difficult but not impossible for a *forma speciales* to evade (Johnson & Lovell, 1994).

Pathotype differentiation

The stripe rust pathogen is known for its ability to over-come resistance genes, resulting in the appearance of numerous pathotypes within a relatively short period of time. Factors that may contribute to the rapid evolution of aggressive pathotypes are increased fecundity, more pathogen generations per season, or a more suitable microclimate for disease development (Coakley *et al.*, 1999).

According to Johnson *et al.* (1972) physiologic specialisation of *P. striiformis* was first demonstrated by Allison & Eisenbeck and Gassner & Straib in 1930. Pathotypes of *P. striiformis* are identified based on their virulence to differential cultivars of wheat (McIntosh *et al.*, 1995). Different differential sets are being used in different regions of the world to identify stripe rust pathotypes. This is due to geographical differences in virulence frequencies, relevance of differential genotypes to deployed resistance, genetic background of differential testers, progressive increases in virulence (resulting in recomposed differential sets), and the occurrence of adult plant resistance (De Vallavieille-Pope & Line, 1990; McIntosh *et al.*, 1995). The North American set of differentials was proposed by Line *et al.* (1970) and revised by Line (1972) and Line *et al.* (1988). The Chinese and North American sets for identifying pathogenic variation in *P. striiformis* f. sp. *tritici* are unique to those regions (McIntosh *et al.*, 1995). The International and European differential sets were proposed by Johnson *et al.* (1972) and revised by Stubbs (1985). The latter system has gained acceptance throughout Europe and with certain additional

differentials, in Australia (Wellings *et al.*, 1988). India is using a system similar to the International and European series (Kumar *et al.*, 1993; McIntosh *et al.*, 1995). Differential sets are open-ended and cultivars or differential lines can be added as new pathotypes appear. Table 1 serves as an example of the ability of the stripe rust pathogen to become established and to form new pathotypes necessary for survival after introduction into Australia.

Prevalence of *Puccinia striiformis* in Africa

Puccinia striiformis is common in most wheat producing countries in Africa, with the most complex pathotypes occurring in Kenya (Njoro) and Ethiopia (Bekoji and Beki) (Louwers *et al.*, 1992). The pathotype introduced in South Africa, 6E16, is common in North and East African countries, including Tunisia, Algeria, Libia, Ethiopia, Kenya, Tanzania, and Burundi. Pathotype 6E16 also occurs in the Middle East including Turkey and Syria and can be distinguished in two sub pathotypes, one with virulence for *YrA* (occurring in East Africa) and one without this virulence factor (occurring in North Africa) (Badebo *et al.*, 1990; Louwers *et al.*, 1992). Virulence has been detected for most known seedling *Yr* genes in Africa except for *Yr4+* (cultivar Hybrid 46), *Yr5* (*Triticum aestivum* sp. *spelta* var. *album*), *YrSp* in Spaldings Prolific and *YrCv* in Carstens V. No information is available on the occurrence of virulence or avirulence to *Yr* genes *11* to *18* in Northern and Eastern African countries. Virulence for *Yr1* has been detected in Kenya in 1987 (Louwers *et al.*, 1992) and in Ethiopia (Badebo *et al.*, 1990), but not since then. Virulence for *Yr3* (cv. Vilmorin 23) was detected in Ethiopia (Badebo *et al.*, 1990) and in Tunisia (Louwers *et al.*, 1992) and for *Yr3+* (cv. Nord Desprez) in Kenya (Louwers *et al.*, 1992) and Ethiopia (Badebo *et al.*, 1990). In 1986 pathotype 166E150, which added virulence for *Yr9,* was detected in Ethiopia (Badebo *et al.*, 1990). Pathotypes virulent on *Yr10* (cv. Moro) occur in Ethiopia (Badebo *et al.*, 1990) and Tanzania (Louwers *et al.*, 1992).

Table 1. Pathogenic changes of *Puccinia striiformis* f. sp. *tritici* in Australasia during 1979-1988 after being introduced in Australia in 1979[a]

Pathotype	First detected	Pathogenicity	
		Avirulence[b]	Virulence[c]
Australia			
104 E137 A-	1979	*Yr1,5,6,7,8,Sp,A*	*Yr2*
104 E137 A+	1981	*Yr1,5,6,7,8,Sp*	*Yr2,A*
108 E141 A-	1983	*Yr1,5,7,8,Sp,A*	*Yr2,6*
108 E141 A+	1983	*Yr1,5,7,8,Sp*	*Yr2,6,A*
360 E137 A-	1984	*Yr1,6,7,8,Sp,A*	*Yr2,5*
360 E137 A+	1984	*Yr1,6,7,8,Sp*	*Yr2,5,A*
104 E153 A-	1985	*Yr1,5,6,7,Sp,A*	*Yr2,8*
104 E153 A+	1985	*Yr1,5,6,7,Sp*	*Yr2,8,A*
108 E205 A+	1985	*Yr1,5,7,8*	*Yr2,6,Sp,A*
110 E143 A+	1986	*Yr1,5,8,Sp*	*Yr2,6,7,A*
104 E9 A-	1988	*Yr1,2,5,6,7,8,Sp,A*	
104 E9 A+	1988	*Yr1,2,5,6,7,8,Sp*	*YrA*
New Zealand			
104 E137 A-	1980	*Yr1,5,6,7,8,Sp,A*	*Yr2*
106 E139 A-	1982	*Yr1,5,6,8,Sp,A*	*Yr2,7*
108 E141 A+	1986	*Yr1,5,7,8,Sp*	*Yr2,6,A*
109 E141 A-	1986	*Yr5,7,8,Sp,A*	*Yr1,2,6*
108 E141 A-	1987	*Yr1,5,7,8,Sp,A*	*Yr2,6*
110 E143 A-	1987	*Yr1,5,8,Sp,A*	*Yr2,6,7*
111 E143 A-	1988	*Yr5,8,Sp,A*	*Yr1,2,6,7*

[a]Wellings & McIntosh (1990).

[b]All cultures avirulent with respect to *Yr9*, *Yr10* and Carstens V.

[c]All cultures virulent with respect to *Yr3*, *Yr4*, Suwon 92/Omar, Strubes Dickkopf and Nord Desprez.

SYMPTOMS

Symptoms of stripe rust usually appear earlier than those of leaf rust or stem rust (*Puccinia graminis* Pers. f. sp. *tritici* Eriks. & Henn.) (Wiese, 1987; Akanda & Mundt, 1997). Small yellowish uredia appear principally on leaves and heads, and are often arranged into conspicuous stripes from a single infection (Wiese, 1987). The linear orientation of pustules between vascular bundles and the development of runner hyphae can result in stripes as long as the leaf (Fig. 1). Urediospores are 20-30 µm in diameter, yellow to orange, and spherical (Wiese, 1987). Individual pustules measure 0.3-0.5 x 0.5-1 µm (Wiese, 1987). In wheat heads, uredia normally occur on the ventral surface of the glumes (Wiese, 1987). Telial pustules, prevalent on leaf sheaths, are persistently subepidermal. Telia are dark-brown and often form long, dark streaks (Wiese, 1987). Teliospores resemble those of *P. triticina* but are able to germinate without cold treatment (Wiese, 1987). Stunting of early-infected plants is common.

EPIDEMIOLOGY

Life cycle

The sexual stage of *P. striiformis* on alternate hosts is unknown (Wiese, 1987). The survival of *P. striiformis* during the non-cropping season, in both the uredial and mycelial stages on cereal and wild grass hosts, is well documented (Sharp & Hehn, 1963; Roelfs & Huerta-Espino, 1994; Marshall & Sutton, 1995; Nazari *et al.*, 1996; Mardoukhi & Torabi, 1998). The ability of the stripe rust pathogen to over-summer on susceptible volunteer wheat plants or accessory hosts adjacent to wheat fields or wheat production areas may influence the onset of epidemics. Shaner & Powelson (1973) observed *P. striiformis* less frequently on grasses at high elevations than on grasses in wheat fields. According to these authors the pathogen over-summers on residual green wheat and grasses within the wheat field rather than on wild grass spp. growing further away in mountainous areas. Wild grass spp. previously reported as susceptible to cereal stripe rust are *Aegilops crassa*, *Agropyron*

Fig 1. Stripe rust symptoms on (A) seedling and (B) flag leaves of wheat.


a


b

cristatum, *A. elongatum*, *A. riparium*, *A. sibiricum*, *A. spicatum*, *A. trachycaulum*, *Bromus marginatus*, *B. mollis*, *B. scoparius*, *B. tectorum*, *B. uniloides*, *Elymus canadensis*, *E. cinereus*, *E. glaucus*, *E. scabrus*, *Hordeum brachyantherum*, *H. glaucum*, *H. hystrix, H. jubatum*, *H. leporinum*, *H. murinum*, *H. marinum*, *H. pusillum*, *H. spontaneum*, *H. vulgare*, *Leymus secalinus*, *Phalaris minor*, *P. paradoxa*, *P. triviales*, and *Secale* spp. (Sharp & Hehn, 1963; Shaner & Powelson, 1973; Line, 1976; Holmes & Dennis, 1985; Stubbs, 1985; Dennis & Brown, 1986; Park, 1990; Roelfs & Huerta-Espino, 1994; Chen *et al.*, 1995b; Marshall & Sutton, 1995; Nazari *et al.*, 1996). Infections similar to stripe rust from *B. rubens*; *D. glomerata*, *Lolium multiflorum*, *P. ampla*, *P. pratensis* and *Polypogon monspeliensis* were previously found to be avirulent on wheat (Shaner & Powelson, 1973; Line, 1976; Holmes & Dennis, 1985). Some of these infections may have been confused with symptoms of oat (*Avena* spp. L.) crown rust caused by *Puccinia coronata* Corda f. sp. *avenae* Eriks. According to Line (1976) the failure of his samples to infect wheat seedlings may have been due to poor viability of the inoculum. *Bromus breviaristatus* was only susceptible under glasshouse conditions (Shaner & Powelson, 1973).

Puccinia striiformis seems to be more sensitive to ultraviolet light and air pollution than the other rusts. This may affect the survival of the pathogen over long distance transport in highly polluted areas (Roelfs *et al.*, 1992). Shaner and Powelson (1973) reported that studies conducted by Hungerford in 1930, Metha in 1923, Raeder & Bever in 1931 and Shaner in 1969 proved that urediospores of *P. striiformis* can survive 4 to 6 weeks on dead leaves and up to 1 month in air-dry soil. According to Sharp & Hehn (1963), the stripe rust fungus survives as dormant mycelium in susceptible wheat up to 159 days during winter. According to Dennis (1987) sporulating infections survived longer than latent infections under repeated exposure to high temperature and could withstand daily exposure of 4 h at 35°C, and 8 h at 30°C. Latent periods increased with increasing temperature and time of exposure at 25°C and above, but did not exceed 25 days. Lesion growth and urediospore production decreased with increasing temperature and time of exposure. Urediospores survived 11 d at 25°C, 9 d at 30°C, 7 d at 35°C and 5 d at 40°C. There was no reduction in the germination of urediospores produced subsequent to periods of exposure to high temperatures.

Inoculum sources

Stripe rust inoculum originates from mycelium that over-winters or over-summers in wheat leaf tissues and especially from urediospores that survive locally or are wind borne from distant hosts (Sharp & Hehn, 1963; Shaner & Powelson, 1973). The amount of over-summering rust depends on the amount of susceptible volunteer wheat which, in turn, is a function of the moisture in the off season (Zadoks, 1961; Bayles *et al.*, 1989; Chilosi & Corazza, 1990; Park, 1990). Wheat at lower elevations may sometimes become infected by urediospores from accessory hosts growing at higher altitude (Nazari *et al.*, 1996). However, Shaner & Powelson (1973) have rejected this. The ability of *P. striiformis* to withstand adverse environmental conditions has been largely underestimated. This is demonstrated by the ability of stripe rust to survive hot dry summers without detection of any host infection (Luig, 1985; McIntosh & Wellings, 1986).

Environmental conditions

Stripe rust is principally a disease of wheat grown in cooler climates, i.e. temperatures generally associated with higher elevations, higher latitudes or cooler years. Minimum, optimum, and maximum temperatures for stripe rust infection are 0, 11 and 23°C, respectively (Roelfs *et al.*, 1992). Free water is needed on the leaf for germination and infection. Park (1990) defined a day as having very favourable temperatures for stripe rust infection when the mean temperature fell within the range of 12.4 to 18.4°C and the minimum temperature fell within the range of 7.3 to 14.6°C. When 15 h of free moisture occur on leaves within each of the latter ranges the infection of inoculated plants may reach 100%. In addition to the well-known temperature requirements of the stripe rust pathogen, light has also been shown to be important. De Vallavieille-Pope *et al.* (2000) reported that a high light intensity prior to inoculation dramatically increased the infection efficiency of *P. striiformis* f. sp. *tritici* in both field and controlled environment experiments.

According to Broers & López-Atilano (1996) the infection process of *P. striiformis* can be divided into: germination, formation of an appressorium, penetration of the stoma, formation of a substomatal vesicle, formation of the first infection hypha, formation of a haustorium, ramification of hyphae, and sporulation.

Other sources have indicated, however, that this fungus does not produce appressoria before penetration or that only weak appressoria are formed (Mares & Cousen, 1977; De Vallavieille-Pope *et al.*, 1995). The latent period may vary from 11 days with mean daily temperatures of 15°C, to perhaps 180 days with near freezing temperatures (Ellison & Murray, 1992). Development of stripe rust on susceptible cultivars follows a sigmoidal pattern over time and stops when the leaves begin to senesce at the late milk to early dough stages of kernel development (Ellison & Murray, 1992).

In areas where the environment is marginally suited for stripe rust, the disease is only severe in years when conditions are unusually favourable and susceptible cultivars are grown (Roelfs *et al.*, 1992). During hot or dry periods sporulation ceases but will often restart with a return to cool moist conditions (Coakley & Line, 1981). Ash *et al.* (1991) found a significant correlation between meteorological parameters and stripe rust severity. They found projected disease severity (DS) negatively correlated with the frequency of days in the preceding calendar year with a maximum temperature in the range of 25 to 30°C. Lower maximum temperatures of 10 to 20°C in the same period were positively correlated with projected disease severity and can compensate for periods of high temperature (Ash *et al.*, 1991). However, the survival and projected disease severity of stripe rust decreased dramatically above 40°C (Dennis, 1987; Ash *et al.*, 1991). According to Park (1990) above average rainfall during the late summer and autumn months benefit the survival of stripe rust on volunteer wheat plants in Australia and may result in early season epidemics.

In the Pacific Northwest (USA) low spring temperatures are strongly correlated with a high disease index in the cultivars Gaines and Omar (Coakley & Line, 1981). Disease development in these two cultivars had the greatest negative correlation with increasing April temperatures. According to Coakley & Line (1981) low spring temperatures would be expected to decrease the effectiveness of resistance in temperature sensitive cultivars and retard growth of the wheat plant, thus providing a longer period for an increase of the rust. Higher summer temperatures limit disease development in June. Stripe rust develops more slowly after the dough stage; therefore, July and August temperatures have little effect on

stripe rust epidemics. The climate during autumn was not well correlated with the occurrence of stripe rust epidemics. Of the winter months January was the most highly correlated with disease index. Higher winter temperatures favoured the survival of over-wintering inoculum. Mycelium is surviving in the host plant unless infected leaves of the host dies off due to low temperatures (Coakley & Line, 1981). Although precipitation was not correlated with stripe rust intensity (Coakley & Line, 1981; Ash *et al.*, 1991), the role that rain plays in either rain-splash or dry-dispersal of spores cannot be excluded (Rapilly, 1979; Geagea *at al.*, 1999). Splash spore dispersal is one important mechanism of spore dispersal beside aerial spore dispersal and contact between plants (Geagea *et al.*, 1997).

ECONOMIC IMPORTANCE

The four basic components of grain yield in wheat are the number of heads or tillers per square meter, the number of spikelets per head, the number of kernels per spikelet, and the mass of individual kernels (Teng & Gaunt, 1980). The number of tillers and spikelets per tiller are determined before booting, the kernels per spikelet from stem elongation to early milk, and the kernel mass after anthesis. Therefore, stress factors influencing the plant at different growth stages affect the respective yield components differentially (Teng & Gaunt, 1980).

Controlled experiments conducted by Bever (1937) showed that the effects of stripe rust depend on the time of onset of the disease relative to the growth stage of the plant. Early infections (starting at seedling, three-leaf and jointing stages) reduced the number of heads, plant height, straw mass and number of kernels, and delayed development of the plants, while these components were not affected when infections began at booting or later stages. Kernel mass and grain yield were reduced irrespective of the time of inoculation. According to Murray *et al.* (1995) stripe rust in southern New South Wales was most severe in susceptible cultivars when the epidemic began before the booting stage and affected more leaf area by the early milk growth stage. Grain yield was reduced by up to 84%, kernel mass by 43%, and kernel number by 72%. Stripe rust did not affect plant height, number of

tillers and stem dry matter at booting and anthesis, or the dates at which these growth stages as well as heading were reached. In some cases stem dry matter was reduced at maturity (Murray *et al.*, 1995). In a more recent study conducted in Uganda rust resistant entries out-yielded susceptible material in control plots untreated with fungicide by 138% (Wagoire *et al.*, 1998).

Ash & Brown (1990) conducted three field trails to quantify the effect of stripe rust on yield of wheat in northern New South Wales. Yield parameters measured were total grain yield per plot, 1000-grain weight, number of grains produced per head, tiller number and grain yield per plant. Factors influencing total yield loss were disease response of the cultivar, onset of the epidemic and the yield potential of the crop. Their results showed that early stripe rust epidemics had a larger effect on yield than late epidemics. Total grain yield and 1000-grain weight were most affected. All the yield parameters were affected by long season epidemics, with losses up to 50% in grain yield being recorded in susceptible cultivars.

Yield losses decreased in cultivars with higher levels of resistance in the adult-plant stage (Park *et al.*, 1988; Murray *et al.*, 1994; Ma & Singh, 1996a). According to Park *et al.* (1988) adult-plant resistance (APR) in the cultivars Cook, Bass, Banks, Kite, and Suneca is generally effective in preventing detectable yield losses due to stripe rust infection. Cultivars with lower levels of APR experienced losses of 15 to 25%, compared with 45 to 50% losses in the susceptible cultivar Teal. Early stripe rust epidemics, starting before the onset of APR, however, can result in significant yield losses in cultivars with seedling susceptibility and moderate to resistant adult-plant reaction (Murray *et al.*, 1994). Ma & Singh (1996a) found that slow rusting resistance conferred by *Yr18* protects grain yield in the range of 36 to 58%, depending on the year and sowing date. Grain yield losses were mainly associated with reductions in kernel weight and kernels per square metre, spikes per square metre and kernels per spike. Reductions in the latter two yield components only occurred in the susceptible line Jupateco S and not in Jupateco R, carrying *Yr18*.

Stripe rust head infections may occur under epidemic conditions and can be destructive, especially when the plant is already stressed by foliar losses. In a study conducted by Cromey (1989b) wheat kernels from infected florets were shriveled

and weighed up to 77% less than kernels from uninfected florets. According to the author this may have resulted from a reduction in the photosynthetic area of florets as well as altered photosynthate translocation patterns due to severe stripe rust infection.

DISEASE CONTROL

Plant disease control strategies should be directed at reducing the probability of epidemics, as well as reducing the magnitude of losses. Integrated cereal rust management, including cultural control practices, disease resistance and fungicide applications, should contribute to the successful control of stripe rust. Due to the airborne nature of stripe rust spores, quarantine measures usually delay, but do not prevent entry of the disease and/or pathotypes with specific virulence combinations into new areas (Wellings *et al.*, 1987; Roelfs *et al.*, 1992).

Cultural practices

Cultural practices provide an alternative measure for reducing risk of wheat rust epidemics. No single practice is effective under all conditions, but using a series of cultural practices greatly enhances the existing resistance (Roelfs *et al.*, 1992). The importance of the green bridge in carrying a disease from one crop to the next has been emphasised by Zadoks & Bouwman (1985). The removal of volunteer plants with strategic animal grazing, tillage or herbicides is an effective control measure for epidemics resulting from endogenous inoculum.

Mundt *et al.* (1996) compared pure stands of a stripe rust susceptible wheat cultivar, pure stands of a resistant cultivar, a 1:1 random mixture of resistant and susceptible cultivars and populations in which strips of the cultivars were alternated. Random mixtures of the two cultivars provided better disease control and increased yield relative to the pure stand mean, while alternating strips failed in this respect. Finckh & Mundt (1992) found that cultivars planted in mixtures yielded between 0 and 5% more than the mean of the pure stands in the absence of disease. In the presence of disease, mixing increased yield between 7 and 13% (Finckh & Mundt,

1992; Akanda & Mundt, 1997). Producers should, however, keep in mind that the performance of a cultivar in a pure stand is not always indicative of its performance in a mixture (Finckh & Mundt, 1992; Akanda & Mundt, 1997). Agronomic traits like short stature may negatively influence the performance of a cultivar in a mixture (Finckh & Mundt, 1992).

Fungicides

Chemical control of cereal diseases is usually not desirable due to high costs of fungicide application as well as potential environmental hazards. However, fungicides are used world-wide to maintain the production levels in wheat cultivars lacking adequate levels of disease resistance (Ireta & Gilchrist, 1994). Additional to effective disease control in susceptible wheat cultivars, fungicides reduce the infection pressure and the possible selection of new more virulent pathotypes which could attack surrounding resistant crops (Rathmell & Skidmore, 1982). Fungicides registered to control stripe rust in South Africa are listed in Table 2.

Foliar application

Incidence of stripe rust on highly susceptible cultivars may still occur despite the application of effective fungicides because weather or human factors frequently do not allow optimal control (Johnson, 1992a). Ash & Brown (1990) found that the most effective chemical control of stripe rust is obtained when application closely coincides with the onset of the epidemic. The time when fungicide application was most effective depended on the appearance of stripe rust, the length of the epidemic and the growth stage at which APR became effective. Late and full season application of fungicides led to a higher 1000-grain weight in all experiments. Grain number per spike tended to be less in the unsprayed than the sprayed treatments. When stripe rust appears late in the season or if the yield potential is low, the losses in moderately susceptible cultivars would be expected to be quite small (Ash & Brown, 1990).

Trials conducted in New Zealand showed that stripe rust infections reduced the grain number after growth stage 32 (2nd node detectable) to 59 (emergence of inflorescence completed), indicating an effect on floret development. Treatments

Table 2. Active components, gram active ingredient, dosage and active ingredient per hectare of fungicides registered to control *Puccinia striiformis* f. sp. *tritici* in South Africa[a]

Active components	Gram active ingredient (g/ℓ)	Dosage for ground applications (ml/ha)	Gram active ingredient per hectare
Foliar applications			
Carbendazim/cyproconazole	300/160	250-375	75/40 or 112.5/60
Carbendazim/epoxiconazole	125/125	900-1000	112.5/112.5 or 125/125
Carbendazim/flusilazole	125/250	400	50/100
Carbendazim/flutriafol	150/94	1200	180/113
Carbendazim/tebuconazole	133/167	750	100/125
Cyproconazole	100	400	40
Bromuconazole	200	700	140
Flusilazole	250	400	100
Flutriafol	125	1000	125
Propiconazole	250	400	100
Tebuconazole	250	750	188
Seed treatment			
Triticonazole	200	120	24

[a]Nel *et al.* (1999)

providing protection only to early infections still produced high grain numbers, but the grain weight was reduced markedly (Gaunt & Cole, 1991). Yield responses of these experiments were indicative of the need for the combining of seed treatment for disease control at early growth stages, supported by foliar application for protection during anthesis. Cromey (1989b) found that the application of the triazole fungicide triadimefon after full head emergence reduced the level of stripe rust head infection from 75 to 20%. The reduction in the number of infected spikelets in sprayed plots resulted in a yield increase of 34%.

Jørgensen & Nielsen (1994) investigated the efficacy of broad-spectrum fungicides at normal and reduced dosages on stripe rust in Denmark and confirmed the effectiveness of ergosterol inhibitors in the control of stripe rust on winter wheat. A longer residual effect was found with higher dosages, with the normal dosage giving 4-5 weeks full effect and lower dosages remaining effective for 3 weeks. Lower dosages were the most effective when applied preventatively. However, when applied on susceptible cultivars, where outbreaks have occurred, lower dosages required additional treatments within a maximum of 3 weeks. This is particularly important during stem elongation ensuring protection of newly developed leaves. It was also found that timing is very important when using both full and reduced dosages. In susceptible cultivars, control is recommended as soon as stripe rust is observed in the field. Application at the end of the latent period, and mistiming the interval between treatments, either by delaying application to the end of the latent period or the beginning of symptom appearance, can result in poor control.

Cyproconazole was found to be one of the most effective fungicides for reduction of stripe rust (Cook *et al.*, 1999). Fenpropimorph was moderately active against stripe rust, highly active against mildew and ineffective against *Septoria* leaf spot. Propiconazole was the most effective against stripe rust and *Septoria* leaf spot. However, Cook *et al.* (1999) found different fungicides to be equally effective against stripe rust when applied just before symptoms were detected. Spray timing thus appeared to be more important than choice of fungicide.

Seed treatment

Important factors to consider before applying seed treatment against stripe rust are inoculum levels on volunteer wheat and accessory hosts in the off-season, susceptibility of cultivar(s) of choice, risk of the area to early stripe rust infections as well as late planting in high risk areas. Seed treatments can be used in combination with foliar sprays when cultivars are very susceptible, alone when yields are too low to justify foliar sprays, and in combination with moderate high-temperature adult-plant resistance (HTAPR) or slow rusting resistance to stripe rust (Everts & Leath, 1993; Line, 1993). Seed treatment with triadimenol is part of the rust control program in the USA (Line, 1993). Trials conducted by Gaunt & Cole (1991) showed that seed treatment of a susceptible cultivar with triadimenol/fuberidazole (Baytan®) increased yield by 23% compared to a carboxin plus thiram treatment, a commercial standard, with no known efficacy for stripe rust control.

In glasshouse and field studies triadimefon (Bayleton®) applied as a slurry to wheat seed at 0.5 g a.i./kg or higher, controlled both stripe and leaf rust from seedling emergence through the boot stage of plant growth (Rakotondradona & Line, 1984). However, at this rate triadimefon significantly reduced the plant stand of some cultivars. According to the authors stand reduction was linearly related to the rates of triadimefon; the higher the rates, the lower the stand. Triadimefon at 0.25 g a.i./kg controlled the rusts through the tillering stage (main shoot and nine or more tillers) (Rakotondradona & Line, 1984). Effective control of stripe rust may thus be obtained by the use of triadimefon as a seed treatment at 0.25 g a.i./kg in combination with adult-plant resistance, or in combination with foliar sprays.

Breeding for resistance

Genetic resistance is the most effective, environment-friendly and economic way to control stripe rust of wheat (Ma & Singh, 1996b). Genetic resistance occurs when a resistance allele is present in the host along with a corresponding avirulence gene in the pathogen (Johnson & Knott, 1992). The value of resistance in crops depends on its level, its stability towards geographical and environmental conditions, and its durability (Broers, 1989). Long-term resistance in wheat to rust diseases depends on the availability and management of durable resistance sources (Bariana &

McIntosh, 1993) or on the continuing use of new sources of resistance and combination of genes for specific resistance (Bariana & McIntosh, 1995). Depending on the phenotype and terminology preferences of the respective observers, several types of resistance to stripe rust in wheat, including seedling resistance, adult-plant resistance, temperature-sensitive resistance, hypersensitive resistance, residual resistance, partial resistance and slow rusting, have been described.

Types of resistance

Seedling resistance. Seedling (qualitative) resistance, also known as complete resistance, usually protects plants against avirulent pathogen isolates during their entire growing period (Cromey, 1989a; Ma & Singh, 1996b). Some disease may, however, develop at post-seedling stages (Bariana & McIntosh, 1995). Seedling resistance is often of the pathotype-specific, major gene type (Johnson, 1988; Johnson, 1992b; Danial, 1994; Ma & Singh, 1996b). When used extensively over time and space, new pathotypes usually circumvent the seedling resistance within three to four years after the release of such cultivars (Danial *et al.*, 1995; Line & Chen, 1995; Broers *et al.*, 1996). However, in Australia stripe rust pathotypes virulent to *Yr5*, *Yr8*, *YrSp*, and *Yr27* have been detected despite the absence of these genes in commercial cultivars (Wellings & McIntosh, 1990; McIntosh & Brown, 1997).

A range of designated and temporarily designated seedling genes, controlling stripe rust resistance, has been detected (Table 3). Most of the seedling genes have become ineffective after their release in commercial agriculture. However, by managing seedling resistance the life span of many cultivars has also been extended. Use of pathotype-specific seedling resistance in a multiline cultivar has provided protection for more than ten years (Line & Chen, 1995). The use of cultivar mixtures also has extended the duration of the effective use of seedling resistance (Line & Chen, 1995).

Adult-plant resistance. Plants with APR are susceptible at the seedling stage and develop resistance in the post-seedling phases. These plants may, however, show moderate to high levels of head infection (Cromey, 1989a). Resistance in wheat to

Table 3. Designated and temporarily designated genes for *Puccinia striiformis* f. sp. *tritici* resistance, genome location, source and tester lines

Yr gene	Genome location	Reference	Source	Tester line	Reference
Designated					
1	2AL	Bariana & McIntosh (1993)	Chinese 166	Chinese 166	Lupton & Macer (1962)
2	7B	Lubrum (1980)[a]	Heines VII	Heines VII	Lupton & Macer (1962)
3a	5BL	Worland (1988)[a]	Capelle-Desprez	Capelle-Desprez	Lupton & Macer (1962)
3c	-	-	Minister	Minister	Lupton & Macer (1962)
4a	3B	Worland (1988)[a]	Capelle-Desprez	Capelle-Desprez	Lupton & Macer (1962)
4b	-	-	Hybrid 46	Hybrid 46	Lupton & Macer (1962)
5	2BL	Law, (1976)[a]	*Triticum spelta album*	*T. spelta album*	Macer (1966)[a]
6	7BS	El-Bedewy & Röbbelen (1982)[a]	Heines Kolben	Heines Kolben	Macer (1966)[a]
7	2BL	Law (1976)[a]	Lumillo durum	Lee	Macer (1966)[a]
8	2A-2D/2M	Riley *et al.* (1968)[a]	*T. comosum*	Compair	Riley *et al.* (1968)[a]
9	1BL-1RS	Zeller (1973)[a]	*S. cereale*	Clement	Macer (1975)[a]
10	1BS	Metzger & Silbaugh (1970)[a]	Moro	Moro	Macer (1975)[a]
11	-	-	Joss Cambier[b]	Joss Cambier[b]	Johnson & Taylor (1972)[a]
12	-	-	Caribo[b]	Mega[b]	Priestley (1978)[a]
13	-	-	Ibis[b]	Maris Huntsman[b]	Priestley *et al.* (1984)[a]

Table 3 (cont.). Designated and temporarily designated genes for *Puccinia striiformis* f. sp. *tritici* resistance, genome location, source and tester lines

Yr gene	Genome location	Reference	Source	Tester line	Reference
Designated					
14	-	-	Falco[b]	Maris Bilo[b]	Priestley (1978)[a]
15	1BS	McIntosh *et al.* (1996)	*T. turgidum* var *dicoc.*	*T. dicoccoides*	Gerechter-Amitai *et al.* (1989)
16	2DL	Worland & Law (1986)[a]	Capelle-Desprez	Capelle-Desprez	Worland & Law (1986)[a]
17	2AS	Bariana & McIntosh (1993)	*T. ventricosa*	Trident	Bariana & McIntosh (1993)
18	7DL	Singh (1992b)	Jupateco R	Jupateco R	Singh (1992b)
19	5B	Chen *et al.* (1995a)	Compair	Compair	Chen *et al.* (1995a)
20	6D	Chen *et al.* (1995a)	Fielder	Fielder	Chen *et al.* (1995a)
21	1B	Chen *et al.* (1995a)	Lemhi	Lemhi	Chen *et al.* (1995a)
22	4D	Chen *et al.* (1995a)	Lee	Lee	Chen *et al.* (1995a)
23	6D	Chen *et al.* (1995a)	Lee	Lee	Chen *et al.* (1995a)
24	1BS	McIntosh & Lagudah (2000)	*T. turgidum*	K733	McIntosh & Lagudah (2000)
25	1D	Calonnec *et al.* (1997)	TP1295	TP1295	Calonnec *et al.* (1997)
26	6AS	Jones (1997)[c]	Yangmai-5	Yangmai-5	Jones (1997)[c]; Yildirim *et al.* (2000)
27	2BS	McIntosh *et al.* (1998)[c]	Selkirk	Selkirk	McIntosh *et al.* (1998)[c]
28	4DS	Singh *et al.* (1998)[c]	*T. tauschii W-219*	Altar 84/T. W-219	Singh *et al.* (1998)[c]

Table 3 (cont.). Designated and temporarily designated genes for *Puccinia striiformis* f. sp. *tritici* resistance, genome location, source and tester lines

Yr gene	Genome location	Reference	Source	Tester line	Reference
Temporarily designated					
A	-	-	Avocet	Avocet	Wellings *et al.* (1988)
Cle	4B	Chen *et al.* (1995c)	Clement	Clement	Chen *et al.* (1995c)
Cv	-	-	Carstens V	Carstens V	McIntosh *et al.* (1995)
D	6A	Chen *et al.* (1994)	Druchamp	Druchamp	Chen *et al.* (1994)
Da1	1A	Chen *et al.* (1995c)	Daws	Daws	Chen *et al.* (1995c)
Da2	5D	Chen *et al.* (1995c)	Daws	Daws	Chen *et al.* (1995c)
Dru	5B	Chen *et al.* (1996)	Druchamp	Druchamp	Chen *et al.* (1996)
Dru2	6A	Chen *et al.* (1996)	Druchamp	Druchamp	Chen *et al.* (1996)
H46	6A	Chen *et al.* (1996)	Hybrid 46	Hybrid 46	Chen *et al.* (1996)
H52	1B	Peng *et al.* (1999)	*T. dicoccoides*	*T. dicoccoides H52*	Peng *et al.* (1999)
HVII	4A	Chen *et al.* (1995c)	Heines VII	Heines VII	Chen *et al.* (1995c)
Mor	4B	Chen *et al.* (1995c)	Moro	Moro	Chen *et al.* (1995c)
Min	4A	Chen *et al.* (1996)	Minister	Minister	Chen *et al.* (1996)
Nd	4A	Chen *et al.* (1996)	Nord Desprez	Nord Desprez	Chen *et al.* (1996)
S	3B	Chen *et al.* (1994)	Stephens	Stephens	Chen *et al.* (1994)

Table 3 (cont.). Designated and temporarily designated genes for *Puccinia striiformis* f. sp. *tritici* resistance, genome location, source and tester lines

Yr gene	Genome location	Reference	Source	Tester line	Reference
Temporarily designated					
Sd	-	-	Strubes Dickkopf	Strubes Dickkopf	McIntosh *et al.* (1995)
Sp	-	-	Spaldings Prolific	Spaldings Prolific	McIntosh *et al.* (1995)
Ste	2B	Chen *et al.* (1996)	Stephens	Stephens	Chen *et al.* (1996)
Ste2	3B	Chen *et al.* (1996)	Stephens	Stephens	Chen *et al.* (1996)
SU	-	-	Suwen 92/Omar	Suwen 92/Omar	McIntosh *et al.* (1995)
Tr1	6D	Chen *et al.* (1995c)	Tres	Tres	Chen *et al.* (1995c)
Tr2	3A	Chen *et al.* (1995c)	Tres	Tres	Chen *et al.* (1995c)
Tye	6D	Chen *et al.* (1995c)	Tyee	Tyee	Chen *et al.* (1995c)
V23	2B	Chen *et al.* (1996)	Vilmorin	Vilmorin	Chen *et al.* (1996)
Yam	4B	Chen *et al.* (1996)	Yamhill	Yamhill	Chen *et al.* (1996)

[a]McIntosh *et al.* (1995).

[b]Roelfs *et al.* (1992).

[c]McIntosh *et al.* (1998).

stripe rust that is only detectable in adult-plants can be pathotype-specific or pathotype-non-specific (Johnson, 1988; Johnson, 1992b). Genes conferring APR are *Yr11* to *Yr14*, *Yr16* and *Yr18*, the latter two genes being pathotype-non-specific (McIntosh *et al.*, 1995). According to Park & Rees (1989) the onset of APR is during tillering to node formation, expressed as chlorosis and/or necrosis in association with rust colonies on the most resistant cultivars.

Temperature-sensitive resistance. Several reports exist proving that some resistance genes to stripe rust are temperature-sensitive (Lewellen & Sharp, 1967; Sharp *et al.*, 1976; Qayoum & Line, 1985; Van Dijk *et al.*, 1988; Line & Chen, 1995; Shang & Shang, 1998). High-temperature adult-plant resistance (HTAPR), which is durable and pathotype-non-specific (quantitative), is the most important method of controlling stripe rust in the Pacific Northwest (Chen & Line, 1995b; Broers *et al.*, 1996). More than 90% of the cultivars grown in this region have HTAPR (Line & Chen, 1995). Cultivars with HTAPR have remained resistant for more than 30 years, even when grown extensively in the region and exposed to numerous pathotypes of *P. striiformis*. High-temperature adult-plant resistance is characterised by a range of infection types influenced mostly by temperature and growth stage of the plant. At high temperatures, the flag leaves are most resistant (Line, 1993; Chen & Line, 1995a). In the field, as the season progresses and temperature increases, infection types become lower, and rust develops slower on these cultivars than on susceptible cultivars. Under controlled conditions, seedlings of these cultivars are susceptible to the prevalent pathotypes over a wide range of temperatures, but as plants mature they become more resistant when incubated at high temperatures (diurnal temperatures of 10 to 30°C or higher). However, at low temperatures (diurnal temperatures of 6 to 21°C or lower) the plants remain susceptible (Qayoum & Line, 1985). High-temperature adult-plant resistance can be considered to be a "slow rusting" type of resistance, since it decreases the rate of rust development (Line & Chen, 1995). According to Broers *et al.* (1996) lower temperatures resulted in reduced expression of HTAPR to stripe rust, therefore, additional resistance genes are required in cooler areas. There is a lack of exploitation of HTAPR outside the Pacific North West mainly due to the dependence of this resistance on high spring

temperatures which is not characteristic of large areas of the world vulnerable to stripe rust.

Hypersensitive resistance. Hypersensitive resistance is characterised by cell collapse around the point of entry of the pathogen, resulting in a low infection type (Danial, 1994). Hypersensitive resistance can be expressed in the seedling and/or adult-plant stage and can be complete (no sporulation) or incomplete (some sporulation). This type of resistance is usually induced by major genes which is vulnerable to genetic adaptation in the pathogen population and, therefore, non-durable (Danial, 1994).

Residual resistance. Residual resistance is obtained by selecting resistance among the progeny of crosses between cultivars rated as susceptible (Johnson, 1984). Residual resistance will not always be pathotype-non-specific. The effect of pathotype-specific genes is often reduced and sometimes completely suppressed when back-crossed into highly susceptible cultivars. A susceptible plant could, therefore, possess pathotype-specific or pathotype-non-specific genes that might become effective when transferred to a new genetic background (Johnson, 1984). Another form of residual resistance occurs when a highly resistant cultivar becomes susceptible due to the introduction or evolution of a new pathotype of the pathogen, but not to the same degree as extremely susceptible cultivars like Morocco (Danial, 1994). Resistance that remains effective in this way is of a quantitative nature (Danial, 1994).

Partial resistance. Parlevliet (1979) introduced the concept of partial resistance (PR) as a form of quantitative resistance. Partial resistance is characterised by a reduction in spore production even though the host plants have a susceptible infection type. Furthermore, no hypersensitive reaction is present. Increasing levels of quantitative resistance are characterised by longer latent period (LP), lower infection frequency and incidence, smaller and slower growing lesions resulting in less leaf area affected (Shaner & Finney, 1980; Broers, 1997). Breeders are interested in PR because it is assumed to be polygenically inherited and durable

(Danial, 1994). In wheat leaf rust, quantitative resistance has been considered partial (Broers, 1989), as it was characterised by a susceptible infection type at both seedling and adult plant stages combined with a slower rate of disease development (Broers, 1997). In wheat, quantitative resistance to stripe rust is not associated with PR (Johnson, 1981; Broers, 1993; Danial, 1993; Broers, 1997). Quantitative resistance in wheat to stripe rust is characterised by a susceptible infection type in the seedling stage (but not in the adult plants in the field) and slow epidemic development (Park *et al.*, 1988; Park & Rees, 1989; Broers *et al.*, 1996; Broers, 1997).

Slow rusting. Slow rusting is incomplete or quantitative resistance that is associated with a reduced rate of epidemic development (Danial, 1994). Slow rusting may be the result of fewer and smaller uredia, longer latent periods and slower growing lesions resulting in less leaf area infected (Danial, 1994; Broers, 1997). It is important to note that environmental effects on the pathogen and host resistance can result in slow rusting. Slow rusting is further a relative measurement against a specific check and must not be confused with resistance due to a non-pathogenic pathotype (Roelfs *et al.*, 1992).

Sources of resistance

Knowledge about the availability and value of sources of resistance is important to the breeder in choosing target genes suitable for breeding for resistance (Johnson, 1992a). There are numerous sources of resistance to rust diseases, although not all are of equal value (McIntosh *et al.*, 1995). Genes conferring resistance to stripe rust were identified in *T. aestivum*, *T. spelta*, *T. turgidum*, *T. ventricosum*, *T. comosum* and *T. cereale* (McIntosh *et al.*, 1995; Friebe, *et al.*, 1996; McIntosh & Lagudah, 2000). Ma *et al.* (1995) evaluated durum wheat (*Triticum turgidum* L. var. *durum*), *T. tauschii*, and synthetic hexaploid wheat (*T. turgidum* x *T. tauschii*) for resistance to stripe rust in the seedling stage in the glasshouse and the adult-plant stage in the field. The synthetic hexaploid wheat showed large variability for disease responses in both glasshouse and field tests, indicating a number of genes for resistance. In general, genotypes with seedling resistance were also found to be resistant as adult-

plants. Genotypes, which were susceptible or moderately susceptible as seedlings but resistant as adult-plants, were present in both *T. turgidum* and the synthetic hexaploids. These sources can be used to incorporate resistance into cultivated hexaploid wheat to increase the existing gene pool of resistance to stripe rust. Ma *et al.* (1997) found that the low seedling infection type of the durum cultivars Kroub 76, Chonta Inia, Sna 3, Syros, and Arena was based on the additive effects of the same two genes. Each of these genes conferred intermediate infection types when present alone. Field resistance of the cultivars was based on the additive effects of the same two seedling genes and one additional, partially effective APR gene. Evaluating 279 *T. tauschii* (syn. *Aegilops squarrosa*) accessions for resistance to stripe rust, Yildirim *et al.* (1995) found 39 accessions resistant to four stripe rust pathotypes, the latter representing all known virulence combinations in the Pacific North West.

Wild emmer (*T. dicoccoides* Koern.), an ancestor of cultivated durum wheat (*Triticum turgidum* L.) and indigenous to the Middle East, is a further valuable source of major genes for stripe rust resistance (Gerechter-Amitai & Stubbs, 1970; Gerechter-Amitai & Grama, 1973; Gerechter-Amitai *et al.*, 1989; Van Silfhout *et al.*, 1989; Singh & Abdalla, 1997; Shang & Shang, 1998). In addition to these Grama *et al.* (1984) showed the existence of additive "minor-effect", temperature-sensitive genes in wild emmer lines. Reinhold *et al.* (1983) found transgressive segregation (when lines are obtained with greater resistance than in both parents), for higher resistance to stripe rust, in segregating populations of crosses of *T.dicoccoides* x *T. aestivum* and *T. dicoccoides* x *T. durum*. This indicates that additive genes for stripe rust resistance can be transferred directly from the wild emmer to cultivated tetraploid and hexaploid wheat.

Genetics of resistance

Knowledge of the genetic bases of resistance to rust pathogens in wheat cultivars is useful in understanding the distribution of pathotypes of the pathogen and also in breeding for resistance to these diseases (Perwaiz & Johnson, 1986). According to Johnson (1992a) Biffen provided first evidence of Mendelian inheritance of resistance to stripe rust in 1905. Lupton & Macer (1962) were the first researchers

to initiate studies on inheritance of resistance in seedlings of wheat cultivars to stripe rust, and introduced the nomenclature of *Yr* genes. The genes identified by Lupton & Macer (1962) were pathotype-specific and assumed to operate on a gene-for-gene basis with the pathogen. The consequences of introducing these genes into breeding programmes were the creation of a succession of cultivars in which the resistance was rapidly overcome by new pathotypes, gaining the corresponding virulence through mutation or recombination (Johnson, 1992a).

Broers & López-Atilano (1996) studied the effect of quantitative resistance in the cultivars Opata 85 (moderately resistant [*Yr18*]), Pavon 76 (moderately to highly resistant), and Parula (highly resistant [*Yr18*]) on the development of *P. striiformis* during the first six days of the infection process. Their results indicated that the major effect of quantitative resistance on *P. striiformis* was an avoidance mechanism that reduced the formation of appressoria, followed by possible disintegration of substomatal vesicles, and delayed development of infection hyphae and subsequent establishment of the fungus. They speculated that the avoidance mechanism might be durable as it is probably a morphological barrier acting before intimate contact between host and parasite occurs.

Krupinsky & Sharp (1979) intercrossed ten commercial cultivars of winter wheat with intermediate or susceptible reactions to stripe rust. When evaluated as seedlings in controlled environment chambers, 38 crosses showed transgressive segregation. Transgressive segregation was also clearly shown in later generations of 17 crosses even though the F_2 and F_3 generations were totally susceptible. Resistance increased with successive selfing, inoculation and selection, with 12% in the F_4, 79% in the F_5, and 61% in the F_6 generation. Wallwork & Johnson (1984) made crosses between wheat varieties Joss Cambier, Nord Desprez and Maris Bilbo, all classified as susceptible to stripe rust in field tests, and between Cappelle Desprez and Maris Huntsman, both classified as moderately and durably resistant. They obtained lines with greater resistance than in both parents from each cross. They suggested that transgressive resistance is more likely to be durable if it is derived from parents that have shown durable resistance.

According to Chen & Line (1995a) the HTAPR cultivars Stephans and Druchamp should be used as female parents in order to obtain the highest HTAPR in

the progeny. The reason is the presence of a significant cytoplasm additive gene interaction in reciprocal crosses of Druchamp with Paha and a significant cytoplasm-dominance interaction when Stephans is crossed with Paha. The HTAPR in the cultivars Nugaines and Luke is partially recessive. Milus & Line (1986a) found significant epistatic gene action for resistance in Nugaines which is higher than in Luke while most gene action among loci is additive.

Selecting for resistance

Breeding for resistance depends largely on an appropriate and reliable method of selection. This may include the selection of seedling genes with avirulent pathotypes, molecular and morphological markers, and by assessing the affected leaf areas and the type of lesion produced (Dyck, 1991; Johnson, 1992a; Singh, 1992a; Singh, 1993; Sun *et al.*, 1997; Irshad *et al.*, 1999; Peng *et al.*, 1999; Robert *et al.*, 1999; Chen & Line, 2000). According to Broers *et al.* (1996) quantitative resistance can be measured in terms of DS, area under the disease progress curve (AUDPC), apparent infection rate, and infection type (IT). However, according to Broers (1989) and Broers *et al.* (1996) the infection rate is an unreliable estimate compared with DS and AUDPC. Infection type is poorly correlated with DS and therefore appears an unsuitable parameter for assessing quantitative resistance (Danial, 1994). In a glasshouse study Broers (1997) found LP and DS to be the best options of selection for quantitative resistance after uniform inoculation.

Infection frequency is a component of resistance that expresses the efficiency by which the fungus is able to complete the whole infection cycle (Broers, 1997). A low level of quantitative resistance, as in Anza, reduced infection frequency substantially (to 47%) and higher levels of resistance as in Pavon 76 reduced infection frequency to less than 10%. According to Broers & López-Atilano (1994) each stripe equals not necessarily one infection. Especially on susceptible cultivars, a single stripe may result from more than one point of infection. This means the infection frequency measured on Morocco is most likely an underestimation and that real genotype differences for infection frequency are probably even larger than reported (Broers, 1997).

Ma & Singh (1996b) conducted glasshouse evaluations of eight wheat cultivars known to carry different levels of APR (HD2258, PBW65, Mexico 82, Pavon 76, Jupateco 73R, Apache 81, Anahuac 75, and Ciano 79) and three susceptible cultivars (Morocco, Avocet S, and Jupateco 73S) at six growth stages. Seedling IT and LP for cultivars with APR were similar to those of the susceptible cultivars. However, as plants matured, resistance increased and was expressed as lower IT's and longer latent periods. The IT and LP at anthesis by resistant cultivars were lowest and highest, respectively, indicating that APR was best expressed at this growth stage. A negative correlation between IT and LP at anthesis suggested that lower IT's of the cultivars were generally associated with longer latent periods Ma & Singh (1996b).

Cultivars with a seedling IT that are consistently lower than that of Morocco (IT= 7 or 8 instead of 9) would still be classified as susceptible. Data presented by Broers (1997) showed that such cultivars carry different levels of quantitative resistance. The cultivars were not susceptible in the adult plant stage, but characterised by a slow epidemic development. Using these characteristics as selection criteria may however lead to the selection of pathotype-specific adult plant resistance genes such as *Yr14* (Broers, 1997).

Achieving durability

Long-term resistance to rust diseases depends on the identification and use of durable resistance (pathotype-non-specific) sources or on the continued use of new resistance sources and combinations of genes for specific resistance (Bariana & McIntosh, 1995). Durable resistance is defined as a resistance source that remained effective after widespread deployment over a considerable period and in the presence of regular disease epidemics (Johnson & Law, 1975; Johnson, 1984). According to McIntosh (1992) a general concept of a durable resistance source is that it may be controlled by more than one gene on several chromosomes, it is more likely to operate in the adult-plant stage, and that it confers a non-hypersensitive response to infection. Similar conclusions, i.e. resistance in wheat cultivars that maintained their level of resistance after wide-spread use appears to be quantitative, sensitive to temperature and based on more than one gene, have been drawn by

others (Sharp *et al.*, 1976; Sharp & Fuchs, 1982; Qayoum & Line, 1985; Milus & Line, 1986a,b; Van Dijk *et al.*, 1988; Shang & Shang, 1998).

In attempting to achieve durable resistance, breeders should be aware of the vulnerability of genes conferring complete resistance. The combination of effective seedling resistance genes can provide longer-lasting protection as this would require pathotypes to undergo multiple simultaneous or step wise changes in order to become virulent (McIntosh & Brown, 1997). McIntosh & Brown (1997) defined the concept of anticipatory resistance breeding. This include the prediction of future pathotypes and the production of resistant germplasm to avert future losses. The latter is obtained through national pathotype surveys and genetic analysis to catalogue the identity and distribution of resistance genes in existing cultivars.

The best breeding prospect for durable resistance 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, 1992a). This requires pathogen isolates that can overcome recognised pathotype-specific components of resistance (Johnson, 1978; Johnson, 1992b). Cultivars given in the literature as having non-specific resistance to stripe rust are summarised in Table 4.

The adult-plant leaf rust resistance gene *Lr34* is tightly linked to the APR stripe rust gene *Yr18* (McIntosh, 1992; Singh, 1992b). The two genes are also known to be linked with gene *Ltn*, which confers leaf tip necrosis in adult-plants, a morphological marker that could be used for the identification of *Lr34* and *Yr18* (Dyck, 1991; Singh, 1992a; Singh, 1993). According to Ma & Singh (1996a) slow rusting resistance conferred by *Yr18* protected grain yield in the range of 36 to 58%, depending on the year and sowing date. The deployment of *Yr18* alone is not recommended in areas with high stripe rust pressure (Viljanen-Rollinson & Cromey, 2000). The pyramiding of *Yr18* with other slow rusting genes and genes for specific resistance should lead to the development of wheat lines with more durable resistance (Ma & Singh, 1996a; Kumar *et al.*, 1999).

Table 4. Wheat cultivars described in the literature as having non-specific resistance to *Puccinia striiformis* f. sp. *tritici*

Cultivar	*Yr* genes	Type of non-specific resistance	Reference
Anahuac 75	-	Intermediate level of APR	Ma & Singh (1996b)
Anza	*A,18*	Intermediate level of QR[a]	Johnson (1988); Broers *et al.* (1996)
Apache 81	-	Intermediate level of APR	Ma & Singh (1996b)
Atou	*3a,4a,16*	Durable[b]	Johnson (1988)
Banks	*Seg. A*	High level of APR	Park *et al.* (1988); Park & Rees (1989)
Bass	-	High level of APR	Park *et al.* (1988); Park & Rees (1989)
Bouquet	*3a,4a,14,16*?	Durable[b]	Johnson (1988)
Capelle-Desprez	*3a,4a,16*	Durable[b]	Lupton *et al.* (1971)[c]
Ciano 79	*Sk*	Intermediate level of APR	McIntosh *et al.* (1995); Ma & Singh (1996b)
Cook	Two genes	High level of APR	Park *et al.* (1988); Park & Rees (1989); Bell & Wellings (1993)[d]
Druchamp	*3a,Dru,Dru2*+	High temperature APR	Chen & Line (1995a,b); Chen *et al.* (1996)
Elite Lepeuple	*2*	Durable[b]	Johnson (1988)
Fan Lui	-	Intermediate level of QR	Broers *et al.* (1996)
Flanders	*1,3a,4a,16*?	Durable[b]	Johnson (1988)
Flinders	One gene	Intermediate level of APR	Bariana & McIntosh (1995)
Flinor	One gene	Durable[b]	Johnson (1988)
Harrier	One gene	Intermediate level of APR	Bariana & McIntosh (1995)

Table 4 (cont.). Wheat cultivars described in the literature as having non-specific resistance to *Puccinia striiformis* f. sp. *tritici*

Cultivar	*Yr* genes	Type of non-specific resistance	Reference
Hybride de Bersee	*3a,4a,16*?	Durable[b]	Johnson (1978); Johnson & Law (1975); Hyde & Elahinia (1990)
Ibis	*1,2,13*	Temperature sensitive?[c]	Stubbs (1977)
Israel	-	High level of QR	Broers *et al.* (1996)
Joss Cambier	*2,3a,11*	Non-durable[b]	Lupton *et al.* (1971)[c]
Jupateco R	*18*	Intermediate level of QR	Broers *et al.* (1996); Ma & Singh (1996b)
Karamu	*A*	Durable[b]	Johnson (1988)
King	Two genes	Intermediate level of APR	Bariana & McIntosh (1995)
Little Joss	-	Durable[b]	Lupton *et al.* (1971)[c]
Luke	Two genes	High temperature APR	Qayoum & Line (1985); Milus & Line (1986a,b)
Maris Huntsman	*2,3a,4a,13,16*?	-	Johnson (1988)
Maris Widgeon	*3a,4a,8,16*?	Durable[b]	Lupton *et al.* (1971)[c]
Mexico 82	-	High level of APR	Ma & Singh (1996b)
Nugaines	Two genes	High temperature APR	Qayoum & Line (1985)
Oxley	*6*, + two genes	High level of APR	Wellings & McIntosh (1990); Bell & Wellings (1993)[d]
Parula	*18*+	High level of APR	Broers *et al.* (1996); Singh (1992b)
Pavon 76	*6,7* + two genes	High level of APR	Ma & Singh (1996b); Dubin *et al.* (1989); Wellings & McIntosh (1990)

Table 4 (cont). Wheat cultivars described in the literature as having non-specific resistance to *Puccinia striiformis* f. sp. *tritici*

Cultivar	*Yr* genes	Type of non-specific resistance	Reference
Stephens	*3a,Ste,Ste2+*	High temperature APR	Chen & Line (1995a,b); Chen *et al.* (1996)
Tonichi 81	*18+* two genes	High level of APR	Singh & Rajaram (1994)
Vilmorin 27	*3a,4a,16?*	Durable[b]	Johnson (1988)

[a]Quantitative resistance.

[b]Johnson (1978); Johnson (1992a); Roelfs *et al.* (1992).

[c]Roelfs *et al.* (1992).

[d]Bariana & McIntosh (1995).

Factors influencing the expression and assessment of resistance

Several factors besides the genome may influence the accurate assessment of resistance to stripe rust (Danial, 1994).

Temperature and light intensity. Stripe rust is a cool-weather disease and the effects of temperature on disease development have been widely reported. According to Sharp (1965) a temperature of 7°C during the dew period is optimum for both *in vivo* germination and infection; 15°C is near maximum. Both prevalence and severity of infection were higher after a dew period at 2°C than one at 13°C. Appressoria developed equally well from 7 to 15°C. Mcgregor & Manners (1985) found that increasing temperature between 7 and 20°C shortened the LP and reduced the longevity of sporulating leaves. Colonisation rate and the frequency of pustules per unit area of infected leaf increased between 7 and 15°C but decreased markedly at 20°C. Spore production reached its peak earlier and declined more rapidly with increasing temperature between 7 and 15°C.

Sharp (1962) found that preinoculation temperature was often critical in determining rust reaction types. Several wheat varieties were susceptible when grown at 15°C prior to inoculation, but resistant when grown at 24°C in the preinoculation phase. Temperature also influences the expression of certain seedling genes. Short exposures (as short as 4 h) to contrasting temperatures, at both pre and post-inoculation phases, may result in significant changes in infection type (Brown & Sharp, 1969). According to Dubin *et al.* (1989) *Yr6* is less effective at extreme glasshouse temperatures, while light intensity may also influence the expression of this gene. According to McIntosh *et al.* (1995) seedlings with *Yr17* are more susceptible at lower temperatures and low light intensities.

Light intensity has an influence on the development of stripe rust. Urediospore production by *P. striiformis* on wheat, per unit leaf area infected, are much lower at low light intensities than at high light intensities (Mcgregor & Manners, 1985) and the daily sporulation rate per pustule increased linearly with increasing light over the range 10-50 W/m^2. According to Stubbs (1967) stripe rust infection type could increase, decrease or remain the same with increasing light intensity, depending on the genotype.

Wellings *et al.* (1988) found that low light intensity suppressed the resistant response of *YrA* and in most instances, approached full compatibility, even after 4 days post-inoculation exposure to high light intensity. However, after 8 days at high light intensity, the response was not noticeably affected by exposure to low light. The high responses were particularly evident on the first leaf, in contrast to the distinctly lower second leaf response. Stubbs (1967) attributed the slow development of high infection types, in some cases approaching a fully compatible reaction indicating pathogen virulence as sometimes observed with Carstens V, in part, to an inability of this differential line to express its resistance in insufficient light conditions. This may occur when leaves become large enough to provide shade (De Vallavieille-Pope & Line, 1990).

Plant nutrition. Changes in disease susceptibility experienced with specific nutrients could be due to changes in the tolerance of the host plant to the disease, disease escape, changes in the physiological resistance of the host, and reduced or enhanced virulence in the pathogen (Huber, 1980). Higher levels of fertilization can lead to a more favourable microclimate in the canopy for certain diseases, especially those caused by biotrophic pathogens, a slower rate of leaf senescence and thus a longer cropping period.

Nutrients differ in their effect on different diseases, and its formulation or availability may be as important as the nutrient itself. Huber & Watson (1974) found that nitrogen (N) applied in the nitrate form increased stripe and stem rust development while the ammonium form resulted in a decrease. The level of disease response observed with a specific form of N is, however, very complex. Factors that may have an influence is the pH of the soil, host response or preference, previous cropping cycle, N-rate and stability, residual N, time of application, soil micro-flora present and the ratio of NH_4 to NO_3 nitrogen (Huber & Watson, 1974).

Several authors reported an increase in stripe rust severity with increased N-rates (Russel, 1978; Daamen *et al.*, 1989; Ash & Brown, 1991). The trend towards increasing the N nutrition of cereals results from the added economic incentive to produce grain with a higher protein content (Ash & Brown, 1991). In glasshouse studies Ash & Brown (1991) found that stripe rust infection reduced wheat yield at all

N application rates. However, in field studies there appeared to be some compensation for stripe rust infection at the higher rates (90 kg N/ha), as the percentage loss in yield resulting from stripe rust infection was similar to the yield losses at lower levels (0-60 kg N/ha) although the disease severity was higher.

Danial & Parlevliet (1995) carried out two experiments to study the effect of nitrogen level on disease severity of stripe rust. Wheat genotypes, varying in quantitative resistance to stripe rust, were exposed to 0, 20, 40 and 80 kg calcium ammonium nitrate (NH_4NO_3 + $CaCO_3$) per ha. The N was divided into two parts, with half the rate applied at tillering and the remainder at booting. In both experiments all genotypes showed a clear increase in disease severity with increased N-rates. The increase in disease severity was associated with a higher infection type with all genotypes responding similarly. Genotype x year, genotype x N-level and genotype x year x N-level interactions occurred, causing some changes in ranking order between years and with changing N-levels for entries that had resistance levels not too far apart. Soil fertility as an environmental factor might thus affect the assessment of resistance in breeding programmes. Breeders must, therefore, screen for quantitative resistance at N-levels similar to those used in the area for which the wheat is intended.

Inter-plot interference. Inter-plot interference could lead to an underestimation of the level of partial or quantitative resistance, especially with wind borne pathogens (Paysour & Fry, 1983; Parlevliet & Van Ommeren, 1984). Screening for resistance carried out in small adjacent plots does not take into account whether lesions developed from inoculum produced by the plot itself or from neighbouring plots. More resistant entries may receive substantial amounts of inoculum from more susceptible neighbours while the latter may receive much smaller amounts from their more resistant neighbours. Disease on more resistant entries may therefore be overestimated and that on the highly susceptible ones underestimated and could lead to errors in the ranking of entries for resistance (Danial *et al.*, 1993). However, results of four wheat stripe rust experiments conducted by Danial *et al.* (1993) did not indicate inter-plot interference. One possible explanation given by the authors is

the higher degree of systemic growth of *P. striiformis* compared to the other cereal rusts where inter-plot interference does occur.

Earliness and observation date. Variation in stripe rust response of certain lines at different sites may be due to observations recorded at different times, hosts at different growth stages, different environments and/or different crop management practices (Bariana & McIntosh, 1995). According to Broers (1989) differences in the developmental rate of cultivars have a large impact on the disease progress. The resistance of early cultivars tended to be underestimated whereas the resistance of late-maturing cultivars tended to be overestimated. When selecting for quantitative resistance in wheat to stripe rust the assessment is based on differences in DS and AUDPC (Broers, 1989; Danial, 1994; Broers *et al.*, 1996). Danial (1994) conducted a study to investigate the influence of earliness, observation date and leaf layer on disease severity. The results showed that when heading dates varied within 2 to 2.5 weeks, the effect of earliness on the assessment of resistance was ignorably small. However, when heading dates differ by longer periods, breeders are advised to classify the entries into growth period groups for reliable comparison. The results further showed that disease severity in the field measured on the flag and second leaf can be reliable, provided the assessment is not done at a too early date. Ranking of the genotypes was affected to some extent by the observation date (Danial, 1994). To avoid bias introduced by variation in growth period, AUDPC can be divided by the duration of the epidemic to provide standardised AUDPC values (Campbell & Madden, 1990).

WHEAT LEAF RUST

Leaf rust of wheat has recently been reviewed by Kloppers (1994), Bender (1995), Jacobs (1996) and Barnard (1999). The purpose of this review is to summarise available literature on losses caused by this disease and chemical control.

Economic importance

Wheat leaf rust is regarded as the most common and most widely distributed of all cereal rusts (Chester, 1946; Roelfs *et al.*, 1992; Ortelli *et al.*, 1996; Sayre *et al.*, 1998; Brahma *et al.*, 1999; Moschini & Pérez, 1999; Manisterski *et al.*, 2000). Losses in grain yield due to leaf rust are primarily attributed to reduced floret set and shrivelling of grain whereas in severe pre-heading epidemics florets, plants and tillers can be killed (Roelfs *et al.*, 1992).

Although leaf rust is considered less devastating when compared to stripe and stem rust, several reports describing its influence on crop productivity exist. Chester (1946) provided evidence of yield reductions of 34% in Kansas in 1938, and even of total losses where no grain could be havested. In Canada leaf rust normally reduces wheat yields by 5 to 10% when widely grown cultivars are susceptible (Samborski, 1985). Early-season leaf rust epidemics on young wheat plants in Mexico reduced yields by as much as 40% during 1976-1977 (Samborski, 1985). More recently in Western Australia, a widespread leaf rust epidemic caused yield losses of up to 37% in susceptible cultivars during 1992 (McIntosh *et al.*, 1995).

In a controlled experiment average losses in grain yield due to leaf rust ranged between 6.6 to 62.7%, (Sayre *et al.*, 1998). The losses in grain yield were mainly attributed to reductions in kernel weight, kernels per square meter and grain fill rate. Singh & Huerta-Espino (1997) evaluated the effect of APR conferred by the durable resistance gene *Lr34* on yield. Leaf rust reduced yield by 15% in the presence of *Lr34*. However, in the absence of *Lr34* the reduction in yield ranged between 43 and 84% depending on planting date and year.

The impact of leaf rust on wheat production was quantified by Smale *et al.* (1998). They estimated the economic benefit of incorporating non-specific leaf rust

resistance genes into bread wheat cultivars in the Yaqui Valley, Mexico, at 17 million U.S. dollars for the period 1970 to 1990.

Chemical control

Yield increases of up to 68% resulting from the chemical control of leaf rust have been documented (Dannenberg *et al.*, 1989; Eversmeyer & Kramer, 1996; Kalappanavar & Patil, 1997; Khan & Trevathan, 1997; Khan *et al.*, 1999; Singh, 1999; Sundin *et al.*, 1999). Fungicides may differ in their ability to control leaf rust and thereby their influence on yield. Kalappanavar & Patil (1997) evaluated the efficacy of propiconazole, triadimefon, hexaconazole, cyproconazole and mancozeb to control leaf rust. They found cyproconazole the most, and mancozeb the least effective. Likewise, the highest yield increase was obtained with cyproconazole. Conner & Kuzyk (1988) found triadimefon less effective in controlling leaf rust than either propiconazole or fenpropimorph. The foliar fungicides oxycarboxin, chlorothalonil, mancozeb and the seed treatment fungicide triadimenol were ineffective in controlling leaf rust and had no effect on yield. However, systemic triazoles, applied as a seed treatment, have been used to control or suppress early season leaf rust infections in other studies (Rakotondradona & Line, 1984; Line, 1993; Everts & Leath, 1993). In a glasshouse experiment difenoconazole (24 g a.i./100 kg seed) suppressed sporulation of *P. triticina* by up to 10% of the levels of control plants for 3 weeks after sowing (Sundin *et al.*, 1999). Spore production was suppressed to 25% of the non-treated control for at least 4.2 weeks, and to 50% for at least 6.5 weeks (Sundin *et al.*, 1999).

In a recent study cyproconazole, fenpropimorph and triadimenol were found to be the most effective fungicides for reduction of leaf rust (Cook *et al.*, 1999). Fungicides were found equally effective against leaf rust when applied just before symptoms were detected (Cook *et al.*, 1999). Timing of fungicide application thus appeared to be more important than choice of fungicide to avoid unacceptable crop losses. According to Rossing *et al.* (1994) the best timing of chemical applications can be calculated in advance when the future course of the population density of the pathogen, the associated damage, the effectiveness of control, the financial revenue

of crop yield and the costs of chemical control are known. Fungicides registered for the control of *P. triticina* in South Africa are listed in Table 5.

CONCLUSIONS

The occurrence of stripe rust epidemics in South Africa depends largely on the ability of *P. striiformis* to over-summer on volunteer wheat and grass hosts, environmental conditions suitable for pathogen survival and growth, and the cultivation of susceptible wheat cultivars. From the literature it is evident that the eastern Free State with its predominant summer rainfall and mild summer temperatures is ideal for the survival of stripe rust between cropping seasons. Similarly the southern Cape occasionally receives summer rain, which may assist in the survival of volunteer wheat and accessory hosts, and thereby the survival of the stripe rust pathogen. Lesotho, with its summer cropping cycle, is also an ideal area for the summer survival of stripe rust. From here stripe rust can easily spread to the Free State, KwaZulu-Natal and the Eastern Cape during the winter cropping cycle. Monitoring the survival of stripe rust in Lesotho and determining the response of their wheat cultivars can be of great value. Since there is no known alternate host for *P. striiformis*, variability in the fungus in South Africa is expected to be determined by mutation, somatic recombination and introduction of exotic pathotypes. The introduction of more virulent pathotypes from central and northern Africa can, however, not be excluded.

At a cost of ± R12/ha in the summer rainfall region, triazole seed treatment seems a viable option for local producers when planting stripe rust susceptible wheat cultivars in the more rust prone areas. Hence they can control stripe rust during early growth stages and prevent the build-up of inoculum, which may contribute significantly to the development of epidemics later in the season. The local cost of foliar application of triazoles is currently ± R90/ha, with an added ± R60/ha for application. With a current wheat price of ± R1200/ha chemical control of stripe rust appears to be viable. However, a sudden drop in the wheat price, lower than predicted yield due to the occurrence of unfavourable environmental conditions

Table 5. Active components, gram active ingredient, dosage and active ingredient per hectare of fungicides registered to control *Puccinia triticina* in South Africa[a]

Active components	Gram active ingredient (g/ℓ)	Dosage for ground applications (ml/ha)	Gram active ingredient per hectare
Foliar applications			
Carbendazim/cyproconazole	300/160	375	112.5/60
Carbendazim/flusilazole	125/250	400	50/100
Carbendazim/flutriafol	150/94	1200	180/113
Cyproconazole	100	400	40
Flutriafol	125	1000	125
Propiconazole	250	400	100
Tebuconazole	250	750	188

[a]Nel *et al.* (1999)

following fungicide application(s), or the repeated need for fungicide application on susceptible cultivars during prolonged epidemics, are some of the risks involved for producers.

Breeding resistant cultivars is the most environment-friendly and cost-effective way to control stripe rust. From the literature reviewed it can be concluded that breeding efforts in South Africa should concentrate on quantitative resistance to obtain durable resistance against stripe rust. Selection for complete resistance (controlled by seedling genes) to stripe rust should be avoided since new pathotypes usually evolve within a few years after the release of cultivars with monogenic seedling resistance, whereas some cultivars may become susceptible even before their release. To increase the possibility of achieving durable resistance, breeders should select parents with satisfactory agronomic traits and proven long-lasting resistance. The deployment of APR genes such as *Yr18* alone is not recommended in areas with high stripe rust pressure. Pyramiding of *Yr18* with other slow rusting genes, and genes for specific resistance, should provide more effective control of stripe rust.

On a global basis leaf rust is best controlled by the use of resistant wheat cultivars. Epidemic outbreaks of this disease, however, still occur mainly due to the appearance of more virulent pathotypes. Although the levels of leaf rust resistance in most South African cultivars are acceptable, seasonal outbreaks, particularly in the Western Cape, still occur. Contributing to the latter is the early season build-up of inoculum on cultivars lacking adequate levels of resistance. Leaf rust thus remains an economically important wheat disease justifying continued research.

To achieve long-lasting rust control in South Africa it is important that research on pathotype distribution and variation, identification of appropriate sources of resistance, genetics of resistance, and more efficient traditional and molecular-based selection techniques, be maintained or developed. Co-operation between breeders nationally, and liaison with rust workers internationally, should also be pursued.

LITERATURE CITED

Akanda, S.I. & Mundt, C.C. 1997. Effect of two-component cultivar mixtures and yellow rust on yield and yield components of wheat. Plant Pathology 46:566-580.

Allan, R.E., Purdy, L.H. & Vogel, O.A. 1963. Inheritance of seedling and adult reaction of wheat to stripe rust. Crop Science 6: 242-245.

Ash, G.J. & Brown, J.F. 1991. Effect of nutrition of the host on the epidemiology of *Puccinia striiformis* f. sp. *tritici* and crop yield in wheat. Australian Plant Pathology 20: 108-114.

Ash, G.J. & Brown, J.F. 1990. Yield losses in wheat caused by stripe rust (*Puccinia striiformis* West.) in northern New South Wales. Australian Journal of Experimental Agriculture 30: 103-108.

Ash, G.J., Brown, J.S. & Rees, R.G. 1991. Prediction of severity of stripe rust on wheat in Australia using regional weather data. Plant Protection Quarterly 6: 43-44.

Aslam, M. Fischbeck, G. 1993. Development of stripe rust in wheat cultivar mixtures. Journal of Agronomy and Crop Science 171: 49-54.

Badebo, A., Stubbs, R.W., Van Ginkel, M. & Gebeyehu, G. 1990. Identification of resistance genes to *Puccinia striiformis* in seedlings of Ethiopian and CIMMYT bread wheat varieties and lines. Netherlands Journal of Plant Pathology 96: 199-210.

Bariana, H.S. & McIntosh, R.A. 1993. Cytogenetic studies in wheat. XV. Location of rust resistance genes in VPM1 and their genetic linkage with other disease resistance genes in chromosome 2A. Genome 36: 476-482.

Bariana, H.S. & McIntosh, R.A. 1995. Genetics of adult-plant stripe rust resistance in four Australian wheats and the French cultivar "Hybride-de-Bersee". Plant Breeding 114: 485-491.

Barnard, J.E. 1999. Adult-plant resistance to *Puccinia recondita* f. sp. *tritici* in a collection of wild *Triticum* species. M.Sc. Agric Thesis, University of the Orange Free State, Bloemfontein, South Africa.

Bayles, R.A., Channell, M.H. & Stigwood, P.L. 1989. New races of *Puccinia striiformis* in the United Kingdom in 1988. Cereal Rusts and Powdery Mildews Bulletin 17: 20-23.

Bender, C.M. 1995. The expression of leaf rust resistance in wheat lines containing *Lr12* and *Lr13*. M.Sc. Thesis, University of the Orange Free State, Bloemfontein, South Africa.

Beresford, R.M. 1982. Stripe rust (*Puccinia striiformis*), a new disease of wheat in New Zealand. Cereal Rusts Bulletin 10: 35-41.

Bever, W.M. 1937. Influence of stripe rust on growth, water economy, and yield of wheat and barley. Journal of Agricultural Research 54: 375-385.

Brahma, R.N., Sivasamy, M. & Saikia, A. 1999: Transfer of alien genes *Lr9*, *Lr24* and *Lr28* to bread wheat cultivars susceptible to leaf rust. Wheat Information Service 88: 21-26.

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

Broers, L.H.M. 1993. Breeding for partial resistance in wheat to stripe rust. In: T. Jacobs & J.E. Parlevliet (eds.), Durability of Disease Resistance, pp. 179-183. Kluwer Academic Publishers, Dordrecht, The Netherlands.

Broers, L.H.M. 1997. Components of quantitative resistance to yellow rust in ten spring wheat cultivars and their relations with field assessments. Euphytica 96: 215-223.

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

Broers, L.H.M. & 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.

Broers, L.H.M., Cuesta Subias, X. & López-Atilano, R.M. 1996. Field assessment of quantitative resistance to yellow rust in ten spring wheat cultivars. Euphytica 90: 9-16.

Brown, J.F. & Sharp, E.L. 1969. Interactions of minor host genes for resistance to *Puccinia striiformis* with changing temperature regimes. Phytopathology 59: 999-1001.

Campbell, C.L. & Madden, L.V. 1990. Introduction to plant disease epidemiology. John Wiley & Sons, New York.

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

Chen, X.M. & Line, R.F. 1995a. Gene action in wheat cultivars for durable, high-temperature, adult-plant resistance and interaction with race-specific, seedling resistance to *Puccinia striiformis*. Phytopathology 85: 567-572.

Chen, X.M. & Line, R.F. 1995b. Gene number and heritability of wheat cultivars with durable, high-temperature, adult-plant (HTAP) resistance and interaction of HTAP and race-specific seedling resistance to *Puccinia striiformis*. Phytopathology 85: 573-578.

Chen, X.M. & Line, R.F. 2000. Molecular mapping of quantitative trait loci for durable resistance to wheat stripe rust. Phytopathology 90: 14 (Abstr.).

Chen, X.M., Line, R.F. & Leung, H. 1993. Relationship between virulence variation and DNA polymorphism in *Puccinia striiformis*. Phytopathology 83: 1489-1497.

Chen, X.M., Line, R.F. & Jones, S.S. 1994. Cromosomal location of genes for resistance to *Puccinia striiformis* in winter wheat cultivars Druchamp, Stephens, and Yamhill. Phytopathology 84: 1116.

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

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

Chen, X.M., Jones, S.S. & 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.

Chester, K.S. 1946. The nature and prevention of the cereal rusts as exemplified in the leaf rust of wheat. Chronica Botanica Publishing Co., Waltham, Massachusetts. 269 pp.

Chilosi, G. & Corazza, L. 1990. Occurrence and epidemics of yellow rust of wheat in Italy. Cereal Rusts and Powdery Mildews Bulletin 18: 1-9.

Coakley, S.M. & Line, R.F. 1981. Quantitative relationships between climatic variables and stripe rust epidemics on winter wheat. Phytopathology 71: 461-467.

Coakley, S.M., Scherm, H. & Chakraborty, S. 1999. Climate change and plant disease managenent. Annual Review of Phytopathology 37: 399-426.

Conner, R.L. & Kuzyk, A.D. 1988. Effectiveness of fungicides in controlling stripe rust, leaf rust, and black point in soft white spring wheat. Canadian Journal of Plant Pathology 10: 321-326.

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

Cromey, M.G. 1989a. Infection and control of stripe rust in wheat spikes. New Zealand Journal of Crops and Horticultural Science 17: 159-164.

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

Daamen, R.A. , Wijnands, F.G. & Van der Vliet, G. 1989. Epidemics of diseases and pests of winter wheat at different levels of agrochemical input. Journal of Phytopathology 125: 305-319.

Danial, D.L. 1994. Aspects of durable resistance in wheat to stripe rust. Ph.D Thesis. Wageningen. 143pp.

Danial, D.L. 1993. Is partial resistance a suitable approach against stripe rust in wheat. In: T. Jacobs & J.E. Parlevliet (eds.), Durability of Disease Resistance, pp. 179-183. Kluwer Academic Publishers, Dordrecht, The Netherlands.

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

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

Danial, D.L. & 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.

Dannenberg, M.D., Eversmeyer, M.D. & Kramer, C.L. 1989: Effect of timing of foliar fungicides on wheat disease control and yield increases. Plant Disease 73: 227-229.

Dennis, J.I. 1987. Effect of high temperatures on survival and development of *Puccinia striiformis* on wheat. Transactions of the British Mycological Society 88: 91-96.

Dennis, J.I. & Brown, J.S. 1986. Summer survival of *Puccinia striiformis* f. sp. *tritici* in Victoria, Australia. Australian Plant Pathology 15: 57-60.

De Vallavieille-Pope, C. & Line, R.F. 1990. Virulence of North American and European pathotypes 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. & 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.

De Vallavieille-Pope, C., Huber, L., Leconte, M. & Bethenod, O 2000. Using controlled and natural conditions to assess infection efficiency of *Puccinia striiformis* and *P. triticina* on wheat. Acta Phytopathologica et Entomologica Hungarica 35: 295-298.

Dubin, H.J., Johnson, R. & Stubbs, R.W. 1989. Postulated genes for resistance to stripe rust in selected CIMMYT and related wheats. Plant Disease 73: 472-475.

Dyck, P.L. 1991. Genetics of adult-plant leaf rust resistance in "Chinese Spring" and "Sturdy" wheats. Crop Science 31: 309-311.

Ellison, P.J. & 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.

Eversmeyer, M.G. & Kramer, C.L. 1996: Modeling winter and early spring survival of *Puccinia recondita* in wheat nurseries during 1980 to 1993. Plant Disease 80: 490-493.

Everts, K.L. & Leath, S. 1993. Effects of triadimenol seed treatment and timing of foliar fungicide applications on onset and extent of powdery mildew and leaf rust epidemics. Phytopathology 83: 557-562.

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

Friebe, B., Jiang, J., Raupp, W.J., McIntosh, R.A. & Gill, B.S. 1996. Characterization of wheat-alien translocations conferring resistance to diseases and pests: current status. Euphytica 91: 59-87.

Gaunt, R.E. & Cole, M.J. 1991. An analysis of yield reduction caused by stripe rust in Rongotea wheat. Australian Journal of Agricultural Research 42: 45-52.

Geagea, L., Huber, L. & Sache, I. 1997. Removal of urediniospores of brown (*Puccinia recondita* f. sp. *tritici*) and yellow (*P. striiformis*) rusts of wheat from infected leaves submitted to a mechanical stress. European Journal of Plant Pathology 103: 785-793.

Geagea, L., Huber, L. & Sache, I. 1999. Dry-dispersal and rain-splash of brown (*Puccinia recondita* f. sp. *tritici*) and yellow (*P. striiformis*) rust spores from infected wheat leaves exposed to simulated raindrops. Plant Pathology 48: 472-482.

Gerechter-Amitai, Z.K. & Grama, A. 1973. Inheritance of resistance to stripe rust (*Puccinia striiformis*) in crosses between wild emmer (*Triticum dicoccoides*)

and cultivated tetraploid and hexaploid wheats. I. *Triticum durum*. Euphytica 23: 387-392.

Gerechter-Amitai, Z.K. & Stubbs, R.W. 1970. A valuable source of stripe rust resistance in Israeli populations of wild emmer, *Triticum dicoccoides* Koern. Euphytica 19: 12-21.

Gerechter-Amitai, Z.K., van Silfhout, C.H., Grama, A. & Kleitman, F. 1989. *Yr15*- a new gene for resistance to *Puccinia striiformis* in *Triticum dicoccoides* sel. G-25. Euphytica 43: 187-190.

Grama, A, Gerechter-Amitai, Z.K. & Van Silfhout, C.H. 1984. Additive gene action for resistance to *Puccinia striiformis* f. sp. *tritici* in *Triticum dicoccoides*. Euphytica 33: 281-287.

Holmes, R.J. & Dennis, J.I. 1985. Accessory hosts of wheat stripe rust in Victoria, Australia. Transactions of the British Mycological Society 85: 159-160.

Huber, D.M. 1980. The role of mineral nutrition in defence. Plant Disease an Advanced Treatise. Volume V. How Plants Defend Themselves. Eds. J.G. Horsfall and E.B. Cowling, Academic Press. N.Y., N.Y. pp. 381-406.

Huber, D.M. & Watson, R.D. 1974. Nitrogen form and plant disease. Annual Review of Phytopathology 12: 139-165.

Hyde, P.M. & 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.

Ireta, M.J. & Gilchrist, S.L. 1994. *Fusarium* head scab of wheat (*Fusarium graminearum* Schwabe). Wheat Special Report No. 21b. Mexico, D.F.: Cimmyt.

Irshad, M., Khan, M.A. & Hussain, M. 1999. Determination of slow rusting response of wheat genotypes to leaf and stripe rust epidemics. Pakistan Journal of Phytopathology 11: 87-92.

Jacobs, A.S. 1996. Histopathological expression and inheritance of wheat leaf rust resistance derived from *Triticum monococcum*. M.Sc. Agric Thesis, University of the Orange Free State, Bloemfontein, South Africa.

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

Johnson, R. 1981. Durable disease resistance. In: J.F. Jenkyn & R.T. Plumb (eds.), Strategies for the Control of Cereal Disease. pp. 55-63.

Johnson, R. 1984. A critical analysis of durable resistance. Annual Review of Phytopathology 22: 309-330.

Johnson, R. 1988. Durable resistance to yellow (stripe) rust in wheat and its implications 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, D.F., 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. & Knott, D.R. 1992. Specificity in gene-for-gene interactions between plants and pathogens. Plant Pathology 41: 1-4.

Johnson, R. & Law, C.N. 1975. Genetic control of durable resistance to yellow rust (*Puccinia striiformis*) in the wheat cultivar Hybride de Bersee. Annals of Applied Biology 81: 385-391.

Johnson, R. & Lovell, N.K. 1994. Genetics of resistance of wheat to barley-attacking races of *Puccinia striiformis*. Cereal Rusts and Powdery Mildews Bulletin 22: 32-40.

Johnson, R., Stubbs, R.W., Fuchs, E. & Chamberlain, N.H. 1972. Nomenclature for physiologic races of *Puccinia striiformis* infecting wheat. Transactions of the British Mycological Society 58: 475-480.

Jørgensen, L.N. & Nielsen, B.J. 1994. Control of yellow rust (*Puccinia striiformis*) on winter wheat by ergosterol inhibitors at full and reduced dosages. Crop Protection 13: 323-330.

Kalappanavar, I.K. & Patil, N.K. 1997: Field evaluation of fungicides against leaf rust of wheat. Journal of Maharashtra Agricultural Universities 22: 335 (Abstr.).

Khan, M.A. & Trevathan, L.E. 1997: Effect of protectant and eradicant fungicide application on leaf rust development on winter wheat and extent of yield and

kernel weight enhancement. Pakistan Journal of Phytopathology 9: 50-63 (Abstr.).

Khan, M.A. & Trevathan, L.E. & Robbins, J.T. 1999: Effect of fungicides on leaf rust, yield and kernel weight of three winter wheat cultivars in Mississippi. Plant Protection Quaterly 14: 147-150 (Abstr.).

Kloppers, F.J. 1994. Characterisation of resistance conferred by selected *Lr* genes with emphasis on histopathology, leaf rust development and associated quality attributes in wheat. Ph.D. Thesis, University of the Orange Free State, Bloemfontein, South Africa.

Krupinsky, J.M. & Sharp, E.L. 1979. Reselection for improved resistance of wheat to stripe rust. Phytopathology 69: 400-404.

Kumar, J., Nayar, S.K., Prashar, M., Bhardwaj, S.C. & Bhatnagar, R. 1993. Virulence survey of *Puccinia striiformis* in India during 1990-1992. Cereal Rusts and Powdery Mildews Bulletin 21: 17-24.

Kumar, J., Singh, R.P., Nagarajam, S. & Sharma, A.K. 1999. Further evidence on the usefulness of *Lr34/Yr18* gene in developing adult plant resistant wheat genotypes. Wheat Information Service 89: 23-29.

Lewellen, R.T. & Sharp, E.L. 1967. Inheritance of minor gene combinations in wheat to *Puccinia striiformis* at two temperature profiles. Canadian Journal of Botany 46: 21-25.

Line, R.F. 1972. Patterns of pathogenicity of *Puccinia striiformis* in the United States. Proceedings of the European and Mediterranean Cereal rusts Conference 3: 181-185.

Line, R.F. 1976. Factors contributing to an epidemic of stripe rust on wheat in the Sacramento Valley of California in 1974. Plant Disease Reporter 60: 312-316.

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

Line, R.F. & Chen, X. 1995. Successes in breeding for and managing durable resistance to wheat rusts. Plant Disease 79: 1254-1255.

Line, R.F. & Chen, X. 1996. Wheat and barley stripe rust in North America. Proceedings of the 9th European and Mediterranean Cereal Rusts and Powdery Mildew Conference. Lunteren, The Netherlands.

Line, R.F., Sharp, E.L. & Powelson, R.L. 1970. A system for differentiating races of *Puccinia striiformis* in the United States. Plant Disease Reporter 54: 992-994.

Line, R.F., Qayoum, A. & Chen, X.M. 1988. Evolution and distribution of *Puccinia striiformis* in North America and inheritance of stripe rust resistance in differential cultivars. Proceedings of the European Cereal Rusts Conference 7: 107-112.

Louwers, J.M., Van Silfhout, C.H. & Stubbs, R.W. 1992. Race analysis of stripe rust in wheat in developing countries. Report 1990-1992. IPO-DLO Report 92-11. 23pp.

Luig, N.H. 1985. Epidemiology in Australia and New Zealand. Pp. 301-328 in A.P. Roelfs and W.R. Bushnell, eds. Cereal Rusts Vol. II. Diseases, Distribution, Epidemiology, and Control, Academic Press, Orlando.

Lupton, F.G.H. & Macer, R.C.F. 1962. Inheritance of resistance to yellow rust (*Puccinia glumarum* Erikss. & Henn.) in seven varieties of wheat. Transactions of the British Mycological Society 45: 21-45.

Ma, H. & Singh, R.P. 1996a. Contribution of adult-plant resistance gene *Yr18* in protecting wheat from yellow rust. Plant Disease 80: 66-69.

Ma, H. & Singh, R.P. 1996b. Expression of adult resistance to stripe rust at different growth stages of wheat. Plant Disease 80: 375-379.

Ma, H., Singh, R.P. & Mujeeb-Kazi, A. 1995. Resistance to stripe rust in *Triticum turgidum*, *T. tauschii* and their synthetic hexaploids. Euphytica 82: 117-124.

Ma, H., Singh, R.P. & Abdalla, O. 1997. Resistance to stripe rust in five durum wheat cultivars. Plant Disease 81: 27-30.

Manisterski, J., Eyal, Z., Ben-Yehuda, P. & Kosman, E. 2000. Comparative analysis of indices in the study of virulence diversity between and within populations of *Puccinia recondita* f. sp. *tritici* in Israel. Phytopathology 90: 601-607.

Mardoukhi, V. & Torabi, M. 1998. Yellow rust on wild grasses in Iran. Seed and Plant 14: 66-73.

Mares, D.J. & Cousen, S. 1977. The interaction of yellow rust (*Puccinia striiformis*) with winter wheat cultivars showing adult plant resistance: macroscopic and microscopic events associated with the resistant reaction. Physiological Plant Pathology 10: 257-274.

Marshall, D. & Sutton, R.L. 1995. Epidemiology of stripe rust, virulence of *Puccinia striiformis* f. sp. *hordei*, and yield loss in barley. Plant Disease 79: 732-737.

Mcgregor, A.J. & Manners, J.G. 1985. The effect of temperature and light intensity on growth and sporulation of *Puccinia striiformis* on wheat. Plant Pathology 34: 263-271.

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

McIntosh, R.A. & Lagudah, E.S. 2000. Cytogenetical studies in wheat. XVIII. Gene *Yr24* for resistance to stripe rust. Plant Breeding 119: 81-83.

McIntosh, R.A. & Wellings, C.R. 1986. The Danial Mcalpine memorial lecture. Wheat rust resistance- the continuing challenge. Australian Plant Pathology 15: 1-8.

McIntosh, R.A., Wellings, C.R. & Park, R.F. 1995. Wheat rusts: An atlas of resistance genes. Kluwer, Dordrecht. 200pp.

McIntosh, S.R., Silk, J. & The, T.T. 1996. Cytogenetic studies in wheat XVII. Monosomic analysis and linkage relationships of gene *Yr15* for resistance to stripe rust. Euphytica 89: 395-399.

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

Milus, E.A. & Line, R.F. 1986a. Gene action for inheritance of durable, high-temperature, adult-plant resistance to stripe rust in wheat. Phytopathology 76: 435-441.

Milus, E.A. & Line, R.F. 1986b. Number of genes controlling high-temperature, adult-plant resistance to stripe rust in wheat. Phytopathology 76: 93-96.

Moschini, R.C. & Pérez, B.A. 1999. Predicting wheat leaf rust severity using planting date, genetic resistance, and weather variables. Plant Disease 83: 381-384.

Mundt, C.C., Brophy, L.S. & Kolar, S.C. 1996. Effect of genotype unit number and spatial arrangement on severity of yellow rust in wheat cultivar mixtures. Plant Pathology 45: 215-222.

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

Murray, G.M., Ellison, P.J. & Watson, A. 1995. Effects of stripe rust on the wheat plant. Australasian Plant Pathology 24: 261-270.

Nazari, K., Torabi, M. & Mardoukhi, V. 1996. Wild grass species as oversummering hosts of *Puccinia striiformis* f. sp. *tritici* in Iran. Cereal Rusts and Powdery Mildews Bulletin 24: 105-107.

Nel, A., Krause, M., Ramautar, N. & van Zyl, K. 1999. A guide for the control of plant diseases. National Department of Agriculture, Pretoria. 1st edition. 122pp.

Newton, A.C., Caten, C.E. & Johnson, R. 1985. Variation for isozymes and double-stranded RNA among isolates of *Puccinia striiformis* and two other cereal rusts. Plant Pathology 34: 235-247.

O'Brien, L., Brown, J.S., Young, R.M. & Pascoe, I. 1980. Occurrence and distribution of wheat stripe rust in Victoria and susceptibility of commercial wheat cultivars. Australian Plant Pathology 9: 14-22.

Ortelli, S., Giezendanner, U., Nösberger, J., Winzeler, H., Keller, B. & Winzeler, M. 1996. Effect of the *Lr9* resistance gene on pathogenesis of the wheat leaf rust fungus. Plant Disease 80: 14-18.

Park, R.F. 1990. The role of temperature and rainfall in the epidemiology of *Puccinia striiformis* f. sp. *tritici* in the summer rainfall area of eastern Australia. Plant Pathology 39: 416-423.

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

Park, R.F., Rees, R.G & Platz, G.J. 1988. Some effects of stripe rust infection in wheats with adult-plant resistance. Australian Journal of Agricultural Research 39: 555-62.

Parlevliet, J.E. 1979. Components of resistance that reduce the rate of epidemic development. Annual Review of Phytopathology 17: 203-222.

Parlevliet, J.E. & Van Ommeren, A. 1984. Interplot interference and the assessment of barley cultivars for partial resistance to leaf rust, *Puccinia hordei.* Euphytica 33: 685-697.

Paysour, R.E. & Fry, W.E. 1983. Interplot interferences: A model for planning field experiments with airily disseminated pathogens. Phytopathology 73: 1014-1020.

Peng, J.H., Fahima, T., Roder, M.S., Li, J.C., Dahan, A., Grama, A., Ronin, J.I., Korol, A.B. & Nevo, E. 1999. Microsatellite tagging of the stripe-rust resistance gene *YrH52* derived from wild emmer wheat, *Trititcum dicoccoides*, and suggestive negative crossover interference on chromosome 1B. Theoretical and Applied Genetics 98: 862-872.

Perwaiz, M.S. & Johnson, R. 1986. Genes for resistance to yellow rust in seedlings of wheat cultivars from Pakistan tested with British isolates of *Puccinia striiformis*. Plant Breeding 97: 289-296.

Pradhanang, P.M. & Sthapit, B.R. 1995. Effect of cultivar mixtures on yellow rust incidence and grain yield of barley in the hills of Nepal. Crop Protection 14: 331-334.

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

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

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

Rapilly, F. 1979. Yellow rust epidemiology. Annual Review of Phytopathology 17: 59-79.

Rathmell, W.G. & Skidmore, A.M. 1982. Recent advances in the chemical control of cereal rust diseases. Outlook on Agriculture 11: 37-43.

Reinhold, M., Sharp, E.L. & Gerechter-Amitai, Z.K. 1983. Transfer of additive "minor-effect" genes for resistance to *Puccinia striiformis* from *Triticum dicoccoides* into *Triticum durum* and *Triticum aestivum*. Canadian Journal of Botany 61: 2702-2708.

Robert, O., Abelard, C. & Dedryver, F. 1999. Identification of molecular markers for the detection of the yellow rust resistance gene *Yr17* in wheat. Molecular Breeding 5: 167-175.

Roelfs, A.P. & Huerta-Espino, J. 1994. Seedling resistance in *Hordeum* to barley stripe rust from Texas. Plant Disease 78: 1046-1049.

Roelfs, A.P., Singh, R.P. & Saari, E.E. 1992. Rust Diseases of Wheat: Concepts and methods of disease management. Mexico, D.F.: Cimmyt. 81pp.

Rossing, W.A.H., Daamen, R.A. & Hendriks, E.M.T. 1994. Framework to support decisions on chemical pest control under uncertainty, applied to aphids and brown rust in winter wheat. Crop protection 13: 25-34.

Russell, G.E. 1978. Some effects of applied sodium and potassium chloride on yellow rust in winter wheat. Annals of Applied Biology 90: 163-168.

Samborski, D.J. 1985. Wheat leaf rust. Pages 39-59 in: The Cereal Rusts, Vol. II. A.P. Roelfs and W.R. Bushnell, eds. Academic press, Inc., New York.

Sayre, K. D., Singh, R.P., Huerta-Espino, J. & Rajaram, S. 1998. Genetic progress in reducing losses to leaf rust in CIMMYT-Derived Mexican wheat cultivars. Crop Science 38: 654-659.

Shan, W.-X., Chen, S.-Y., Kang, Z.-S., Wu, L.-R. & Li, Z.-Q. 1999. Genetic diversity in *Puccinia striiformis* Westend. f. sp. *tritici* revealed by pathogen genome-specific repetitive sequence. Canadian Journal of Botany 76: 587-595.

Shaner, G. & Finney, R. 1980. New sources of slow leaf rusting resistance in wheat. Phytopathology 70: 1183-1186.

Shaner, G. & Powelson, R.L. 1973. The oversummering and dispersal of inoculum of *Puccinia striiformis* in Oregon. Phytopathology 63: 13-17.

Shang, H. & Shang, H.S. 1998. High temperature resistance of wheat to stripe rust. Scientia Agricultura Sinica 31: 46-50.

Sharp, E.L. 1962. Effects of preinoculation and postinoculation host temperatures on infection of wheat seedlings by *Puccinia striiformis*. Phytopathology 52. 751-752. (Abstr.)

Sharp, E.L. 1965. Prepenetration and postpenetration environment and development of *Puccinia striiformis* on wheat. Phytopathology 55: 198-203.

Sharp, E.L. & Fuchs, E. 1982. Additive genes in wheat for resistance to stripe (yellow) rust (*Puccinia striiformis* Westend.). Crop Protection 2: 181-189.

Sharp, E.L. & Hehn, E.R. 1963. Overwintering of stripe rust in winter wheat in Montana. Phytopathology 53: 1239-1240.

Sharp, E.L., Sally, B.K. & Taylor, G.A. 1976. Incorporation of additive genes for stripe rust resistance in winter wheat. Phytopathology 66: 794-797.

Singh, D.P. 1999. Yield losses due to brown rust in two popular cultivars of wheat. Plant Disease Research 14: 60-62 (Abstr.).

Singh, M.H. & Abdalla, O. 1997. Resistance to stripe rust in five durum wheat cultivars. Plant Disease 81: 27-30.

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

Singh, R.P. 1992b. 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. 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, R.P. & Huerta-Espino, J. 1997. Effect of leaf rust resistance gene *Lr34* on grain yield and agronomic traits of spring wheat. Crop Science 37: 390-395.

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

Smale, M., Singh, R.P., Sayre, K., Pingali, P., Rajaram, S. & Dubin, H.J. 1998. Estimating the economic impact of breeding nonspecific resistance to leaf rust in modern bread wheats. Plant Disease 82: 1055-1061.

Stubbs, R.W. 1967. Influence of light intensity on the reactions of wheat and barley seedlings to *Puccinia striiformis*. Phytopathology 57: 615-617.

Stubbs, R.W. 1977. Observations on horizontal resistance to yellow rust (*Puccinia striiformis* f. sp. *tritici*). Cereal Rusts Bulletin 5: 27-32.

Stubbs, R.W. 1985. Stripe rust. Pages 61-101 in: The Cereal Rusts II: Diseases, Distribution, Epidemiology and Control. Roelfs A.P. and Bushnell, W.R. eds. Academic Press, New York.

Sun, G.L., Fahima, T., Korol, A.B., Turpeinen, T., Grama, A., Ronin, Y.I. & Nevo, E. 1997. Identification of molecular markers linked to the *Yr15* stripe rust resistance gene of wheat originated in wild emmer wheat, *Triticum dicoccoides*. Theoretical and Applied Genetics 95: 622-628.

Sundin, D.R., Bockus, W.W. & Eversmeyer, M.G. 1999. Triazole seed treatments suppress spore production by *Puccinia recondita*, *Septoria tritici*, and *Stagonospora nodorum* from wheat leaves. Plant Disease 83: 328-332.

Teng, P.S. & Guant, R.E. 1980. Modelling systems of disease and yield loss in cereals. Agricultural Systems 6: 131-154.

Van Dijk, P., Parlevliet, J.E., Kema, G.H.J., Zeven, A.C. & Stubbs, R.W. 1988. Characterization of the durable resistance to yellow rust in old winter wheat cultivars in the Netherlands. Euphytica 38: 149-158.

Van Silfhout, C.H., Grama, A., Gerechter-Amitai, Z.K. & Kleitman, F. 1989. Resistance to yellow rust in *Triticum dicoccoides*. I. Crosses with susceptible *Triticum durum*. Netherlands Journal of Plant Pathology 95: 73-78.

Viljanen-Rollinson, S.L.H. & Cromey, M.G. 2000. Effects of the gene *Yr18* on yield and disease severity of wheat under different stripe rust epidemics. Acta Phytopathologica et Entomologica Hungarica 35: 3-9.

Wagoire, W.W., Stølen, O. & Ortiz, R. 1998. Is there a 'cost' for wheat cultivars with genes for resistance to yellow rust caused by *Puccinia striiformis*. Crop Protection 17: 337-340.

Wallwork, H. & Johnson, R. 1984. Transgressive segregation for resistance to stripe rust in wheat. Euphytica 33: 123-132.

Wellings, C.R., McIntosh, R.A. & Hussain, M. 1988. A new source of resistance to *Puccinia striiformis* f. sp. *tritici* in spring wheats (*Triticum aestivum*). Plant Breeding 100: 88-96.

Wellings, C.R. & McIntosh, R.A. 1990. *Puccinia striiformis* f. sp. *tritici* in Australia: pathogenic changes during the first 10 years. Plant Pathology 39: 316-325.

Wellings, C.R., McIntosh, R.A. & Walker, J. 1987. *Puccinia striiformis* f. sp. *tritici* in Eastern Australia-possible means of entry and implications for plant quarantine. Plant Pathology 36: 239-241.

Wiese, M.V. 1987. Compendium of Wheat Diseases, Second Edition. St Paul, Minnesota: APS Press.

Yildirim, A., Jones, S.S., Murray, T.D., Cox, T.S. & Line, R.F. 1995. Resistance to stripe rust and eyespot diseases of wheat in *Triticum tauschii*. Plant Disease 79: 1230-1236.

Yildirim, A., Jones, S.S., Murray, T.D. & Line, R.F. 2000. Evaluation of *Dasypyrum villosum* populations for resistance to cereal eyespot and stripe rust pathogens. Plant Disease 84: 40-44.

Zadoks, J.C. 1961. Stripe rust on wheat. Studies in epidemiology and physiologic specialization. Netherlands Journal of Plant Pathology 67: 69-76.

Zadoks, J.C. & Bouwman, J.J. 1985. Epidemiology in Europe. Pp 329-369 in The Cereal Rusts Vol. II; Diseases, Distribution, Epidemiology, and Control. A.P. Roelfs & W.R. Bushnell, eds. Academic Press, Orlando.

OCCURRENCE AND PATHOGENICITY OF *PUCCINIA STRIIFORMIS* f. sp. *TRITICI* IN SOUTH AFRICA

ABSTRACT

Stripe rust, caused by *Puccinia striiformis* Westend. f. sp. *tritici* Eriks., has become an endemic disease of wheat (*Triticum aestivum* L.) in South Africa after being observed for the first time near Moorreesburg, Western Cape during August 1996. Results of surveys conducted during 1996-1999 revealed that rainfed wheat produced in the Western Cape, Eastern Cape, and the eastern Free State, as well as irrigated wheat produced in KwaZulu-Natal and the Free State, are most likely to be affected by stripe rust epidemics. Pathotypes (pts.) detected were 6E16A- with virulence to *Yr2*, *6*, *7*, *8*, *11*, *14*, *17* and *Yr19* and pt. 6E22A- with added virulence to *Yr25*. The occurrence of pt. 6E22A- is currently restricted to KwaZulu-Natal and the Free State. Stripe rust isolates found on *Hordeum murinum* L. in the Western Cape were identified as pt. 6E16A- whereas both pts. 6E16A- and 6E22A- were collected from *Bromus catharticus* Vahl (=*B. unioloides* H.B.K.) in the eastern Free Sate. Urediospores from infections similar to stripe rust found on the grass species *Dactylis glomerata* L. (Eastern Cape), *Poa pratensis* L. (=*P. bidentata* Stapf) (Western Cape), and *P. annua* and *P. triviales* L. (eastern Free State) failed to infect Morocco seedlings in the glasshouse. The possible role that grass spp. may play in the over-summering of the stripe rust pathogen has not yet been established. However, stripe rust infections have been found on summer-sown wheat in the south Western Cape during 1998, volunteer wheat growing in the summer and autumn months in the eastern Free State from 1998 to 2000, and on summer-sown wheat in Lesotho.

INTRODUCTION

Stripe rust, caused by the obligate parasite *Puccinia striiformis* Westend. f. sp. *tritici* Eriks., is one of the most important diseases of bread wheat (*Triticum aestivum* L.) (Stubbs, 1985). This *forma speciales* infects numerous wheat cultivars as well as a few barley varieties and certain grass species (Stubbs, 1985). Historically, stripe rust has been more important in areas with cool and wet environmental conditions and, therefore, occurs regularly in northern Europe, the Mediterranean region, Middle East, north western USA, Australia, East African highlands, China, the Indian subcontinent, New Zealand and the Andean regions of South America (Danial, 1994). Stripe rust is also important in more tropical areas of higher altitude such as North African countries, the Himalayan foothills of India and Pakistan, and Mexico (McIntosh, 1980).

In comparison with leaf rust (*P. triticina* (Eriks.) =*P.* recondita f. sp. tritici) and stem rust (*P. graminis* Pers. f. sp. *tritici*) pathogens of wheat, the global distribution of *P. striiformis* f. sp. *tritici* has been more restricted. Stripe rust did not occur in Australia until 1979 (O'Brien *et al.*, 1980), and was not found in New Zealand until 1980 (Beresford, 1982). In central Africa stripe rust was first reported in northern Zambia in 1958 (Angus, 1965). According to Bonthuis (1978) stripe rust is rarely observed south of the 15 S latitude, whereas the disease becomes progressively more important towards the equator.

The sexual stage of *P. striiformis* has not been encountered and so far no alternate hosts have been found (Stubbs, 1985). Numerous, highly specified pathotypes of *P. striiformis* occur (Johnson, 1992a), and are probably formed by mutation and somatic recombination (Stubbs, 1985). Selection for virulence by growing hosts with pathotype-specific resistance thus plays a major role in determining the pathotype structure of the pathogen. Shan *et al.* (1999) used DNA fingerprinting to examine genetic variation among 160 stripe rust isolates. They found 97 phenotypes and phenotypic diversity varied among different regions in China. Chen *et al.* (1993) found a low association between virulence and RAPD patterns of *P. striiformis* f. sp. *tritici* isolates which suggested that DNA polymorphisms are independent of virulence.

The epidemic that occurred in the Western Cape during 1996 showed that stripe rust could potentially become the most important foliar disease of wheat in both the summer and winter rainfall areas of South Africa. The occurrence of crop losses and higher input costs due to the repeated application of fungicides necessitated the development of more affordable control strategies against this disease. Under local wheat growing conditions of predominantly dry-land wheat production under uncertain rainfall, low average yields of 2.05 t/ha (Anonymous, 2000), breeding for host resistance represents the most cost-effective means of controlling stripe rust. Effective breeding strategies are, however, dependent on an understanding of genetic variation in both pathogen and host. Of further importance in an effective disease control strategy is an understanding of the epidemiology of the pathogen. For stripe rust this includes the ability of the pathogen to survive through the non-crop season and the probability of the disease occurring in the different wheat growing areas of South Africa. The latter will have a limiting influence on the release of susceptible cultivars in areas of high risk and will be an important factor in cultivar choice by wheat producers.

The objectives of this research, conducted from August 1996 to February 2000, were (1) to determine the contribution of weather conditions on the successful establishment and subsequent epidemic outbreak of *P. striiformis* in the Western Cape during 1996, (2) to monitor the development, occurrence and distribution of the stripe rust pathogen in local wheat producing areas and to detect possible pathogenic changes, and (3) to determine the susceptibility of grass species to stripe rust infection.

MATERIALS AND METHODS

Disease surveys

Wheat fields and disease nurseries (rust trap nurseries) were surveyed periodically from 1996 to 1999 by researchers of the Small Grain Institute (SGI) to monitor the occurrence, development and distribution of stripe rust in South Africa. Disease nurseries, planted annually in all the important wheat production areas of South Africa, were visited at least once during the wheat season, as well as selected commercial

wheat fields in the different production areas. Disease nurseries included the two spreader lines McNair (stem and stripe rust susceptible) and Morocco (leaf, stem, and stripe rust susceptible), a stripe rust differential and supplemental set, several lines carrying different stem and leaf rust genes, a number of commercial wheat cultivars, and advanced breeding lines. Stripe rust severity was recorded on disease nursery entries and in commercial wheat fields in the different wheat producing areas. The latter data were used to map the occurrence, distribution and incidence of the stripe rust pathogen from 1996 to 1999.

A survey was conducted during February 1997 in the Western Cape to determine the role of volunteer wheat and wild grass spp. in the survival of the stripe rust pathogen during the hot summer months. Similar surveys were conducted in the south Western Cape during March 1998 and 1999 and in the eastern Free State during February to May in 1998 and 1999. The occurrence of stripe rust on summer-sown wheat in Lesotho was monitored from 1998 to 2000 and the susceptibility of wheat cultivars commonly grown in Lesotho was determined in both seedling and adult plant stages. Procedures followed for the seedling and adult plant evaluation of the Lesotho cultivars are as described under pathotype determination.

Pathotype determination

During field surveys leaves containing sporulating uredia were sampled from wheat fields, disease nurseries, and triticale (X *Triticosecale* Wittmack), placed in glassine bags, stored in a portable refrigerator (7°C), and transported to the SGI at Bethlehem. Furthermore, rust collections were sent to the SGI by co-workers responsible for the planting and maintenance of disease nurseries. Details were recorded of the date of collection, location, cultivar (if known) and rust severity. Urediospores were collected from the leaves using a cyclone collector, suspended in a mineral oil (Soltrol 170), and sprayed onto the primary leaves of seven-day-old seedlings, of the wheat cultivar Morocco. Morocco seedlings were grown in 10 cm diam. plastic pots filled with steam-sterilised soil, in a disease-free room kept at 22± 1°C. Upon emergence these seedlings were treated with 50 ml/pot of a $0.3gl^{-1}$ maleic hydrazide solution to retard plant growth and stimulate uredospore production. Inoculations were done in an

enclosed inoculating booth. The booth was rinsed with water between isolates to eliminate contamination. After drying for 2 h in an air-conditioned room, inoculated seedlings were placed in a dew chamber at 11± 1°C and >96% relative humidity for 30 h. Seedlings were then moved to a glasshouse cubicle where a day/night cycle of 16/8 h was maintained. Day light was supplemented with cool-white fluorescent tubes emitting photosynthetic active radiation of 120 $\mu E/m^2$. Day and night temperatures were kept at 17± 2°C. Morocco seedlings, inoculated with different stripe rust isolates, were placed in separate glass cages.

After 14 to 16 days urediospores of each isolate were inoculated onto the primary leaves of the World and European differentials, placed in the dew chamber, and then moved to a glasshouse cubicle as described above. The cultivars Clement (*Yr9*, decanery value 2^7=128) and *Triticum spelta album* (*Yr5*, decanery value 2^8=256) were added to the World set as proposed previously (Johnson *et al.*, 1972; Johnson & Taylor, 1976; Wellings & McIntosh, 1990). In addition the seedling reaction of the cultivar Wembley and 25 supplementary wheat lines was determined. Seeds of the differential lines and supplementary testers were kindly provided by C.R. Wellings, Plant Breeding Institute, Cobbitty, Australia. Rust reaction of seedlings in the differential cultivars and supplemental sets was assessed 14 to 16 days following inoculation on the primary leaves, using the 0 to 4 scale (Appendix 1) (McIntosh *et al.*, 1995). Pathotype classification was determined according to Johnson *et al.* (1972) and Wellings & McIntosh (1990). For adult plant evaluation in the field, differential cultivars and supplemental lines were planted in 1 m rows with 30 cm inter row spacing. Spreader rows consisting of a mixture of Morocco and McNair were planted perpendicular to the differentials. Disease assessments of the differential cultivars and supplemental lines were determined at different field localities using the modified Cobb scale for measuring rust severity (Peterson *et al.*, 1948) and infection type scale (McIntosh *et al.*, 1995) (Appendix 1).

Susceptibility of wild grass species

Stripe rust samples were collected from infected grasses during surveys and tested in the glasshouse for pathogenicity on Morocco seedlings. When infections occurred, procedures followed for pathotype determination were the same as described above. In addition, 17 grass spp. belonging to six genera were tested in the glasshouse for their susceptibility to an isolate of *P. striiformis* f. sp. *tritici* from wheat.

Influence of weather conditions

Data on temperature and precipitation collected by the ARC-Institute for Soil, Climate and Water in Pretoria were used to determine the influence of weather conditions on the successful establishment, spread, and subsequent epidemic outbreak of stripe rust in the Western Cape during 1996.

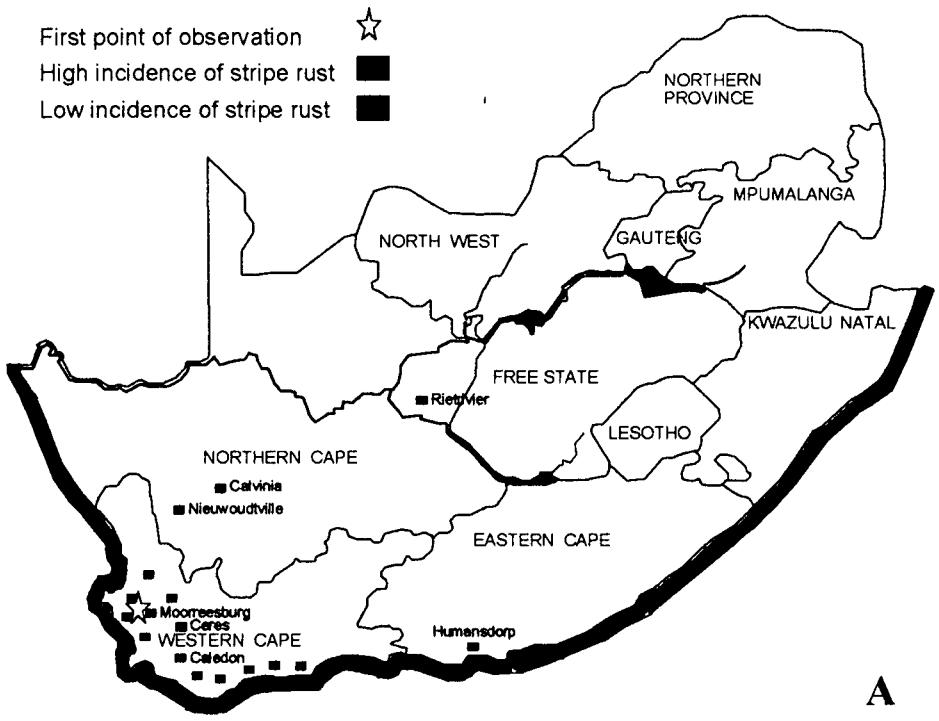
RESULTS

Disease surveys

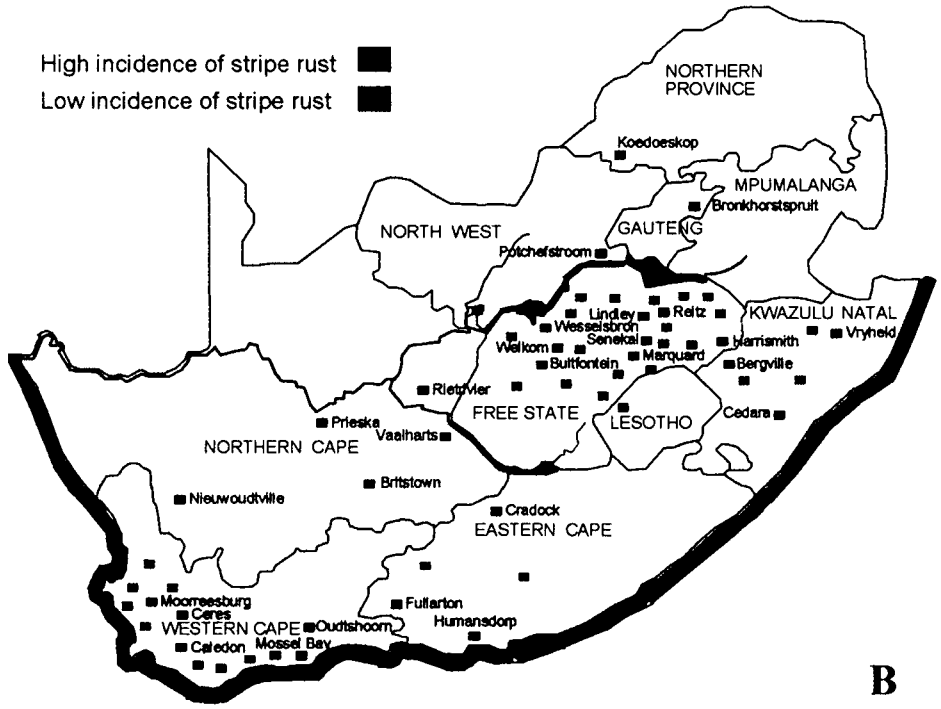
Outbreak, occurrence and distribution during 1996. Stripe rust was first observed on 9 August 1996 on the bread wheat cultivar Palmiet in the winter rainfall region near Moorreesburg in the Western Cape (Fig. 1A). Subsequent surveys showed, however, that at the time of detection, the disease was well established in wheat fields in the western and northern parts of the Western Cape Province as well as at Nieuwoudtville in the Northern Cape Province. The initial point and time of outbreak could, therefore, not be determined.

During 1996 stripe rust was most severe in the western and northern parts of the Western and Northern Cape Provinces where prolonged cool and wet conditions favoured epidemic development of the disease and necessitated extensive and often repeated applications of triazole fungicides. Despite these efforts head infection and destruction of foliage often resulted in significant losses in grain quantity and quality. Producers spent an estimated R28 million on fungicides to control the stripe rust epidemic during 1996 in the Western Cape Province.

Fig. 1. (A) Occurrence and incidence of *Puccinia striiformis* f. sp. *tritici* in South Africa during 1996 and (B) 1997.


First point of observation
High incidence of stripe rust
Low incidence of stripe rust
NORTHERN PROVINCE
MPUMALANGA
NORTH WEST
GAUTENG
KWAZULU NATAL
FREE STATE
Rietvlei
LESOTHO
NORTHERN CAPE
Calvinia
Nieuwoudtville
EASTERN CAPE
Moorreesburg
Ceres
WESTERN CAPE
Caledon
Humansdorp

A


High incidence of stripe rust
Low incidence of stripe rust
NORTHERN PROVINCE
Koedoeskop
MPUMALANGA
Bronkhorstspruit
NORTH WEST
GAUTENG
Potchefstroom
Lindley
Reitz
Wesselsbron
KWAZULU NATAL
Vryheid
Senekal
Harrismith
Welkom
Marquard
Bultfontein
Bergville
Rietvlei
FREE STATE
LESOTHO
Cedara
Prieska
Vaalharts
NORTHERN CAPE
Britstown
Nieuwoudtville
Cradock
EASTERN CAPE
Moorreesburg
Ceres
Fullarton
WESTERN CAPE
Oudtshoorn
Humansdorp
Caledon
Mossel Bay

B

The disease also spread to wheat producing areas in the southern parts of the Western Cape Province and the western and southern areas of the Eastern Cape Province. Due to hot and dry weather conditions in the latter areas the incidence of stripe rust was low and fungicide applications were restricted mostly to the districts of Caledon and Humansdorp. During November 1996 stripe rust was observed on irrigated wheat at Rietrivier in the summer rainfall area south of Kimberley, proving that inoculum had been disseminated to the interior of the country.

Occurrence and distribution during 1997. During the 1997 wheat season stripe rust was first observed (early June) near Mossel Bay in the southern and Moorreesburg in the western regions of the Western Cape (Fig. 1B). Unlike the 1996 season, environmental conditions were more favourable (wet and cold conditions until flowering) for stripe rust development in the southern areas of the Western Cape Province and the western and southern areas of the Eastern Cape. This necessitated the extensive use of fungicides to control stripe rust on susceptible cultivars. The low incidence of stripe rust in the western and northern parts of the Western Cape Province during the 1997 season may be attributed to the widespread cultivation of the stripe rust resistant cultivar SST57, estimated at >60% of the cultivated area. Unfavourable weather, i.e. dry and hot conditions for most of the season, and farmers spraying susceptible cultivars prophylacticly, contributed further to the low incidence of stripe rust.

Stripe rust was observed during August 1997 near Wesselsbron in the western Free State from where it spread to the rest of the province, and to KwaZulu-Natal, Gauteng, the North-West and Northern Province. Epidemic outbreaks of stripe rust reported during September on the spring cultivar Palmiet, grown under centre pivot irrigation near Bloemfontein, was the most southerly reported epidemic of the disease in the Free State. During October stripe rust occurred on irrigated wheat at Rietrivier, Vaalharts and Prieska in the Northern Cape Province. Higher day temperatures towards the end of October and early November, however, restricted rust development on irrigated wheat in the Northern Cape. Except for parts of the western, central, and eastern Free State, the incidence of stripe rust on dry-land wheat in the summer rainfall

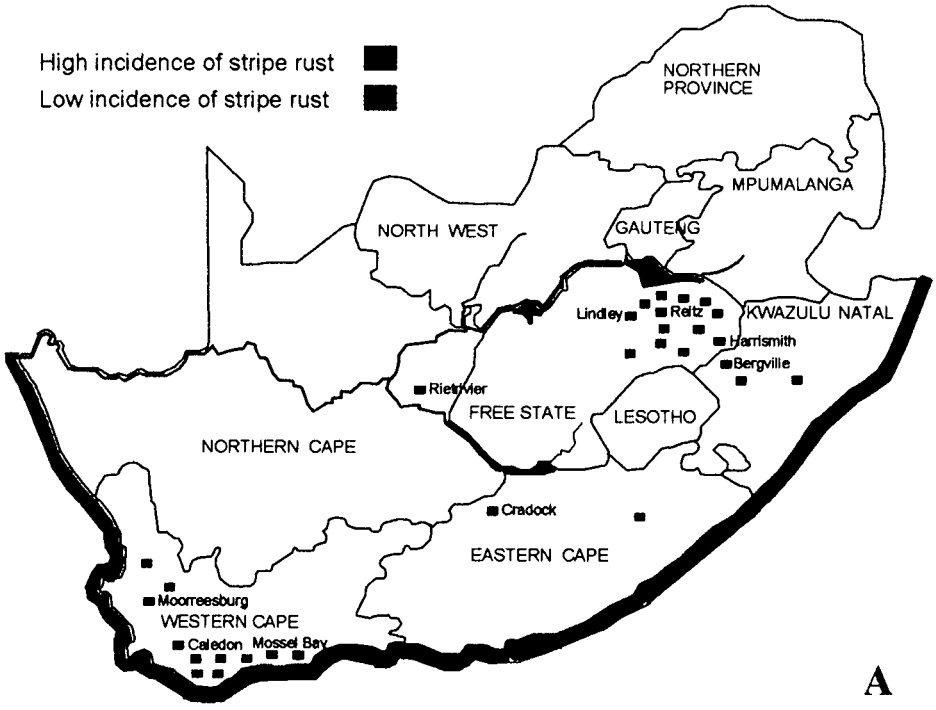
region was low due to dry and hot weather conditions. Producers in the eastern Free State spent an estimated R18 million to control stripe rust during 1997.

Occurrence and distribution during 1998. During the 1998 season stripe rust incidence was low in the Western and Eastern Cape Provinces (Fig. 2A). The first symptoms were observed during the last week of July near Swellendam in the south Western Cape. This low incidence could be attributed mainly to a shift to resistant cultivars, preventing the rapid build-up and spread of the disease. Furthermore, environmental conditions were not conducive for stripe rust development during most of the season. Only trace symptoms of stripe rust were observed near Cradock in the Eastern Cape and at Rietrivier in the Northern Cape.

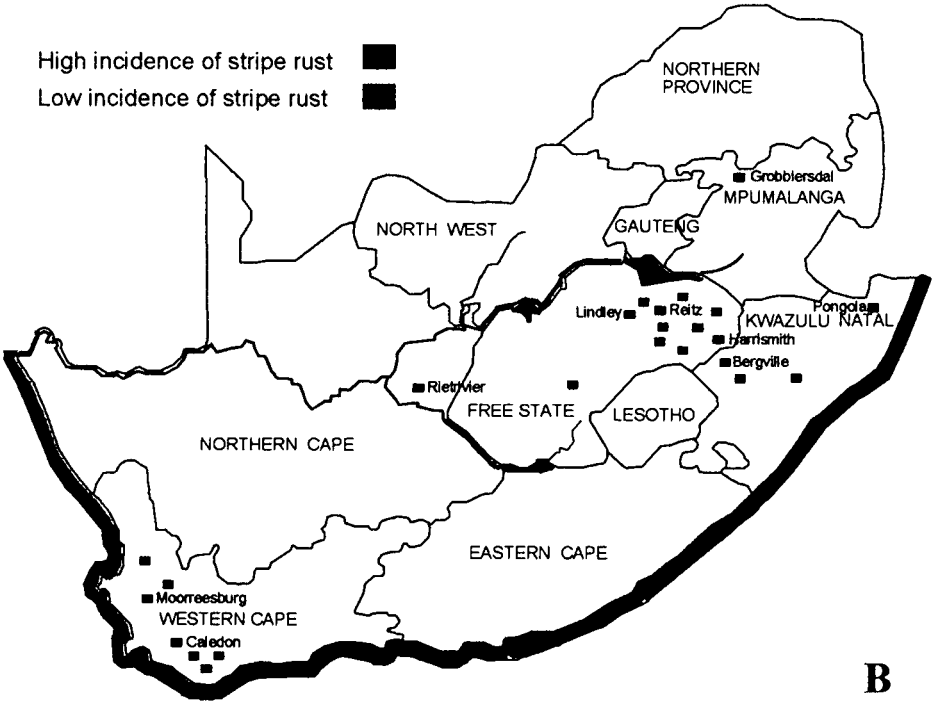
In the summer rainfall region stripe rust was observed during mid-September in KwaZulu-Natal and later near Vrede in the northeastern Free State. The development of a new stripe rust pathotype in the eastern Free State was mainly responsible for the stripe rust epidemic experienced in this region during 1998. The new pathotype, identified as 6E22A-, differed from the introduced pathotype, 6E16A- (discussed below), in its ability to infect the cultivars Hugenoot and Carina in both seedling and adult plant growth stages. In 1998 these two cultivars were grown extensively on an estimated 20% (42 000 ha) of the total wheat hectarage in the eastern Free State, mainly due to their resistance to pathotype 6E16A-. Farmers spent an estimated R8 million on chemical control of stripe rust in the eastern Free State during 1998 whereas yield losses were estimated to amount to a further R5 million.

Occurrence and distribution during 1999. Similar to 1998, stripe rust was observed in the Western Cape later during the 1999 season. In the western parts the first symptoms were observed near Porterville and Piketberg at the end of July. Isolated epidemics occurred on SST 66 in the Piketberg area during mid-September (Fig. 2B). In this region chemical control of stripe rust was required in commercial fields planted to Adam Tas, SST 66, SST 75 and the moderately susceptible cultivar SST 65. Throughout the rest of the western and northern areas of the Western Cape the incidence of stripe rust was low and restricted to Morocco included in disease nurseries.

Fig. 2. (A) Occurrence and incidence of *Puccinia striiformis* f. sp. *tritici* in South Africa during 1998 and (B) 1999.


High incidence of stripe rust
Low incidence of stripe rust
NORTHERN PROVINCE
MPUMALANGA
NORTH WEST
GAUTENG
Lindley
Reitz
KWAZULU NATAL
Harrismith
Bergville
Rietrivier
FREE STATE
LESOTHO
NORTHERN CAPE
Cradock
EASTERN CAPE
Moorreesburg
WESTERN CAPE
Caledon
Mossel Bay

A


High incidence of stripe rust
Low incidence of stripe rust
NORTHERN PROVINCE
Grobblersdal
MPUMALANGA
NORTH WEST
GAUTENG
Lindley
Reitz
Pongola
KWAZULU NATAL
Harrismith
Bergville
Rietrivier
FREE STATE
LESOTHO
NORTHERN CAPE
EASTERN CAPE
Moorreesburg
WESTERN CAPE
Caledon

B

In the southern parts of the Western Cape the first stripe rust symptoms were found during the first week of August, occurring in commercial fields near Bredasdorp and Caledon. This region experienced the worst drought in decades with abnormally high temperatures during June and July. Chemical applications in this region were restricted to SST 75 fields near Caledon. Similar to other areas, the occurrence of *P. striiformis* f. sp. *tritici* remained restricted to susceptible entries in disease nurseries planted near Caledon, Riviersonderend and Napier.

In the summer rainfall areas stripe rust infections were observed during the third week of June on breeding lines of the SGI planted near Pongola in northeastern KwaZulu-Natal. During the second week of July stripe rust was found on the lower leaves of breeding lines, included in field trials of the SGI, near Bloemfontein. In the eastern Free State stripe rust infections were common from September onwards on lower leaves of susceptible cultivars in the districts of Reitz, Kranzfontein, and Harrismith. Chemical control was restricted to wheat fields planted to Hugenoot near Reitz and Kranzfontein. Stripe rust was also observed for the first time near Groblersdal in the north of Mpumalanga where Morocco was multiplied under irrigation. In the Northern Cape the incidence of stripe rust on irrigated wheat was low. The first symptoms were reported during the first week of October at Rietrivier with the disease being restricted mainly to Morocco entries used as spreaders in SGI trials.

Survival during summer and autumn. Attempts at finding stripe rust symptoms on summer-sown wheat, volunteer wheat, and wild grass spp. in the western and southern parts of the Western Cape during February 1997 were unsuccessful. However, the pathogen over-summered successfully and the first stripe rust symptoms were observed in June 1997 on wheat seedlings (three leaf stage) near Mossel Bay and during early July on volunteer wheat near Moorreesburg.

During March 1998 stripe rust infections were observed in a summer-sown wheat trial of the SGI planted near Riviersonderend in the Western Cape. In addition, many volunteer wheat plants and thus potential bridging hosts, occurred in the latter region due to above average summer rains during the 1997/1998 season. Stripe rust was, furthermore, observed on volunteer wheat near Bethlehem during the autumn of

1998, indicating that the pathogen also successfully over-summered in the summer rainfall region.

During a survey conducted in March 1999 in the Western Cape no stripe rust symptoms were found on summer-sown and volunteer wheat plants. In the eastern Free State, however, volunteer wheat plants showing stripe rust symptoms were commonly observed during March and April. Infected wheat plants were found in the districts of Petrus Steyn, Reitz, Kranzfontein, Harrismith, Danielsrus, and Bethlehem.

During a survey conducted in Lesotho during March 1999 stripe rust was commonly found on wheat planted by subsistence farmers. In Lesotho wheat is sown during October and November and harvested towards the end of March. Furthermore, volunteer wheat infected with stripe rust was found during June 1999 in Lesotho. During February 2000 a stripe rust epidemic occurred on wheat in the Mokhotlong district, Lesotho, with the cultivar Moholotsane being affected worst. The seedling and field reactions of Lesotho wheat cultivars are summarised in Table 1 (Fig. 3A). From the data it is evident that the cultivar Moholotsane is highly susceptible. Although stripe rust development was slower on the cultivars Bolane, Telu Nts'o, and Mants'a, the pathogen sporulated actively, which is necessary for survival, on all wheat cultivars grown in Lesotho.

Virulence of *Puccinia striiformis* f. sp. *tritici*

Over the 4-year study period a total of 231 *P. striiformis* isolates were tested on the differential set (Table 2). Isolates were collected from commercial wheat fields, disease nurseries, triticale cultivars and lines, the barley (*Hordeum vulgare* L.) cultivar Clipper (Fig. 3B), barley breeding lines, and several wild grass species. Most isolates originated from the Western Cape (124), followed by the Free State (65), Northern Cape (16), Lesotho (13), Eastern Cape (7), and KwaZulu-Natal (6).

Puccinia striiformis samples established from the initial epidemic in the Western Cape in 1996 were identified as pt. 6E16A- (Table 3). This pathotype is characterised by avirulence to the differential cultivars Chinese 166 (*Yr1*), Vilmorin 23 (*Yr3a,4a*), Moro (*Yr10,Mor*), Strubes Dickkopf (*YrSd,25*), Suwon 92/Omar (*Yr4,Su*), Clement

Table 1. Seedling infection type and field response of wheat cultivars planted in Lesotho to pathotypes 6E16A- and 6E22A- of *Puccinia striiformis* f. sp. *tritici*

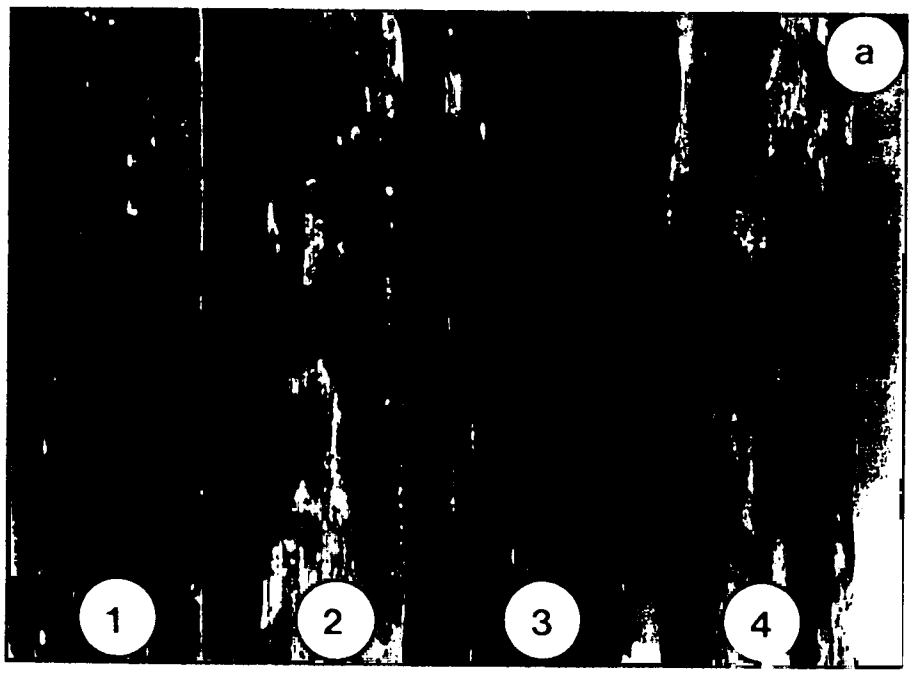
Cultivar	Seedling reaction		Field response
	pt. 6E16A-	pt. 6E22A-	Bethlehem 1999 (pt. 6E22A-)
Moholotsane (Mother of the birds)	4	4	80MS[a]
Bolane (Ou Boland)	3	3	30MS
Mant's Tlala (Tugela)	;, 1C	3+[b]	T,5MS[c]
Telu Nts'o (Black beard)	3	3	20MS

[a]Field response reactions represent the highest percentage infection recorded for each entry during the growth season.

[b]Susceptible to pathotype 6E22A- in the seedling stage which indicates the presence of *Yr25*.

[c]Low field reaction indicating the presence of adult plant resistance.

Fig. 3. (A) Field reaction of four wheat cultivars planted in Lesotho to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici*: 1, Bolane; 2, Moholotsane; 3, Mant's Tlala; 4, Telu Nts'o, (B) Stripe rust symptoms on flag leaves of the barley cultivar Clipper.


a
1
2
3
4


b

Table 2. Pathotypes of *Puccinia striiformis* f. sp. *tritici* detected in South Africa from 1996 to 1999

Year and region	Number of isolates	Pathotype	
		6E16A-	6E22A-
1996			
Western Cape	47	47	0
Northern Cape	8	8	0
Total number	55	100%	
1997			
Western Cape	42	42	0
Northern Cape	2	2	0
Eastern Cape	6	6	0
KwaZulu-Natal	2	2	0
Free State	14	14	0
Lesotho	11	11	0
Total number	77	100%	
1998			
Western Cape	15	15	0
Eastern Cape	1	1	0
Northern Cape	1	1	0
KwaZulu-Natal	2	0	2
Free State	21	7	14
Total number	40	60%	40%
1999			
Western Cape	20	20	0
Northern Cape	5	5	0
KwaZulu-Natal	2	0	2
Lesotho	2	2	0
Free State	30	6	24
Total number	59	56%	44%

Table 3. 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*

Cultivar	*Yr* gene(s)	Low infection type[a]	Seedling response	
			pt. 6E16A-	pt. 6E22A-
Differentials				
1. Chinese 166	*1*	0;	0;	;
2. Lee	*7*	;N, 1N	4	4
3. Heines Kolben	*2*[b]*,6*	;, N1	4	4
4. Vilmorin 23	*3a,4a*[c]	;	;N	;N, 1C
5. Moro	*10,Mor*[d]	0;	;	;
6. Strubes Dickkopf	*Sd,25*[e]	-[f]	;C, 1CN	;C, 1CN
7. Suwon 92/Omar	*4,Su*	;	0;	0;, 1C
8. Clement	*2,9,25*[e]*,Cle*[d]	0;	0;	;
9. *T. spelta album*	*5*	0;, ;	0;	;
10. Hybrid 46	*4b*[g]	;	;	;
11. Reichersberg 42	*7,25*[e]	;N, 1N	;1CN	4
12. Heines Peko	*2,6,25*[e]	;N, N1	;N	4
13. Nord Desprez	*3a,4a*	;	;	;, ;C
14. Compair	*8,19*[h]	0;, ;	4	4
15. Carstens V	*Cv*	-	;	;C
16. Spaldings Prolific	*Sp*	-	0;	0;
17. Heines VII	*2,25*[e]*,HVII*[d]	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 3 (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*

Cultivar	*Yr* gene(s)	Low infection type[a]	Seedling response	
			pt. 6E16A-	pt. 6E22A-
Supplemental set				
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*[i]	-	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*[c]	-	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. Jupateco S	-	-	4	4

[a]McIntosh *et al.* (1995); [b]Calonnec *et al.* (1997b).

[c]McIntosh *et al.* (1998); [d]Chen *et al.*, (1995b).

[e]Calonnec *et al.* (1997a); [f]not available.

[g]Chen *et al.* (1996); [h]Chen *et al.*, (1995a).

[i]Hyde & Elahinia (1990).

(*Yr2,9,25,Cle*), *Triticum aestivum* ssp. *spelta* var. *album* (*Yr5*), Hybrid 46 (*Yr4b*), Reichersberg 42 (*Yr7,25*), Heines Peko (*Yr2,6,25*), Nord Desprez (*Yr3a,4a*), Carstens V (*YrCv*), Spaldings Prolific (*YrSp*), Heines VII (*Yr2,25,HVII*), Avocet R (*YrA*), and virulence to Heines Kolben (*Yr2,6*), Lee (*Yr7*), Compair (*Yr8,19*), and Trident (*Yr17*). Pathotype identity was confirmed by G.H.J. Kema at IPO-DLO, Wageningen, the Netherlands, using one South African isolate of *P. striiformis* f. sp. *tritici*.

Data obtained from stripe rust surveys conducted in the major wheat producing areas in South Africa showed that only the introduced pt. (6E16A-) was detected during 1996 and 1997. During 1998 stripe rust infection reached epidemic proportions on the extensively grown cultivars Hugenoot and Carina in the eastern Free State. The latter two cultivars are resistant to pt. 6E16A-. Avirulence/virulence characteristics of *P. striifomis* f. sp. *tritici* isolates collected from Hugenoot and Carina were determined on the standard stripe rust differential wheat lines and 25 supplementary testers. Additionally, the wheat lines TP981 and TP1295 (supplied by R. Johnson, Cambridge, UK), both of which have a major resistance gene in common with the differentials Heines Peko, Reichersberg 42, Strubes Dickkopf, Clement and Heines VII, were included (Calonnec *et al.*, 1997a; McIntosh *et al.*, 1997). Isolates obtained from Hugenoot and Carina differed from pt. 6E16A- based on virulence to Reichersberg 42 (*Yr7, 25*), Heines Peko (*Yr2, 6, 25*) (Fig. 4,5), TP981 (*Yr25*), and TP1295 (*Yr25*). The new variant, designated as 6E22A-, was also identified in collections from KwaZulu-Natal. Seedling tests with pathotypes 6E16A- and 6E22A- have shown that Hugenoot, Carina, Tugela, and Tugela-DN are the only local cultivars expected to be affected by pathotype 6E22A-. The data presented in Table 2 further proved a regional difference in pathotype distribution giving evidence that stripe rust pathotypes survives independently at several locations in South Africa during the summer months.

The seedling infection types of pathotypes 6E16A- and 6E22A- on the World (1 to 9) and European (10 to 17) differentials as well as on a set of supplemental lines (18 to 42) are presented in Table 3. The field response of these lines recorded at different localities to both pathotypes (Fig. 6A,B) is presented in Table 4. Disease severity ratings represent the maximum development of the disease in the adult plant stage. From the latter data it is evident that the differential cultivars Compair, Kalyansona, and

Fig. 4. Field reaction of four differential cultivars to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici*: 1, Lee; 2, Heines Kolben; 3, Reichersberg 42; 4, Heines Peko. Pathotype 6E16A- is avirulent on the differentials Reichersberg 42 and Heines Peko.

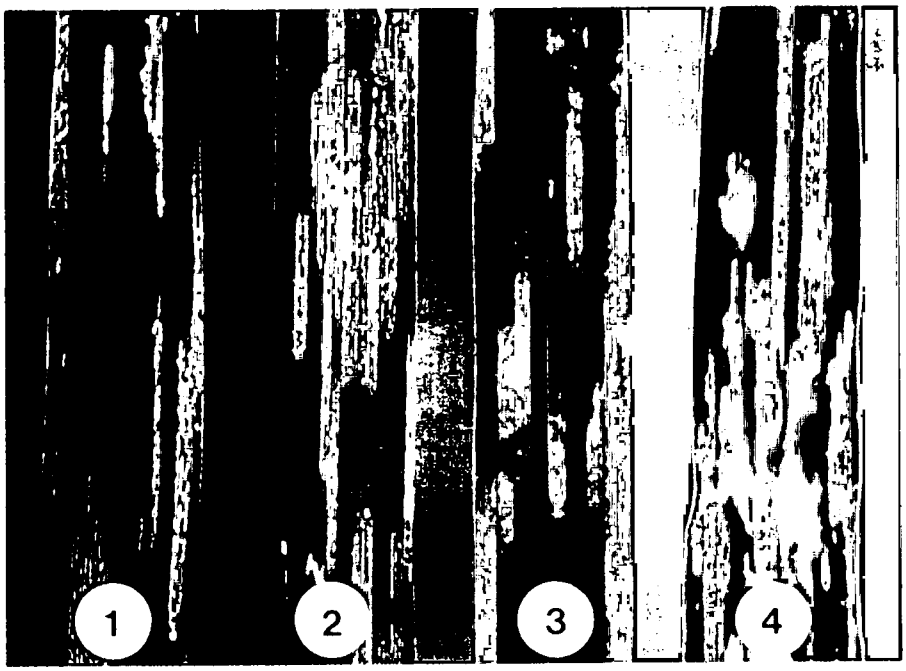
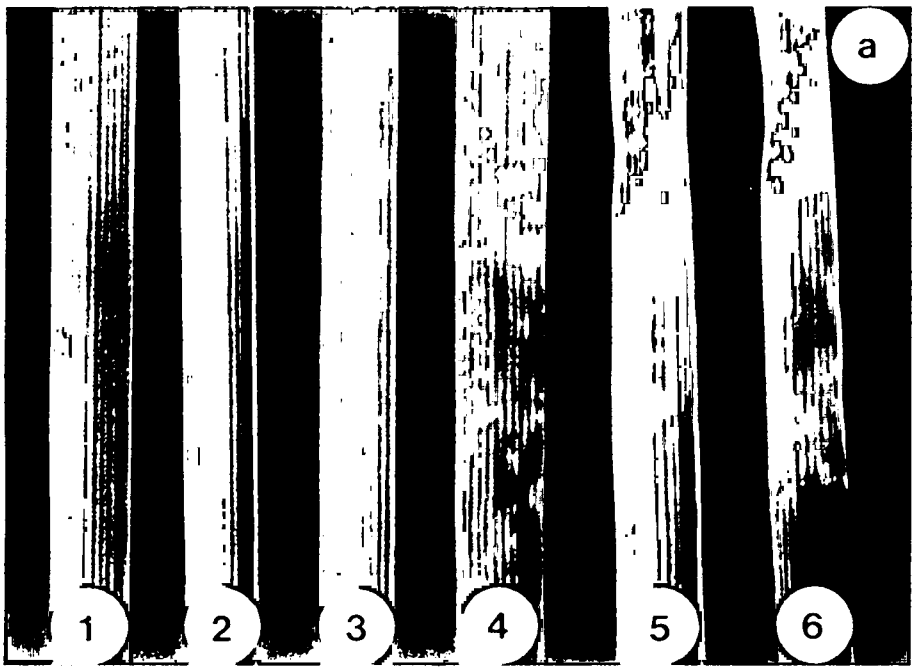

1
2
3
4

Fig. 5. Seedling reaction of six differential cultivars to pathotypes 6E16A- (A) and 6E22A- (B) of *Puccinia striiformis* f. sp. *tritici*: 1, Suwon 92/Omar; 2, Clement; 3, *Triticum aestivum* spp. *spelta* var. *album*; 4, Hybrid 46; 5, Reichersberg 42; 6, Heines Peko.


a
1
2
3
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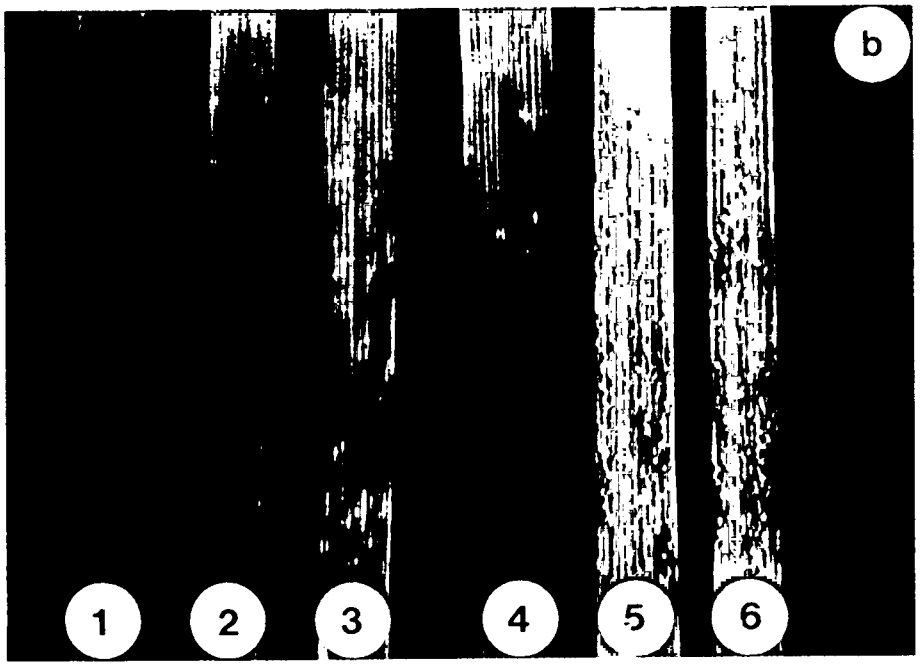
6


b
1
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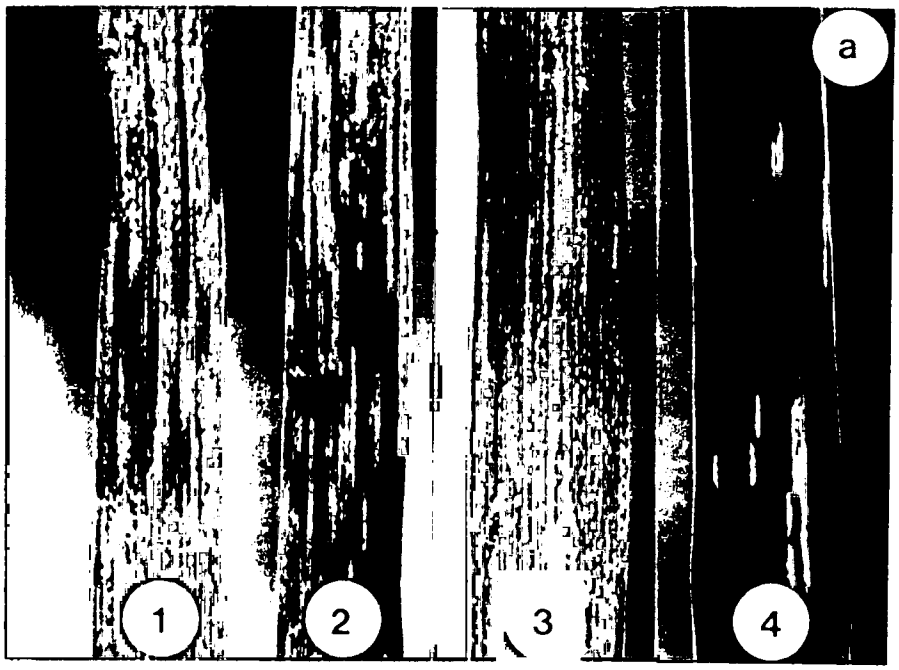
6

Fig. 6. Field reaction of seven supplemental lines to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici*. (A) **Lines:** 1, *Yr6*/6*Avocet-S; 2, *Yr7*/6*Avocet-S; 3, Avocet-S; 4, *Yr8*/3*Avocet-S; (B) **Lines:** 1, *Yr11*/3*Avocet-S; 2, *Yr17*/3*Avocet-S; 3, *Yr18*/3*Avocet-S. Reactions may differ from the data in Table 4 since the latter represents the highest percentage infection recorded for each entry during the growth season.


a
1
2
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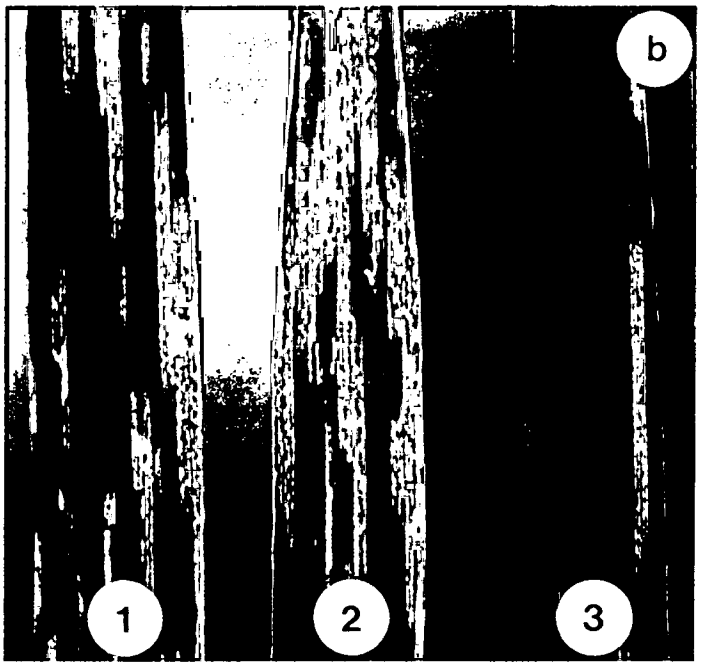

b
1
2
3

Table 4. Field response of the World (1 to 9) and European (10 to 17) differentials as well as supplemental lines (18 to 42) to pathotypes 6E16A- and 6E22A- of *Puccinia striiformis* f. sp. *tritici* at different localities in South Africa during 1998 and 1999

Cultivar/line	*Yr* gene(s)	Bethlehem 1998 (pt. 6E16A-)	Greytown 1998 (pt. 6E22A-)	Bethlehem 1999 (pt. 6E22A-)	Riviersonderend 1999 (pt. 6E16A-)	Burgershall 1999 (pt. 6E16A-)	Greytown 1999 (pt. 6E22A-)
Differentials							
1. Chinese 166	*1*	0[a]	0	0	0	0	0
2. Lee	*7*	90MSS	30MS	80S	10MS	20S	50MS
3. Heines Kolben	*2,6*	80MSS	50MRMS	80S	10MS	20S	50R
4. Vilmorin 23	*3a,4a*	0	10R	5MS	0	0	TR
5. Moro	*10,Mor*	0	-	0	0	0	-
6. Strubes Dickkopf	*Sd,25*	0	-	0	0	0	-
7. Suwon 92/Omar	*4,Su*	0	0	0	0	0	0
8. Clement	*2,9,25,Cle*	0	0	0	0	0	0
9. *T. spelta album*	*5*	0	10R	0	0	0	0
10. Hybrid 46	*4b*	0	0	0	0	0	0
11. Reichersberg 42	*7,25*	0	10MR	20MS	0	0	0
12. Heines Peko	*2,6,25*	0	15R	20MS	0	0	10R
13. Nord Desprez	*3a,4a*	0	TR	TR	0	0	0

Table 4 (cont.). Field response of the World (1 to 9) and European (10 to 17) differentials as well as supplemental lines (18 to 42) to pathotypes 6E16A- and 6E22A- of *Puccinia striiformis* f. sp. *tritici* at different localities in South Africa during 1998 and 1999

Cultivar/line	*Yr* gene(s)	Bethlehem 1998 (pt. 6E16A-)	Greytown 1998 (pt. 6E22A-)	Bethlehem 1999 (pt. 6E22A-)	Riviersonderend 1999 (pt. 6E16A-)	Burgershall 1999 (pt. 6E16A-)	Greytown 1999 (pt. 6E22A-)
Differentials							
14. Compair	*8,19*	0	0	5MS	0	0	0
15. Carstens V	*Cv*	0	0	0	0	0	0
16. Spaldings Prolific	*Sp*	0	0	0	0	0	0
17. Heines VII	*2,25,HVII*	0	0	0	0	0	0
Supplemental set							
18. *Yr1*/6*AvS	*1*	0	0	0	0	0	0
19. Kalyansona	*2*	5MS	10R	10R	5MS	0	5R
20. *Yr5*/6*AvS	*5*	0	0	0	0	0	0
21. *Yr6*/6*AvS	*6*	-	-	100S	80S	100S	100S
22. *Yr7*/6*AvS	*7*	100S	70S	100S	60S	100S	100S
23. *Yr8*/6*AvS	*8*	30MR	15R	20MR,80MS	5MS	TMS	TR
24. Federation 4/Kavkaz	*9*	0	0	0	0	0	0
25. *Yr9*/6*AvS	*9*	5R,40MS	0	0,20MS	0	20MR	30MRMS

Table 4 (cont.). Field response of the World (1 to 9) and European (10 to 17) differentials as well as supplemental lines (18 to 42) to pathotypes 6E16A- and 6E22A- of *Puccinia striiformis* f. sp. *tritici* at different localities in South Africa during 1998 and 1999

Cultivar/line	*Yr* gene(s)	Bethlehem 1998 (pt. 6E16A-)	Greytown 1998 (pt. 6E22A-)	Bethlehem 1999 (pt. 6E22A-)	Riviersonderend 1999 (pt. 6E16A-)	Burgershall 1999 (pt. 6E16A-)	Greytown 1999 (pt. 6E22A-)
Supplemental set							
26. *Yr10/6*AvS*	*10*	0	0	0	0	0	0
27. *Yr11*/3*AvS	*11*	-	-	80S	50S	80S	60MR-MS
28. Wembley	*14*	90MRMS	30MR	80MS	-	40S	30MR
29. *Yr15*/6*AvS	*15*	0	0	0	0	0	0
30. Trident	*17*	20MS	20R	40MS	5MS	20MS	30MR
31. *Yr17*/3*AvS	*17*	90MS	40MSS	90MS	20S	40MS	80MSS
32. Jupateco R	*18*	20MR	5R	20MRR	10MR	10MRMS	20R
33. *Yr18*/3*AvS	*18*	-	-	30,80MS	-	20MS	20MR
34. *Yr24*/3*AvS	*24*	-	-	20R,70R	-	-	70MR
35. *Yr26*/3*AvS	*26*	-	-	20R	-	-	5R
36. Selkirk	*27*	10R	40RMR	20R	10R	TR	10R
37. *Yr27*/3*AvS	*27*	-	-	10R,80S	0	0,100S	0,20MS
38. Avocet R	*A*	20MS	5R	30MR,80MS	0	10MS	TR

Table 4 (cont.). Field response of the World (1 to 9) and European (10 to 17) differentials as well as supplemental lines (18 to 42) to pathotypes 6E16A- and 6E22A- of *Puccinia striiformis* f. sp. *tritici* at different localities in South Africa during 1998 and 1999

Cultivar/line	*Yr* gene(s)	Bethlehem 1998 (6E16A-)	Greytown 1998 (6E22A-)	Bethlehem 1999 (6E22A-)	Riviersonderend 1999 (6E16A-)	Burgershall 1999 (6E16A-)	Greytown 1999 (6E22A-)
Supplemental set							
39. *YrSp*/3*AvS	*Sp*	-	-	0	0	0	0
40. Federation 1221	-	80S	80S	100S	30S	80S	80S
41. Avocet S	-	-	-	100S	60S	100S	100S
42. Jupateco S	-	100S	-	100MS	10S	100S	90MSS

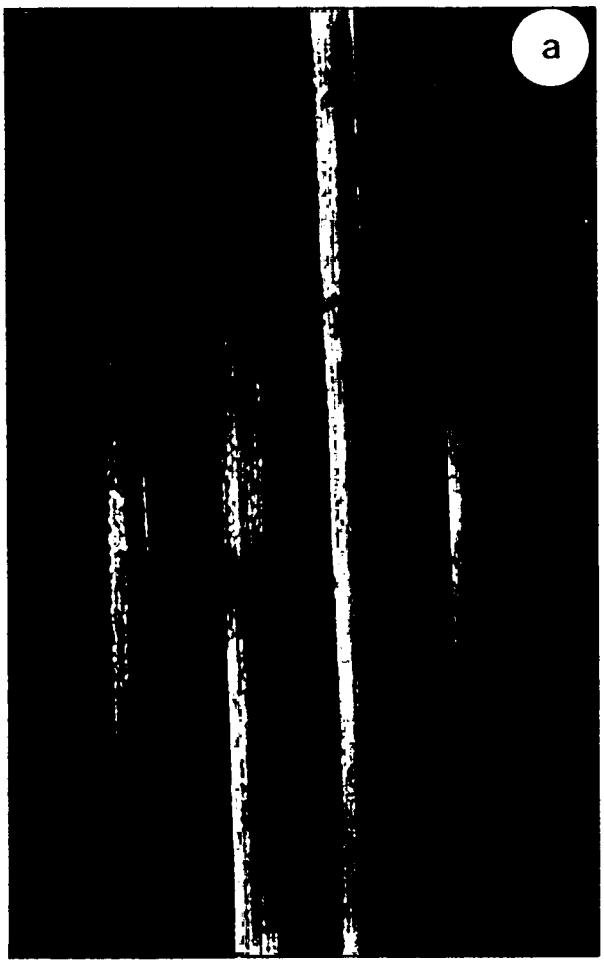
[a]Field response reactions represent the highest percentage infection recorded for each entry during the growth season.

Trident, and the supplemental line *Yr8*/6*Avocet-S carry additional gene(s) resulting in varying levels of adult plant resistance against these pathotypes. The low adult plant response obtained for the differential cultivars Reichersberg 42 and Heines Peko at Greytown and Bethlehem, where pathotype 6E22A- was present during 1999, may be attributed to their long growth period and subsequent escape of infection at the end of the season. However, the presence of additional adult plant resistance in Reichersberg 42 and Heines Peko can not be excluded. The gene *Yr27* present in Selkirk produced a compatible (3) seedling reaction when scored 12 to 14 days after inoculation, but this infection type became incompatible (necrotic) when scored 3 to 4 days later. From the field data it is clear that *Yr27* is still effective in the adult plant stage. The adult plant gene *Yr18* present in Jupateco R and *Yr18*/3*Avocet-S exhibit intermediate to high levels of resistance under South African conditions. Although the adult plant genes *Yr11* and *Yr14*, present in *Yr11*/3*Avocet-S and the cultivar Wembley, respectively, showed a level of resistance relative to Avocet S, their responses are not expected to provide adequate crop protection. Supplemental lines exhibiting heterogeneous field reactions are *Yr8*/6*Avocet-S, *Yr9*/6*Avocet-S, *Yr18*/3*Avocet-S, *Yr24*/3*Avocet-S, *Yr27*/3*Avocet-S, and Avocet-R. Since only original seed sources were used for these trials the source of susceptible plants are unknown.

Susceptibility of wild grass species

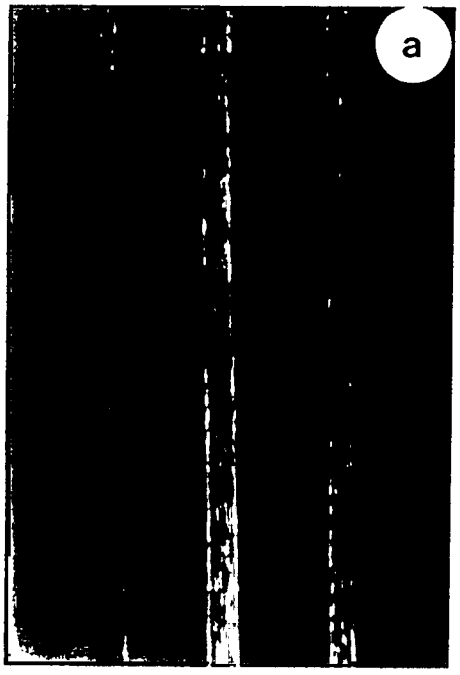
During 1997 stripe rust was found on *Bromus catharticus* Vahl (=*B. unioloides* H.B.K.) (Fig. 7) in the eastern Free State and on *Hordeum murinum* L. (Fig. 8) in the Western Cape. Isolates sampled from both spp. were identified as pt. 6E16A-. During 1999 stripe rust sampled from *B. catharticus* in the eastern Free State was identified as pt. 6E22A-. Uredospores from infections similar to stripe rust sampled from leaves of *Dactylis glomerata* L. (Eastern Cape), *Poa pratensis* L. (=*P. bidentata* Stapf) (Western Cape), and *P. annua* and *P. triviales* L. (eastern Free State) failed to infect Morocco seedlings. Seedling infection types of 17 grass spp. to an isolate of *P. striiformis* f. sp. *tritici* are presented in Table 5. *Bromus arenarius* and *B. oxydon* showed compatible seedling reactions and *B. trinii* appeared heterogeneous.

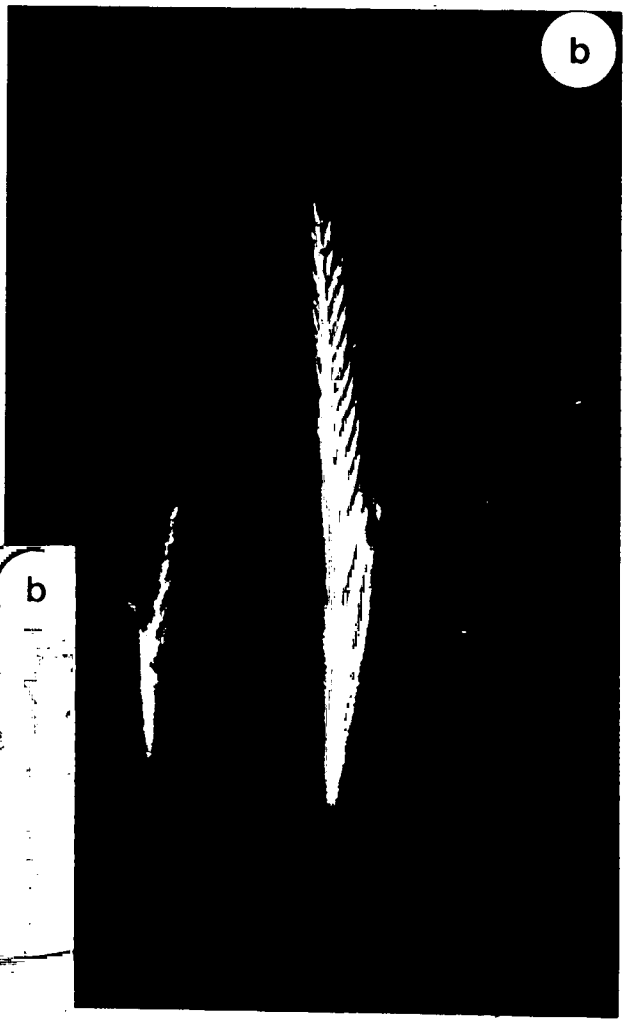
Fig. 7. Stripe rust symptoms on (A) seedling and (B) adult plant leaves of *Bromus catharticus*.


a


b

Fig. 8. Stripe rust symptoms on (A) seedling and (B) adult plant leaves of *Hordeum murinum*.


a


b


b

Table 5. Pathogenicity of an isolate of *Puccinia striiformis* f. sp. *tritici* (pt. 6E16A-) from wheat to 17 grass species belonging to six genera occurring naturally or used for grazing purposes

Grass spp.	Origin	Infection type
Bromus arenarius acc. W209[a]	DPQC[b]	3
Bromus carinatus acc. 1599[a]	DPQC	0
Bromus carinatus	Riviersonderend	0
Bromus catharticus Vahl (=*B. unioloides* H.B.K.)[c]	Bethlehem	3
Bromus catharticus acc. 80270[a]	DPQC	3
Bromus diandrus Roth	Malmesbury	;N
Bromus japonicus Sensu (=*B. pectinatus* Thunb)[c]	DPQC	0
Bromus mollis L. cv Catapico[a]	DPQC	;
Bromus oxydon acc. 28124[a]	DPQC	2+3
Bromus sp. [a]	DPQC	;N
Bromus sp. cv 14789[a]	DPQC	1N, 2
Bromus trinii cv 25531[a]	DPQC	1N, 2
Bromus trinii cv 25528[a]	DPQC	1C, 4
Bromus unioloides H.B.K.	DPQC	3
Dactylis glomerata L.	Eastern Cape	0
Hordeum capense Thunb. (=*H. nodosum* auctt., non L.)[c]	Porterville	;N, 1N

Table 5 (cont.). Pathogenicity of an isolate of *Puccinia striiformis* f. sp. *tritici* (pt. 6E16A-) from wheat to17 grass species belonging to six genera occurring naturally or used for grazing purposes

Grass spp.	Origin	Infection type
Hordeum murinum L.	Elsenburg	0;
Hordeum murinum L.	Hopefield/Piketberg	4
Hordeum murinum L.	Gouda	;c
Lolium multiflorum Lam.	Malmesbury	0
Lolium sp.	Poterville	0
Poa pratensis L. (=*P. bidentata* Stapf)[c]	DPQC	0
Poa triviales L.	Bethlehem	0
Poa annua L.	Bethlehem	0
Secale cereale L. cv Cool Grazer[a]	DPQC	0, ;C
Secale cereale L. cv M/71/128[a]	DPQC	0, ;C

[a]Used only for grazing purposes.

[b]Seed obtained from the Directorate of Plant and Quality Control, South Africa.

[c]Gibbs Russel *et al.* (1991).

Influence of weather conditions

The monthly rainfall (mm), average minimum and maximum temperatures (°C) from 1996 to 1999 and an 11 year (1989-1999) average for the Moorreesburg district are summarised in Table 6. From this data it is evident that the above average rainfall together with lower than average minimum and maximum temperatures recorded during August and September 1996, contributed significantly to the establishment, spread and subsequent epidemic outbreak of stripe rust in the Western Cape. A further contributing factor was that almost all the cultivars grown in this area in 1996 were susceptible to the introduced pathotype. During 1997 and 1998 a major shift to the stripe rust resistant cultivars SST 57, SST 825, and Kariega, accompanied by less favourable weather conditions contributed to the lower incidence of stripe rust in this region. Although the rainfall during August and September 1999 was nearly double the expected average, along with conducive minimum and maximum temperatures, the deployment of resistant cultivars significantly reduced epidemic development.

DISCUSSION

Following the detection of *P. striiformis* f. sp. *tritici* for the first time on bread wheat in the Western Cape during August 1996, the pathogen has spread to all the important wheat production areas in South Africa. Stripe rust has subsequently become established as an endemic disease with the biggest impact on wheat production in the western and southern areas of the Western Cape, the Eastern Cape, areas of the eastern Free State, and KwaZulu-Natal. The average hectarage of wheat planted in the latter regions over the last six years are 500 000 ha, accounting for approximately 47% of the total area sown to wheat in South Africa annually. Wheat produced under dry-land conditions in the western, central, south-eastern and southern Free State largely escaped stripe rust epidemics. Contributing factors to the latter are low seeding rates (15- 30 kg/ha) and thus less dense stands, low humidity, and drought conditions during early growth stages, followed by high day temperatures after the first spring rains. Wheat produced under irrigation in the Northern Cape, North-West, Gauteng,

Table 6. Monthly rainfall (mm) and average minimum and maximum temperatures (°C) from April to November recorded at Moorreesburg where *Puccinia striiformis* f. sp. *tritici* was first detected during August 1996

		Month							
	Year	April	May	June	July	August	Sept.	Oct.	Nov.
Rainfall[a]	1996	32.8	38.4	84.2	88.5	90.1	100.2	25.3	41.0
	1997	23.6	55.4	86.1	25.1	60.4	4.4	2.7	4.7
	1998	19.4	79.8	46.3	58.7	37.8	23.4	27.4	49.7
	1999	28.3	43.3	38.5	57.9	83.3	97.0	0.4	11.5
11 year average		35.2	53.3	78.6	69.5	48.5	43.4	25.6	16.7
Minimum temperature									
	1996	15.1	12.4	8.4	7.3	7.2	8.6	10.9	12.3
	1997	12.6	11.9	8.3	8.8	8.5	11.7	13.7	14.0
	1998	14.5	11.6	9.1	9.0	9.0	10.5	12.3	13.9
	1999	15.4	12.4	9.9	7.7	8.0	8.3	13.0	14.3
11 year average		14.1	11.4	8.8	7.9	7.9	9.7	11.8	13.8
Maximum temperature									
	1996	27.0	23.1	17.7	16.0	16.9	17.9	22.2	22.9
	1997	24.3	22.2	16.6	18.8	17.7	24.0	27.6	25.7
	1998	26.4	20.7	17.8	17.1	19.4	20.8	25.4	26.5
	1999	27.0	21.7	20.3	18.3	19.2	19.5	27.2	28.2
11 year average		25.6	21.4	17.8	17.1	18.2	20.9	24.7	26.7

[a]Data supplied by the Institute of Soil, Climate and Water, Pretoria, South Africa.

Northern Province and Mpumalanga also escaped stripe rust epidemics. High day temperatures experienced during October and November in the irrigation areas together with high levels of resistance present in most of the cultivars grown in those areas may have contributed to the latter.

Park (1990) defined days as being stripe rust-favourable when the mean temperature falls within the range 12.4 to 18.4°C, and the minimum between 7.3 and 14.6°C. Both minimum and mean temperatures recorded for Moorreesburg during August and September 1996, when the stripe rust epidemic was at its peak in the Western Cape, fall within these ranges. Contributing to the establishment and epidemic development of stripe rust was the above average rainfall (highest in 11 years) recorded for Moorreesburg during August and September 1996. The higher rainfall not only contributed to lower temperatures and prolonged leaf wetness, but may have played a significant role in the spread of the disease as rain-splash has been proved an important mechanism of stripe rust dispersal (Geagea *et al.*, 1999).

Results of the survey revealed the presence of two stripe rust pathotypes in South Africa. Yield losses caused by pt. 6E16A- in the eastern Free State during 1997 resulted in a major shift to the cultivation of the resistant cultivars Hugenoot and Carina the next year. The high selection pressure evidently resulted in the development of pt. 6E22A- that appears to be a single-step mutation event with additional virulence for *Yr25*, from 6E16A-. Pathotype 6E16A- was previously detected in East and North Africa, the Middle East, and Western Asia (Badebo *et al.*, 1990; Louwers *et al.*, 1992). With several resistance genes not being reflected in the classification of pathotypes globally, it is possible some variation within this pathotype may occur elsewhere. It is therefore not possible to speculate as to the likely origin of the introduced pathotype.

Virulence was detected for the seedling genes *Yr2*, *6*, *7*, *8*, *17*, *19* and *25,* as well as for the adult plant genes *Yr11* and *Yr14*. The relatively small pathogen sample size examined through the four year period does not justify an assumption that virulence is absent for the remaining seedling genes. Furthermore, the current absence of most of the effective seedling genes from commercial cultivars (Pretorius, 1998), and the resulting low selection pressure preclude any predictions of their durability to local stripe rust pathotypes. However, seedling genes with a hypersensitive response to

avirulent isolates of *P. striiformis* are well known for their vulnerability (Wellings & McIntosh, 1990; Danial *et al.*, 1995; Ma & Singh, 1996; McIntosh & Brown, 1997; Shan *et al.*, 1999). Over a 10 yr period 15 different stripe rust pathotypes were detected in Australia and New Zealand adding virulence to seven seedling genes, initially effective to the pathotype introduced to Australia in 1979 (Wellings & McIntosh, 1990). Furthermore, the seedling genes *Yr5*, *8*, *27*, and *YrSp* have become ineffective due to changes in the pathogen population without the presence of the genes in Australasian wheats (Wellings & McIntosh, 1990; McIntosh & Brown, 1997). Adult plant resistance against *P. striiformis* has been known to last longer (Park & Rees, 1989; Johnson, 1992b; Broers *et al.*, 1996; Ma & Singh, 1996; McIntosh & Brown, 1997).

The ability of the stripe rust pathogen to survive near wheat fields through the non-cropping season will play an important role in disease onset and build-up of inoculum during early growth stages. The data presented proved that stripe rust survives independently during the summer months in the summer and winter rainfall areas of South Africa. The summer and autumn survival of the stripe rust fungus in foreign countries is dependent on susceptible volunteer or self-sown wheat plants and to a lesser extent of grass spp. (Sharp & Hehn, 1963; Shaner & Powelson, 1973; Wellings & McIntosh, 1981; Stubbs, 1985; Dennis & Brown, 1986; Nazari *et al.*, 1996). Virulence on grasses provides greater opportunity for the survival and increase of stripe rust (McIntosh & Brown, 1997) and may play a role in the occurrence of epidemics (Mardoukhi & Torabi, 1998). In this study two accessory hosts of stripe were found in the field with a further two spp. being seedling susceptible and one showing a heterogeneous seedling reaction. *Bromus catharticus* is commonly found during the spring, summer and autumn months along river banks, roads and agricultural fields throughout all the wheat growing areas. This *Bromus* sp. is perennial to weekly annual, dependent on the growing conditions (Gibbs Russel *et al.*, 1991; Van Oudtshoorn *et al.*, 1991). *Hordeum murinum* found along commercial fields, roads and waste areas in the Western Cape as well as in the mountainous southern parts of Lesotho is a winter-active annual and uncommon during the summer months in the Western Cape (Gibbs Russel *et al.*, 1991; Van Oudtshoorn *et al.*, 1991). Sporulation of *P. striiformis* on leaves of both these grasses is abundant during environmental conditions conducive

to stripe rust development. Attempts at finding the stripe rust pathogen during the summer and autumn months on susceptible grass spp. have failed. However, the possibility that susceptible grasses plays an important role in the over-summering of stripe rust in the mountainous region of the Western Cape, Eastern Cape, eastern Free State, KwaZulu-Natal, and Lesotho cannot be excluded and warrants further investigation.

Similar to previous studies uredospores obtained from infections on *P. pratensis* and *D. glomerata* were avirulent on wheat (Line, 1976; Holmes & Dennis, 1985). *Hordeum murinum* (=*Hordeum leporinum* Link), *Bromus unioloides*, and *P. trivialis* have also previously been found susceptible to stripe rust pathotypes originating from wheat (Line, 1976; Holmes & Dennis, 1985; Nazari *et al.*, 1996). The seedling reaction of *B. arenarius*, *B. oxydon*, and *B. trinii* showed that these spp. could also serve as accessory hosts of local *P. striiformis* pathotypes. The latter spp. currently do not occur naturally in South Africa but are planted for grazing purposes (Gibbs Russel *et al.*, 1991).

Unusually mild and rainy weather during the off-season promote the build-up of stripe rust inoculum and is closely associated with subsequent levels of stripe rust observed in commercial crops (Bayles *et al.*, 1989; Chilosi & Corazza, 1990; Ellison & Murray, 1992; Park, 1990). In the eastern Free State stripe rust infection on susceptible volunteers is currently serving as an inoculum source for the onset of early season infections. The higher elevation areas of Lesotho, with its growing period differing from the main wheat areas in South Africa, is further providing a zone for the over-summering of stripe rust. These sources of inoculum can be minimised by an increase of resistant cultivars, especially in the eastern Free State and Lesotho where summer rain and mild summer temperatures can significantly contribute to the summer survival of stripe rust.

Factors that may lead to the rapid evolution of aggressive pathogen races are increased fecundity, and thus more pathogen generations per season, or a more conducive microclimate for disease development (Coakley *et al.*, 1999). Stripe rust control strategies in South Africa should be directed at reducing the probability of epidemics and reducing the magnitude of losses. The latter may be obtained by avoiding the release of cultivars containing only genes for seedling resistance and the

recommendation of only stripe rust resistant cultivars in the more rust-prone areas. It is important to continue monitoring the stripe rust population for pathotype changes so that new pathotypes with the potential to overcome resistance genes currently deployed can be detected early. The latter is justified by research proving that stripe rust can cause substantial losses to grain yield and quality under local environmental conditions given a susceptible cultivar.

LITERATURE CITED

Angus, A. 1965. Annotated list of plant pests and diseases in Zambia. Parts 1-7 and supplements. Mount Makulu Reseasch Station, Zambia.

Anonymous, 2000. Abstract of Agricultural Statistics, National Department of Agriculture, Pretoria, South Africa. 112pp.

Badebo, A., Stubbs, R.W., Van Ginkel, M. & Gebeyehu, G. 1990. Identification of resistance genes to *Puccinia striiformis* in seedlings of Ethiopian and Cimmyt wheat varieties and lines. Netherland Journal of Plant Pathology 96: 199-210.

Bayles, R.A., Channell, M.H. & Stigwood, P.L. 1989. New races of *Puccinia striiformis* in the United Kingdom in 1988. Cereal Rusts and Powdery Mildews Bulletin 17: 20-23.

Beresford, R.M. 1982. Stripe rust (*Puccinia striiformis*) a new disease of wheat in New Zealand. Cereal Rust Bulletin 10: 35-41.

Bonthuis, H. 1978. Stripe rust on wheat in Zambia. An epidemiological analysis. Cimmyt Regional Program for East Africa. 15pp.

Broers, L.H.M., Cuesta Subias, X. & López Atilano, R.M. 1996. Field assessment of quantitative resistance to yellow rust in ten spring bread wheat cultivars. Euphytica 90: 9-16.

Calonnec, A., Johnson, R. & 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. & 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: 373-386.

Chen, X.M., Line, R.F. & Leung, H. 1993. Relationship between virulence variation and DNA polymorphism in *Puccinia striiformis*. Phytopathology 83: 1489-1497.

Chen, X.M., Jones, S.S. & 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. & Leung, H. 1995b. Virulence and polymorphic DNA relationships of *Puccinia striiformis* f. sp. *hordei* to other rusts. Phytopathology 85: 1335-1342.Chen, X.M., Jones, S.S. & Line, R.F. 1996. Chromosomal location of genes for resistance to *Puccinia striiformis* in seven wheat cultivars with resistance genes at the *Yr*3 and *Yr*4 loci. Phytopathology 86: 1228-1233.

Chen, X.M., Jones, S.S. & Line, R.F. 1996. Chromosomal location of genes for resistance to *Puccinia striiformis* in seven wheat cultivars with resistance genes at the *Yr*3 and *Yr*4 loci. Phytopathology 86: 1228-1233.

Chilosi, G. & Corazza, L. 1990. Occurrence and epidemics of yellow rust of wheat in Italy. Cereal Rusts and Powdery Mildews Bulletin 18: 1-9.

Coakley, S.M., Scherm, H. & Chakraborty, S. 1999. Climate change and plant disease. Annual Review of Phytopathology 37: 399-426.

Danial, D.L. 1994. Aspects of durable resistance in wheat to yellow rust. Ph.D. Thesis. Wageningen. 143pp.

Danial, D.L., Kirigwi, F.M. & 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.

Dennis, J.I. & Brown, J.S. 1986. Summer survival of *Puccinia striiformis* f. sp. *tritici* in Victoria, Australia. Australian Plant Pathology 15: 57-60.

Ellison, P.J. & 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.

Geagea, L., Huber, L. & Sache, I. 1999. Dry-dispersal and rain-splash of brown (*Puccinia recondita* f. sp. *tritici*) and yellow (*P. striiformis*) rust spores from infected wheat leaves exposed to simulated raindrops. Plant Pathology 48: 472-482.

Gibbs Russell, G.E., Watson, L., Koekemoer, M., Smook, L., Barker, N.P., Anderson, H.M. & Dallwitz, M.J. 1991. Grasses of southern Africa. 437 pp.

Holmes, R.J. & Dennis, J.I. 1985. Accessory hosts of wheat stripe rust in Victoria, Australia. Transactions of the British Mycology Society 85: 159-160.

Hyde, P.M. & 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. 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 eye spot of wheat. Plant Pathology 41: 239-254.

Johnson, R. & Taylor, A.J. 1976. Yellow rust of wheat. Plant Breeding Institute Cambridge, Annual Report. pp. 106-109.

Johnson, R., Stubbs, R.W. & Fuchs, E. 1972. Nomenclature for physiologic races of *Puccinia striiformis* infecting wheat. Transactions of the British Mycological Society 58: 475-480.

Line, F.L. 1976. Factors contributing to an epidemic of stripe rust on wheat in the Sacramento valley of California in 1974. Plant Disease Reporter 60: 312-316.

Louwers, J.M., Van Silfhout, C.H. & Stubbs, R.W. 1992. Race analysis of yellow rust in wheat in developing countries, Report 1990- 1992, IPO-DLO report 92-11. 23 pp.

Ma, H. & Singh, R.P. 1996. Expression of adult resistance to stripe rust at different growth stages of wheat. Plant Disease 80: 375-379.

Mardoukhi, V. & Torabi, M. 1998. Yellow rust on wild grasses in Iran. Seed-and-Plant 14: 66-73.

McIntosh, R.A. 1980. Stripe rust - A new wheat disease for Australia. The University

of Sydney Plant Breeding Institute, Castle Hill. Bulk Wheat: 65-67.

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

McIntosh, R.A., Wellings, C.R. & Park, R.F. 1995. Wheat rusts: An atlas of resistance genes. Kluwer, Dordrecht. 200 pp.

McIntosh, R.A., Hart, G.E., Devos, K.M. & Gale, M.D. 1997. Catalogue of gene symbols for wheat: 1997 Supplement. Wheat Information Service 85: 56-81.

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

Nazari, K., Torabi, M. & Mardoukhi, V. 1996. Wild grass species as oversummering hosts of *Puccinia striiformis* f. sp. *tritici* in Iran. Cereal Rusts and Powdery Mildews Bulletin 24: 105-107.

O'Brien, L., Brown, J.S., Young, R.M. & Pascoe, I. 1980. Occurrence and distribution of wheat stripe rust in Victoria and susceptibility of commercial wheat cultivars. Australian Plant Pathology 9: 14.

Park, R.F. 1990. The role of temperature and rainfall in the epidemiology of *Puccinia striiformis* f. sp. *tritici* in the summer rainfall area of eastern Australia. Plant Pathology 39: 416-423.

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

Peterson, R.F., Campbell, A.B. & 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. Unpublished report, Department of Plant Pathology, University of the Orange Free State, Bloemfontein, South Africa. 72pp.

Shan, W.-X., Chen, S.-Y., Kang, Z.-S., Wu, L.-R. & Li, Z.-Q. 1999. Genetic diversity in *Puccinia striiformis* Westend. f. sp. *tritici* revealed by pathogen genome-specific

repetitive sequence. Canadian Journal of Botany 76: 587-595.

Shaner, G. & Powelson, R.L. 1973. The oversummering and dispersal of inoculum *of Puccinia striiformis* in Oregon. Phytopathology 63: 13-17.

Sharp, E.L. & Hehn, E.R. 1963. Overwintering of stripe rust in winter wheat in Montana. Phytopathology 53: 1239-1240.

Stubbs, R.W. 1985. Stripe Rust. Pages 61-101 in: The Cereal Rusts II: Diseases, Distribution, Epidemiology and Control. A.P. Roelfs and W.R. Bushnell, eds. Academic Press, Inc., New York.

Van Oudtshoorn, F.P., Trollope, W.S.W., Scotney, D.M. & McPhee, P.J. 1991. Gids tot grasse van Suid-Afrika. Briza publikasies, Arcadia, South Africa. 301pp.

Wellings, C.R. & McIntosh, R.A. 1990. *Puccinia striiformis* f. sp. *tritici* in Australasia: pathogenic changes during the first 10 years. Plant Pathology 39: 316-325.

Wellings, C.R. & McIntosh, R.A. 1981. Stripe rust - a new challenge to the wheat industry. Reprinted from the Agricultural Gazette of the New South Wales Department of Agriculture. 4pp.

RESISTANCE IN SOUTH AFRICAN AND FOREIGN WHEAT CULTIVARS TO PATHOTYPES 6E16A- AND 6E22A- OF *PUCCINIA STRIIFORMIS* F. SP. *TRITICI*

ABSTRACT

The reaction of 55 South African and 18 foreign *Triticum aestivum* L. cultivars was determined to pathotypes 6E16A- and 6E22A- of *Puccinia striiformis* Westend. f. sp. *tritici* Eriks. in the seedling and adult plant stage. The occurrence of stripe rust head infections was studied in 16 spring wheat cultivars and 17 supplemental lines. Six of the 55 local wheat cultivars expressed seedling resistance (infection type <2+), 18 appeared heterogeneous and 31 were susceptible (infection type ≥2+). The mean area under the disease progress curve (AUDPC) determined in the field for 42 cultivars over a three year period showed that 11 cultivars expressed high levels of complete or adult plant resistance (AUDPC <200). Twelve cultivars displayed intermediate levels of resistance (AUDPC 200 to 500) and 19 displayed AUDPC values of 500 to 1598. Terminal severity ratings were highly correlated with AUDPC for both winter (R^2=0.91, P<0.001) and spring (R^2=0.82, P<0.001) wheat cultivars. Stripe rust resistance expressed by local wheat cultivars appeared stable over five different environments. The percentage head infection was positively correlated (R^2=0.78, P<0.001 during 1997 and R^2=0.84, P<0.001 during 1999) to stripe rust severity on flag leaves. Cultivars and lines with seedling resistance showed no or a very low percentage (0 to 2%) head infection, whereas cultivars susceptible in both seedling and adult plant stages were severely infected in the heads. Cultivars and lines expressing adult plant resistance showed intermediate to low percentages of head infection. Of the 18 foreign cultivars evaluated 10 were resistant in both seedling and adult plant stages. The remaining eight cultivars were susceptible as seedlings but showed high levels of adult plant resistance in the field.

INTRODUCTION

Stripe rust, caused by *Puccinia striiformis* Westend. f. sp. *tritici* Eriks., is an important disease of bread wheat (*Triticum aestivum* L.) in parts of the world where cool and moist environmental conditions prevail (McIntosh, 1980; Stubbs, 1985; Danial, 1994). Under severe epidemic conditions yield losses as high as 84% have been recorded (Murray *et al.*, 1994). The application of fungicides (Ash & Brown, 1990; Gaunt & Cole, 1991; Jørgensen & Nielsen, 1994), and to a lesser extent the use of cultivar mixtures (Finckh & Mundt, 1992; Mundt *et al.*, 1996; Akanda & Mundt, 1997), have been successfully deployed in controlling stripe rust outbreaks. However, genetic resistance is still considered the most cost-effective and environment-friendly control strategy (Ma *et al.*, 1995; Ma & Singh, 1996a).

The value of genetic resistance to diseases in crops depends largely on its level, its stability towards geographical and environmental conditions, and its durability (Broers, 1989). Long-term resistance in wheat to rust diseases depends on the availability and management of durable resistance sources (Bariana & McIntosh, 1993), on the continuing use of new sources of resistance, and on the combination of genes for specific resistance (Bariana & McIntosh, 1995). Resistance to stripe rust can broadly be classified in types that are expressed in seedlings or adult plants. Seedling resistance, which usually is effective throughout the life-span of the plant, is mostly pathotype-specific (Danial, 1994) whereas adult plant resistance can be pathotype-specific or non-specific (Johnson, 1992a; Ma & Singh, 1996a; McIntosh & Brown, 1997). Seedling resistance to *P. striiformis* is easily detected in glasshouse studies, whereas adult plant resistance is better expressed in the field.

When stripe rust was observed for the first time on spring wheat grown under rain-fed conditions in the Western Cape during August 1996 (Pretorius *et al.*, 1997), no data on the reaction of South African wheats to this disease were available. During 1997 the situation deteriorated when the stripe rust pathogen spread to the summer rainfall region affecting winter wheat cultivars grown under dry-land conditions and spring wheat cultivars grown under irrigation. Within two years *P.*

striiformis became established as an endemic disease in the major wheat producing areas of South Africa.

The rapid dispersal of *P. striiformis* during the 1996 and 1997 wheat seasons, the occurrence of yield losses due to the destruction of foliage and severe head infections, increased production costs resulting from fungicide applications, and favourable climatic conditions in most wheat growing areas in South Africa necessitated the development of co-ordinated control strategies. The main objective of this study was to determine which South African commercial bread wheat cultivars possess *Yr* genes for seedling and/or adult plant resistance to pathotypes 6E16A- and 6E22A- of *P. striiformis* f. sp. *tritici*. The susceptibility of certain cultivars and lines to head infections, and the stability of resistance over different environments were also studied. Furthermore, selected international wheat cultivars, described in the literature as having high levels of pathotype-non-specific resistance, were evaluated for possible use in local breeding programmes.

MATERIALS AND METHODS

Seedling evaluations

Seedling infection types of 22 spring and 33 winter wheat cultivars were determined using one isolate each of pathotypes 6E16A- and 6E22A- of *P. striiformis* f. sp. *tritici*. Of each cultivar 10-15 seeds were sown in a plastic pot (10 cm diam.), filled with steam-sterilised soil, and grown in a disease-free room at 22± 1°C. Uredospores of each pathotype were multiplied in advance on the susceptible cultivar Morocco. Seven-day old seedlings were inoculated by spraying primary leaves with a suspension of freshly collected stripe rust spores in mineral oil (Soltrol 170). The inoculation booth was rinsed with water after application of each pathotype to avoid contamination. After drying for 2 h in an air-conditioned room, inoculated seedlings were placed in a dew chamber at 11± 1°C and >96 % relative humidity for 30 h. After incubation seedlings were moved to a glasshouse cubicle where a day/night cycle 16/8 h was maintained. Day light was supplemented with cool-white fluorescent tubes emitting photosynthetic active radiation of 120 $\mu E/m^2$. Day and

night temperatures were kept at 17± 2°C. Infection types were recorded 14 to 16 days after inoculation, using a 0 to 4 scale (Appendix 1) (McIntosh *et al.*, 1995). All seedling infection types were confirmed at least twice in independent experiments.

Adult plant evaluations

The response of 22 spring and 33 winter wheat cultivars was determined in field nurseries at Bethlehem from 1997 to 1999. During the 1997 and 1998 seasons plots were naturally infected with pathotype 6E16A-. In 1999 a stripe rust epidemic was initiated by inoculating spreader rows with pathotype 6E22A-. On 1 July 1997 trial entries were planted in 1 m rows spaced 30 cm apart. In 1998 and 1999 trials were split in an early (first week of June) and late (first week of July) planting date. All experiments were arranged according to a randomised block design with two replications. Spreader rows consisted of a mixture of Morocco and McNair and were planted perpendicular to both sides of the row plots. Disease assessments were carried out on six consecutive dates each year, with three to eight day intervals. Disease severity for each cultivar was assessed from visual scores according to the modified Cobb Scale (0-100%) (Peterson *et al.*, 1948) combined with a field reaction type (Appendix 1). Data were recorded separately for the lower (flag leaf –1 and flag leaf –2) and flag leaves during 1997, and only for flag leaves during the following years. Disease severity data were used to calculate the area under the disease progress curve (AUDPC) for trial entries as well as susceptible checks (Schultz & Line, 1992). The total leaf area affected (modified Cobb scale) was used for statistical analyses due to the rapid influence of temperature on stripe rust disease response (McIntosh *et al.*, 1995).

Thirty-seven wheat cultivars included in disease nurseries (rust trap nurseries) during the 1998 and 1999 wheat seasons were evaluated at five different localities to determine the stability of resistance over environments. Cultivars included in disease nurseries were planted in 1 m rows with every 10th row consisting of Morocco. The pathotype(s) occurring at each locality was identified as described (Chapter 2).

Head infections

Stripe rust head infection was determined for 15 spring types in 1997 and for 17 spring wheat cultivars and 17 lines in 1998. For each entry 10 wheat heads were randomly selected, five per replication. A wheat spikelet was regarded as infected when stripe rust pustules were visible on the lemma, gluma, palea or on the seed coat. Stripe rust incidence was then expressed as the percentage infected spikelets per wheat head. The growth stage of cultivars and lines during the determination of percentage head infection varied between 73 and 83 (Zadoks *et al.*, 1974). The relationship between AUDPC and flag leaf severity was also determined.

Evaluation of international cultivars

Selected wheat cultivars described in the literature as having high levels of race non-specific resistance to stripe rust were evaluated in both the seedling and adult plant stages to pts. 6E16A- and 6E22A-. Methods used were similar to those given above.

Statistical analysis

AUDPC and head infection data obtained in each year were analysed for variance (Appendix 3 to 5). Spring and winter wheat cultivars were analysed separately due to differences in growth stage resulting in a slower onset of disease development on flag leaves of those with a winter growth habit. Means obtained for percentage head infection and AUDPC were separated using the Bonferroni multiple comparison test. This test has been recommended for pair-wise comparisons of means (User's Guide, NCSS 97 Statistical System). The statistical programme Genstat 5, 4 th ed. for Windows, was used for all statistical analyses.

RESULTS

Cultivar evaluation

The seedling reaction and field response of 55 South African wheat cultivars to pts. 6E16A- and 6E22A- of *P. striiformis* f. sp. *tritici* are presented in Table 1. The

Table 1. Seedling reaction and field response of 55 South African bread wheat cultivars to pathotypes 6E16A- and 6E22A- of *Puccinia striiformis* f. sp. *tritici*

Wheat cultivar	Yr *gene(s)*	Seedling response[a]		Disease severity[b]		
		pt. 6E16A-	pt. 6E22A-	1997 (pt. 6E16A-)	1998 (pt. 6E16A-)	1999 (pt. 6E22A-)
Spring cultivars						
Adam Tas	*6*[c]	4	4	100MRMS	100MSS	100MS
Chokka	*9*[c]	0, 1p=3	0	0,100MS	0,70MRMS	0,80MRMS
Gamka	-	4	4	-	100MSS	100S
Gamtoos	*9*[c]	0;, 1p=4	0;	0,100S	0,100S	0
Inia	seg *A*[c]	;, 1p=3+	;, 1p=4	0,30MR	0,40MRMS	0,40MS
Kariega	-	2+3	2+3	0,5MR	0,10R	0,5R
Marico	-	3+, 1p=1	3+, 1p=;cn	0,20MR	0,40R	0,20R
Nantes	*6*[c]	3+	3+	100MRMS	100S	100MS
Palmiet	-	4	4	100MRMS	100MSS	100MS
SST 16	seg *6*[c]	4	4	100MS	100MSS	100S
SST 33	-	4, 2p=;	4, 1p=1CN	0,60MR,100MS	0,60MR,100MS	0,100MS
SST 38	-	4, 1p=2	4	5MR,100MS	100MSS	100MS
SST 44	-	3	3	-	5R,60MRMS	30MRMS
SST 55	*6,7*[c]	3+	3	100MRMS	100MRMS	100MRMS
SST 57	-	3	3	15R	30R	30MR

Table 1 (cont.). Seedling reaction and field response of 55 South African bread wheat cultivars to pathotypes 6E16A- and 6E22A- of *Puccinia striiformis* f. sp. *tritici*

Wheat cultivar	Yr *gene(s)*	Seedling response[a]		Disease severity[b]		
		pt. 6E16A-	pt. 6E22A-	1997 (pt. 6E16A-)	1998 (pt. 6E16A-)	1999 (pt. 6E22A-)
Spring cultivars						
SST 65	-	2+3, 3p=1CN	3, 3p=1CN	0,50MR,100S	0,50MR,100S	0,40MR,100S
SST 66	*6*[c]	4	4	100MS	100MSS	100MSS
SST 75	-	4	4	-	-	100MRMS
T4	*A*[c]	;, 1, 2	;, 2	0,5MS	0	0,5R
SST 822	*A*[c]	;	;	0,20MR	0,20MRMS	0,20R,40R
SST 825	*6*[c]	1C, 2p=3	1CN, 1p=3	0,TR	0,TR	TR
SST 876	-	;C, 1p=4	;C, 1p=4	10MR,30MRMS	0,15R,80MRMS	0,20MR,40MR
Winter cultivars						
Belinda	-	2	2	-	40MRR	30MRR
Betta	-	3	3	40MR	40R	30MR
Betta DN	-	3	3	70MRMS	70MRMS	50MRMS
Caledon	-	3	3	40MRMS,80MRMS	40MRR,80MRR	30MRR
Carina	seg *6*[c],*25*[d]	;c, 1p=4	4	0,5MR,100MRMS	0,5MR,90MSS	100MSS
Caritha	-	3, 1p=1CN	3, 1p=1CN	0,20MR,40MS	0,30MR,100MS	0,5R,30MR
Carol	-	4, 1p=;	4, 2p=1CN	0,20MR,100S	0,30MS,100S	10R,100S

Table 1 (cont.). Seedling reaction and field response of 55 South African bread wheat cultivars to pathotypes 6E16A- and 6E22A- of *Puccinia striiformis* f. sp. *tritici*

Wheat cultivar	Yr *gene(s)*	Seedling response[a]		Disease severity[b]		
		pt. 6E16A-	pt. 6E22A-	1997 (pt. 6E16A-)	1998 (pt. 6E16A-)	1999 (pt. 6E22A-)
Winter cultivars						
Elands	-	3	3	-	90MRMS	60MR
Gariep	-	3	3	100MRMS	100MSS	90S
Hugenoot	*25*[d]	;, 1+	3+	0,15MR	0,20MR	100S
Karee	-	4	4	90MRMS	70MSS	80MSS
Letaba	*9*[c]	;, 1p=4	;, 1p=4	0,40MS	0,100MSS	0,60MRMS
Limpopo	-	2+3	2+3	40MRMS	50MRMS	30MRR
Molen	-	4	4	0,30MRMS	5R,50MSS	5R,20R
Molopo	-	3	3	30MRMS	40R	30R
Oom Charl	*9*[c]	0;, 1p=4	0;, 1p=4	0,90MRMS	0,80MRMS	0,70MRMS
PAN 3211	-	3	3	50MRMS	70MRR	50R
PAN 3232	-	2+3	2+3	40MS	40MRR	30R
PAN 3235	-	2+3	2+3	50MRMS	50MRR	30MR
PAN 3349	-	;, 1CN, 2	1CN, 2	0,TR	0,TMS	0,TMR
PAN 3377	-	4	4, 1p=1C	-	0	0,TR

Table 1 (cont.). Seedling reaction and field response of 55 South African bread wheat cultivars to pathotypes 6E16A- and 6E22A- of *Puccinia striiformis* f. sp. *tritici*

Wheat cultivar	Yr *gene(s)*	Seedling response[a]		Disease severity[b]		
		pt. 6E16A-	pt. 6E22A-	1997 (pt. 6E16A-)	1998 (pt. 6E16A-)	1999 (pt. 6E22A-)
Winter cultivars						
SST 102	*6*[c]	3+	3+	50R,70MS	40R	-
SST 107	-	2+3	2+3	50RMS	40R	-
SST 124	-	1CN	1CN, 1p=4	20R	20R	20R
SST 333	-	4	4	40R,80MRMS	40R	20R,40R
SST 363	-	4	4	90MSS	100S	100S
SST 367	-	;	;, 1p=2	5R,15MR	0,15R,40MSS	0,20R,40MR
SST 936	-	;1, 4	;, 4	0,50MR,100S	0,5R,30MRR,60MR	0,30MRMS
SST 966	-	;1, 2, 3	;C, 3	0,10R,20MS	15R	0,20R
SST 972	-	;, 1C, 2	;, 2	0	0	0
Tugela	*25*[d]	;, 1C	3+	-	0	5R
Tugela DN	*25*[d]	;, 1C	3+	0	0	5R

[a]Mean of at least two replications.

[b]Disease severity scores represent the highest percentage flag leaf infection recorded at Bethlehem each year.

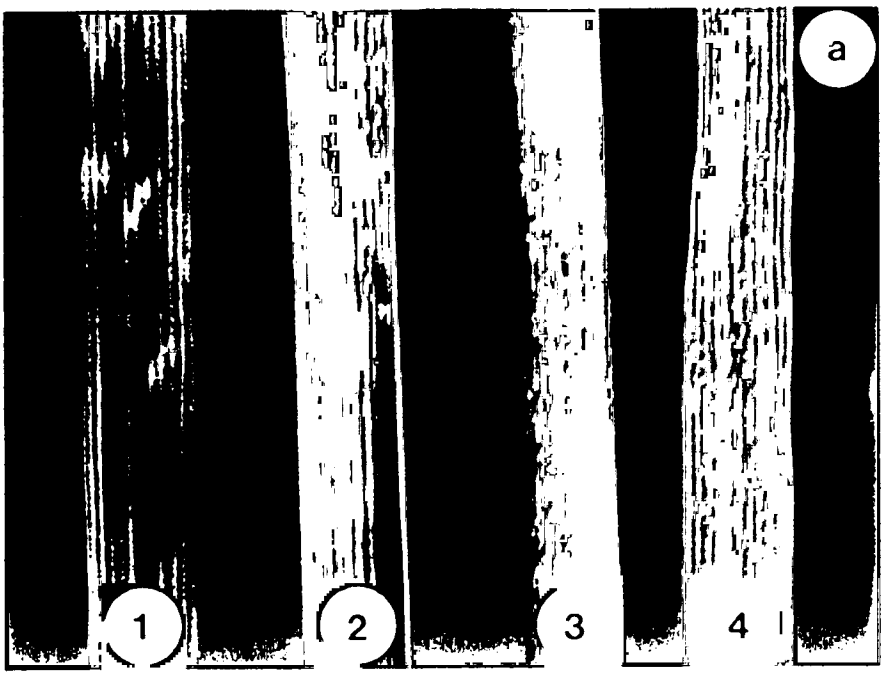
[c]According to Pretorius (1998).

[d]Susceptible to 6E22A- in the seedling stage which indicates the presence of *Yr25*.

cultivars Belinda, PAN 3349, SST 367, SST 822, SST 972 and T4 showed low seedling infection types (<2+) against both pathotypes (Fig. 1). Eighteen cultivars, including SST 825, SST 876, SST 936 and SST 966, appeared heterogeneous for their seedling response to one or both pathotypes, with 31 cultivars being susceptible (≥2+) to either pathotype. The cultivars Hugenoot, Tugela, Tugela DN, and Carina displayed susceptible seedling reactions only to pathotype 6E22A- (Fig. 2), indicating that they carry the gene *Yr25*. The low adult plant response recorded for the cultivars Tugela and Tugela DN indicates the presence of adult plant resistance.

Field responses rated on flag leaves ranged between highly susceptible and highly resistant (Fig. 3). Ten winter and nine spring wheat cultivars expressed AUDPC values ranging from 500 to 1598 (Table 2). Although the latter group of cultivars represents a significantly wide variation in AUDPC they are not expected to provide adequate crop protection based on their disease severity scores presented in Table 1. AUDPC further showed that eight winter and four spring wheat cultivars expressed intermediate levels of resistance (AUDPC 200 to 500). High levels of complete or adult plant resistance were expressed by the winter cultivars Letaba, Caritha, PAN 3349, PAN 3377, SST 367, SST 972, and Tugela DN, as well as by the spring types Kariega, SST 57, SST 822, and SST 825 (AUDPC <200). Cultivars with a flag leaf AUDPC significantly ($P<0.001$) lower than the flag-1 and flag-2 leaves were Betta DN, Caledon, Carol, Gariep, Inia, PAN 3211, PAN 3232, SST 65, SST 936, and SST 966. This may indicate the presence of adult plant resistance. Spring wheats reacting significantly ($P<0.05$) to planting date during 1998 were Adam Tas, Nantes, and SST 38 and during 1999 SST 75, Palmiet, and Nantes. Winter cultivars significantly ($P<0.001$) influenced by planting date in 1998 were Betta DN, Carol, Gariep, PAN 3211, and SST 363. A strong positive correlation was found between AUDPC and terminal severity ratings. These relationships determined over the three year study period were highly significant for both winter ($R^2=0.91$ [$P<0.001$] and spring ($R^2=0.82$ [$P<0.001$] wheat cultivars. Field data recorded at five different localities (Table 3) did not deviate greatly from the data presented in Table 1. High day temperatures and dry conditions resulted in the low disease levels recorded at Bainsvlei.

Fig. 1. (A) Seedling reaction of four spring (1, SST 822; 2, SST 825; 3, Marico; 4, Kariega) and (B) four winter (1, Molen; 2, PAN 3349; 3, SST 124; 4, SST 367) wheat cultivars to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici*.


a
1
2
3
4


b
1
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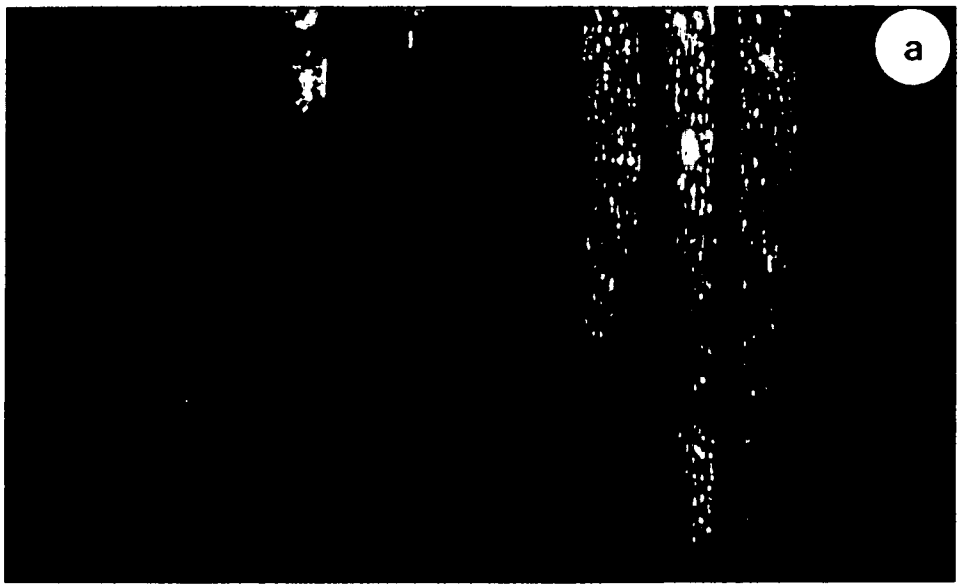
4

Fig. 2. (A) Seedling reaction of Hugenoot, (B) Tugela DN, and (C) Carina to pathotypes 6E16A- (left) and 6E22A- (right) of *Puccinia striiformis* f. sp. *tritici*.


a


b

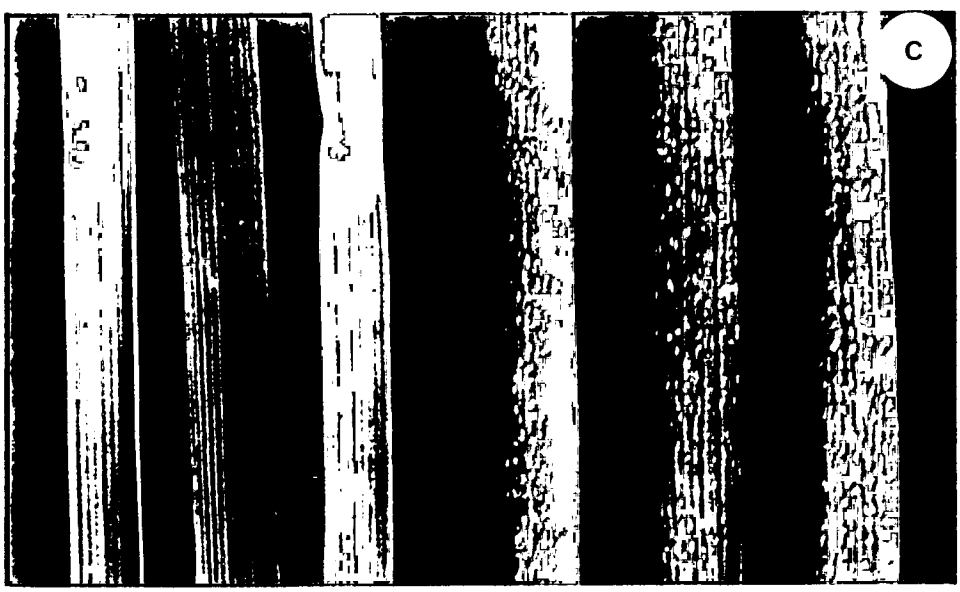

c

Fig 3. (A) Field reaction of spring (1, SST 38; 2, SST 55; 3, SST 57; 4, SST 822) and (B,C) winter (1, Hugenoot; 2, Betta DN; 3, Molen; 4, PAN 3377; (C): (1, Carol; 2, Carina; 3, Caritha; 4, SST 966) wheat cultivars to pathotype 6E22A- of *Puccinia striiformis* f. sp. *tritici*.

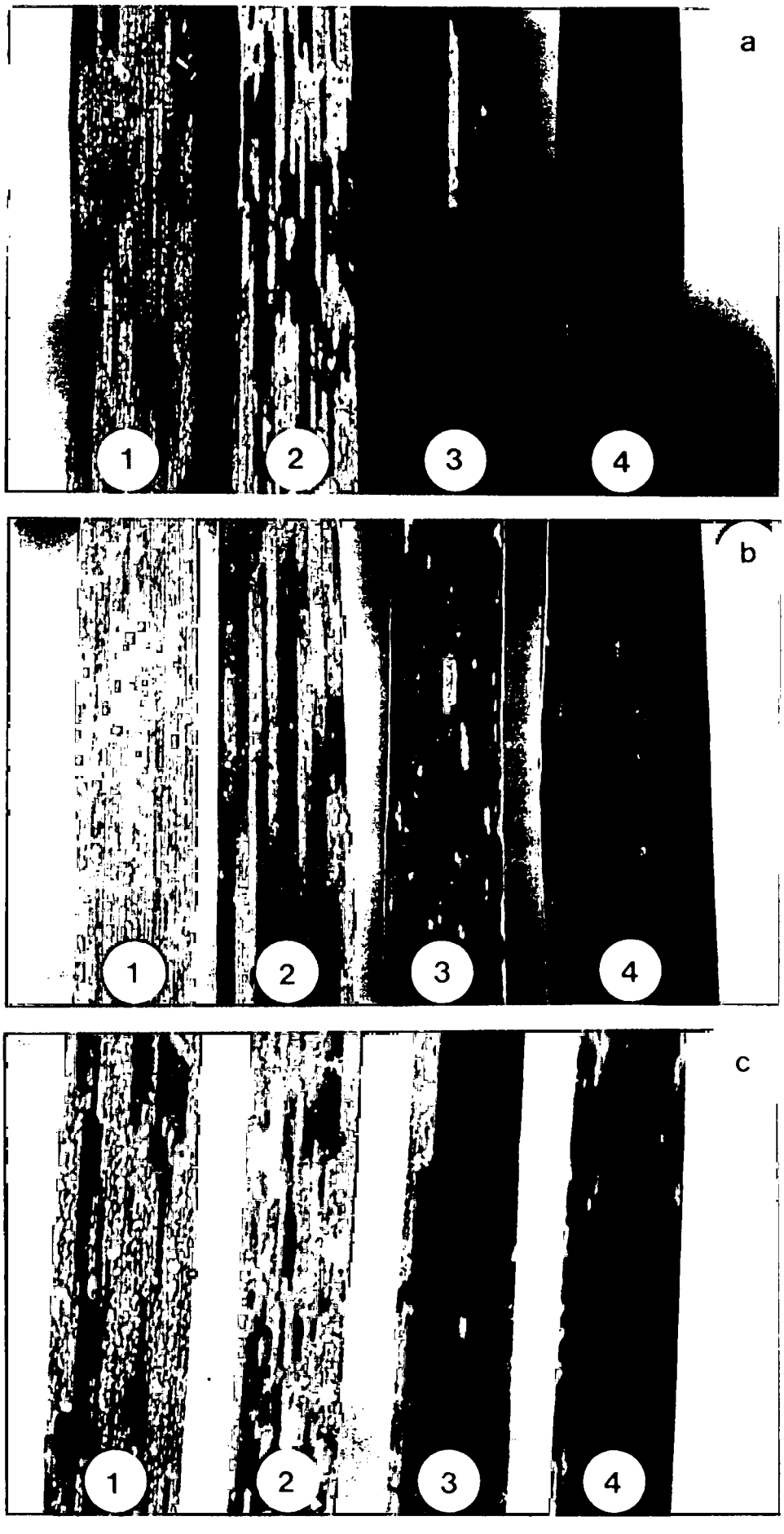

a
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Table 2. Area under the disease progress curve determined for 42 South African bread wheat cultivars and two spreader lines to pathotypes 6E16A- and 6E22A- of *Puccinia striiformis* f. sp. *tritici*

Wheat cultivar	Mean AUDPC	Area under the disease progress curve calculated for trials planted at different dates and years [a]					
		1 July 1997[b] pt. 6E16A-	1 July 1997[c] pt. 6E16A-	1 June 1998[c] pt. 6E16A-	1 July 1998[c] pt. 6E16A-	1 June 1999[c] pt. 6E22A-	1 July 1999[c] pt. 6E22A-
Spring cultivars							
SST825	25.5	0a	39.4a	0a	15.6a	46.2a	51.9a
Kariega	61.7	55a	70a	31.3a	102.5ab	57.5a	53.7a
SST822[d]	147.6	139.4ab	165ab	165.6ab	26.3a	168.8ab	220.6abc
SST57	161.8	99.4ab	220ab	129.4ab	76.2ab	213.1abc	232.5abc
Inia[d]	236.8	400.6b	241.9ab	206.2ab	117.5ab	217.5abc	236.9abc
Marico[d]	325.8	331.3b	365.6b	225ab	250ab	383.8abc	398.8abc
SST876[d]	339.7	-	-	270ab	302.5ab	458.8bc	327.5abc
SST65[d]	417.9	550b	238.1ab	386.9b	301.3ab	558.7cd	472.5bc
SST75	1016.3	-	-	-	-	1150efg	882.5def
Palmiet	1070	907.5c	1207.5def	808.7cd	858.7cde	1202.5fgh	1435ghij
Adam Tas	1144.7	1178.1def	1186.2def	946.9cdef	759.4c	1405ghij	1392.5ghij
Nantes	1181.6	1142.5cde	1358.7ef	755c	958.5cdefg	1327.5ghi	1547.5hijkl
SST33[d]	1191.7	1096.2cd	1295def	803.1cd	870.6cde	1562.5hijkl	1522.5hijk
SST38	1320.8	1171.2de	1418.7fg	1137.5efgh	942.5cdef	1565hijkl	1690ijklm

Table 2 (cont.). Area under the disease progress curve determined for 42 South African bread wheat cultivars and two spreader lines to pathotypes 6E16A- and 6E22A- of *Puccinia striiformis* f. sp. *tritici*

Wheat cultivar	Mean AUDPC	Area under the disease progress curve calculated for trials planted at different dates and years[a]					
		1 July 1997[b] pt. 6E16A-	1 July 1997[c] pt. 6E16A-	1 June 1998[c] pt. 6E16A-	1 July 1998[c] pt. 6E16A-	1 June 1999[c] pt. 6E22A-	1 July 1999[c] pt. 6E22A-
Spring cultivars							
SST16	1543	1657.5gh	1755h	1105.6defgh	1115defgh	1732.5jklm	1892.5lmn
SST55	1594.2	1670h	1707.5h	1216.2fghi	1298.7hij	1852.5klmn	1820klmn
SST66	1597.9	1627.5gh	1785hi	1274.4ghij	1285.6hij	1747.5jklmn	1867.5klmn
Morocco[e]	1901.7	2162.5i	2027.5i	1565j	1520ij	2035mn	2100n
L.s.d.[f]		244.78 (P=0.00005)		323.42 (P=0.00004)		367.1 (P=0.00004)	
Winter cultivars							
Letaba[d]	0	0a	0a	0a	0a	0a	0a
SST 972	0	-	-	0a	0a	0a	b
PAN 3377	7.2	-	-	0a	0a	0a	28.8ab
Tugela DN	19.2	0a	0a	0a	0a	57.5abc	57.5abc
PAN 3349	30.15	-	-	11.2a	0a	51.9ab	57.5abc
SST 367[d]	164.4	156.3abcd	115abcd	151.3abcdef	145abcdef	203.1abcd	215.6abcd
Caritha[d]	168.85	272.5abcde	72.5abc	98.1abcd	106.2abcd	205abcd	258.8abcdef
SST 966[d]	223.4	368.8bcdef	145abcd	168.1abcdefg	140abcde	289.4bcdef	228.8abcde

Table 2 (cont.). Area under the disease progress curve determined for 42 South African bread wheat cultivars and two spreader lines to pathotypes 6E16A- and 6E22A- of *Puccinia striiformis* f. sp. *tritici*

Wheat cultivar	Mean AUDPC	Area under the disease progress curve calculated for trials planted at different dates and years [a]					
		1 July 1997[b] pt. 6E16A-	1 July 1997[c] pt. 6E16A-	1 June 1998[c] pt. 6E16A-	1 July 1998[c] pt. 6E16A-	1 June 1999[c] pt. 6E22A-	1 July 1999[c] pt. 6E22A-
Winter cultivars							
SST 124	255.1	313.1abcde	273.8abcde	158.8abcdefg	172.5abcdefg	290bcdefg	322.5cdefgh
Molen[d]	286.6	435defgh	305abcde	216.9abcdefgh	241.9abcdefghi	230.6abcde	290bcdefg
SST 333[d]	387.6	388.8bcdef	420cdefgh	305.6defghij	396.3ghijk	400defghi	415defghi
PAN 3232	412.8	752.5hij	350abcdef	303.7defghij	281.9cdefghij	401.3defghi	387.5defghi
Limpopo	419.5	408.8bcdefgh	397.5bcdefgh	476.3ijkl	385.6fghijk	361.3defghi	487.5efghi
PAN 3235	474.5	668.7efghi	657.5efghi	381.9efghijk	288.8cdefghij	441.3defghi	408.8defghi
SST 936[d]	479.9	985ijkl	375bcdef	346.9efghij	283.8cdefghij	450defghi	438.8defghi
Hugenoot	565.6	0a	0a	23.1ab	26.2ab	1627.5n	1717.5n
Elands	573.8	-	-	-	-	557.5hi	590i
Caledon[d]	588.4	1132.5klm	950ijk	361.3efghijk	263.8bcdefghij	391.3efghi	431.3defghi
Carina[d]	603	195.6abcd	57.5ab	55abc	70abcd	1657.5n	1582.5mn
Betta DN	657.5	967.5ijkl	692.5fghi	658.1lmn	499.4jkl	515fghi	612.5i
PAN 3211	681.55	1310klm	627.5defghi	603.1klm	436.2hijkl	557.5hi	555ghi
Karee	807.2	740ghij	1145klm	479.4ijkl	473.7ijkl	1037.5jk	967.5j

Table 2 (cont.). Area under the disease progress curve determined for 42 South African bread wheat cultivars and two spreader lines to pathotypes 6E16A- and 6E22A- of *Puccinia striiformis* f. sp. *tritici*

Wheat cultivar	Mean AUDPC	Area under the disease progress curve calculated for trials planted at different dates and years [a]					
		1 July 1997[b] pt. 6E16A-	1 July 1997[c] pt. 6E16A-	1 June 1998[c] pt. 6E16A-	1 July 1998[c] pt. 6E16A-	1 June 1999[c] pt. 6E22A-	1 July 1999[c] pt. 6E22A-
Winter cultivars							
Gariep	1107.7	1437.5mn	1207.5klm	1005o	873.7no	997.5jk	1125jkl
SST 363	1164.8	1253.7klm	1317.5lm	873.7no	996.2o	1197.5jkl	1350lm
McNair[e]	1256.9	1202.5klm	1271.9klm	1403.7p	1315.6p	1257.5kl	1090jkl
Carol[d]	1391.9	1778.7n	1061.3jkl	1407.5p	786.9mno	1657.5n	1660n
L.s.d.[f]		360.72 (P=0.00003)		244.47 (P=0.00002)		265.39 (P=0.00008)	

[a]AUDPC values followed by the same letter do not differ significantly according to the Bonferroni multiple comparison test.

[b]AUDPC calculated from disease ratings of the lower leaves (flag leaf -1 and flag leaf -2).

[c]AUDPC calculated from disease ratings on flag leaves.

[d]Cultivars regarded as heterogeneous for their stripe rust field reaction (Table 1); the most representative field reactions were used to calculate the AUDPC.

[e]Rust spreader lines.

[f]Least significant differences as determined by the Bonferroni multiple comparison test.

Table 3. Field response[a] of 37 South African wheat cultivars to pathotypes 6E16A- and 6E22A- of *Puccinia striiformis* f. sp. *tritici* evaluated at five different localities in South Africa during 1998 and 1999

Wheat cultivar	Locality					
	Cradock 1998 pt. 6E16 A-	Kranzfontein 1998 pt. 6E22 A-	Burgershall 1998 pt. 6E16A-	Bainsvlei 1998 pt. 6E16A-	Burgershall 1999 pt. 6E16A-	Greytown 1998 pt. 6E22A-
Spring cultivars						
Adam Tas	60MRMS	100S	80MS	0	10S	50MRMS
Inia	0	30MS	0	0	0	0,70S
Kariega	0	0,5R,40MR	0	0	0	TR
Marico	30MRMS	0,50MS	5MR	0	5S	10R
Nantes	80MRMS	100S	90MS	0	10S	70MSS
Palmiet	50MRMS	100S	40MRMS	0	5S	90S
SST16	90S	100S	80MS	0	60MS	70MSS,30MRR
SST 38	100S	100S	50MRMS,80MS	TS	40S	50MSS
SST 55	90MR	100S	80MRMS	TS	50S	70MRMS
SST 57	15MR	20R	TR,15MR	0	5S	5R
SST 65	0,40MR	0,40MR,100S	0,10MR,20MS	0	0	10RMR,30MS
SST 66	100S	100S	80MRMS	0	10S	80MSS
SST 822	0,5MS	10R	TMR	0	5S	TR
SST 825	0	0	0	0	0	TR

Table 3 (cont.). Field response[a] of 37 South African wheat cultivars to pathotypes 6E16A- and 6E22A- of *Puccinia striiformis* f. sp. *tritici* evaluated at five different localities in South Africa during 1998 and 1999

Wheat cultivar	Locality					
	Cradock 1998	Kranzfontein 1998	Burgershall 1998	Bainsvlei 1998	Burgershall 1999	Greytown 1998
	pt. 6E16A-	pt. 6E22A-	pt. 6E16A-	pt. 6E16A-	pt. 6E16A-	pt. 6E22A-
Spring cultivars						
SST 876	0,30MS	0,40MRR	0,5MRMS	0	5S	TR,20MR
Winter cultivars						
Betta DN	20MRMS	40MR	30MRR	0	0	60MRMS
Caledon	20MRMS	30MRR	10R	0	0	40MR
Carina	0,20S	90MSS	0,5MRMS	0	0	30MRMS
Caritha	0,20S	0,70S	5R,30MRMS	0	0	40MR
Carol	0,40S	0,100S	0,50MRMS	TS	40S	30MR,100S
Gariep	30MRMS	80MRMS	30MRMS	TMS	10MS	60MSMR
Hugenoot	0	90S	0	0	0	60MRMS
Letaba	0	0,90S	0	0	0,10S	0,30MR
Limpopo	10MRMS	20MR	10MRMS	0	0	40RMR
Molen	20MRMS	30S	10MR	0	0	30RMR
PAN 3211	10MS	20MRR	10MR	0	5MR	50MRMS
PAN 3232	TMS	15MRR	5MR	0	0	50MR

Table 3 (cont.). Field response[a] of 37 South African wheat cultivars to pathotypes 6E16A- and 6E22A- of *Puccinia striiformis* f. sp. *tritici* evaluated at five different localities in South Africa during 1998 and 1999

Wheat cultivar	Locality					
	Cradock 1998 pt. 6E16A-	Kranzfontein 1998 pt. 6E22A-	Burgershall 1998 pt. 6E16A-	Bainsvlei 1998 pt. 6E16A-	Burgershall 1999 pt. 6E16A-	Greytown 1998 pt. 6E22A-
Winter cultivars						
PAN 3235	TMS	15MRR	10MR	0	5S	50MR
PAN 3349	0	0	0	0	0	5R,30MR
SST 124	10R	20R	0,10MS	0	0	40RMR
SST 333	30R	0,40R	TMR	0	0	40MR
SST 363	70S	60R	40MRMS	0	5S	60S
SST 367	0	0	0	0	0	20R
SST 936	0,40S	0,20R	5MR	0	0	10R,30MR
SST 966	0,20S	10R	5MR	0	0	5R
SST 972	0	0	0	0	0	TR
Tugela DN	0	5R	0	0	0	5R,30MRMS

[a]Disease severity scores represent the highest percentage flag leaf infection recorded at each locality.

Head infections

A high percentage head infection was observed for some cultivars (Fig. 4). Percentage head infection varied from 0 to 95% (Table 4). Cultivars and lines expressing high levels of resistance in both seedling and adult plant stages showed no or very low percentages of head infection. For example, the Avocet lines carrying major genes effective against stripe rust pathotypes 6E16A- and 6E22A-, were highly resistant to head infections. In contrast, the percentage head infection obtained for susceptible Avocet lines varied between 48 and 55%. SST 57, Kariega, and Marico, which are susceptible in the seedling stage but expressing moderate to high levels of adult plant resistance, varied in their susceptibility to head infection. The mean stripe rust infection for SST 57 over the two years was 6% followed by Kariega with 8% and Marico with 18%. Head infections were common in cultivars and lines susceptible in both seedling and adult plant stages. The mean percentage head infection recorded over two seasons for this group was 39%. This resulted in a positive relationship between head infection and AUDPC (R^2=0.78, P<0.001 [1997] and R^2=0.84, P<0.001 [1998]).

Evaluation of international cultivars

Of the 18 cultivars evaluated 10 showed low infection types (<2+) in the seedling stage, indicating the presence of major gene(s) effective against pathotypes 6E16A- and 6E22A- of *P. striiformis* f. sp. *tritici* (Table 5). The presence of seedling resistance in the cultivars Elite Lepeule and Flinor has not been reported previously and should therefore be confirmed using a different seed source. In the remaining cultivars compatible seedling reactions were contrasted by high levels of adult plant resistance in the field.

DISCUSSION

The 55 commercial cultivars evaluated in this study reflect the present status of stripe rust resistance in South African bread wheat. Considering the recent introduction of stripe rust in South Africa it is not surprising that 73% of the cultivars

Fig 4. Wheat spikelet of SST66 severely infected with *Puccinia striiformis* f. sp. *tritici*.

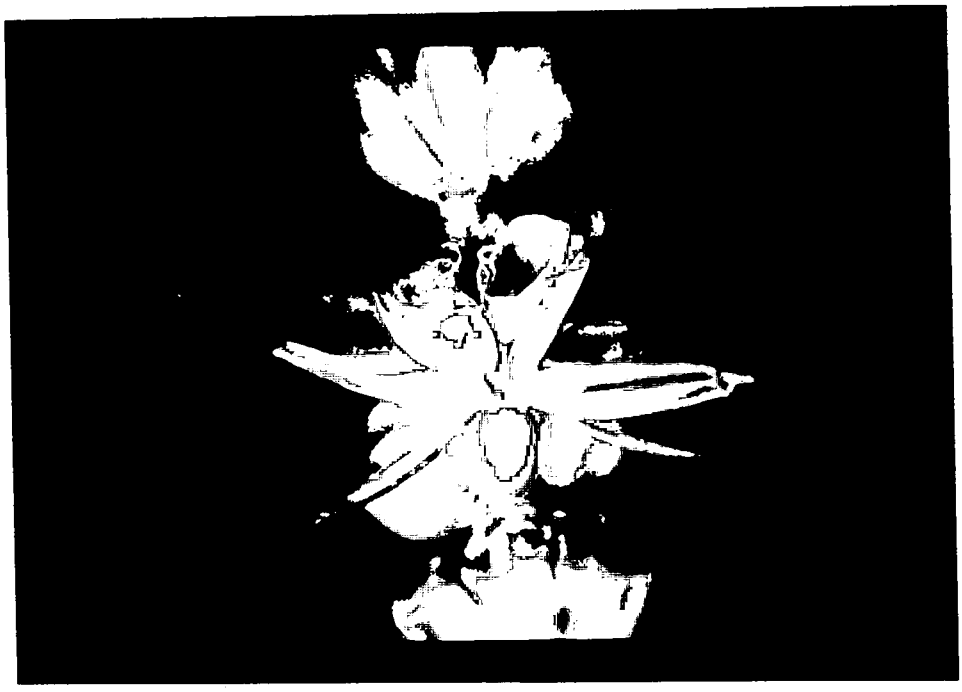

Table 4. Percentage wheat head infection and AUDPC determined for 16 spring wheat cultivars and 17 lines in field trials at Bethlehem to *Puccinia striiformis* f. sp. *tritici* in 1997 and 1998

Wheat cultivar/line	*Yr* gene(s)	Seedling reaction (pt. 6E22A-)	% Head infection[a,b,c] 1997 (pt. 6E16A-)	AUDPC 1997	% Head infection[c] 1999 (pt. 6E22A-)	AUDPC 1999
Wheat cultivars						
Chokka	*9*[d]	0	0a	0a	0a	0a
Gamtoos	*9*[d]	0;	0a	0a	0a	0a
Inia	seg *A*[d]	;	0a	321a (112)	2a (0.25)	227abc (14)
SST822	*A*[d]	;	0.85ab (0.06)	152a (18)	2.29a (0.28)	195abc (37)
SST825	*6*[d]	1C, 3	-	-	3.64a (0.18)	49ab (4)
SST65	-	3, 1CN	2.51ab (0.78)	394a (221)	15.68cdef (0.31)	515c (61)
SST57	-	3	5.44bc (0.56)	160a (85)	6.16ab (0.04)	223abc (14)
Kariega	-	2+3	13.02de (0.84)	60a (14)	3.9a (0.08)	56ab (3)
SST75	-	4	-	-	12.98bcde (1.21)	1016de (189)
Adam Tas	*6*[d]	4	4.8abc (1.92)	1182b (6)	21.67efg (4.82)	1399fg (9)
Marico	-	3+	16.63e (0.03)	348a (24)	19.98defg (6.74)	393bc (93)
Nantes	*6*[d]	3+	8.9cd (1.36)	1251bc (153)	24.44fgh (3.89)	1438fg (156)
Palmiet	-	4	31.88f (0.18)	1133b (106)	46.81lmop (0.81)	1319efg (164)
SST55	*6,7*[d]	3+	32.27f (2.28)	1689cd (27)	26.38ghi (0.47)	1836ij (23)
SST66	*6*[d]	4	33.81f (0.22)	1706cd (111)	31.96hijk (0.09)	1808hij (85)

Table 4 (cont.). Percentage wheat head infection and AUDPC determined for 16 spring wheat cultivars and 17 lines in field trials at Bethlehem to *Puccinia striiformis* f. sp. *tritici* in 1997 and 1998

Wheat cultivar/line	*Yr* gene(s)	Seedling reaction (pt. 6E22A-)	% Head infection[a,b,c] 1997 (pt. 6E16A-)	AUDPC 1997	% Head infection[c] 1999 (pt. 6E22A-)	AUDPC 1999
Wheat cultivars						
SST38	-	4	52.11g (1.37)	1295bc (175)	38.12jkl (0.86)	1628ghi (88)
Supplemental lines						
*Yr*1/6*AvS	*1*	;	-	-	0a	0a
Federation/4*Kavkaz	*9*	;	-	-	0a	0a
*Yr*5/6*AvS	*5*	0;	-	-	0a	0a
*Yr*9/6*AvS	*9*	0;	-	-	0a	0a
*Yr*10/6*AvS	*10*	0;	-	-	0a	0a
*Yr*15/6*AvS	*15*	;	-	-	0a	0a
Yr27/3*AvS	*27*	;	-	-	0a	0a
YrSp/3*AvS	*Sp*	;	-	-	0a	43ab (60)
Kalyansona	*2+*	4	-	-	6.69abc (1.24)	228abc (17)
Trident	*17*	4	-	-	15.64cdef (1.27)	900d (127)
Federation 1221	-	4	-	-	35.25ijk (5.36)	1188def (244)
Jupateco R	*18*	4	-	-	40.43klm (2.19)	361bc (51)
Avocet S	-	4	-	-	47.74mnop (2.04)	1848ij (32)

Table 4 (cont.). Percentage wheat head infection and AUDPC determined for 16 spring wheat cultivars and 17 lines in field trials at Bethlehem to *Puccinia striiformis* f. sp. *tritici* in 1997 and 1998

Wheat cultivar/line	*Yr* gene(s)	Seedling reaction (pt. 6E22A-)	% Head infection[a,b,c] 1997 (pt. 6E16A-)	AUDPC 1997	% Head infection[c] 1999 (pt. 6E22A-)	AUDPC 1999
Supplemental lines						
*Yr*6/6*AvS	*6*	3	-	-	49.65nop (0.91)	1618ghi (67)
Jupateco S	-	4	-	-	52.49op (2.49)	1378fg (18)
*Yr*7/6*AvS	*7*	3	-	-	54.9p (2.27)	1618ghi (67)
Morocco	-	4	94.53h (2.01)	2095d (96)	94.89q (0.91)	2051j (23)
L.s.d.[e]			4.88 (P=0.0002)	470.65 (P=0.0002)	9.19 (P=0.00005)	355.49 (P=0.00005)

[a]Mean percentage head infection and AUDPC values followed by the same letter do not differ significantly according to the Bonferroni multiple comparison test.

[b]Standard deviations are given in brackets.

[c]Percentage wheat head infection and AUDPC were highly correlated during 1997 (R^2=0.78, P=0.001)=0.57 and during 1999 (R^2=0.84, P=0.001)=0.34.

[d]According to Pretorius (1998).

[e]Least significant differences as determined by the Bonferroni multiple comparison test.

Table 5. Seedling and field response of international wheat cultivars, known for high levels of non-specific resistance to pathotypes 6E16A- and 6E22A- of *Puccinia striiformis* f. sp. *tritici*

Wheat cultivar	Given *Yr* gene(s)	Seedling response		Field response[a]	
		pt. 6E16A-	pt. 6E22A-	1998 (pt. 6E16A-)	1999 (pt. 6E22A-)
Spring cultivars					
Anza	*A,18*[b]	;	;	5R	TR
Bass	-[c]	3	3	10R	5R
Cook	-[c]	4	4	10R	5R
Harrier	-[d]	3	3+	10R	5R
Oxley	*6*+[e]	3	3+	10R	5R
Parula	*18*+[f]	2+3	3	0	0
Pavon 76	*6,7*+[g]	2+3	3	15R	20R
Winter cultivars					
Bouqet	*3a,4a,14,16*?[b]	;	;	0	0
Capelle Desprez	*3a,4a,16*[b]	;	;	0	0
Druchamp	*3a,4a,Dru,Dru2*[h]	;	;	0	0
Elite Lepeule	*2*[b]	;	;	0	0
Flinor	-[b]	;	;	0	0
Ibis	*1,2,13*[b]	1	1C	0	0
Joss Cambier	*2,3a,11*[b]	;, 1p=2	;	0	0
Luke	-[b]	4	3+	15R	20R
Maris Huntsman	*2,3a,4a,13,16*?[b]	;	;	0	0
Nugaines	-[b]	3+	4	20R	20R
Stephens	*3a,Ste,Ste2*[h]	;	;	0	0

[a]Disease severity values represent the highest percentage flag leaf infection recorded for each entry in field trials at Bethlehem during 1998 and 1999.

[b]Roelfs *et al.* (1992); Park *et al.* (1988); [c]Park & Rees (1989).

[d]Bariana & McIntosh (1995); [e]Wellings & McIntosh (1990); [f]Broers *et al.* (1996).

[g]Dubin *et al.* (1989); Wellings & McIntosh (1990); Ma & Singh (1996b).

[h]Chen & Line (1995a,b); Chen *et al.* (1996).

were susceptible or appeared heterogeneous for their response to pathotypes 6E16A- and 6E22A-. Single gene resistance, controlled by the pathotype-specific genes *YrA* (incomplete) and *Yr9* (complete) (Pretorius, 1998), was expressed by the cultivars Chokka, Gamtoos, Inia, Letaba, Oom Charl, SST 822, and T4, most which gave heterogeneous seedling and field reactions. The primary leaf reactions of the cultivars PAN 3349, Scheepers 69, SST 124, SST 367, SST 825, SST 876, SST 936, SST 966, and SST 972 indicate the presence of unidentified seedling genes. The high mutation potential of the stripe rust pathogen could alter the current resistance status in these cultivars. Seedling genes identified in local cultivars that are not effective to one or both of the local pathotypes are *Yr6, 7* and *Yr25* (Pretorius, 1998; Boshoff & Pretorius, 1999).

Intermediate to high levels of adult plant resistance was expressed by Caritha, Kariega, Limpopo, Marico, Molen, PAN 3232, PAN 3235, PAN 3377, SST 57, SST 65, SST 333, Tugela and Tugela DN. In addition the cultivars Betta DN, Caledon, Carol, Gariep, Inia, PAN 3211, SST 966, and SST 936 showed a significant decrease in AUDPC on their flag leaves in comparison to the AUDPC determined for the two lower leaves in 1997. Difference in growth stages is one factor other than the presence of adult plant resistance genes that may have contributed to the latter. Short-season wheats such as Inia and Gariep reached the flag leaf stage in advance of the 1997 epidemic whereas flag leaves of those with a longer growing period, e.g. Carol, SST 936, and SST 966 escaped the onset of epidemic development. Adult plant resistance to stripe rust is present in many bread wheat cultivars (Park *et al.*, 1988; Singh & Rajaram, 1994; Bariana & McIntosh, 1995; Chen & Line, 1995b; Broers *et al.*, 1996; Ma & Sing, 1996b). Some adult plant resistance genes to stripe rust, e.g. *Yr18*, have remained durable (Chen & Line, 1995b; Line & Chen, 1995; Ma & Singh, 1996a; McIntosh & Brown, 1997), whereas others such as *Yr11, 12, 13 and Yr14*, are race-specific (Johnson, 1992b; McIntosh *et al.*, 1995; McIntosh & Brown, 1997). Whether the adult plant resistance detected in local cultivars will be durable remains unknown. Assumptions about durability are only possible if the genetics of resistance in such cultivars have been studied in detail and if pathogenicity is monitored regularly in the region of interest.

Knowledge of the genetic basis of resistance is also useful in understanding the distribution of pathotypes (Perwaiz & Johnson, 1986). The current absence of local stripe rust pathotypes with complex virulence factors that can overcome most seedling genes confounds selection for adult plant resistance. This is especially apparent during early generations when seedling tests are not conducted. However, this problem may in part be overcome by determining the genetic basis of resistance in the parents of crosses (Singh & Rajaram, 1994). The cultivars Bass, Cook, Harrier, Oxley, Parula, Pavon 76, Luke, and Nugaines showed susceptible seedling reactions but high levels of adult plant resistance, qualifying them as useful donor parents in crosses with local susceptible cultivars. The value of adult plant resistance was emphasized by Ma & Singh (1996a) who showed that *Yr18* can reduce the percentage loss in wheat grain yield between 36 and 58%, depending on the year and sowing date. Selection for gene *Ltn*, which confers leaf tip necrosis in adult plants (Singh, 1992), can be used as a morphological marker for the identification of the adult plant gene *Yr18* (Singh, 1993) and should thus be used during selection.

Resistance expressed by the cultivars evaluated appeared stable over different environments. Environmental differences, especially temperature, may have a dramatic influence on the expression of stripe rust resistance (Broers *et al.* 1996; Khan *et al.*, 1998; Shang & Shang, 1998). Factors other than temperature that may explain differences in stripe rust response of wheat cultivars, evaluated at different field localities, may be different sowing dates, growth stages, observation dates and crop management practices, and pathogenic variation (Daamen *et al.*, 1989; Ash & Brown, 1991; Ellison & Murray, 1992; Danial & Parlevliet, 1995; Bariana & McIntosh, 1995). Differences in growth stage are most likely responsible for the small percentage of cultivars influenced by planting date in this study. South African wheat cultivars have different vernalisation requirements resulting in differences in growth stage. In this study the AUDPC and final disease severity ratings were strongly correlated for both spring and winter cultivars. This implies that stripe rust responses recorded at the end of the respective epidemic were a good indication of the levels of resistance or susceptibility in local cultivars. However, a complex of

foliar diseases may negate clear differentiation of stripe rust responses late in the season.

The infection of wheat heads by *P. striiformis* f. sp. *tritici* can be destructive, resulting in yield losses of more than 34% in susceptible cultivars (Cromey, 1989a). Furthermore, grain from infected florets can weigh up to 77% less than grain from uninfected florets (Cromey, 1989b). Purdy & Allan (1965) reported that cultivars susceptible in the seedling stage, but resistant as adult plants, endured yield losses of up to 20%. Similar to the results of the present study Cromey (1989a) found a positive correlation between leaf and head resistance for most wheat cultivars. Cultivars and lines with seedling resistance showed no or very low levels of head infection, whereas seedling-susceptible cultivars had moderate to high levels of head infection. Compared to SST 57, Kariega, Marico and Jupateco R exhibited more head infection than would have been expected from their AUDPC scores. The opposite was found for Adam Tas and Nantes during 1997 when large AUDPC values did not correlate with less head infection. However, this could not be confirmed in 1998. Cromey (1989a) suggested that differences in head infection of the same cultivar at different localities could be due to the influence of environmental differences. Inoculum pressure, viability of inoculum, and differences in growth period among cultivars could further contribute to differences in head infections at different localities. However, stripe rust spores required for head infections are most likely to come from infected leaves within the crop (Rapilly, 1979; Cromey, 1989a). Cultivars with high levels of foliar resistance, but which are susceptible to head infection, should therefore not be severely infected in commercial fields unless nearby epidemics can serve as inoculum sources.

The susceptibility of several wheat cultivars, favourable climatic conditions in the most important wheat growing areas and the additional costs of fungicide application qualify stripe rust as an important disease with strong impact on South African wheat production. This presents a new challenge to the local wheat industry. Only a low percentage of the cultivars evaluated expressed acceptable levels of resistance, with an even lower percentage expressing adult plant resistance that may be durable. There are, however, several sources of resistance available that can be utilised in breeding programmes. If these sources are deployed with the

necessary responsibility, the control of stripe rust in South Africa through the use of resistant varieties is possible. Future breeding efforts should be directed towards strategies to obtain effective and durable resistance against stripe rust. The release of cultivars containing pathotype-specific resistance genes only should be strongly discouraged. Selection of resistance sources for application in breeding programmes should focus almost entirely on genotypes with known durability.

LITERATURE CITED

Akanda, S.I. & Mundt, C.C. 1997. Effect of two-component cultivar mixtures and yellow rust on yield and yield components of wheat. Plant Pathology 46: 566-580.

Ash, G.J. & Brown, J.F. 1990. Yield losses in wheat caused by stripe rust (*Puccinia striiformis* West.) in northern New South Wales. Australian Journal of Experimental Agriculture 30: 103-108.

Ash, G.J. & Brown, J.F. 1991. Effects of nitrogen nutrition of the host on the epidemiology of *Puccinia striiformis* f.sp. *tritici* and crop yield in wheat. Australasian Plant Pathology 20: 108-114.

Bariana, H.S. & McIntosh, R.A. 1993. Cytogenetic studies in wheat. XV. Location of rust resistance genes in VPM1 and their genetic linkage with other disease resistance genes in chromosome 2A. Genome 36: 476-482.

Bariana, H.S. & McIntosh, R.A. 1995. Genetics of adult-plant stripe rust resistance in four Australian wheats and the French cultivar 'Hybride-de-Bersee'. Plant Breeding 114: 485-491.

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

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 Subias, X. & López-Atilano, R.M. 1996. Field assessment of quantitative resistance to yellow rust in ten spring wheat cultivars. Euphytica 90: 9-16.

Chen, X.M. & Line, R.F. 1995a. Gene action in wheat cultivars for durable, high-temperature, adult-plant resistance and interaction with race-specific, seedling resistance to *Puccinia striiformis*. Phytopathology 85: 567-572.

Chen, X.M. & Line, R.F. 1995b. Gene number and heritability of wheat cultivars with durable, high-temperature, adult-plant (HTAP) resistance and interaction of HTAP and race-specific seedling resistance to *Puccinia striiformis*. Phytopathology 85: 573-578.

Chen, X.M., Jones, S.S. & Line, R.F. 1996. Chromosomal location of genes for resistance to *Puccinia striiformis* in seven wheat cultivars with resistance genes at the *Yr*3 and *Yr*4 loci. Phytopathology 86: 1228-1233.

Cromey, M.G. 1989a. Infection and control of stripe rust in wheat spikes. New Zealand Journal of Crops and Horticultural Science 17: 159-164.

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

Daamen, R.A., Wijnands, F.G. & Van der Vliet, G. 1989. Epidemics of diseases and pests of winter wheat at different levels of agrochemical input. Journal of Phytopathology 125: 305-319.

Danial, D.L. 1994. Aspects of durable resistance in wheat to yellow rust. Ph.D Thesis. Wageningen. 143pp.

Danial, D.L. & 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.

Dubin, H.J., Johnson, R. & Stubbs, R.W. 1989. Postulated genes for resistance to stripe rust in selected CIMMYT and related wheats. Plant Disease 73: 472-475.

Ellison, P.J. & 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.

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

Gaunt, R.E. & Cole, M.J. 1991. An analysis of yield reduction caused by stripe rust in Rongotea wheat. Australian Journal of Agricultural Research 42: 45-52.

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.

Jørgensen, L.N. & Nielsen, B.J. 1994. Control of yellow rust (*Puccinia striiformis*) on winter wheat by ergosterol inhibitors at full and reduced dosages. Crop Protection 13: 323-330.

Khan, M.A., Irshad, M., Ilyas, M.B. & Hussain, M. 1998. Relationship of environmental conditions with leaf and stripe rust development on wheat. Pakistan Journal of Agricultural Sciences 35: 34-38.

Line, R.F. & Chen, X. 1995. Successes in breeding for and managing durable resistance to wheat rusts. Plant Disease 79: 1254-1255.

Ma, H. & Singh, R.P. 1996a. Contribution of adult plant resistance gene *Yr18* in protecting wheat from yellow rust. Plant Disease 80: 66-69.

Ma, H. & Singh, R.P. 1996b. Expression of adult resistance to stripe rust at different growth stages of wheat. Plant Disease 80: 375-379.

Ma, H., Singh, R.P. & Mujeeb-Kazi, A. 1995. Resistance to stripe rust in *Triticum turgidum*, *T. tauschii* and their synthetic hexaploids. Euphytica 82: 117-124.

McIntosh, R.A. 1980. Stripe rust – A new wheat disease for Australia. The University of Sydney Plant Breeding Institute, Castle Hill. Bulk Wheat. pp65-67.

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

McIntosh, R.A., Wellings, C.R. & Park, R.F. 1995. Wheat rusts: An atlas of resistance genes. Kluwer, Dordrecht. 200pp.

Mundt, C.C., Brophy, L.S. & Kolar, C. 1996. Effect of genotype unit number and spatial arrangement on severity of yellow rust in wheat cultivar mixtures. Plant Pathology 45: 215-222.

Murray, G.M., Ellison, P.J., Watson, A. & Cullis, B.R. 1994. The relationship between wheat yield and stripe rust as affected by length of epidemic and

temperature at the grain development stage of crop growth. Plant Pathology 43: 397-405.

Park, R.F. & 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., Rees, R.G & Platz, G.J. 1988. Some effects of stripe rust infection in wheats with adult-plant resistance. Australian Journal of Agricultural Research 39: 55-62.

Perwaiz, M.S. & Johnson, R. 1986. Genes for resistance to yellow rust in seedlings of wheat cultivars from Pakistan tested with British isolates of *Puccinia striiformis*. Plant Breeding 97: 289-296.

Peterson, R.F., Campbell, A.B. & 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. Unpublished report, Department of Plant Pathology, University of the Orange Free State, Bloemfontein, South Africa. 72pp.

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

Purdy, L.H. & Allan, R.E. 1965. Stripe rust head infection in five Pacific northwest wheats. Plant Disease Reporter 49: 335-338.

Rapilly, F. 1979. Yellow rust epidemiology. Annual Review of Phytopathology 17: 59-73.

Roelfs, A.P., Singh, R.P. & Saari, E.E. 1992. Rust Diseases of Wheat: Concepts and methods of disease management. Mexico, D.F.: Cimmyt. 81pp.

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

Shang, H. & Shang, H.S. 1998. High temperature reisistance of wheat to stripe rust. Scientia Agricultura Sinica 31: 46-50.

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.

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 rusts in bread wheat. Plant Disease 77: 1103- 1106.

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

Stubbs, R.W. 1985. Stripe rust. Pages 61-101 in: The Cereal Rusts II: Diseases, Distribution, Epidemiology and Control. Roelfs A.P. and Bushnell, W.R. eds. Academic Press, New York.

Wellings, C.R. & McIntosh, R.A. 1990. *Puccinia striiformis* f. sp. *tritici* in Australia: pathogenic changes during the first 10 years. Plant Pathology 39: 316-325.

Zadoks, J.C., Chang, T.T. & Konzak, C.F. 1974. A decimal code for the growth stages of cereals. Weed Research 14: 415-426.

CHEMICAL CONTROL OF FOLIAR RUSTS OF BREAD WHEAT IN SOUTH AFRICA

ABSTRACT

Field trials were conducted from 1997 to 1999, in both the winter and summer rainfall areas of South Africa, to determine the effect of *Puccinia striiformis* Westend. f. sp. *tritici* Eriks. and *P. triticina* Eriks. epidemics on yield and quality of *Triticum aestivum* L. Furthermore, information on the efficacy of different seed and foliar treatments in controlling these diseases was obtained. Five triazole fungicides applied in double applications (growth stage [GS] 16 to 19 and GS 49 to 59), on three cultivars, during 1997 in the south Western Cape resulted in a mean decrease of 31% in the area under the disease progress curve (AUDPC) calculated for stripe rust infection. The application of fungicides closely to, or just after head emergence (GS 49 to 59), resulted in a 65 to 74% decrease in the occurrence of stripe rust head infections. In contrast, head infection was reduced by only 8% when fungicides were applied at the seven leaf (GS 16 to 19) stage. Combined seven and flag leaf treatments, over the three cultivars, resulted in a 56% yield increase with the application of propiconazole, followed by 49%, 44%, 39% and 25% for tebuconazole, flutriafol, bromuconazole, and flusilazole, respectively. Yield was negatively correlated with the AUDPC (R=-0.5 (23% of variance accounted for). In the absence of disease during 1998 no fungicide treatment resulted in a significant yield or hectolitre mass increase in any of the trials.

The application of eight fungicides at both seven and flag leaf stages (GS 16 and GS 37) at Langgewens in the Western Cape, resulted in a mean decrease of 65% in the AUDPC, calculated for leaf rust severity during 1999. Combined seven and flag leaf treatments resulted in a mean yield increase of 56%, followed by 50% and 15% for the flag leaf and seven leaf treatments, respectively. Over treatments applied, yield increases varied from 24% for bromuconazole to 53% for epoxiconazole/carbendazim. Yield was negatively correlated with the AUDPC (R=-0.88 (77% of variance accounted for). The application of the flag leaf and combined

seven and flag leaf treatments resulted in a significant ($P<0.001$) increase in the hectolitre mass (kg $h\ell^{-1}$).

During 1999 the combination of triticonazole seed treatment with a propiconazole flag leaf treatment (GS 37) on the cultivar Gariep in the eastern Free State resulted in a 91% decrease in the AUDPC calculated for stripe rust infection, and a 36% yield increase. Hectolitre mass increased by 3% and protein content decreased by 4% for the latter treatment. Triticonazole seed treatment reduced AUDPC by 54%, resulting in 16 and 2% yield and hectolitre mass increase, respectively. 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. The latter two treatments resulted in a 49% yield increase. Yield was negatively correlated with the AUDPC ($R=-0.92$ (85% of variance accounted for)).

Significant differences ($P<0.001$) were found among the fungicides carboxin/thiram, flutriafol/thiabendazole, tebuconazole, triadimenol, and triticonazole, applied as seed treatments on the cultivar Gariep, in the eastern Free State during 1999. The AUDPC decreased by 61 and 25% in plots where the seed treatment fungicides triadimenol and triticonazole were applied, respectively. The remaining three seed treatments did not differ significantly from the control.

INTRODUCTION

Stripe and leaf rust, respectively caused by *Puccinia striiformis* Westend. f. sp. *tritici* Eriks. and *P. triticina* Eriks., are foliar pathogens of wheat (*Triticum aestivum* L.) with the potential to cause extensive losses in grain yield (Wiese, 1987; Conner & Kuzyk, 1988; Dannenberg *et al.*, 1989; Yang & Zeng, 1989; Murray *et al.* 1995; Torabi *et al.*, 1995; Khan & Trevathan, 1997; Sayre *et al.*, 1998; Wagoire *et al.*, 1998; Cook *et al.*, 1999). The effect of stripe rust on susceptible wheat cultivars is most severe under prolonged epidemic conditions, in particular when the onset of such epidemics occurs before the booting stage of growth. Grain yield losses of up to 84%, a reduction in kernel mass of up to 43%, and a decrease in kernel number of up to

72% have been reported for stripe rust (Murray *et al.*, 1995). Under epidemic conditions wheat leaf rust infection may cause reductions in yield components including kernel weight, kernels per square meter, and grain fill rate, which individually or collectively may result in mean yield losses of between 7 and 63%, depending on cultivar susceptibility (Conner & Kuzyk, 1988; Dannenberg *et al.*, 1989; Eversmeyer & Kramer, 1996; Sayre *et al.*, 1998; Singh, 1999).

The control of foliar rusts on susceptible wheat cultivars has cost South African wheat farmers millions of rands from 1996-2000. Approximately 500 000 ha of wheat are grown annually under moderate to high risk environmental conditions for stripe and leaf rust development. Although the levels of leaf rust resistance in most South African wheat cultivars are acceptable, fungicide applications are required when susceptible varieties are grown in environmental conditions conducive to epidemic development (Anonymous, 2000a,b). Following the observation of stripe rust for the first time in South Africa during August 1996 (Pretorius *et al.*, 1997), wheat producers spent an estimated R28 million on fungicides to control this disease in the Western Cape that year. Despite the widespread application of chemicals, significant crop losses, varying from 5 to 50%, still occurred. These losses can be ascribed to the rapid increase in epidemic potential in the region, an initial reluctance among farmers to invest in expensive fungicide applications against a new and unknown disease, and the fact that weather conditions and the availability of spray equipment did not always allow optimal control.

During 1997 the stripe rust pathogen spread to winter wheat planted under dry land conditions in the eastern Free State. Subsequent epidemic outbreaks resulted in the extensive use of fungicides in this region, at an estimated cost of R18 million. The appearance of pathotype 6E22A- during 1998 in the eastern Free State resulted in epidemic outbreaks of stripe rust on the previously resistant cultivars Hugenoot and Carina. The cost to control stripe rust on the 42 000 ha planted under the latter two cultivars, excluding losses in yield and quality, was estimated at more than R6 million.

Chemical control of cereal diseases is usually not desirable due to the high costs of fungicide application. However, fungicides present an effective short-term alternative for the control of foliage diseases, particularly when adequate resistance

is not available and production levels must be maintained (Ireta & Gilchrist, 1994). Demethyl inhibiting (DMI) fungicides, including several triazole compounds, dominate fungicide use on both spring and winter wheat in South Africa. Currently one triazole seed treatment, six triazole fungicides and five triazole/benzimidazole mixtures are registered for the control of stripe rust (Nel *et al.*, 1999). For the control of leaf rust four triazole fungicides and three triazole/benzimidazole mixtures are registered (Nel *et al.*, 1999).

The main objective of this study was to quantify the potential impact of stripe and leaf rust on yield and quality of wheat planted in the winter and summer rainfall areas of South Africa. Additionally, the efficacy of different triazole compounds, applied as foliar and seed treatments in controlling stripe and leaf rust, was investigated.

MATERIALS AND METHODS

Foliar application of fungicides

In 1997 a field trial was established at Tygerhoek experimental farm in the south Western Cape. The trial was planted on 20 May 1997 at a site that lay fallow during the previous year. The seeding rate was 90 kg ha^{-1} and seedbed fertilizer was applied at a rate of 90 kg N ha^{-1}. The fungicides bromuconazole (160 g active ingredient (a.i.) ha^{-1}), flusilazole (100 g a.i. ha^{-1}), flutriafol (125 g a.i. ha^{-1}), propiconazole (200 g a.i. ha^{-1}), and tebuconazole (187.5 g a.i. ha^{-1}) were applied on three stripe rust susceptible wheat cultivars. A randomized split-block design was used with each cultivar, fungicide and treatment combination replicated eight times. Sub-plots were 1.2-m wide, consisting of three 10-m rows of the cultivars Palmiet, Nantes, and SST 55, respectively. Two spreader rows, consisting of a mixture of the cultivars Palmiet, Nantes and SST55, were planted among sub-plots to prevent fungicide drift. Fungicides were applied in single applications at growth stages (GS, Zadoks *et al.*, 1974) 16 to 19 and GS 49 to 59, respectively, and in double applications at both GS 16 to 19 and GS 49 to 59. Fungicides were applied with a pressurized knapsack sprayer at 160 kPa using 3.5 psi cone nozzles and water

volumes of approximately 300 ℓ ha^{-1}. Disease assessment was carried out on two consecutive occasions at GS 49 to 59 and GS 75 to 77, assessing the percentage disease severity on the flag leaves of each sub-plot using a modified Cobb scale (Peterson *et al.*, 1948). The data was used to calculate the area under the disease progress curve (AUDPC) for each treatment. Stripe rust head infections were visually scored for each entry at GS 75 to 77, using a 0-3 scale with 0= no head infection, 1 = a low percentage (approximately <10% of spikelets) of heads infected, 2 = intermediate percentage (approximately 10- 30% of spikelets) of heads infected, and 3 = a high percentage (approximately >30% of spikelets) of heads infected. Plots were harvested with a plot combine. Seed were air-dried, cleaned and weighed to determine grain yield. The yield obtained from a 10-m row was not always enough to determine the hectolitre mass (kg $h\ell^{-1}$). The latter was, therefore, determined by combining grain samples from two 10-m rows with similar treatments. Protein content (% w/w) was determined using a near-infrared reflectance spectrophotometer (Infra-Alyser 360R).

The fungicides bromuconazole (140 g a.i. ha^{-1}), epoxiconazole/carbendazim (112.5/112.5 g a.i. ha^{-1}), flusilazole/carbendazim (100/50 g a.i. ha^{-1}), flutriafol (125 g a.i. ha^{-1}), propiconazole (100 g a.i. ha^{-1}), cyproconazole (40 g a.i. ha^{-1}), tebuconazole (187.5 g a.i. ha^{-1}), and tebuconazole/carbendazim (125/100 g a.i. ha^{-1}) were evaluated in two field trials during 1998. Trials were planted at Langgewens and Small Grain Institute (SGI) experimental farms in the Western Cape and in the eastern Free State, respectively. Both trails were arranged according to a randomized block design with four replicates on sites that had been fallowed the previous season. The cultivar Palmiet was planted at Langgewens on 22 May 1998 at a seeding rate of 90 kg ha^{-1}, with seedbed fertiliser applied at a rate of 90 kg N/ha. Individual plots were 1.02 m wide and 5 m long, consisting of 6 rows spaced 0.17 m apart. Plots were spaced 1 m from each other to minimize the effect of fungicide drift during the application of treatments.

The cultivar Gariep was planted at SGI on 24 June 1998 at a seeding rate of 25 kg ha^{-1} (normal seeding rate for dry land wheat in the summer rainfall area), with seedbed fertilizer applied at a rate of 45 kg N ha^{-1}. Individual plots were 1.35 m wide and 5 m long, consisting of 3 rows spaced 0.45 m apart. Plots were separated by

three untreated rows (1.35 m wide) of the cultivar Gariep. In both trials fungicides were applied in single applications at GS 16 and GS 39, respectively and in repeated applications at both GS 16 and GS 39, using a CO_2 backpack sprayer at 200 kPa with flat fan nozzles and water volumes of approximately 300 ℓ ha^{-1}. Disease assessment was carried out on whole plots three weeks after the application of treatments using a modified Cobb scale. Plots were harvested with a plot combine. Seed were air-dried, cleaned and weighed to determine grain yield. Yield obtained from each plot was used to determine the hectolitre mass.

The Langgewens trial was repeated during 1999 at the same site, using the same fungicide and treatment combinations. The cultivar SST 75 was used and the trial was planted on 17 May 1999. The seeding rate and fertiliser application were as described above. Fungicides were used in single applications at GS 16 and GS 37, respectively and in double applications at both GS 16 and 37. Disease assessment was carried out at GS 61 and GS 75, assessing the percentage leaf rust severity on the top two leaves of each plot using a modified Cobb scale. The data was used to calculate the AUDPC for each fungicide and treatment combination. After harvesting grain obtained from each plot was air-dried, cleaned and sieved to remove siftings (seed <1.5 mm). Thereafter the siftings and grain obtained from each plot were weighed to determine the percentage siftings and grain yield obtained for each plot. Hectolitre mass and protein content (% w/w, using a near-infrared reflectance spectrophotometer (Infra-Alyser 360R)) were determined using only the sieved grain (seed >1.5 mm).

Seed treatment

Seed treatment combined with foliar fungicides. During 1999 the foliar fungicide propiconazole, and the seed treatment fungicide triticonazole, were applied on the cultivar Gariep, planted at SGI in the eastern Free State. A four-replicate randomized block design was used. Treatments included were an untreated control, triticonazole seed treatment (0.24 g a.i. kg^{-1} seed), triticonazole seed treatment combined with two single foliar applications of propiconazole (100 g a.i. ha^{-1}) at GS 16 and GS 37, respectively, triticonazole seed treatment combined with two foliar applications of propiconazole at both GS 16 and GS 37, two single foliar applications

of propiconazole at GS 16 and GS 37, respectively, and two combined foliar applications of propiconazole (at both GS16 and 37). The trial was planted on 5 July 1999 at a seeding rate of 25 kg ha^{-1}, with seedbed fertiliser applied at a rate of 60 kg N ha^{-1}. Individual plots were 1.02 m wide and 5 m long, consisting of 6 rows spaced 0.17 m apart. Plots were bordered on both sides with 1 m wide plots of McNair 701 and Gariep and with 1.2 m alleys on each end to reduce fungicide drift. Fungicides were applied by using a CO_2 backpack sprayer at 200 kPa, flat fan nozzles and water volumes of approximately 300 ℓ ha^{-1}. Supplemental irrigation was applied when needed to improve plant development as well as environmental conditions for rust development. Disease assessment was carried out on whole plots every fifth day, for three consecutive weeks, using a modified Cobb scale. The data were used to calculate the AUDPC for each seed treatment and/or fungicide combination. Plots were harvested with a plot combine. Seed were air-dried, cleaned and weighed to determine grain yield. Yield obtained from each plot was used to determine the hectolitre mass and protein content.

Evaluation of different seed treatment fungicides. During 1999 five seed treatment fungicides, mainly registered to control bunt diseases, were evaluated for their efficacy to control stripe rust during early growth stages of the cultivar Gariep. Treatments included an untreated control, carboxin/thiram (0.6/0.6 g a.i. kg^{-1} seed), flutriafol/thiabendazole (0.05/0.05 g a.i. kg^{-1} seed), tebuconazole (0.013 g a.i. kg^{-1} seed), triadimenol (0.23 g a.i. kg^{-1} seed), and triticonazole (0.24 g a.i. kg^{-1} seed). A four-replicate randomized block design was used. The trial was planted on 9 July 1999 at SGI in the eastern Free State. The assessment of the percentage stripe rust severity on the flag leaf –1 and flag leaf was carried out from appearance of the first stripe rust symptoms (GS 29) until GS 51, using a modified Cobb scale. The data were used to calculate the respective AUDPC values.

Statistical analysis

AUDPC values calculated for rust development over time, occurrence of head infection, grain yield, hectolitre mass and protein content data obtained in the different experiments in different years, were analysed for variance using the

statistical software Genstat 5, 4 th ed. for Windows (Appendices 6-11). Means were compared using Fisher's least significant difference test (P=0.05). Data were further subjected to regression analyses (using NCSS 2000 Statistical System for Windows) to determine the relationship between the different yield parameters and disease (AUDPC).

RESULTS

Fungicide trials

The occurrence of stripe and leaf rust infection and the onset of epidemics differed from year to year.

Tygerhoek 1997. Stripe rust symptoms were observed during the application of the seven leaf treatment and reached epidemic levels at the flag leaf stage. No other foliar diseases were observed in this trial. When the flag leaf treatments were applied, four weeks after the seven leaf treatments, stripe rust severity varied between 5 to 10% for the plots sprayed at the seven leaf growth stage, and between 20 to 40% for the unsprayed plots. These high levels of infection resulted in a significant percentage of leaf damage in all the flag leaf treatments. High temperatures and moisture stress experienced during flowering (GS 65) also contributed to premature leaf necrosis.

Cultivar means calculated over fungicides and treatments showed that SST 55 exhibited the most leaf damage, resulting in the highest AUDPC (Fig. 1). AUDPC calculated for the combined seven and flag leaf treatments was 31% lower than the control, whereas the seven leaf and flag leaf treatments decreased leaf damage by 24 and 14%, respectively (Fig. 2). Fungicides reduced AUDPC between 19% for propiconazole to 13% for flusilazole (Fig. 3).

Differences were found among cultivars, fungicides, and treatments applied for the percentage head infection scored between GS 75 and 77. The cultivar Palmiet was the worst affected, over fungicides and treatments applied, by stripe rust head infection (Fig. 4). Flag leaf treatments reduced head infection by 74%, followed

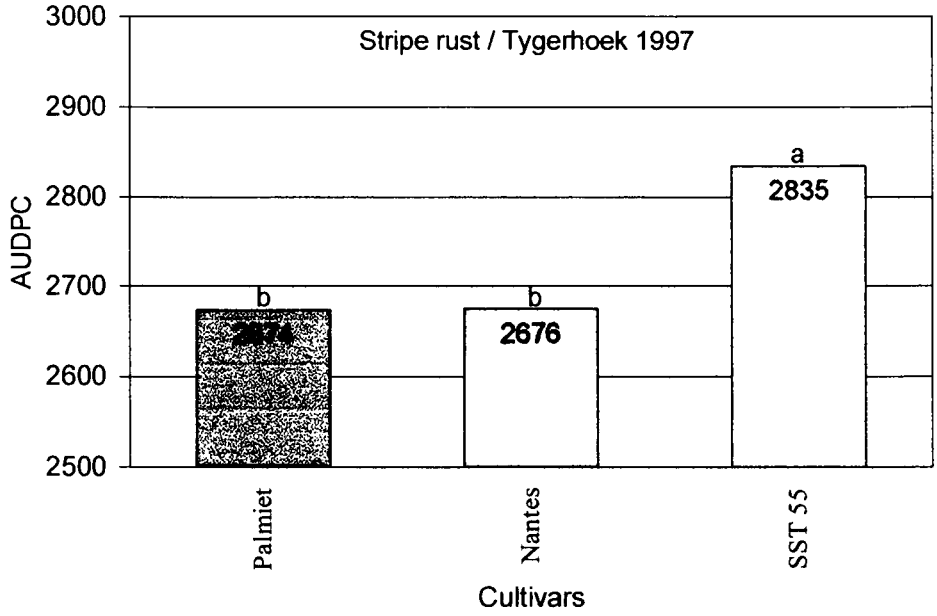

Fig. 1. Mean area under the disease progress curve (AUDPC) calculated for each cultivar, over fungicides and treatments applied.

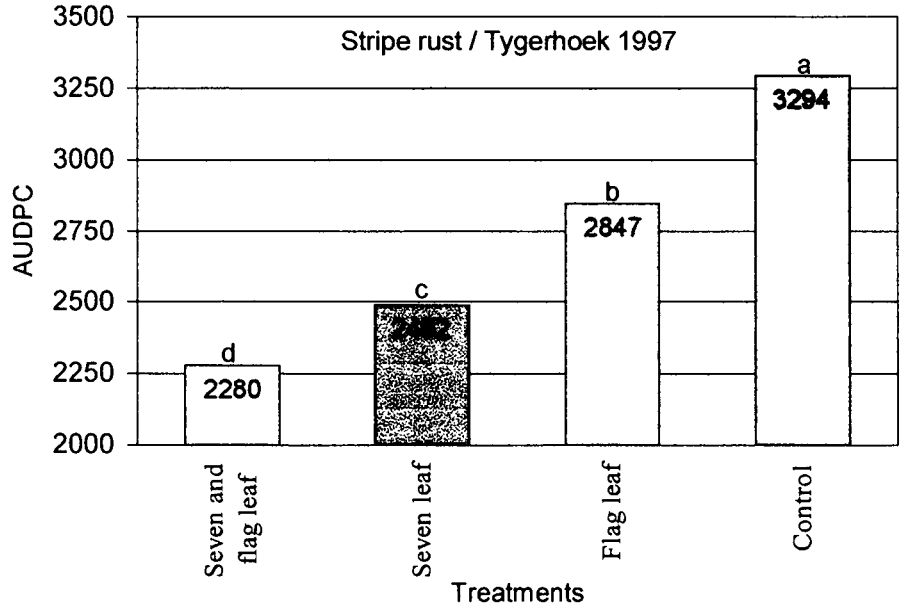

Fig. 2. Mean area under the disease progress curve calculated for each treatment, over cultivars (Palmiet, Nantes and SST 55) and fungicides applied.

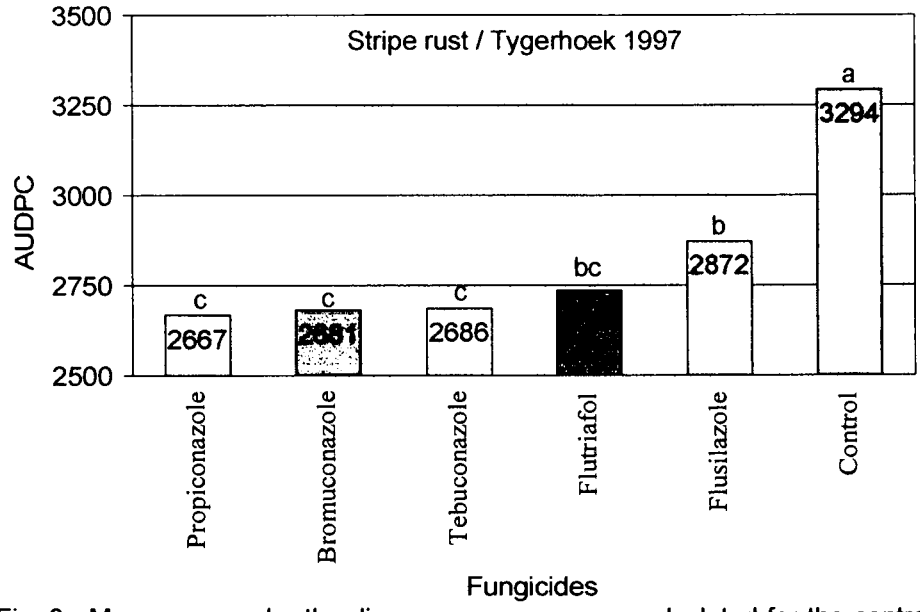

Fig. 3. Mean area under the disease progress curve calculated for the control treatment and five fungicides, averaged over cultivars (Palmiet, Nantes and SST 55) and treatments applied.

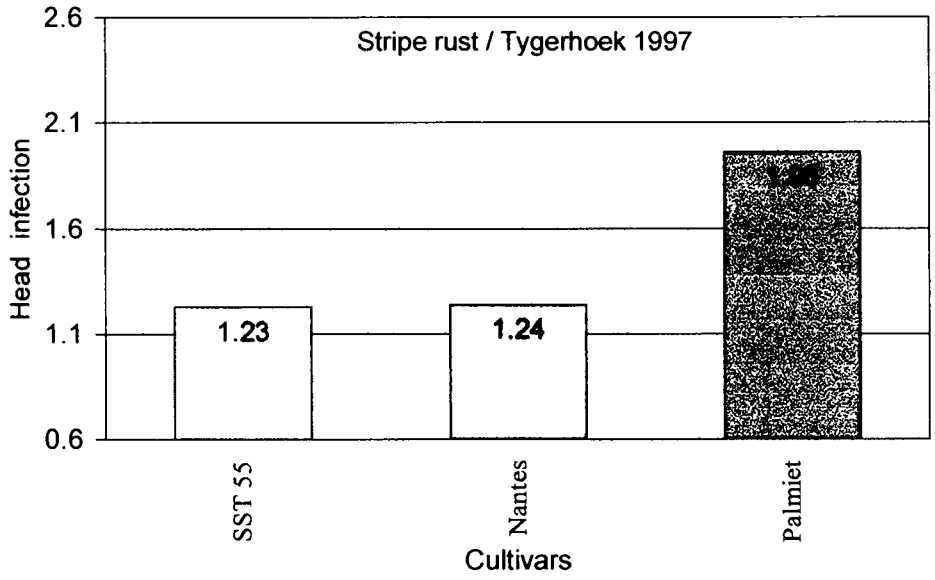

Fig. 4. Mean prevalence of stripe rust head infection determined for each cultivar, over fungicides and treatments applied (0= no head infection; 1= a low percentage of heads infected; 2= intermediate percentage of heads infected; 3= a high percentage of heads infected).

by the combined seven and flag leaf treatments and the seven leaf treatments with 65 and 8% reductions, respectively (Fig. 5). Decreases in head infection obtained with the different fungicides, over cultivars and treatments applied, varied from 49% for propiconazole to 9% for flusilazole (Fig. 6). Head ratings of 2 and 3 were associated with higher AUDPC values (Fig. 7).

The highest yield (1.29 t/ha) was obtained with Palmiet (Fig. 8). Yields were below average for the south Western Cape region. Extreme conditions including epidemic levels of stripe rust up to flowering, non-optimal timing of the flag leaf fungicide treatments, and prolonged drought conditions after flowering resulted in a high percentage of leaf necrosis. The latter contribute to the unexpected low relationship of AUDPC to yield obtained (Fig. 9) ($R=-0.5$ (23% of variation accounted for) $P<0.001$). However, treatments significantly ($P<0.001$) influenced yield. The combined seven and flag leaf treatments resulted in a mean yield increase of 43%, over cultivars and fungicides applied, followed by a mean yield increase of 29% and 10% for the seven leaf treatments and flag leaf treatments, respectively (Fig. 10). Considering the seven leaf treatment, over cultivars, a yield increase of 39% was obtained with propiconazole followed by tebuconazole (35%), bromuconazole (28%), flutriafol (26%), and flusilazole (15%) (Fig. 11). The highest yield increase with the flag leaf application of fungicides, over cultivars, was obtained with propiconazole (16%), followed by flutriafol (11%), tebuconazole (10%), flusilazole (9%), and bromuconazole (7%) (Fig. 12). With the combined seven and flag leaf treatment, over cultivars, the highest yield increase was obtained with propiconazole (56%), followed by tebuconazole (49%), flutriafol (44%), bromuconazole (39%), and flusilazole (25%) (Fig. 13). Over cultivars and treatments applied the best yield was obtained with propiconazole (37%), followed by tebuconazole (32%), flutriafol (27%), bromuconazole (25%), and flusilazole (16%) (Fig. 14).

Hectolitre mass and protein content were poorly correlated with stripe rust infection (Figs. 15 and 16). Over fungicides and treatments applied, cultivars differed significantly ($P<0.001$) in hectolitre mass (Fig. 17). Hectolitre mass obtained with the flag leaf and combined seven and flag leaf treatments was significantly higher ($P<0.001$) than those obtained for the seven leaf fungicide treatments and the control plots (Fig. 18). Application of flutriafol, propiconazole and tebuconazole, over

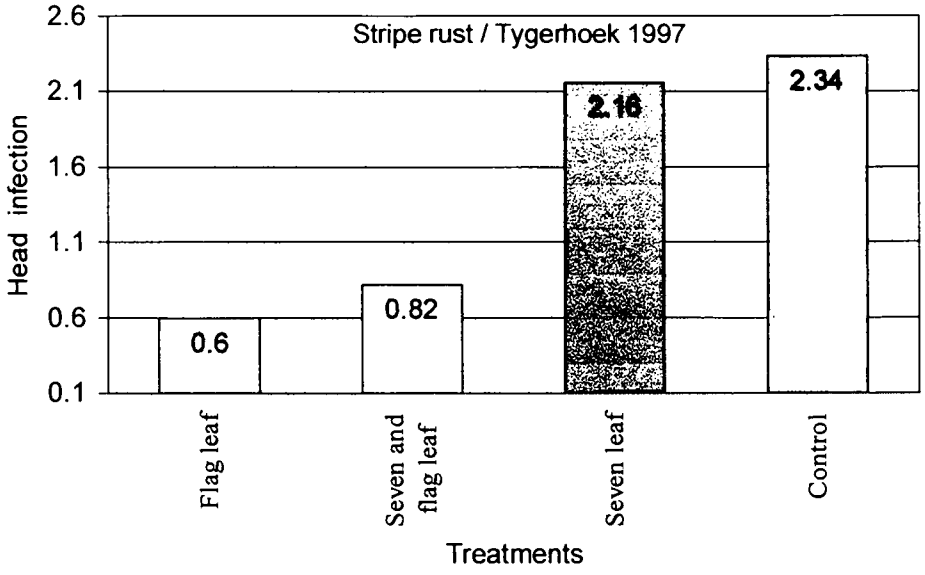

Fig. 5. Mean prevalence of stripe rust head infection determined for each treatment, over cultivars (Palmiet, Nantes and SST 55) and fungicides applied (0= no head infection; 1= a low percentage of heads infected; 2= intermediate percentage of heads infected; 3= a high percentage of heads infected).

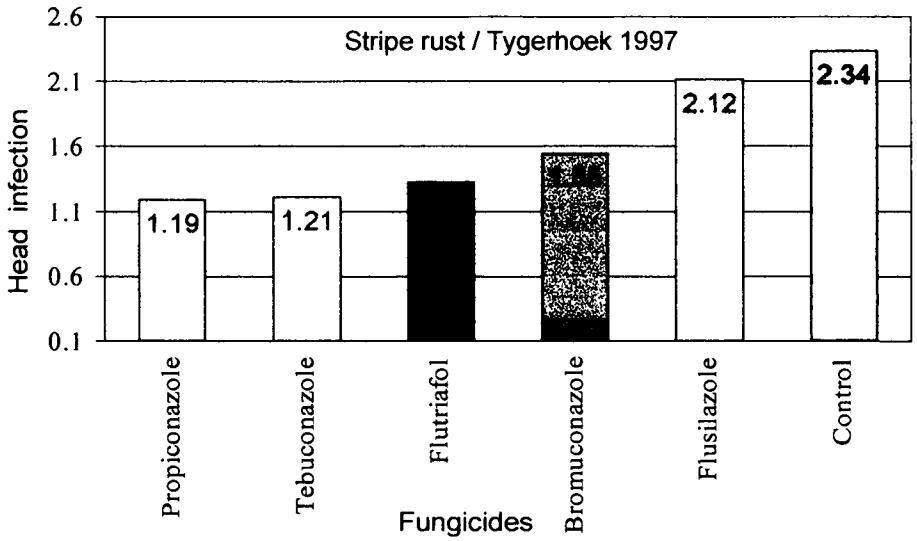

Fig. 6. Mean prevalence of stripe rust head infection determined for the control treatment and each fungicide, over cultivars (Palmiet, Nantes and SST 55) and treatments applied (0= no head infection; 1= a low percentage of heads infected; 2= intermediate percentage of heads infected; 3= a high percentage of heads infected).

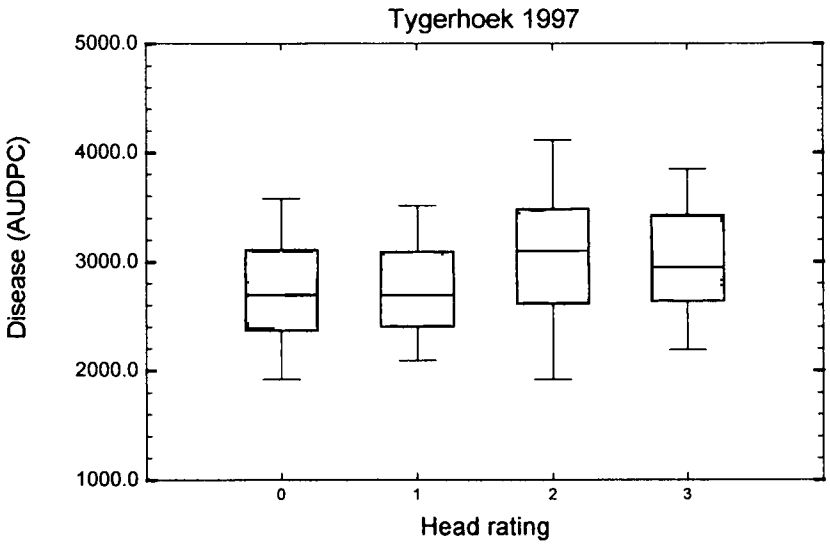

Fig. 7. Relationship of stripe rust head ratings (0 to 3 scale) to the area under the disease progress curve calculated for the cultivars Palmiet, Nantes and SST 55. The top and bottom of each box represent the 25th and 75th percentiles, and the line through the middle the median.

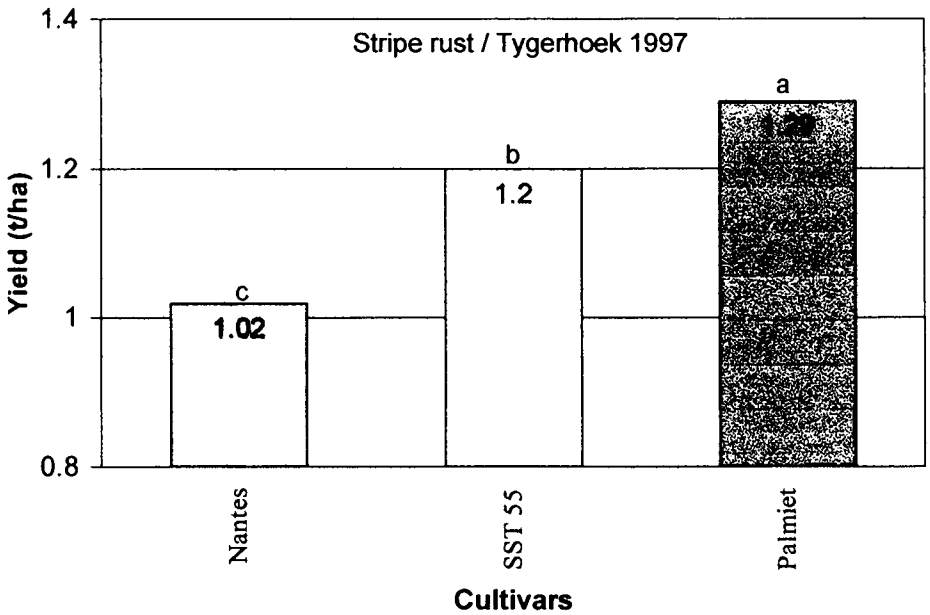

Fig. 8. Mean yield (t/ha) obtained for the cultivars Nantes, SST 55 and Palmiet, over fungicides and treatments applied.

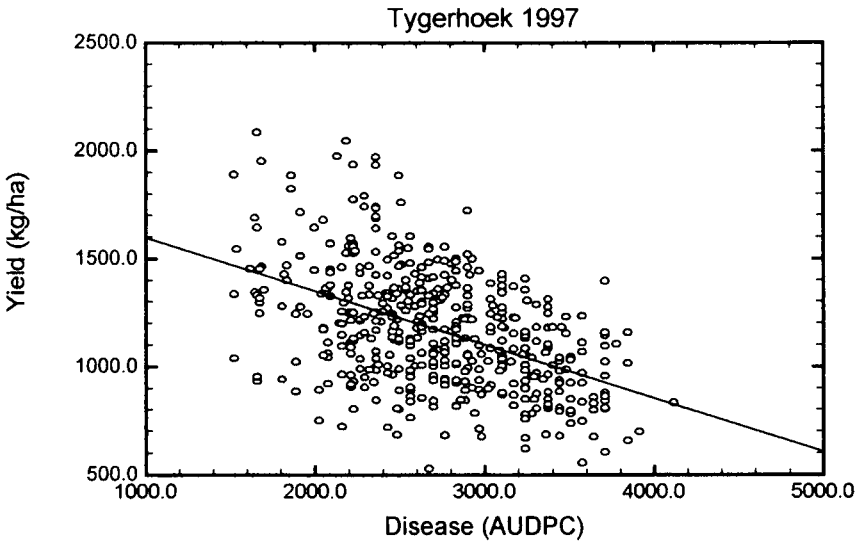

Fig. 9. Relationship between the area under the disease progress curve, calculated for stripe rust development on the cultivars Palmiet, Nantes and SST 55, and yield (R^2=0.23).

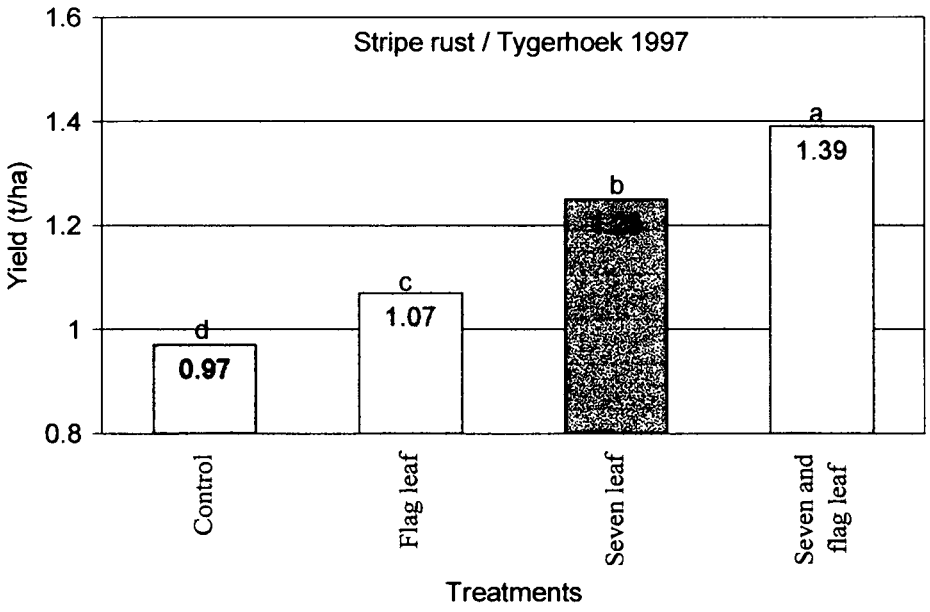

Fig. 10. Mean yield (t/ha) obtained for each treatment, over cultivars (Palmiet, Nantes and SST 55) and fungicides applied.

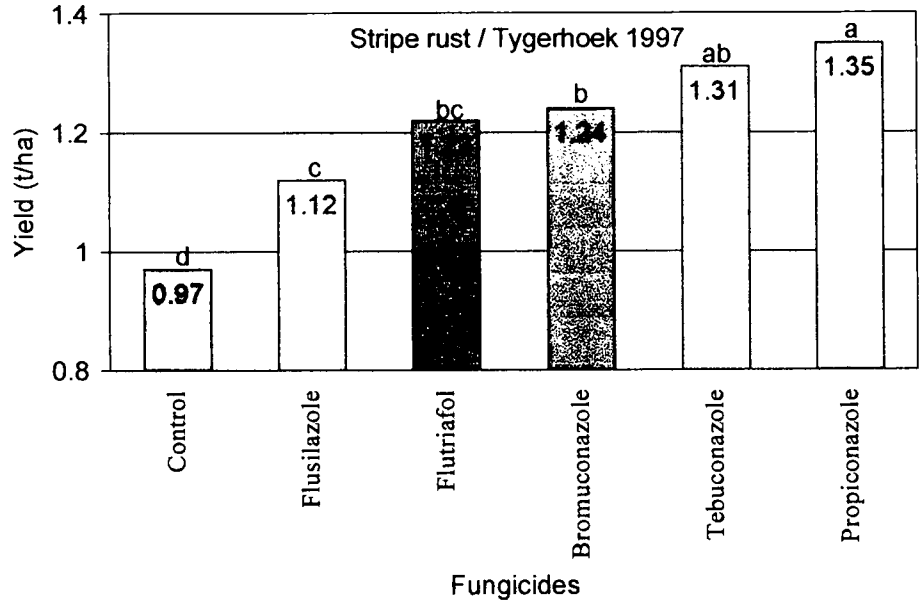

Fig. 11. Mean yield (t/ha) obtained for the control treatment and each fungicide applied during the seven leaf growth stage, averaged over cultivars (Palmiet, Nantes and SST 55).

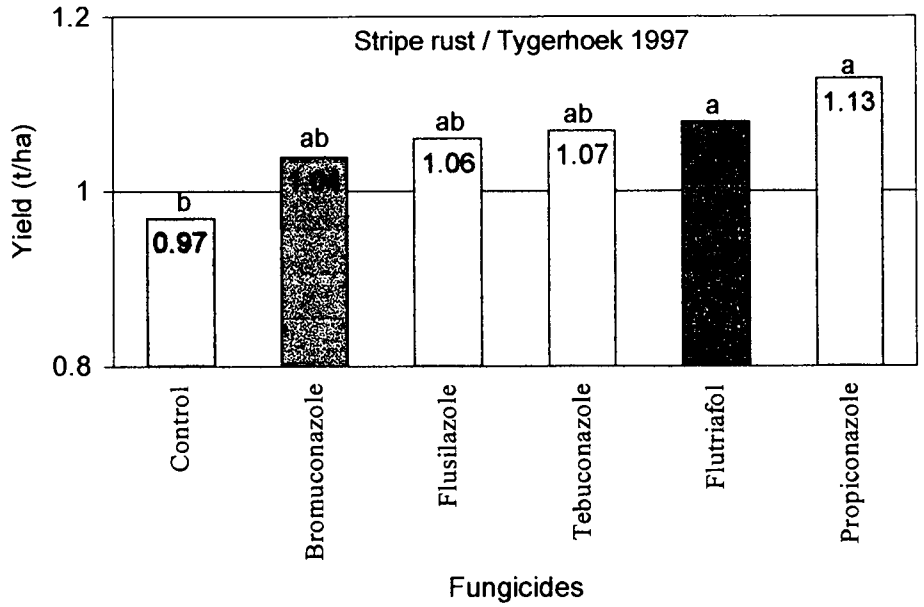

Fig. 12. Mean yield (t/ha) obtained for the control treatment and each fungicide applied during the flag leaf growth stage, averaged over cultivars (Palmiet, Nantes and SST 55).

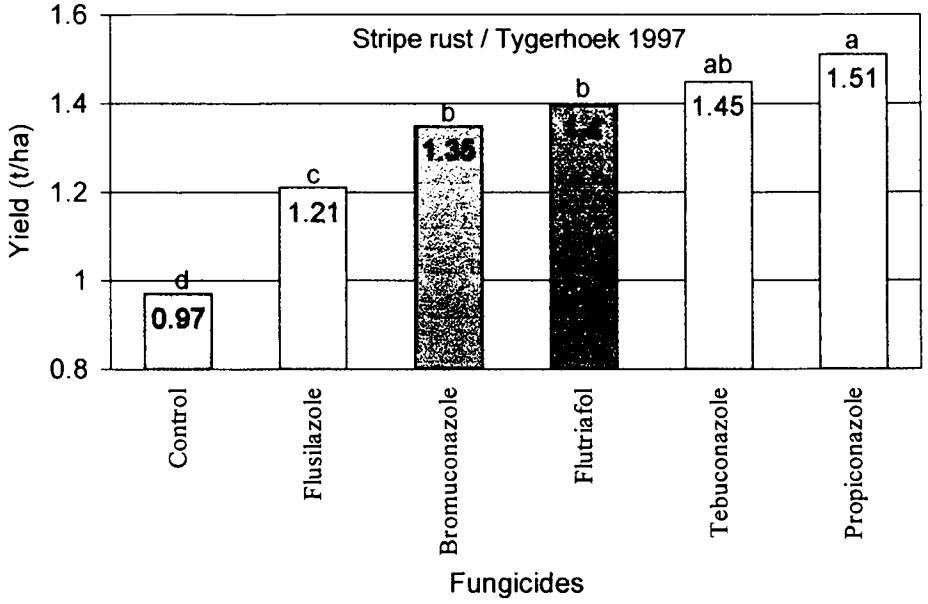

Fig. 13. Mean yield (t/ha) obtained for the control treatment and each fungicide applied at both the seven and flag leaf growth stages, averaged over cultivars (Palmiet, Nantes and SST 55).

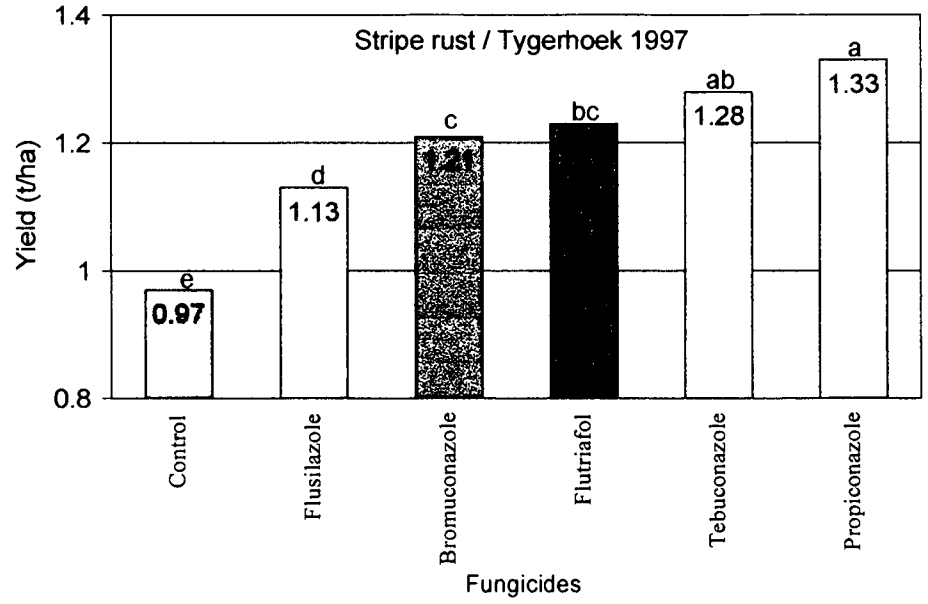

Fig. 14. Mean yield (t/ha) obtained for the control and each fungicide treatment, averaged over cultivars (Palmiet, Nantes and SST 55) and timing of applications.

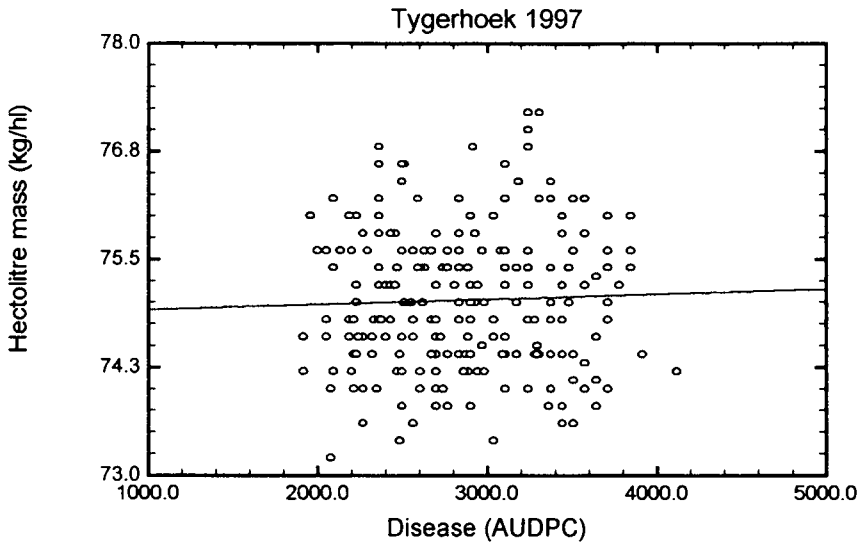

Fig. 15. Relationship between the area under the disease progress curve, calculated for stripe rust development on the cultivars Palmiet, Nantes and SST 55, and hectolitre mass (R^2=0.0014).

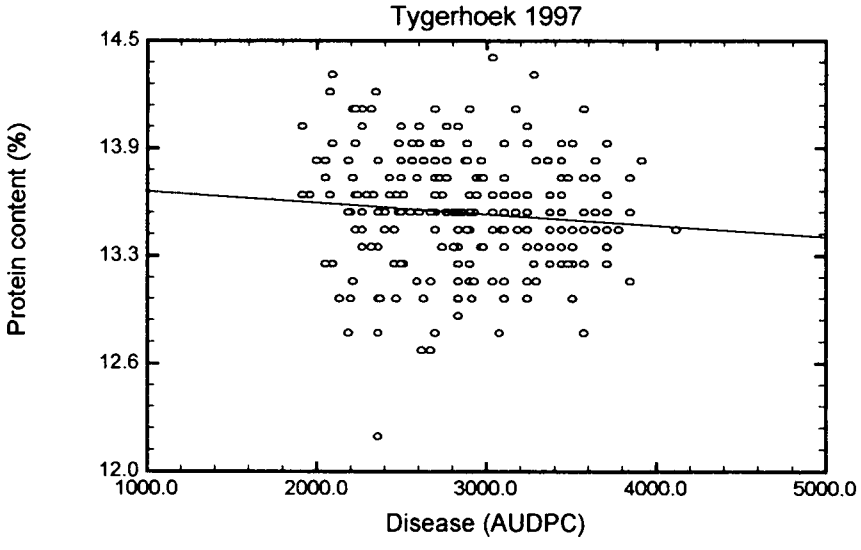

Fig. 16. Relationship between the area under the disease progress curve, calculated for stripe rust development on the cultivars Palmiet, Nantes and SST 55, and protein content (R^2=0.0098).

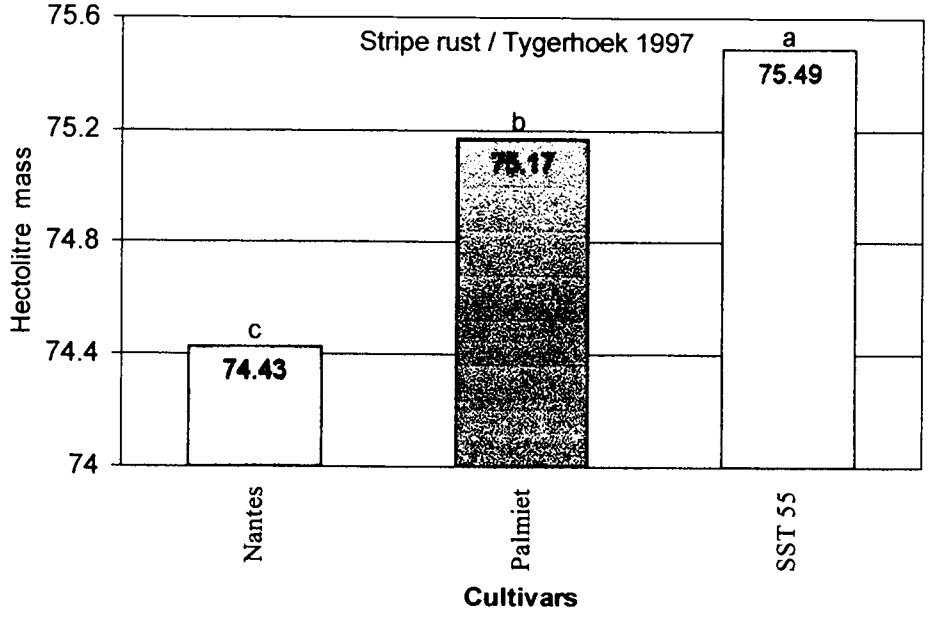

Fig. 17. Mean hectolitre mass (kg/hℓ) obtained for the cultivars Nantes, Palmiet and SST 55, over fungicides and treatments applied.

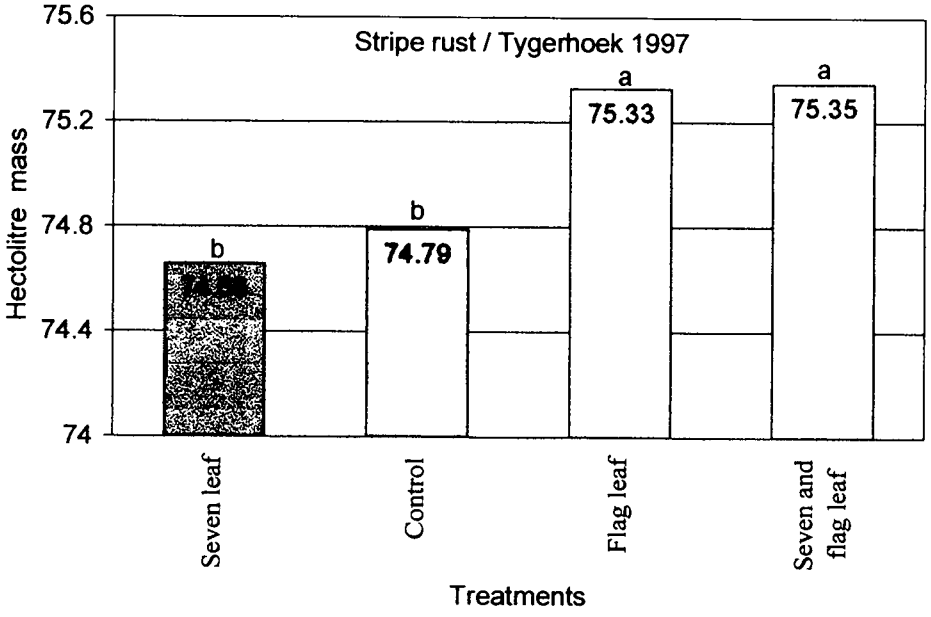

Fig. 18. Mean hectolitre mass (kg/hℓ) obtained for each treatment, over cultivars (Palmiet, Nantes and SST 55) and fungicides applied.

cultivars and treatments applied, resulted in significantly higher ($P<0.007$) hectolitre mass values of >75 kg $h\ell^{-1}$ (Fig. 19). Cultivars differed significantly ($P<0.001$) in their protein content (Fig. 20). The highest protein content was obtained with the cultivar Nantes (13.7), followed by SST 55 (13.5), and Palmiet (13.3). The protein content of the seven leaf treatment was significantly higher ($P<0.01$) than those of the other three treatments (Fig. 21).

Langgewens and Small Grain Institute 1998. Due to prolonged hot and dry environmental conditions experienced from flag leaf to the end of the growing season, only traces of rust infection occurred in both the trials planted at Langgewens and SGI during 1998. In the absence of disease no fungicide treatment resulted in a significant yield or hectolitre mass increase in any of the trials. Protein content was therefore not measured. The mean yield and hectolitre mass obtained in these trials are presented in Figures 22 to 25.

Langgewens 1999. Environmental conditions in the Western Cape were conducive for the development of leaf rust during 1999. The first symptoms of natural leaf rust infection were observed on plots during the application of the seven leaf treatments. With the application of the flag leaf treatments, three weeks after the seven leaf treatments, traces of leaf rust were observed on the flag leaves of the control plots as well as on plots receiving only flag leaf treatments. Leaf rust infections reached epidemic levels two weeks after the flag leaf treatments were applied. Only trace symptoms of stripe rust were observed after flag leaf emergence. Timing of the flag leaf treatments was better than those obtained at Tygerhoek during 1997. The latter resulted in high levels of leaf rust control and consequently lower AUDPC values for treated plots.

Leaf rust was best controlled in the combined seven and flag leaf treatment, resulting in a mean decrease (calculated over fungicides) of 65% in AUDPC. The flag leaf treatment reduced AUDPC by 57% followed by the seven leaf treatment with 18% (Fig. 26). Significant differences ($P<0.001$) were found among fungicides in the control of leaf rust. The percentage decrease in the AUDPC, over treatments,

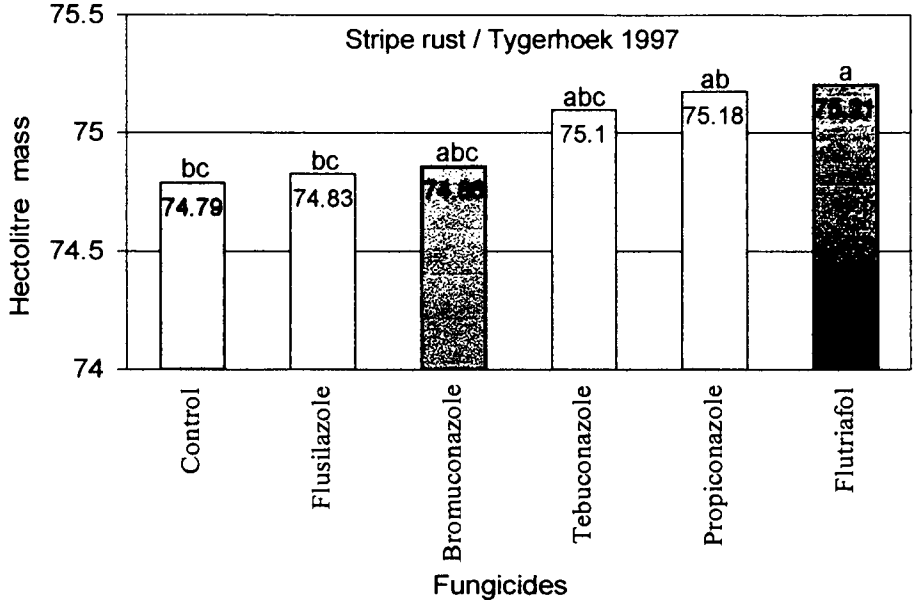

Fig. 19. Mean hectolitre mass (kg/hℓ) obtained for the control and each fungicide treatment, averaged over cultivars (Palmiet, Nantes and SST 55) and timing of applications.

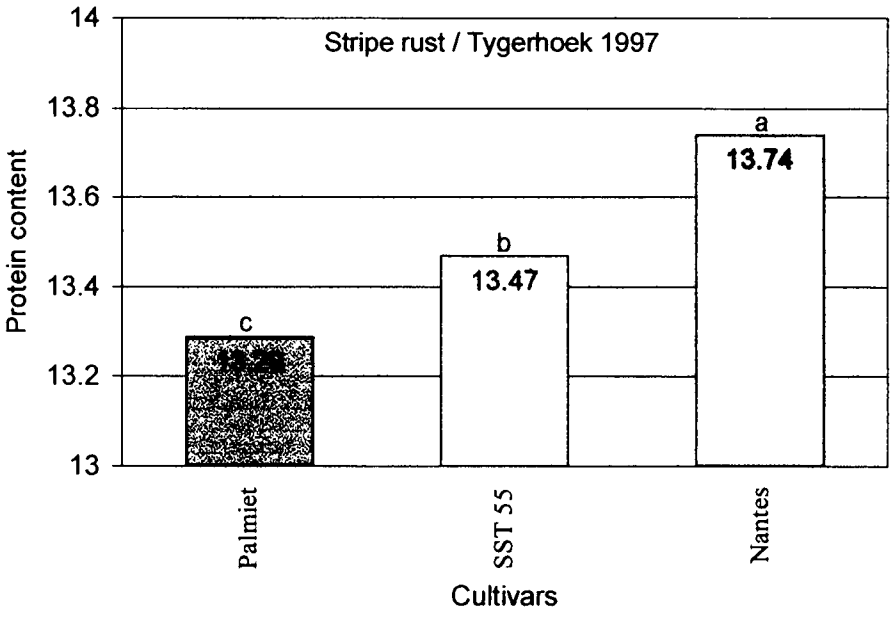

Fig. 20. Mean protein content measured for the cultivars Palmiet, SST 55 and Nantes, over fungicides and treatments applied.

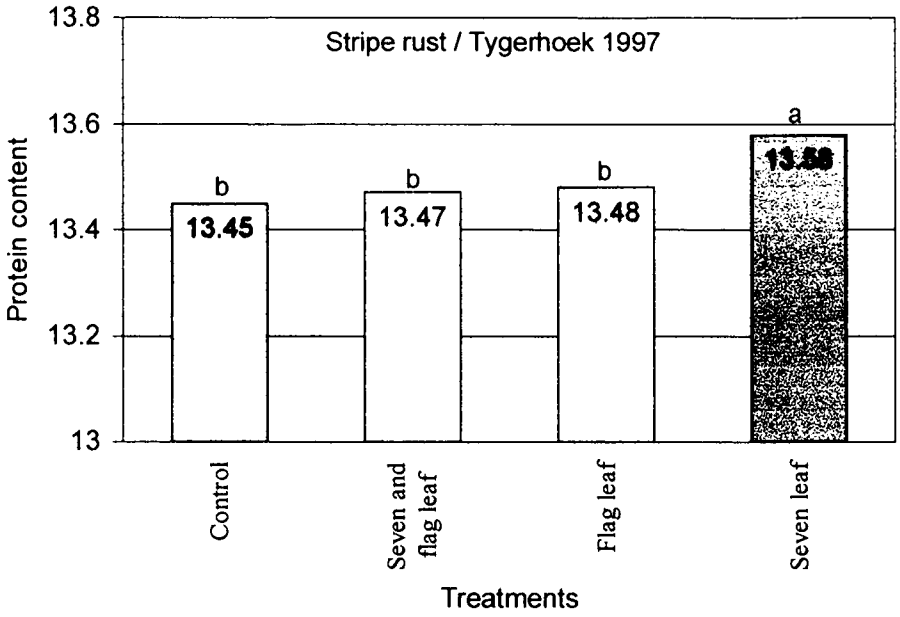

Fig. 21. Mean protein content measured for each treatment, over cultivars (Palmiet, Nantes and SST 55) and fungicides applied.

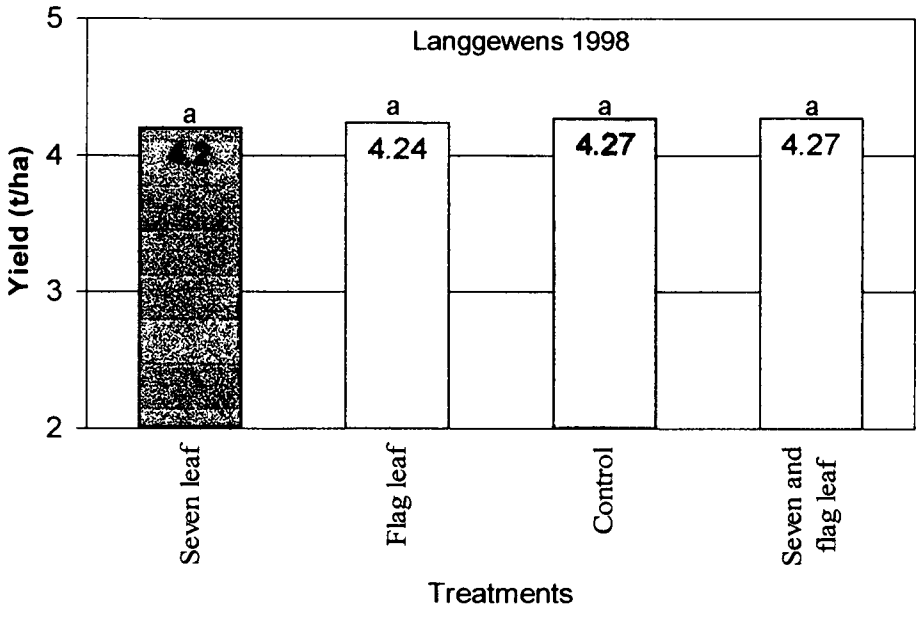

Fig. 22. Mean yield (t/ha) of the cultivar Palmiet obtained for each treatment, over fungicides applied.

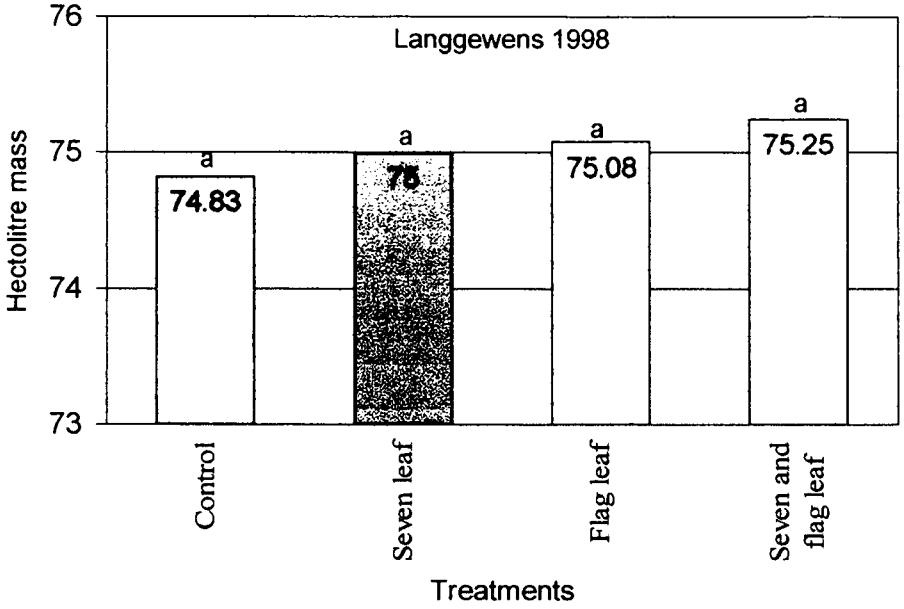

Fig. 23. Mean hectolitre mass (kg/hℓ) of the cultivar Palmiet obtained for each treatment, over fungicides applied.

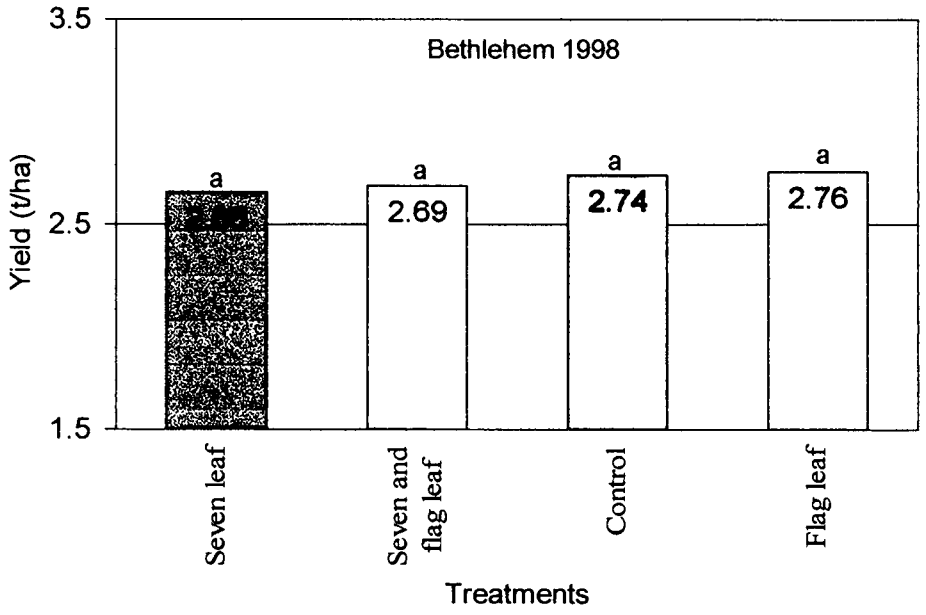

Fig. 24. Mean yield (t/ha) of the cultivar Gariep obtained for each treatment, over fungicides applied.

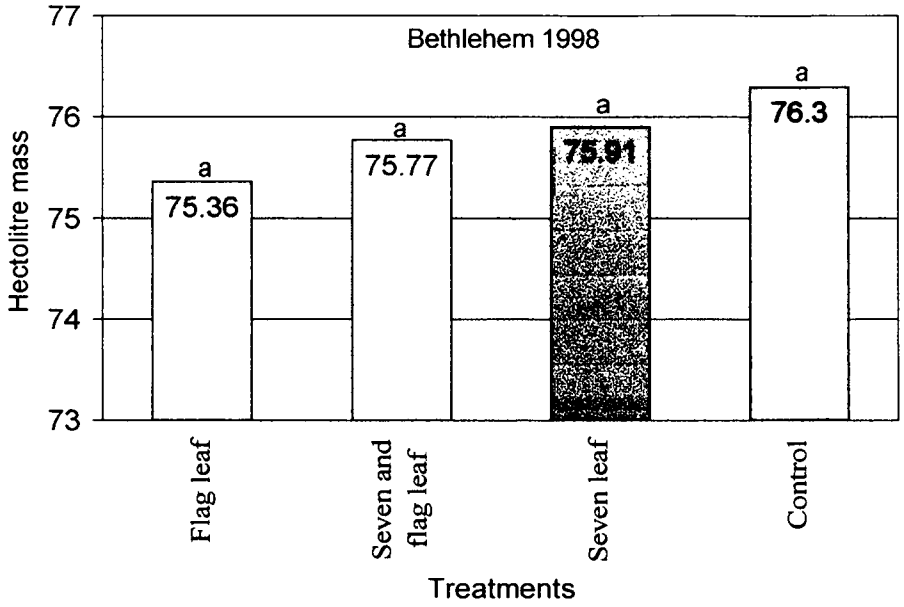

Fig. 25. Mean hectolitre mass (kg/hℓ) of the cultivar Gariep obtained for each treatment, over fungicides applied.

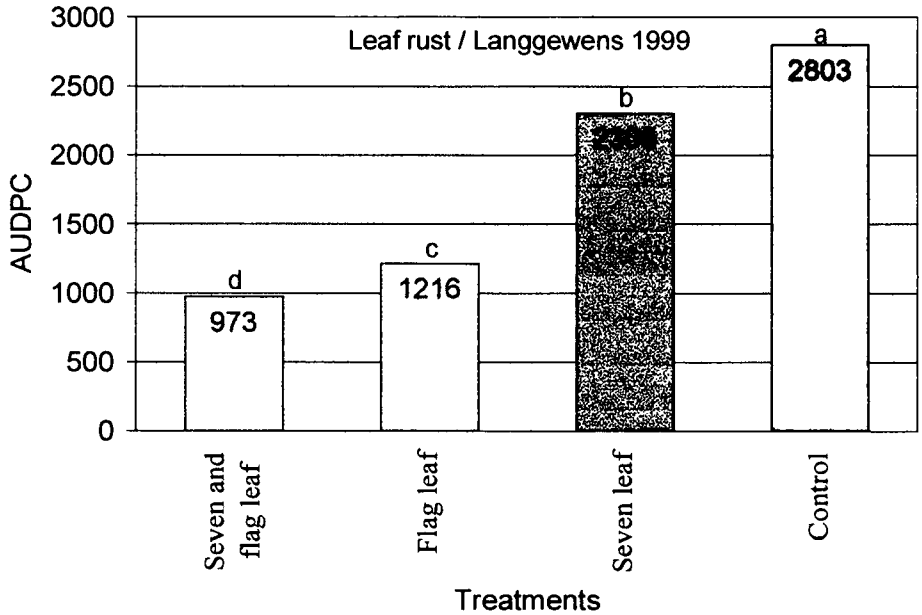

Fig. 26. Mean area under the disease progress curve calculated for each treatment on the cultivar SST 75, over fungicides applied.

varied from 53% for epoxiconazole/carbendazim to 39% for bromuconazole (Fig. 27). Leaf rust infection had a significant influence on the percentage siftings obtained. In the control plots 23% siftings were obtained, followed by 20%, 13%, and 12% for the seven leaf, flag leaf, and combined seven and flag leaf treatments, respectively (Fig. 28). The percentage siftings recovered for each fungicide, over treatments, varied from 12% for epoxiconazole/carbendazim to 18% for bromuconazole (Fig. 29). Percentage siftings were positively correlated with AUDPC (R^2=0.68, P<0.001) (Fig. 30).

The combined seven and flag leaf treatment, over fungicides applied, resulted in a 56% yield increase, followed by the flag leaf treatment with 50% and the seven leaf treatment with 15% (Fig. 31). Yield increases attained with the different fungicides, for the seven leaf treatment, varied from 4% for tebuconazole/carbendazim to 17% for cyproconazole (Fig. 32). With the flag leaf treatment differences in yield increase varied from 33% for bromuconazole to 78% for tebuconazole (Fig. 33). Similar differences were found with the combined seven and flag leaf treatments where the application of bromuconazole resulted in a 34% yield increase and cyproconazole in 70% (Fig. 34). Over treatments applied yield increase obtained with the different fungicides varied from 24% for bromuconazole to 53% for epoxiconazole/carbendazim (Fig. 35). Yield was negatively correlated with the AUDPC (Fig. 36) (R^2=0.77, P<0.001).

A significant (P<0.001) increase in the hectolitre mass was obtained with the flag leaf and combined seven and flag leaf treatments (Fig. 37). Hectolitre mass obtained with the application of fungicides, over treatments, varied from 78.1 kg hl^{-1} for propiconazole to 79.3 k hl^{-1} for tebuconazole/carbendazim (Fig. 38). Hectolitre mass was negatively correlated with AUDPC (Fig. 39) (R^2=0.60, P<0.001). Protein content, over fungicides applied, varied from 12.5% for the combined seven and flag leaf treatments to 12.8% for the seven leaf treatment (Fig. 40). Protein content was positively but weakly correlated with AUDPC (Fig. 41) (R^2=0.15).

Seed treatment trials

Small Grain Institute 1999. The use of seed treatment in combination with foliar applications of propiconazole resulted in high levels of stripe rust control at SGI

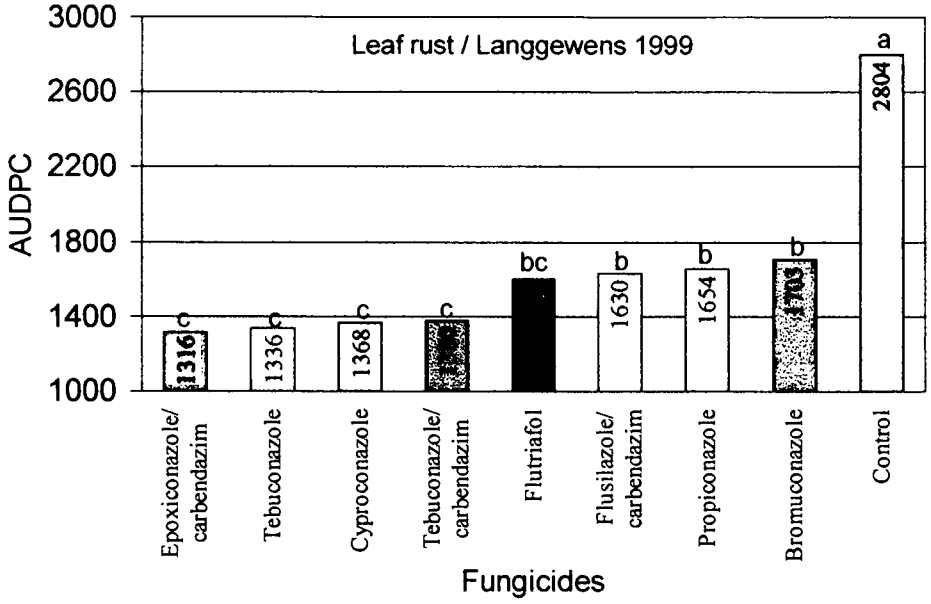

Fig. 27. Mean area under the disease progress curve calculated for the control and for each fungicide treatment on SST 75, averaged over the timing of applications.

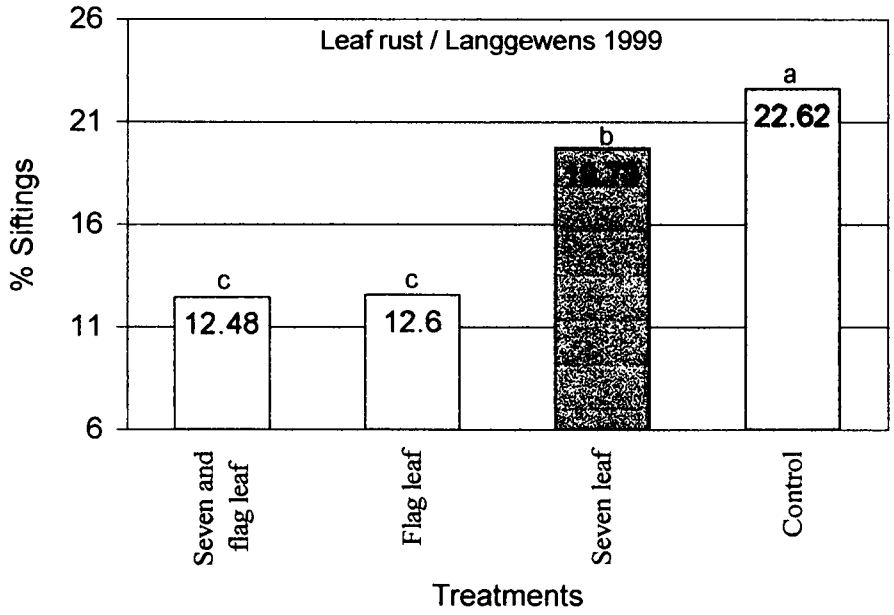

Fig. 28. Mean percentage siftings (kernels <1.5 mm) of SST 75 obtained for the control and timing of applications, averaged over fungicides.

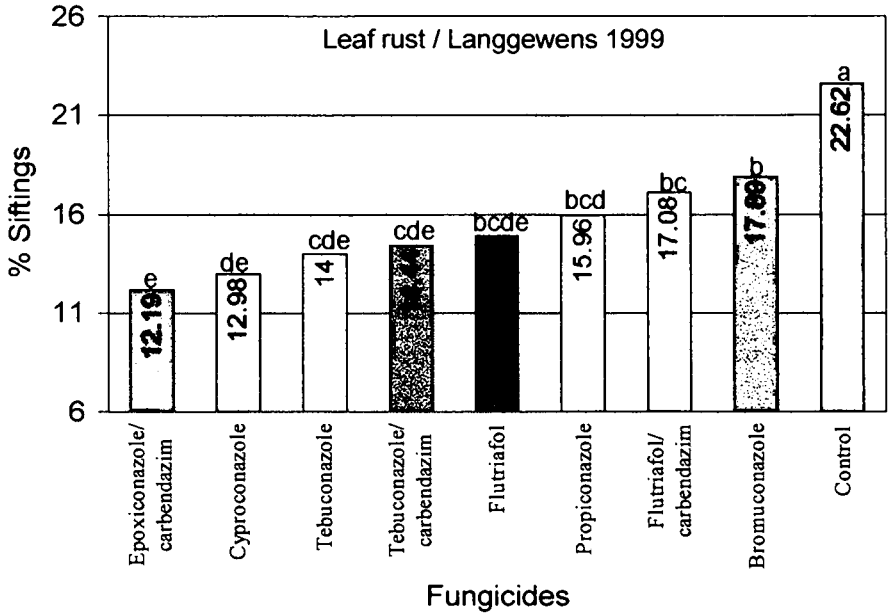

Fig. 29. Mean percentage siftings (kernels <1.5mm) of SST 75 obtained for the control and each fungicide treatment, averaged over timing of applications.

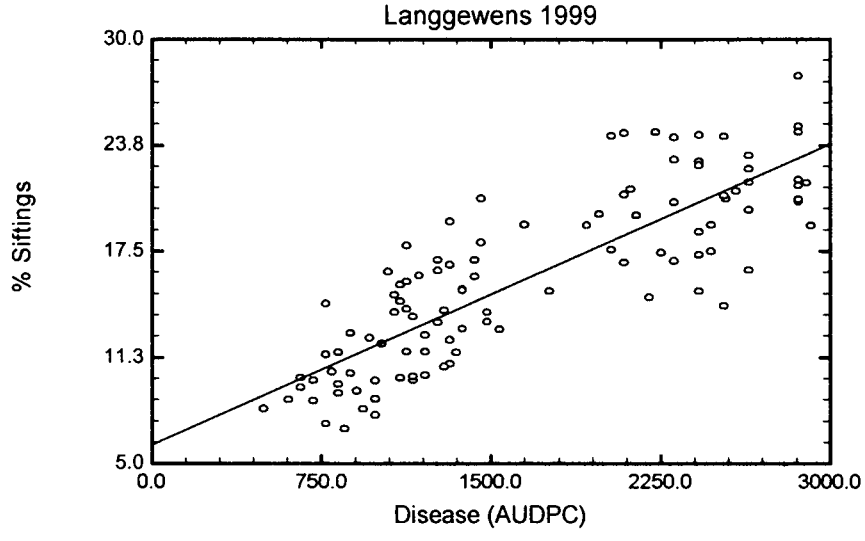

Fig. 30. Relationship between the area under the disease progress curve, calculated for leaf rust development on the cultivar SST 75, and the percentage siftings (R^2=0.68).

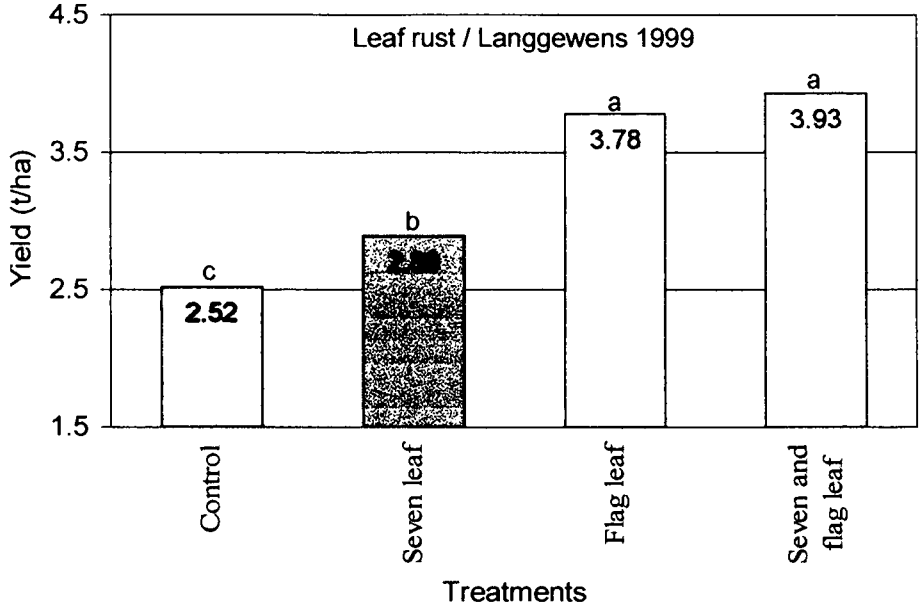

Fig. 31. Mean yield (t/ha) of SST 75 obtained for each treatment, averaged over fungicides applied.

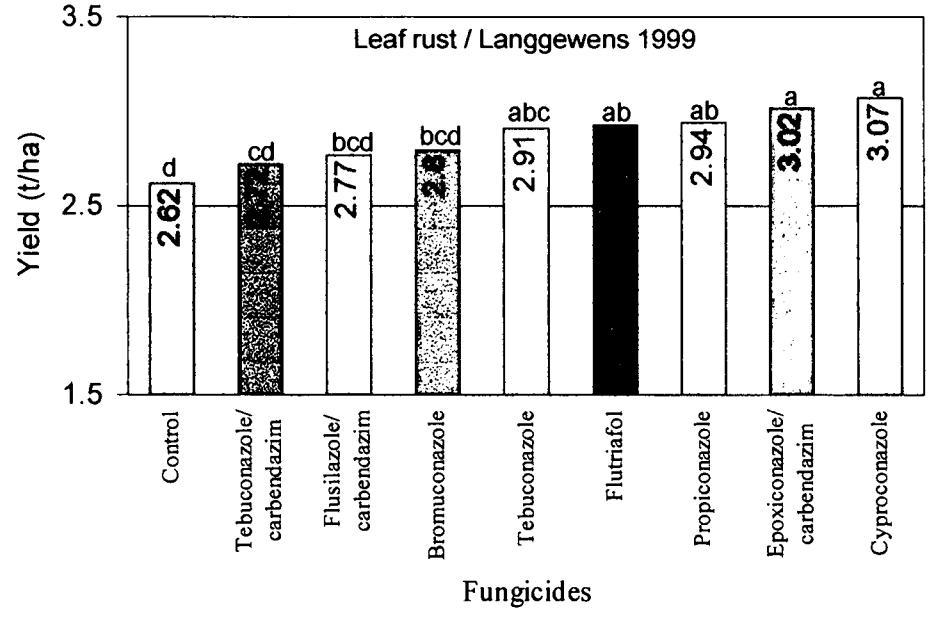

Fig. 32. Mean yield (t/ha) of SST 75 obtained for the control and each fungicide treatment applied during the seven leaf growth stage.

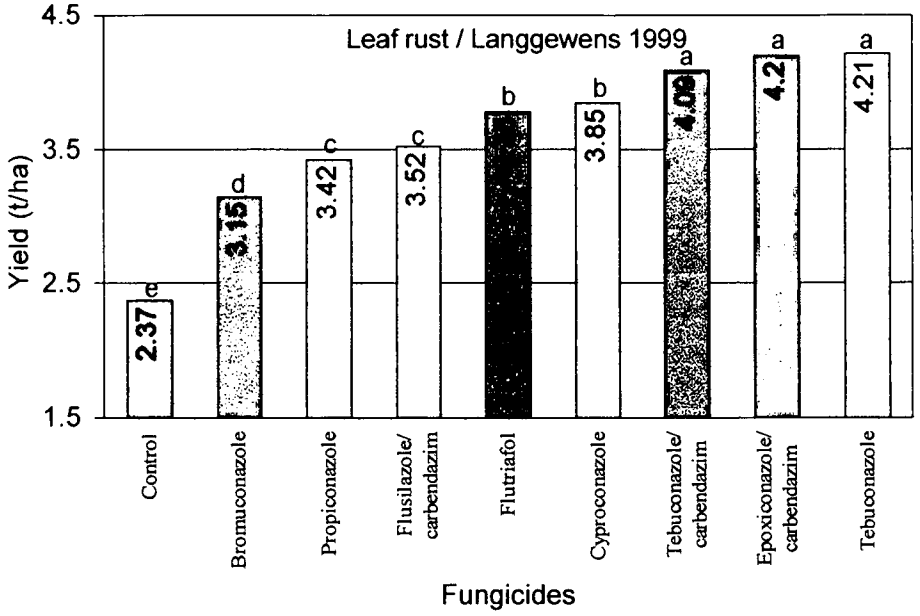

Fig. 33. Mean yield (t/ha) of SST 75 obtained for the control and each fungicide treatment applied during the flag leaf growth stage.

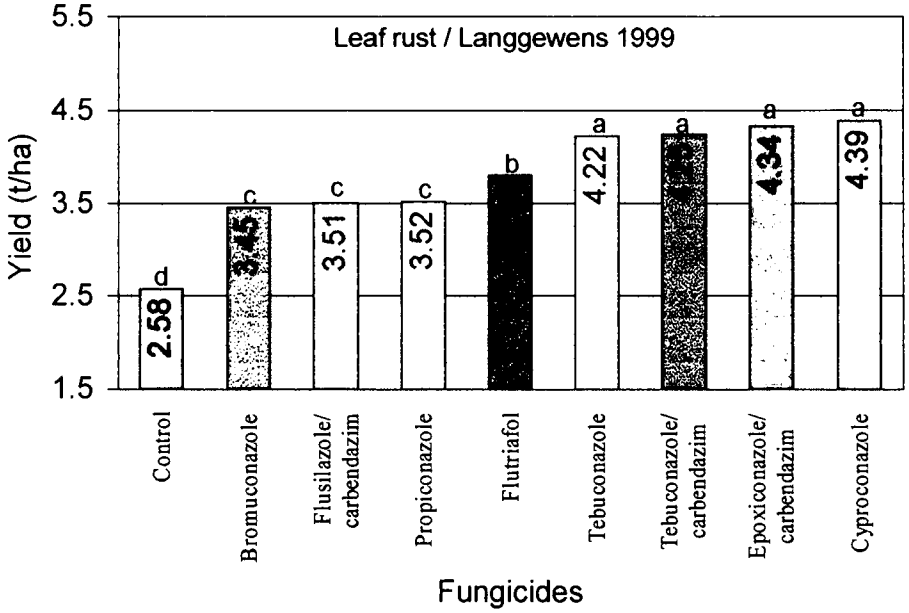

Fig. 34. Mean yield (t/ha) of SST 75 obtained for the control and each fungicide treatment applied at both the seven and flag leaf growth stages.

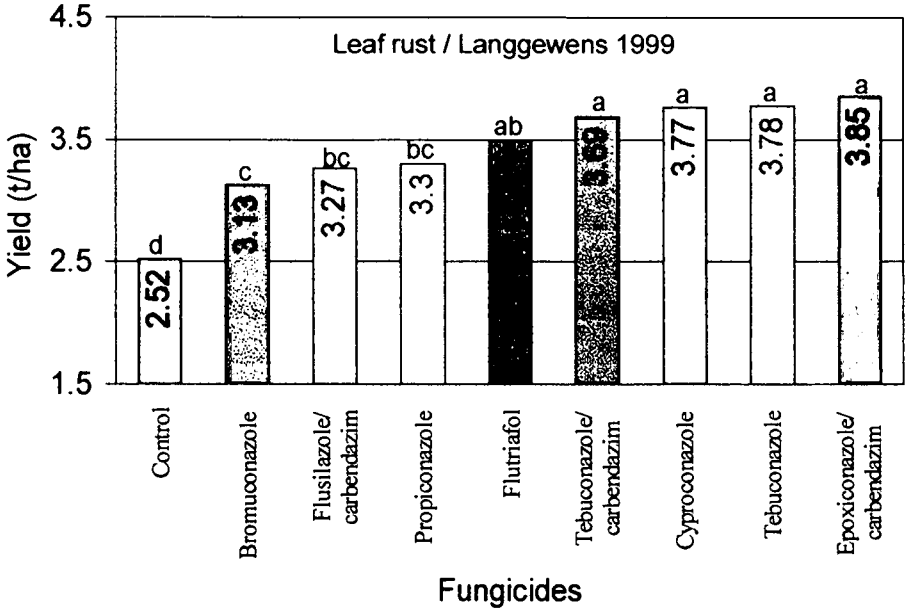

Fig. 35. Mean yield (t/ha) of SST 75 obtained for the control and each fungicide treatment, averages over timing of applications.

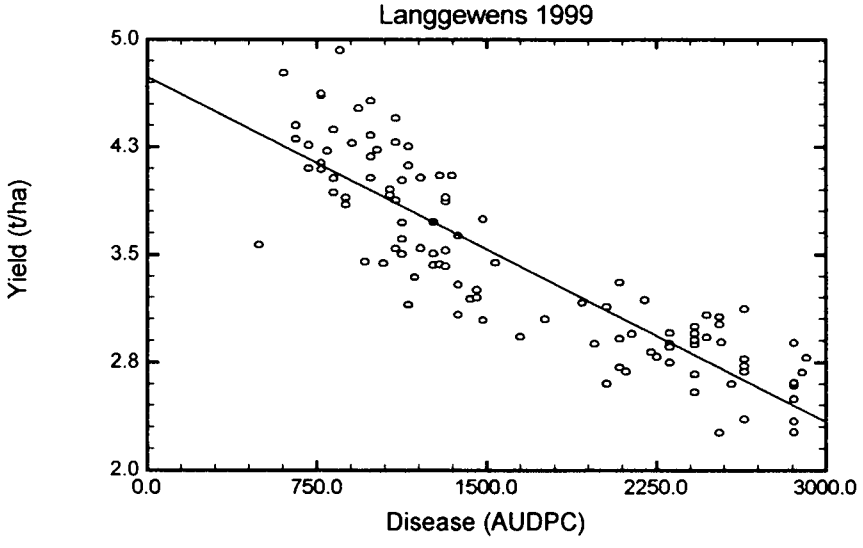

Fig. 36. Relationship between the area under the disease progress curve, calculated for leaf rust development on the cultivar SST 75, and yield (R^2=0.77).

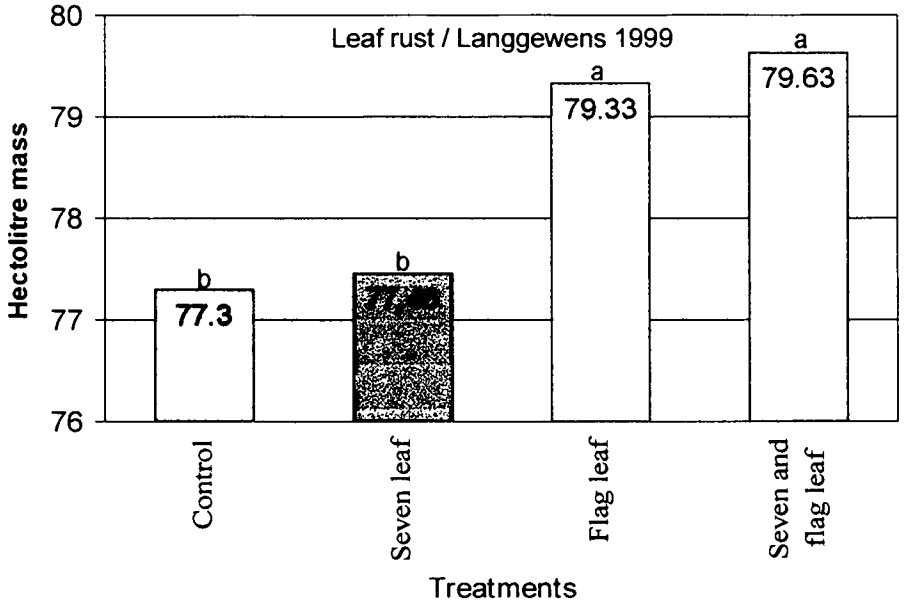

Fig. 37. Mean hectolitre mass (kg/hℓ) of SST 75 obtained for each treatment, averaged over fungicides applied.

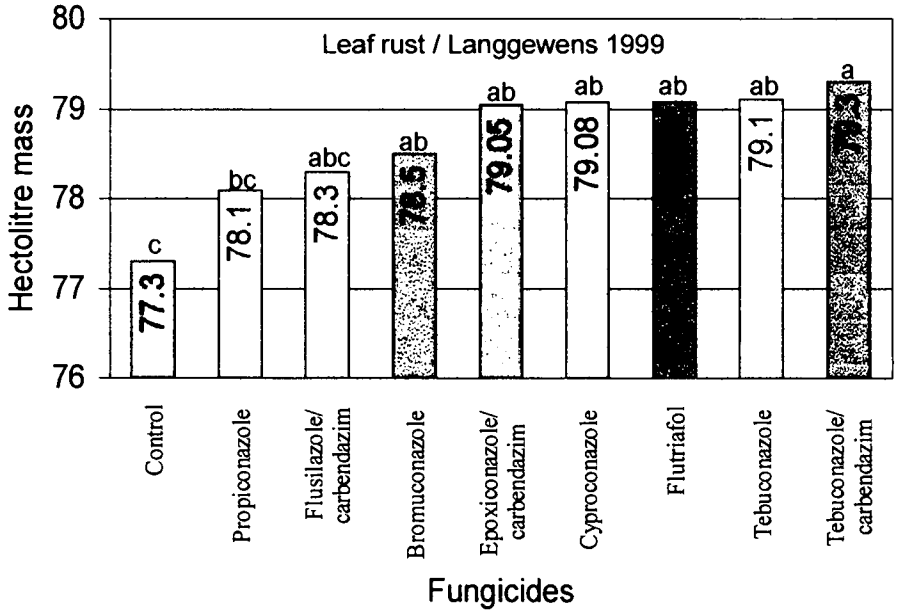

Fig. 38. Mean hectolitre mass (kg/hℓ) of SST 75 obtained for the control and each fungicide treatment, averaged over timing of applications.

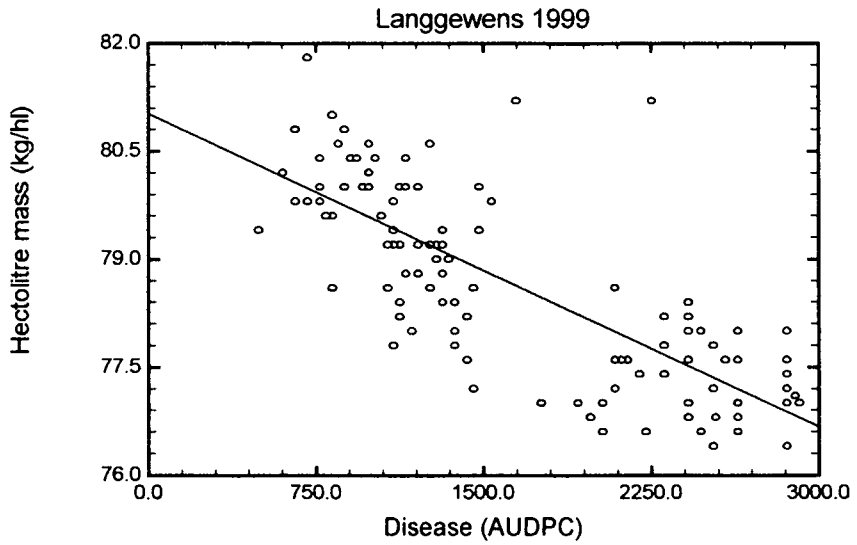

Fig. 39. Relationship between the area under the disease progress curve, calculated for leaf rust development on the cultivar SST 75, and hectolitre mass (R^2=0.60).

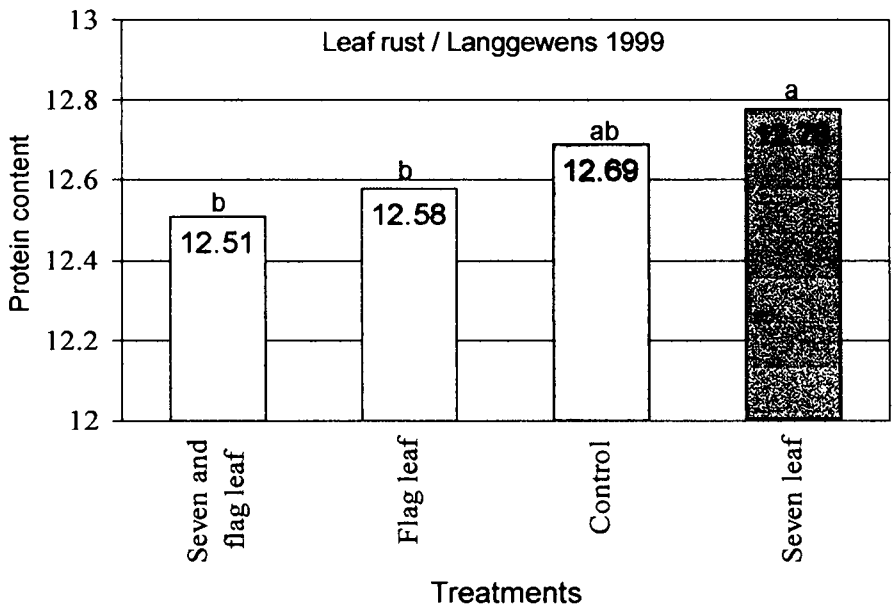

Fig. 40. Mean protein content of SST 75 measured for each treatment, averaged over fungicides applied.

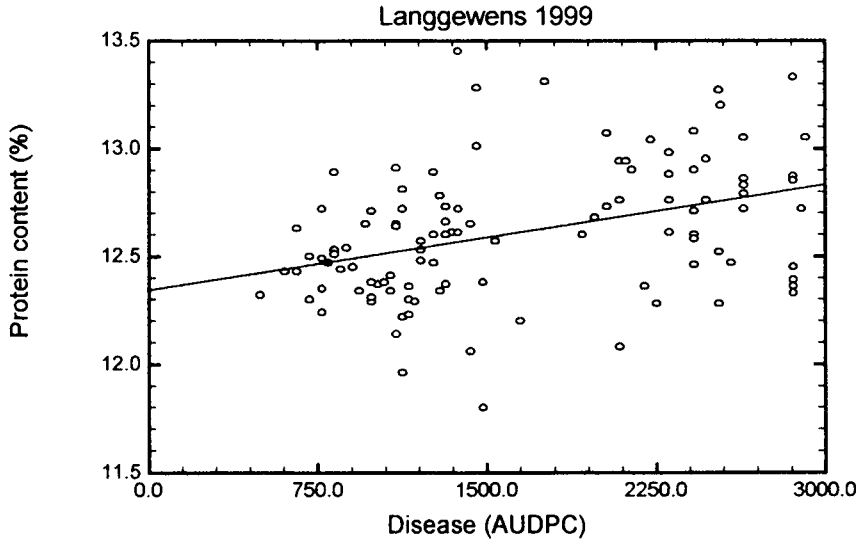

Fig. 41. Relationship between the area under the disease progress curve, calculated for leaf rust development on the cultivar SST 75, and protein content (R^2=0.15).

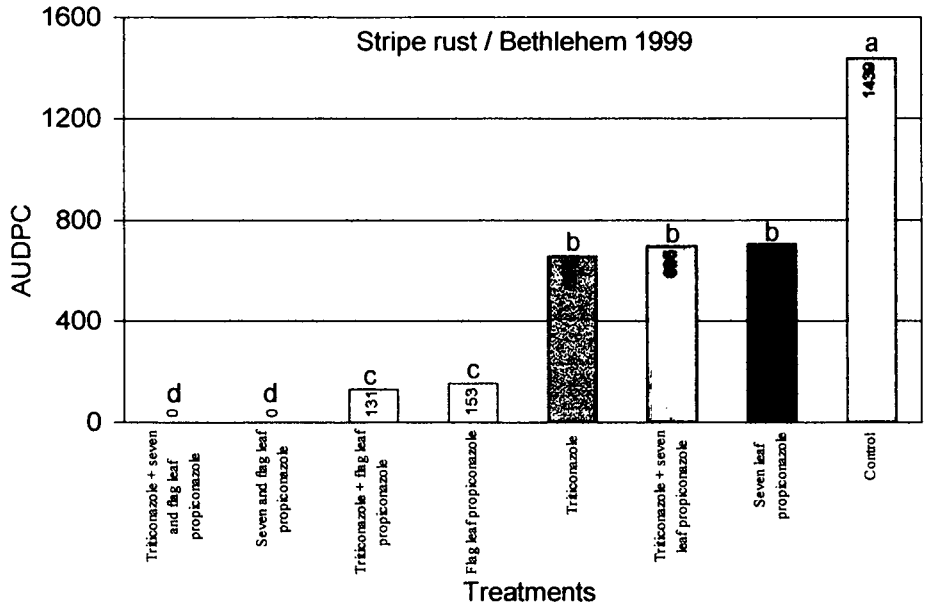

Fig. 42. Mean area under the disease progress curve, calculated for stripe rust infection of the cultivar Gariep, for the control and each foliar fungicide and/or seed treatment applied.

during 1999. The first stripe rust symptoms appeared before the application of the seven leaf treatments and reached epidemic levels a week after the application of the flag leaf treatments. No other foliar diseases occurred in the trial. Optimum timing of the flag leaf treatments resulted in high levels of stripe rust control (Fig. 42). Triticonazole seed treatment, triticonazole seed treatment combined with a seven leaf propiconazole treatment, and the seven leaf propiconazole treatment resulted in a decrease in the AUDPC of between 51 and 54%. The flag leaf propiconazole treatment, and triticonazole combined with a flag leaf application of propiconazole, resulted in a decrease in the AUDPC of 91 and 89%, respectively. No stripe rust infections occurred in plots sprayed with propiconazole at both the seven and flag leaf stages, or where triticonazole was applied in addition to the latter treatments.

Yield increased by 13% when a triticonazole seed treatment was supplemented with propiconazole sprayed at the seven leaf stage, and with 49% when the seed treatment was followed by propiconazole at both seven and flag leaf stages (Fig. 43). The flag leaf propiconazole treatment and the triticonazole seed treatment combined with a flag leaf application of propiconazole resulted in yield increases of 36% and 38%, respectively. Triticonazole seed treatment resulted in a yield increase of 16%. AUDPC was negatively correlated with yield (Fig 44) (R^2=0.85 P<0.001). Hectolitre mass varied from 72.2 kg $h\ell^{-1}$ for the control to 74.7 kg $h\ell^{-1}$ for the flag leaf propiconazole treatment (Fig. 45). Hectolitre mass was negatively correlated with AUDPC (Fig. 46) (R^2=0.62, P<0.001). Protein content varied from 16.5% for the combined seven and flag leaf propiconazole treatment to 17.3% for the control (Fig. 47). Protein content was positively correlated with the AUDPC (Fig 48) (R^2=0.28).

Significant differences (P<0.001) occurred among the different fungicides applied as seed treatments on the cultivar Gariep at SGI during 1999. The highest decrease in the AUDPC, calculated for stripe rust infection between GS 29 and GS 51, was obtained for triadimenol (61%), followed by triticonazole which resulted in a 25% decease (Fig. 49).

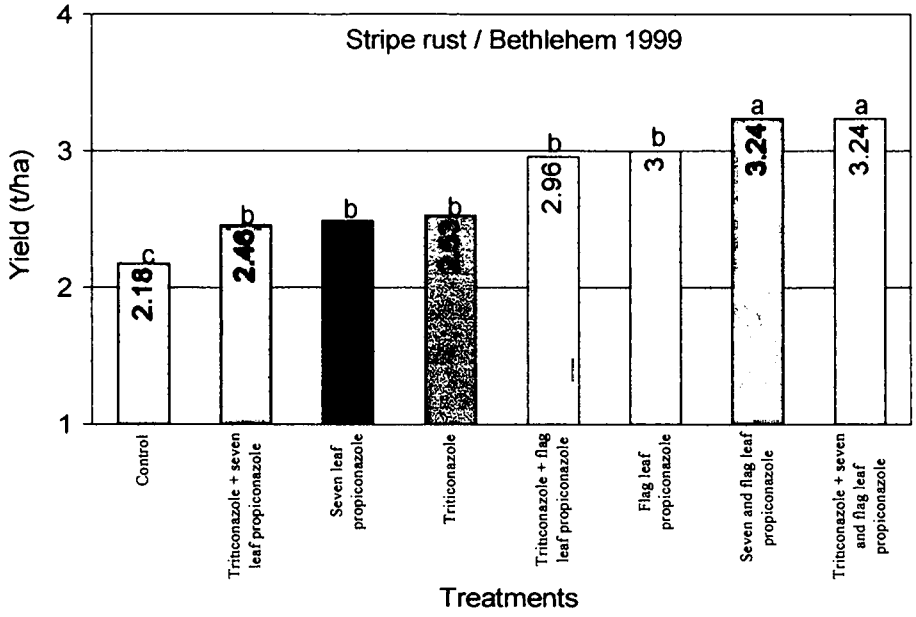

Fig. 43. Mean yield (t/ha) of Gariep obtained for the control and each foliar fungicide and/or seed treatment applied.

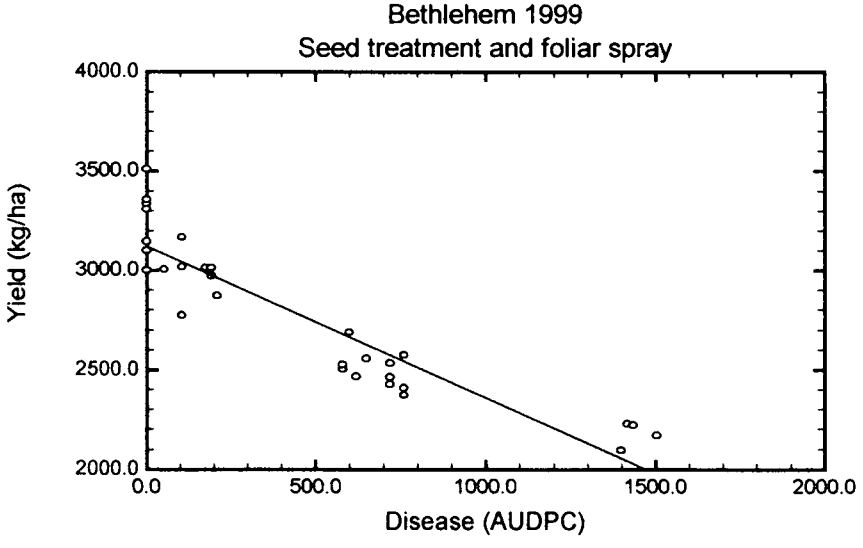

Fig. 44. Relationship between the area under the disease progress curve, calculated for stripe rust development on the cultivar Gariep, and yield (R^2=0.85).

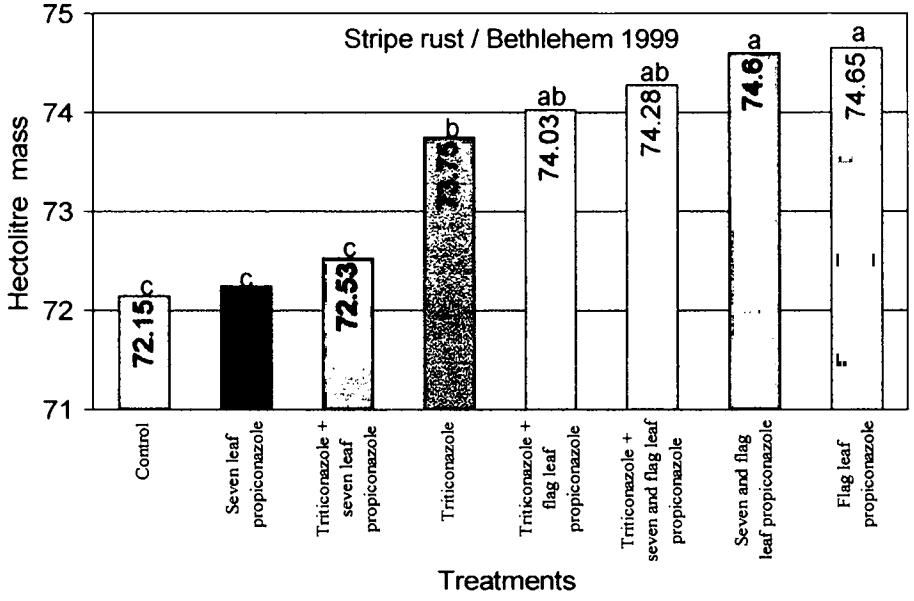

Fig. 45. Mean hectolitre mass (kg/hℓ) of Gariep obtained for the control and each foliar fungicide and/or seed treatment applied.

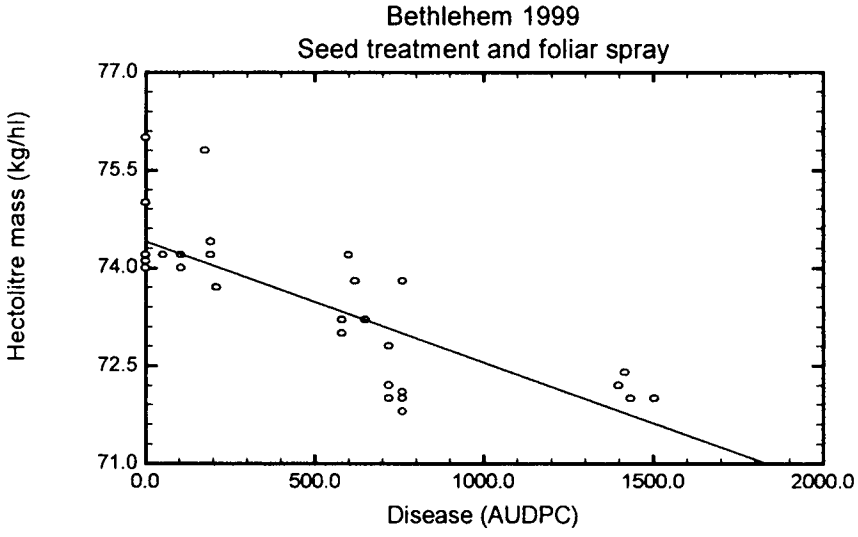

Fig. 46. Relationship between the area under the disease progress curve, calculated for stripe rust infection of Gariep, and hectolitre mass (R^2=0.62).

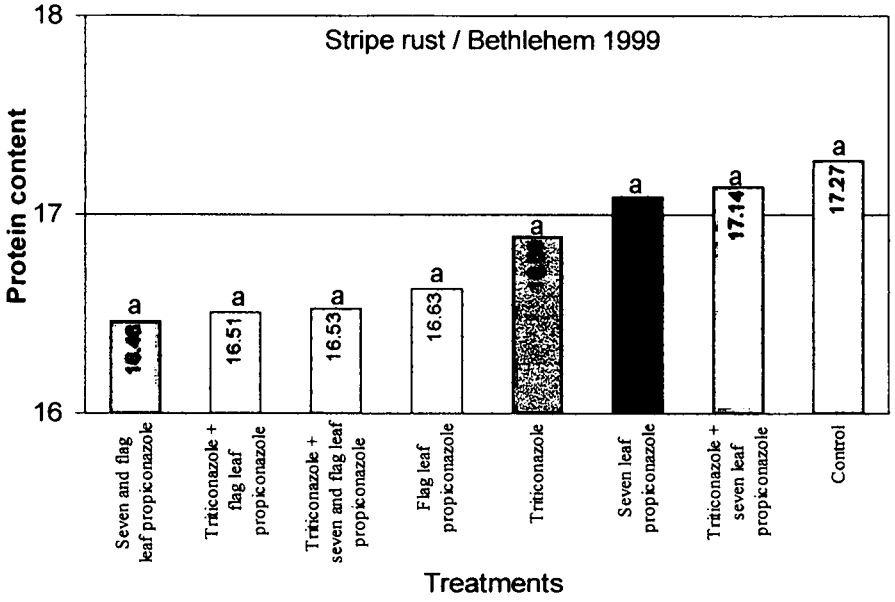

Fig. 47. Mean protein content of Gariep measured for the control and each foliar fungicide and/or seed treatment applied.

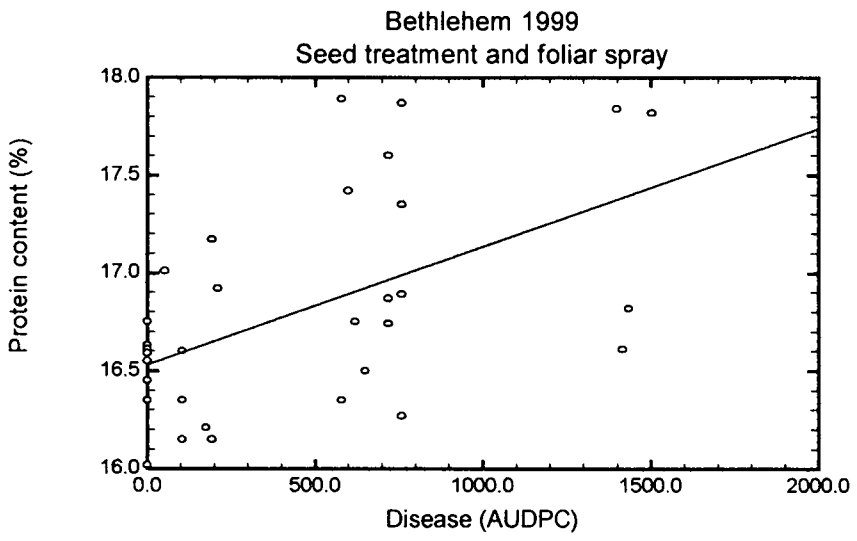

Fig. 48. Relationship between the area under the disease progress curve, calculated for stripe rust development on the cultivar Gariep, and protein content (R^2=0.28).

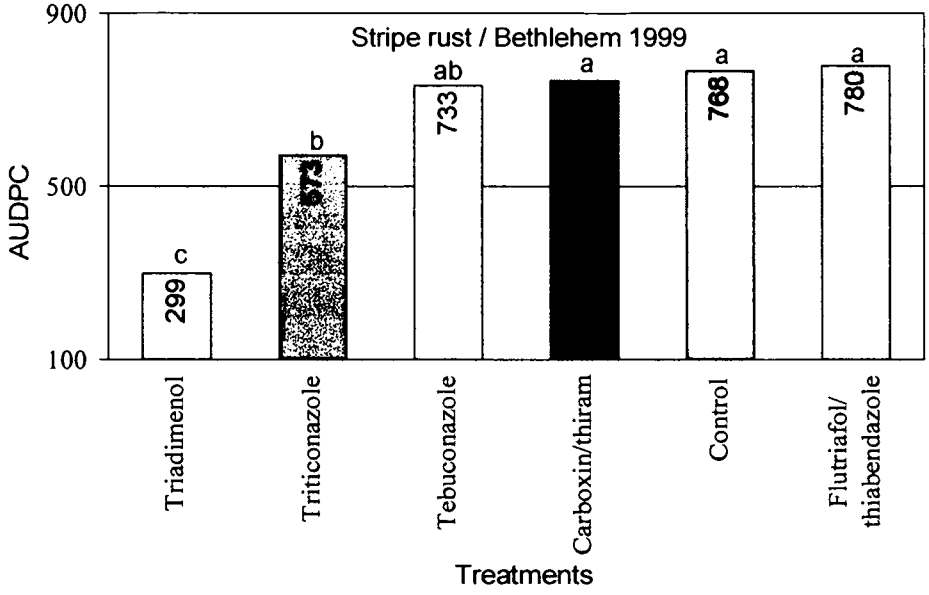

Fig. 49. Mean area under the disease progress curve, calculated for stripe rust infection of Gariep from growth stage 29 to 51, for the control and different fungicide seed treatments.

DISCUSSION

The data presented in this chapter confirm the damage potential of foliar rusts of wheat in South Africa. It is clear that the environment in the major wheat producing areas is conducive for rust epidemics and that significant economic losses will be experienced should a virulent pathotype coincide with a susceptible host and favourable climatic conditions. Depending on the time of fungicide application and active ingredient, mean yield of sprayed plots were increased by 7 to 56% on three stripe rust-susceptible spring wheat cultivars. In field studies of the leaf rust-susceptible spring wheat cultivar SST 75, mean yield of plots sprayed with eight fungicides, applied as three treatments, were increased between 4% and 78%. In the summer rainfall area the control of stripe rust on the cultivar Gariep resulted in yield increases of between 13% and 49%, depending on the treatments applied. However, in the absence of foliar rust diseases during 1998 the application of fungicides on the rust susceptible cultivars Palmiet and Gariep did not result in any yield increases. This is important especially in view of the fact that wheat farmers often follow a fixed spraying programme, irrespective of whether diseases are present or not.

In general the application of fungicides during the flag leaf growth stage resulted in higher hectolitre mass. Control of leaf and stripe rust with combined seven and flag leaf treatments, as well as with single flag leaf fungicides treatments, resulted in lower grain protein contents in comparison to the seven leaf fungicide treatments and the control plots.

Previous studies showed that the effect of foliar rusts on plant development and wheat quality depends on the onset of disease, yield potential and the level of cultivar resistance (Bever, 1937; Ash & Brown, 1990; Gaunt & Cole, 1991; Schultz & Line, 1992; Murray *et al.*, 1995; Ma & Singh, 1996; Sayre *et al.*, 1998). Ash & Brown (1990) found that early stripe rust epidemics had a larger effect on yield than late epidemics. Total grain yield and 1000-grain mass were most affected. All the yield parameters were affected by long season epidemics, with losses up to 50% in grain yield being recorded in plots of susceptible cultivars. Yield losses decreased in cultivars with higher levels of resistance in the adult-plant stage (Park *et al.*, 1988;

Murray *et al.*, 1994). According to Park *et al.* (1988) adult-plant resistance (APR) in the cultivars Cook, Bass, Banks, Kite, and Suneca is generally effective in preventing detectable yield losses due to stripe rust infection. Cultivars with lower levels of APR experienced losses of 15 to 25%, compared with 45 to 50% in the susceptible cultivar Teal. Early stripe rust epidemics, starting before the onset of APR, however, can result in significant yield losses in cultivars with seedling susceptibility and moderate to resistant adult-plant reactions (Murray *et al.*, 1994). When stripe rust appears late in the season, or when yield potential is low, the losses in moderately susceptible cultivars would be expected to be small (Ash & Brown, 1990). Ma & Singh (1996) found that slow rusting conferred by *Yr18* protected grain yield in the range of 36 to 58%, depending on the season and sowing date.

Significant differences were found among triazole and triazole/benzimidazole mixtures in their control of stripe and leaf rust. Lower AUDPC values calculated for stripe rust infection during 1997, as well as a lower percentage head infection observed with the application of the fungicides flutriafol, propiconazole, and tebuconazole, resulted in higher yield and hectolitre mass values obtained. Application of cyproconazole, epoxiconazole/carbendazim, flutriafol, tebuconazole, and tebuconazole/carbendazim, resulted in lower AUDPC values determined for leaf rust infection during 1999, fewer siftings, with consequently higher yields and hectolitre mass. Previous studies showed that fungicides may differ in their efficacy against different wheat diseases (Hardwick *et al.*, 1994; Mercer & Ruddock, 1996; Conner & Kuzyk, 1988; Turner *et al.*, 1996; Kalappanavar & Patil, 1997; Boshoff *et al.*, 1999; Cook *et al.*, 1999). This may be attributed to differences in the efficacy of their mechanism of action, and features such as net-uptake or degradation, and systemic ability of fungistatic compounds (Lyr, 1995; Parry *et al.*, 1995).

Although the timing of flag leaf treatments during 1999 was optimal, prolonged environmental conditions conducive for leaf rust development as well as a highly susceptible cultivar resulted in re-infection of the sprayed plots after only three weeks. Differences observed among the eight fungicides applied were mainly due to differences in their duration of effect. Contributing to the shorter effectivity period observed in this study are the lower recommended rates at which fungicides are registered in South Africa. This has also been recognised by Jørgensen & Nielsen

(1994) when they found that the application of fungicides at lower rates shortened their duration of effect. Applied at full dosage the duration of effect was found to be four to five weeks, whereas lower doses gave protection for three weeks only. Lower dosages are the most effective when applied preventatively, or when cultivars contain partial resistance. The application of lower doses on susceptible cultivars further requires additional treatments within a maximum of three weeks (Jørgensen & Nielsen, 1994). Lower dosages may result in insufficient levels of control, especially when timing of application is poor, when the cultivar grown is highly susceptible, or when prolonged epidemic conditions occurred (Dannenberg *et al.*, 1989; Ash & Brown, 1990; Jørgensen, 1994; Jørgensen & Nielsen, 1994; Cook *et al.*, 1999).

Previous reports indicated that fungicides have growth regulating characteristics which may increase yield in the absence of disease (Carver & Griffiths, 1981; Jordan, 1981; Priestley, 1981; Entz *et al.*, 1990; Scott, 1996). In this study no increase in yield or hectolitre mass was obtained with the application of fungicides in the absence of disease. Environmental factors, restricting optimal plant development, may have contributed to the latter. According to Davies *et al.* (1984) yield components are not necessarily affected by an increase in green leaf area due to fungicide applications.

Infection of wheat heads by stripe rust was widespread in commercial fields, planted under susceptible cultivars, in the Western Cape and eastern Free State during 1996 and 1997, respectively. Cromey (1989b) proved the economic importance of head infections in New Zealand. Kernels from infected florets weighed up to 77% less than kernels from uninfected florets. Factors that influenced the reduction in grain weight included environmental conditions, timing and severity of infection (Cromey, 1989b). The results obtained at Tygerhoek during 1997 as well as in Chapter 3 showed that wheat cultivars differ in their susceptibility to head infection. In this study the best control of head infection was obtained with the application of triazole fungicides closely to, or just after head emergence. Stripe rust head infection was reduced by 65 and 74% with the combined seven and flag leaf treatments and the flag leaf treatments, respectively. Although the application of all triazole fungicides resulted in a decrease of head infection, considerable variation

was found among fungicides in the control of head infections. In a similar study a single application of triadimefon at full head emergence reduced stripe rust floret infection from 75 to 20% (Cromey, 1989a).

Currently only triticonazole is registered as a seed treatment to control stripe rust up to the eight-leaf growth stage on wheat cultivars in South Africa. Results of the present study showed that triadimenol provided protection for a longer period. Triazole seed treatment can be used in combination with foliar sprays when cultivars are highly susceptible to rust, alone when yields are too low to justify foliar sprays, and in combination with partial (slow rusting) resistance (Rakotondradona & Line, 1984; Line, 1993). Other reports have also shown that the application of foliar fungicides can be delayed when wheat seed has been treated with a triazole (Everts & Leath, 1993, Sundin *et al.*, 1999). Triadimefon applied as a seed treatment at 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 & Line, 1984). Trials conducted by Gaunt & Cole (1991) showed that seed treatment of a susceptible cultivar with triadimenol/fuberidazole increased yield by 23% compared to a carboxin/thiram treatment, a commercial standard, with no known efficacy for stripe rust. Although the application of triadimenol/fuberidazole, as a seed treatment in New Zealand, did not always result in a yield increase, reduction in severity of stripe rust leaf infection up to ear emergence was found (Beresford, 1982).

The results of this study showed that stripe and leaf rust could severely reduce the yield of susceptible wheat cultivars in both the winter and summer rainfall areas of South Africa, especially when prolonged environmental conditions, conducive to rust development, persist. Although hectolitre mass and protein content were not influenced to the same extent as yield, significant differences occurred between treated and untreated plots. By making use of triazole seed treatment to prevent the build up of rust inoculum on susceptible cultivars during early growth stages farmers can not only lower their input costs in comparison to foliar sprays, but also the risk of epidemic outbreaks.

LITERATURE CITED

Anonymous, 2000a. Production guidelines for the production of small grains in the summer rainfall area, Small Grain Institute, Bethlehem.

Anonymous, 2000b. Production guidelines for the production of small grains in the winter rainfall area, Small Grain Institute, Bethlehem.

Ash, G.J. & Brown, J.F. 1990. Yield losses in wheat caused by stripe rust (*Puccinia striiformis* West.) in northern New South Wales. Australian Journal of Experimental Agriculture 30: 103-108.

Beresford, R.M. 1982. Stripe rust (*Puccinia striiformis*), a new disease of wheat in New Zealand. Cereal Rusts Bulletin 10: 35-41.

Bever, W.M. 1937. Influence of stripe rust on growth, water economy, and yield of wheat and barley. Journal of Agricultural Research 54: 375-385.

Boshoff, W.H.P., Pretorius, Z.A. & Swart, W.J. 1999. In vitro differences in fungicide sensitivity between *Fusarium graminearum* and *Fusarium crookwellense*. African Plant Protection 5: 65-71.

Carver, T.L.W. & Griffiths, E. 1981. Relationship between powdery mildew infection, green leaf area and grain yield of barley. Annals of Applied Biology 99: 255-266.

Conner, R.L. & Kuzyk, A.D. 1988. Effectiveness of fungicides in controlling stripe rust, leaf rust, and black point in soft white spring wheat. Canadian Journal of Plant Pathology 10: 321-326.

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

Cromey, M.G. 1989a. Infection and control of stripe rust in wheat spikes. New Zealand Journal of Crops and Horticultural Science 17: 159-164.

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

Dannenberg, M.D., Eversmeyer, M.G. & Kramer, C.L. 1989. Effect of timing of foliar fungicides on wheat disease control and yield increases. Plant Disease 73: 227-229.

Davies, W.P., Kettlewell, P.S., Green H.J., Hocking, T.J. & Jarai, M.M. 1984. Senescence and net photosynthesis of the flag leaf and grain growth of wheat in response to fungicide. Annals of Applied Biology 105: 303-312.

Entz, M.H., Van den Berg, C.G.J., Lafond, G.P., Stobbe, E.H., Rossnagel, B.G. & Austenson, H.M. 1990. Effect of late season fungicide application on grain yield and seed size distribution in wheat and barley. Canadian Journal of Plant Science 70: 699-706.

Eversmeyer, M.G. & Kramer, C.L. 1996. Modelling winter and early spring survival of *Puccinia recondita* in wheat nurseries during 1980 to 1993. Plant Disease 80: 490-493.

Everts, K.L. & Leath, S. 1993. Effect of triadimenol seed treatment and timing of foliar fungicide applications on onset and extent of powdery mildew and leaf rust epidemics. Phytopathology 83: 557-562.

Gaunt, R.E. & Cole, M.J. 1991. An analysis of yield reduction caused by stripe rust in Rongotea wheat. Australian Journal of Agricultural Research 42: 45-52.

Hardwick, N.V., Jenkins, J.E.E., Collins, B. & Groves, S.V. 1994. Powdery mildew (*Erisiphe graminis*) on winter wheat: control with fungicides and the effects on yield. Crop Protection 13: 93-98.

Ireta, M.J. & Gilchrist, S.L. 1994. *Fusarium* head scab of wheat (*Fusarium graminearum* Schwabe). Wheat Special Report No. 21b. Mexico, D.F.: Cimmyt.

Jordan, V.W.L. 1981. Effect of fungicides on *Septoria nodorum* and wheat green leaf area. EPPO Bulliton 11: 355-356.

Jørgensen, L.N. 1994. Duration of effect of EBI-fungicides when using reduced rates in cereals. In Proceedings of the Brighton Crop Protection Conference, Pests and Diseases 2: 703-710.

Jørgensen, L.N. & Nielsen, B.J. 1994. Control of yellow rust (*Puccinia striiformis*) on winter wheat by ergosterol inhibitors at full and reduced dosages. Crop Protection 13: 323-330.

Kalappanavar, I.K. & Patil, N.K. 1997. Field evaluation of fungicides against leaf rust of wheat. Journal of Maharashtra Agricultural Universities 22: 335 (Abstr.).

Khan, M.A. & Trevathan, L.E. 1997. Economical analysis of fungicide application to manage leaf rust on winter wheat in Mississippi. Pakistan Journal of Phytopathology 9: 34-40.

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

Lyr, H. 1995. Selectivity in modern fungicides and its basis. In: *Modern selective fungicides*- Properties: applications, mechanisms of action, 2nd edition, 13-22 (Ed. H Lyr). Gustav Fischer Verlag, Stuttgart.

Ma, H. & Singh, R.P. 1996. Contribution of adult plant resistance gene *Yr18* in protecting wheat from yellow rust. Plant Disease 80: 66-69.

Mercer, P.C. & Ruddock, A. 1996. Evaluation of fungicide dosage on yield, *Septoria tritici* and senescence in winter wheat in Northern Ireland. Tests of Agrochemicals and Cultivars No. 16. Annuals of Applied Biology 128: 22-23.

Murray, G.M., Ellison, P.J., Watson, A., & 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.

Murray, G.M., Ellison, P.J. & Watson, A. 1995. Effects of stripe rust on the wheat plant. Australasian Plant Pathology 24: 261-270.

Nel, A., Krause, M., Ramautar, N. & van Zyl, K. 1999. A guide for the control of plant diseases. National Department of Agriculture, Pretoria. 122pp.

Park, R.F., Rees, R.G & Platz, G.J. 1988. Some effects of stripe rust infection in wheats with adult-plant resistance. Australian Journal of Agricultural Research 39: 55-62.

Parry, D.W., Jenkinson, P. & Mcleod, L. 1995. *Fusarium* ear blight (scab) in small grain cereals – a review. Plant Pathology 44: 317-319.

Peterson, R.F., Campbell, A.B. & 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. & Kema, G.H.J. 1997. First report of *Puccinia striiformis* f. sp. *tritici* on wheat in South Africa. Plant Disease 81: 424.

Priestley, R.H. 1981. Fungicide treatment increases yield of cereal cultivars by reducing disease and delaying senescence. EPPO Bulliton 11: 357-363.

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

Sayre, K.D., Singh. R.P., Huerta-Espino, J. & Rajaram, S. 1998. Genetic progress in reducing losses to leaf rust in CIMMYT-Derived Mexican spring wheat cultivars. Crop Science 38: 654-659.

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

Scott, D.B. 1996. Disease control and yield improvement obtained with fungicides applications to spring wheat in South Africa. Crop Protection 15: 167170.

Singh, D.P. 1999. Yield losses due to brown rust in two popular cultivars of wheat. Plant Disease Research 14: 60-62. (Abstr.).

Sundin, D.R., Bockus, W.W. & Eversmeyer, M.G. 1999. Triazole seed treatments suppress spore production by *Puccinia recondita*, *Septoria tritici*, and *Stagonospora nodorum* from wheat leaves. Plant Disease 83: 328-332.

Torabi, M., Mardoukhi, V., Nazari, K. Afshari, F., Forootan, A.R., Ramai, M.A., Golzar, H. & Kashani, A.S. 1995. Effectiveness of wheat yellow rust resistance genes in different parts of Iran. Cereal Rusts and Powdery Mildews Bulletin 23: 9-12.

Turner, J.A., Elcock, S.J. & Hilms, M.J. 1996. Monitoring fungicide sensitivity in populations of *Septoria tritici*. In Proceedings of the Brighton Crop Protection Conference. Pests and Diseases 2: 695-700.

Wagoire, W.W., Stølen, O. Hill, J. & Ortiz, R. 1998. Is there a 'cost' for wheat cultivars with genes for resistance to yellow rust caused by *Puccinia striiformis*. Crop Protection 17: 337-340.

Wiese, M.V. 1987. Compendium of Wheat Diseases, Second Edition. St Paul, Minnesota: APS Press.

Yang, X.B. & Zeng, S.M. 1989. Effect of yellow rust on yield components of winter wheat in China. Plant Pathology 38: 1-8.

Zadoks, J.C., Chang, T.T. & Konzak, C.F. 1974. A decimal code for the growth stages of cereals. Weed Research 14: 415-426.

SUMMARY

Stripe rust, caused by *Puccinia striiformis* Westend. f. sp. *tritici* Eriks., has become an endemic disease of wheat (*Triticum aestivum* L.) in South Africa after being observed for the first time near Moorreesburg, Western Cape, during August 1996. Pathotypes (pts.) detected in surveys were 6E16A- with virulence to *Yr2,6,7,8,11,14,17* and *Yr19,* and 6E22A- with added virulence to *Yr25.* Stripe rust isolates found on *Hordeum murinum* L. in the Western Cape were identified as pt. 6E16A- whereas both pts. 6E16A- and 6E22A- were collected from *Bromus catharticus* Vahl in the eastern Free Sate. The possible role that grass species may play in the over-summering of the stripe rust pathogen has not yet been fully established. However, stripe rust infections have been found on summer-sown wheat in the south Western Cape during 1998, volunteer wheat growing in the summer and autumn months in the eastern Free State from 1998 to 2000, and on summer-sown wheat in Lesotho.

The reaction of 55 South African and 18 foreign wheat cultivars was determined to pts. 6E16A- and 6E22A- in both the seedling and adult plant stage. The occurrence of stripe rust head infections was studied using 16 spring wheat cultivars and 17 supplemental lines. Six of the 55 local wheat cultivars expressed seedling resistance, 18 appeared heterogeneous and 31 were susceptible. The mean area under the disease progress curve (AUDPC) determined in the field for 42 cultivars over a three year period showed that 11 cultivars expressed high levels of complete or adult plant resistance (AUDPC <200). Twelve cultivars expressed intermediate levels of resistance (AUDPC 200 to 500) and 19 displayed AUDPC values of 500 to 1598. The percentage head infection was positively correlated to stripe rust severity on flag leaves. Of the 18 foreign cultivars evaluated 10 were resistant in both seedling and adult plant stages. The remaining eight cultivars were susceptible as seedlings but showed high levels of adult plant resistance in the field.

Field trials were conducted from 1997 to 1999 to determine the effect of stripe and leaf rust (*P. triticina* Eriks.) epidemics on yield and quality of wheat. Five triazole fungicides, applied at two growth stages on three cultivars in the south Western Cape during 1997, resulted in a mean decrease of 31% in the AUDPC calculated for

stripe rust infection. The application of fungicides closely to, or just after head emergence, resulted in a 65 to 74% decrease in the occurrence of stripe rust head infections. In contrast, head infection was reduced by only 8% when fungicides were applied at the seven leaf stage. Combined seven and flag leaf treatments with propiconazole, averaged over the three cultivars, resulted in a 56% yield increase, followed by increases of 49%, 44%, 39% and 25% with tebuconazole, flutriafol, bromuconazole, and flusilazole, respectively. In the absence of disease during 1998 no fungicide treatment resulted in a significant yield or hectolitre mass increase in any of the trials.

The application of eight fungicides at both seven and flag leaf stages at Langgewens in the Western Cape during 1999, resulted in a mean decrease of 65% in the AUDPC, calculated for leaf rust severity. Combined seven and flag leaf treatments resulted in a mean yield increase of 56%, followed by 50 and 15% for the flag leaf and seven leaf treatments, respectively. Over treatments applied, yield increases varied from 24% for bromuconazole to 53% for epoxiconazole/carbendazim. Furthermore, the application of a flag leaf, and combined seven and flag leaf treatments, resulted in a significant increase in hectolitre mass.

During 1999 the combination of triticonazole seed treatment with a propiconazole flag leaf spray on the cultivar Gariep in the eastern Free State resulted in a 91% decrease in the stripe rust AUDPC, and an associated 36% yield increase. Hectolitre mass increased by 3% and protein content decreased by 4% for the latter treatment. Triticonazole seed treatment had a 54% decrease in the AUDPC resulting in 16 and 2% yield and hectolitre mass increase, respectively. 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. The latter two treatments resulted in a 49% yield increase.

The results obtained in yield loss studies emphasise the importance of research aimed at the genetic control of rust diseases of wheat in South Africa. Effective and longlasting genetic control can only be obtained by coordinating future research, continuous monitoring of changes in the pathotype population, the regular

collection of germ plasm carrying new or unused sources of resistance, characterising current sources of resistance, and by deploying available sources of resistance in a responsible manner. The result of successful genetic control is not only aimed at preventing the repeated application of fungicides, but also at reducing risk in wheat production, thereby ensuring more stable yields of high quality.

Keywords: *Puccinia striiformis*, *Puccinia triticina*, stripe rust, leaf rust, pathotypes, cultivar resistance, chemical control, seed treatment

OPSOMMING

Streeproes, veroorsaak deur *Puccinia striiformis* Westend. f. sp. *tritici* Eriks., het 'n endemiese siekte van koring (*Triticum aestivum* L.) in Suid-Afrika geword na die eerste waarneming daarvan naby Moorreesburg in die Wes-Kaap gedurende Augustus 1996. Patotipes (pts.) gevind in gereelde opnames was 6E16A- met virulensie vir *Yr2,6,7,8,11,14,17* en *Yr19* en 6E22A- met bykomende virulensie vir *Yr25.* Streeproesisolate verkry vanaf *Hordeum murinum* L. in die Wes-Kaap is geïdentifiseer as pt. 6E16A-, terwyl beide pts. 6E16A- en 6E22A- geïdentifiseer is vanaf *Bromus catharticus* Vahl in die Oos-Vrystaat. Die moontlike rol wat grasspesies speel in die oorsomering van die streeproespatogeen kon nie volledig uitgeklaar word nie. Streeproesinfeksies is egter gevind op someraanplantings van koring in die suidelike Wes-Kaap gedurende 1998, asook gedurende 1998-2000 op opslagkoring in die somer- en herfsmaande in die Oos-Vrystaat, sowel as op someraanplantings van koring in Lesotho.

Die reaksie van 55 Suid-Afrikaanse en 18 buitelandse koringkultivars is bepaal teen pts. 6E16A- en 6E22A- in die saailing- en volwasseplantstadiums. Die voorkoms van streeproesaarinfeksies is bestudeer deur gebruik te maak van 16 lentekoringkultivars en 17 bykomstige lyne. Ses van die 55 plaaslike kultivars het saailingweerstand getoon, 18 het heterogeen voorgekom en 31 was vatbaar. Die gemiddelde area onder die siekte-ontwikkelingskurwe (AOSOK), soos bepaal in die veld vir 42 kultivars oor 'n driejaar periode, het getoon dat 11 kultivars oor hoë vlakke van algehele of volwasseplantweerstand beskik (AOSOK <200). Twaalf kultivars het intermediêre vlakke van weerstand getoon (AOSOK 200 tot 500) met 19 kultivars waarvan die AOSOK tussen 500 en 1598 gevarieer het. Die persentasie aarinfeksie was positief gekorreleer met streeproesintensiteit op vlagblare. Van die 18 buitelandse kultivars was 10 weerstandbiedend in beide saailing- en volwasseplantstadiums. Die oorblywende agt kultivars was vatbaar as saailinge met hoë vlakke van volwasseplantweerstand in die veld.

Veldproewe is uitgevoer vanaf 1997 tot 1999 om die effek van streep- en blaarroesepidemies (*P. triticina* Eriks.) op opbrengs en kwaliteit van koring te bepaal. Vyf triasoolswamdoders, toegedien tydens twee groeistadiums op drie kultivars in

die suid Wes-Kaap gedurende 1997, het aanleiding gegee tot 'n 31% afname in die streeproes AOSOK. Die toediening van swamdoders, naby of net na aarverskyning, het 'n 65- tot 74% afname in die voorkoms van streeproes-aarinfeksies meegebring. In kontras hiermee het aarinfeksie met 8% afgeneem wanneer swamdoders slegs op die seweblaargroeistadium toegedien is. Gekombineerde sewe- en vlagblaarbehandelings met propikonasool, oor die drie kultivars, het 'n 56% opbrengstoename gerealiseer, gevolg deur opbrengsverhogings van 49%, 44%, 39% en 25% met tebukonasool, flutriafol, bromukonasool en flusilasool, onderskeidelik. In die afwesigheid van siekte gedurende 1998 kon geen swamdoderbehandeling 'n betekenisvolle opbrengs of hektolitermassaverhoging realiseer in enige van die proewe nie.

Met die toediening van agt swamdoders op beide sewe- en vlagblaargroeistadiums op Langgewens in die Wes-Kaap gedurende 1999, is 'n gemiddelde afname van 65% in die blaarroes AOSOK verkry. Die resultaat van die gekombineerde sewe- en vlagblaartoedienings was 'n gemiddelde toename van 56% in opbrengs, gevolg deur 50% en 15% vir die vlagblaar- en seweblaartoedienings, onderskeidelik. Oor behandelings toegepas het opbrengstoenames gevarieer vanaf 24% vir bromukonasool tot 53% vir epoksikonasool/karbendasim. Die toediening van die vlagblaar- en gekombineerde sewe- en vlagblaartoedienings het 'n betekenisvolle toename in hektolitermassa gerealiseer.

Gedurende 1999 het die kombinasie van 'n tritikonasool saadbehandeling met 'n propikonasool vlagblaarbehandeling op die kultivar Gariep in die Oos-Vrystaat 'n 91% afname in die AOSOK bereken vir streeproes teweeggebring, met 'n gevolglike 36% opbrengstoename. Hektolitermassa het toegeneem met 3% en proteïeninhoud het afgeneem met 4% vir laasgenoemde behandeling. Tritikonasool saadbehandeling het 'n 54% afname in die AOSOK tot gevolg gehad wat 16% en 2% opbrengs- en hektolitermassatoenames onderskeidelik teweeggebring het. Die beste beheer van streeproes is verkry met 'n gekombineerde sewe- en vlagblaarbehandeling van propikonasool, sowel as met tritikonasool saadbehandeling gekombineer met 'n sewe- en vlagblaartoediening van propikonasool. Laasgenoemde twee behandelings het in 'n 49% opbrengsverhoging gerealiseer.

Oesverliesstudies het die belangrikheid van navorsing wat daarop gerig is om roessiektes van koring geneties te beheer in Suid-Afrika baie sterk beklemtoon. Effektiewe genetiese beheer kan slegs bereik word deur die koördinering van toekomstige navorsing, deur volgehou patogeenmonitering, die versameling van nuwe of onbenutte weerstandsbronne, die karaktarisering van bestaande weerstandsbronne, en deur beskikbare weerstand op 'n verantwoordelike wyse te ontplooi. Die resultaat van suksesvolle genetiese beheer het nie net ten doel om die herhaaldelike toediening van swamdoders te voorkom nie, maar ook om die risiko van koringverbouing te verlaag en om by te dra tot 'n meer stabiele jaarlikse opbrengs van hoë kwaliteit.

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

Seedling infection types	Description
0	no visible symptoms
;	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
Field response	
0	no visible 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, generally with little or no chlorosis and no necrosis
Severity (Field response)	Severity is recorded as the percentage (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
60S	60% severity with a susceptible field response

[a]McIntosh, R.A., Wellings, C.R. & Park, R.F. 1995. Wheat rusts: An atlas of resistance genes. Kluwer, Dordrecht. 200 pp.

Appendix 2. Names and pedigrees of South African cultivars

Cultivar	Pedigree
Adam Tas	SST16*3//5*T4/S67-336
Chokka	SST16/Aurora
Belinda	Ottowa*2/Cheyenne (Nebraska 62384)
Betta	Klein Lucero/Klein 157//Klein Orgullo (Klein Impacto)
Betta DN	Betta*4/SA 1648
Caledon	Betta//Monon/Arthur.Oh130/3/*3Gaudam 1/Fisai
Carina	Hybrid
Caritha	Hybrid
Carol	Hybrid
Elands	Betta//Monon/Arthur.Oh130/3/*3Gaudam 1/Fisai
Gamka	*T. timopheevi* der. P6297/Agent
Gamtoos	Kavkaz/Buho "S"//Kalyansona/Bluebird (Veer #3)
Gariep	Betta//Monon/Arthur.Oh130/3/*3Gaudam 1/Fisai
Hugenoot	Betta//Flamink/Amigo
Inia	Lerma Rojo 64/Sonora 64
Karee	Betta//Triumph/CI 13523
Kariega	SST44//K4500.2/Sapsucker "S"
Letaba	Warrior*5/Agent//Kavkaz
Limpopo	Betta*4//Gaudum 1/Fisai
Marico	Clement/Moclis 73//Torum (Broadbill)
Molen	Betta/3/Yaktana//N10B/Mazoe
Molopo	Betta//Monon/Arthur.Oh130
Nantes	SST16*3//5*T4/S67-336
Oom Charl	Betta//MN*1972
Palmiet	SST3*2//Scout*5/Agent
PAN 3211	Confidential
PAN 3232	Confidential
PAN 3235	Confidential
PAN 3349	Confidential
PAN 3377	Confidential

Appendix 2 (cont.). Names and pedigrees of South African cultivars

Cultivar	Pedigree
Scheepers 69	Unknown, selection from Scheepers ex India
SST 16	Inia 66/Calidad
SST 33	Reward/CI 12632//3*Flameks/3/3*SST3
SST 38	Palmiet/4/SST16*4/3/SST3*3//Fm3/H441
SST 44	T4*5/S67-336
SST 55	SST16*3//5*T4/S67-336/3/4*SST16/Eagle
SST 57	SST16*3//5*T4/S67-336/3/A2398
SST 65	Nantes/4/Palmiet/A2398/3/SST66*2//Pat25/Alondra sib
SST 66	LD 398/LD357//St 464/3/3*Flameks/4/3*SST16
SST 75	NTS/4/A2398/3/SST16*3//T4*5/S67-336
SST 102	Betta*2/Agent
SST 107	Triumph/Agent//4*Scheepers69/3/Schprs69/Tifton412*Schprs69
SST 124	Bezostaya//Betta/Line W
SST 333	SST124*4/ENT1
SST 363	SST124*3/RWA-R
SST 367	PI 137739/SST102//Hugenoot/PI 262660
SST 936	Hybrid
SST 966	Hybrid
SST 972	Hybrid
SST 822	SST86*3/3/SST16*3//5*T4/S67-336//3*Nana//T4/Aurora
SST 825	Kavkaz/Buho//Kalyansona/Bluebird/3/Hermosilo 77/Sapsucker (Tui "S")
SST 876	Palmiet/A2398//Adam Tas/3/SST 825
Tugela	Kavkaz/Jaral
Tugela DN	Tugela*4/SA 1684
T4	Lerma Rojo/N10B//*3Ane

Appendix 3. Analysis of variance for AUDPC calculated for stripe rust infection of spring type cultivars

Year: 1997

Source	d.f.	s.s.	m.s.	F-value	Pr>F
Lower/flag leaves	1	49785	49785	1499.4	
Residual	2	66	33	0.01	
Cultivar	15	29185437	1945696	651.69	<0.001
Lower/flag/cultv	15	382379	25492	8.54	<0.001
Residual	30	89568	2986		
Total	63	29707234			

l.s.d. lower/flag leaves = 78.91; l.s.d. cultv = 108.08; l.s.d. lower/flag/cultv = 111.59

cv% lower/flag leaves = 0.2; cv% lower/flag leaves /rep./units = 6.0

Year: 1998

Source	d.f.	s.s.	m.s.	F-value	Pr>F
Planting date	1	2995	2995	0.21	
Residual	2	28425	14212	2.73	
Cultivar	16	16285318	1017832	195.39	<0.001
Planting date/cultv	16	172261	10766	2.07	<0.039
Residual	32	166696	5209		
Total	67	16655694			

l.s.d. planting date = 103.96; l.s.d. cultv = 154.73; l.s.d. planting date/cultv = 147.02

cv% planting date/rep. = 4.5; cv% planting date/rep./units = 11.2

Year: 1999

Source	d.f.	s.s.	m.s.	F-value	Pr>F
Planting date	1	11756	11756	0.75	
Residual	2	31239	15619	2.33	
Cultivar	17	35641674	2096569	312.8	<0.001
Planting date/cultv	17	253298	14900	2.22	<0.023
Residual	34	227889	6703		
Total	71	36165855			

l.s.d. planting date = 117.65; l.s.d. cultv = 172.55; l.s.d. planting date/cultv = 166.38

cv% planting date/rep. = 3.0; cv% planting date/rep./units = 8.2

Appendix 4. Analysis of variance for AUDPC calculated for stripe rust infection of winter type cultivars

Year: 1997

Source	d.f.	s.s.	m.s.	F-value	Pr>F
Lower/flag leaves	1	502710	502710	138.24	
Residual	2	7273	3637	0.57	
Cultivar	21	18511689	881509	137.24	<0.001
Lower/flag/cultv	21	1477253	70345	10.95	<0.001
Residual	42	269761	6423		
Total	87	20768686			

l.s.d. lower/flag leaves = 114.36; l.s.d. cultv = 159.96; l.s.d. lower/flag/cultv = 161.74

cv% lower/flag leaves = 2.2; cv% lower/flag leaves /rep./units = 13.5

Year: 1998

Source	d.f.	s.s.	m.s.	F-value	Pr>F
Planting date	1	68121	68121	543.61	
Residual	2	251	125	0.04	
Cultivar	24	13452053	560502	191.39	<0.001
Planting date/cultv	24	451273	18803	6.42	<0.001
Residual	48	140574	2929		
Total	99	14112272			

l.s.d. planting date = 76.94; l.s.d. cultv = 106.7; l.s.d. planting date/cultv = 108.81

cv% planting date/rep. = 0.6; cv% planting date/rep./units = 15.3

Year: 1999

Source	d.f.	s.s.	m.s.	F-value	Pr>F
Planting date	1	7552	7552	0.57	
Residual	2	26349	13175	1.57	
Cultivar	25	27693410	1107736	131.9	<0.001
Planting date/cultv	25	120513	4821	0.57	0.933
Residual	50	419923	8398		
Total	103	28267748			

l.s.d. cultivar = 130.16

cv% planting date/rep. = 3.9; cv% planting date/rep./units = 15.8

Appendix 5. Analysis of variance for percentage head infection and AUDPC calculated for stripe rust infection of spring type cultivars

Analysis of variance for percentage head infection during 1997

Source	d.f.	s.s.	m.s.	F-value	Pr>F
Replication	1	2.08	2.08	1.79	
Cultivar	14	19132.24	1366.59	1173.36	<0.001
Residual	14	16.31	1.17		
Total	29	19150.63			

l.s.d.= 2.32; cv% replication = 1.9; cv% replication/units = 5.5

Analysis of variance for AUDPC during 1997

Source	d.f.	s.s.	m.s.	F-value	Pr>F
Replication	1	5655	5655	0.52	
Cultivar	14	14489981	1034999	95.65	<0.001
Residual	14	151483	10820		
Total	29	14647119			

l.s.d.= 223.1; cv% replication = 2.5; cv% replication/units = 13.2

Analysis of variance for percentage head infection during 1999

Source	d.f.	s.s.	m.s.	F-value	Pr>F
Replication	1	1.49	1.49	0.35	
Cultivar	32	34104.93	1065.78	251.21	<0.001
Residual	32	16.31	1.17		
Total	65	34242.18			

l.s.d.= 4.2; cv% replication = 1.1; cv% replication/units = 10.4

Analysis of variance for AUDPC during 1999

Source	d.f.	s.s.	m.s.	F-value	Pr>F
Replication	1	6525	6525	1.03	
Cultivar	32	34615212	1081725	170.28	<0.001
Residual	32	203290	6353		
Total	65	34825027			

l.s.d.= 162.32; cv% replication = 2.0; cv% replication/units = 11.3

Appendix 6. Analysis of variance for AUDPC calculated for stripe rust infection at Tygerhoek during 1997

Source	d.f.	s.s.	m.s.	F-value	Pr> F
Replication	7	4.492×10^5	6.418×10^4	0.48	
Treatment	3	7.082×10^7	2.361×10^7	177.93	<0.001
Residual	21	2.786×10^6	1.327×10^5	1.07	
Fungicide	4	2.741×10^6	6.853×10^5	5.51	<0.001
Treatment/Fung	12	2.686×10^6	2.238×10^5	1.8	<0.056
Residual	112	1.393×10^7	1.244×10^5	0.99	
Cultivar	2	2.749×10^6	1.374×10^6	10.89	<0.001
Treatment/Cultivar	6	2.452×10^5	4.086×10^4	0.32	
Fungicide/Cultivar	8	8.367×10^4	1.046×10^4	0.08	
Treatment/Fung/Cultv.	24	4.140×10^5	1.725×10^4	0.14	
Residual	280	3.535×10^7	1.262×10^5		
Total	479	1.323×10^8			

l.s.d. treatment = 97.8; l.s.d. fungicide = 100.9; l.s.d. cultivar = 78.2

cv% replication = 1.2

cv% replication/treatment = 3.4;

cv% replication/treatment/fung = 7.5

cv% replication/treatment/fung/cultv = 13.0

Appendix 6 (cont.). Analysis of variance for yield obtained after the application of fungicides to control stripe rust infection at Tygerhoek during 1997

Source	d.f.	s.s.	m.s.	F-value	Pr> F
Replication	7	348471	49782	2.11	
Treatment	3	12099394	4033131	171.07	<0.001
Residual	21	495105	23576	0.7	
Fungicide	4	1269249	317312	9.38	<0.001
Treatment/Fungicide	12	809381	67448	1.99	<0.031
Residual	112	3788069	33822	0.96	
Cultivar	2	5970159	2985080	84.67	<0.001
Treatment/Cultivar	6	589150	98192	2.79	<0.012
Fungicide/Cultivar	8	301493	37687	1.07	
Treatment/Fung/Cultv.	24	488280	20345	0.58	
Residual	280	9871716	35256		
Total	479	36030466			

l.s.d. treatment = 41.2; l.s.d. fungicide = 52.6; l.s.d. cultivar = 41.3;

l.s.d. teatment/fungicide = 101.78; l.s.d. treatment/cultivar = 78.09

cv% replication = 2.5

cv% replication/treatment = 3.4

cv% replication/treatment/fungicide = 9.1

cv% replication/treatment/fungicide/cultivar = 16.1

Appendix 6 (cont.). Analysis of variance for hectolitre mass obtained after the application of fungicides to control stripe rust infection at Tygerhoek during 1997

Source	d.f.	s.s.	m.s.	F-value	Pr> F
Replication	3	1.3515	0.4505	1.14	
Treatment	3	23.2995	7.7665	19.64	<0.001
Residual	9	3.5582	0.3954	1.02	
Fungicide	4	6.1723	1.5431	3.97	<0.007
Treatment/Fungicide	12	4.0484	0.3374	0.87	
Residual	48	18.6353	0.3882	1.12	
Cultivar	2	47.3976	23.6988	68.46	<0.001
Treatment/Cultivar	6	3.8697	0.6450	1.86	
Fungicide/Cultivar	8	1.0632	0.1329	0.38	
Treatment/Fung/Cultv.	24	4.2161	0.1757	0.51	
Residual	120	41.54	0.3462		
Total	239	155.1518			

l.s.d. treatment = 0.2597; l.s.d. fungicide = 0.2557; l.s.d. cultivar = 0.1842

cv% replication = 0.1

cv% replication/treatment = 0.2

cv% replication/treatment/fungicide = 0.5

cv% replication/treatment/fungicide/cultivar = 0.8

Appendix 6 (cont.). Analysis of variance for grain protein content measured after the application of fungicides to control stripe rust infection at Tygerhoek during 1997

Source	d.f.	s.s.	m.s.	F-value	Pr> F
Replication	3	0.0405	0.0135	0.5	
Treatment	3	0.5475	0.1825	6.74	<0.011
Residual	9	0.24383	0.02709	0.41	
Fungicide	4	0.03542	0.00885	0.13	
Treatment/Fungicide	12	1.81458	0.15122	2.3	<0.021
Residual	48	3.16067	0.06585	0.7	
Cultivar	2	8.19633	4.09817	43.29	<0.001
Treatment/Cultivar	6	0.785	0.13083	1.38	
Fungicide/Cultivar	8	0.34658	0.04332	0.46	
Treatment/Fung/Cultv.	24	0.92542	0.03856	0.41	
Residual	120	11.36	0.09467		
Total	239	27.45583			

l.s.d. treatment = 0.068; l.s.d. treatment/fungicide = 0.197; l.s.d. cultivar = 0.0963

cv% replication = 0.1

cv% replication/treatment = 0.3

cv% replication/treatment/fungicide = 1.1

cv% replication/treatment/fungicide/cultivar = 2.3

Appendix 7. Analysis of variance for yield obtained after the application of fungicides to control foliar rusts at Langgewens during 1998

Source	d.f.	s.s.	m.s.	F-value	Pr> F
Replication	3	1.85657	0.61886	4.0	
Treatment	2	0.08722	0.04361	0.28	0.764
Residual	6	0.92759	0.1546	1.58	
Fungicide	8	1.21833	0.15229	1.56	0.154
Treatment/Fungicide	16	1.29111	0.08069	0.82	0.654
Residual	72	7.04833	0.09789		
Total	107	12.42917			

cv% replication = 3.6

cv% replication/treatment = 3.1

cv% replication/treatment/fungicide = 7.4

Analysis of variance for hectolitre mass obtained after the application of fungicides to control foliar rusts at Langgewens during 1998

Source	d.f.	s.s.	m.s.	F-value	Pr> F
Replication	3	4.4444	1.4815	1.84	
Treatment	2	1.1667	0.5833	0.72	0.523
Residual	6	4.8333	0.8056	0.9	
Fungicide	8	11.5	1.4375	1.61	0.137
Treatment/Fungicide	16	18.5	1.1562	1.3	0.224
Residual	72	64.2222	0.8920		
Total	107	104.6667			

cv% replication = 0.3

cv% replication/treatment = 0.4

cv% replication/treatment/fungicide = 1.3

Appendix 8. Analysis of variance for yield obtained after the application of fungicides to control foliar rusts at SGI during 1998

Source	d.f.	s.s.	m.s.	F-value	Pr> F
Replication	3	2595190	865063	4.06	
Treatment	2	166227	83113	0.39	0.693
Residual	6	1279551	213259	2.39	
Fungicide	8	583969	83424	0.93	0.486
Treatment/Fungicide	16	1412833	100917	1.13	0.35
Residual	72	5621327	89227		
Total	107	11659096			

cv% replication = 7.0

cv% replication/treatment = 6.0

cv% replication/treatment/fungicide = 11.1

Analysis of variance for hectolitre mass obtained after the application of fungicides to control foliar rusts at SGI during 1998

Source	d.f.	s.s.	m.s.	F-value	Pr> F
Replication	3	7.293	2.431	0.55	
Treatment	2	5.243	2.622	0.59	0.583
Residual	6	26.597	4.433	2.17	
Fungicide	8	15.320	2.189	1.07	0.391
Treatment/Fungicide	16	20.41	1.458	0.71	0.751
Residual	72	128.47	2.039		
Total	107	203.33			

cv% replication = 0.4

cv% replication/treatment = 1.0

cv% replication/treatment/fungicide = 1.9

Appendix 9. Analysis of variance for AUDPC calculated for different fungicide treatments applied to control leaf rust at Langgewens during 1999

Source	d.f.	s.s.	m.s.	F-value	Pr> F
Replication	3	195668	65223	1.74	
Treatment	2	29164177	14582088	389.06	0.001
Residual	6	224881	37480	1.67	
Fungicide	8	20386211	2548276	113.48	0.001
Treatment/Fungicide	16	3955515	247220	11.01	0.001
Residual	72	1616789	22455		
Total	107	55543241			

l.s.d. treatment = 111.7; l.s.d. fungicide = 122.0; l.s.d. treatment/fungicide = 219.1

cv% replication = 3.0

cv% replication/treatment = 3.9

cv% replication/treatment/fungicide = 9.1

Analysis of variance for percentage siftings obtained after the application of different fungicide treatments to control leaf rust at Langgewens during 1999

Source	d.f.	s.s.	m.s.	F-value	Pr> F
Replication	3	17.364	5.788	0.59	
Treatment	2	886.013	443.007	44.81	0.001
Residual	6	59.313	9.885	1.86	
Fungicide	8	952.796	119.1	22.38	0.001
Treatment/Fungicide	16	459.805	28.738	5.4	0.001
Residual	72	383.099	5321		
Total	107	2758.39			

l.s.d. treatment = 1.813; l.s.d. fungicide = 1.877; l.s.d. treatment/fungicide = 3.408

cv% replication = 2.9

cv% replication/treatment = 6.6

cv% replication/treatment/fungicide = 14.6

Appendix 9 (cont.). Analysis of variance for yield obtained after the application of different fungicide treatments to control leaf rust at Langgewens during 1999

Source	d.f.	s.s.	m.s.	F-value	Pr> F
Replication	3	0.48448	0.16149	1.17	
Treatment	2	17.32833	8.66417	62.93	0.001
Residual	6	0.82614	0.13769	2.29	
Fungicide	8	17.28004	2.160	35.96	0.001
Treatment/Fungicide	16	5.85551	0.36597	6.09	0.001
Residual	72	4.32454	0.06006		
Total	107	46.09904			

l.s.d. treatment = 0.214; l.s.d. fungicide = 0.1995; l.s.d. treatment/fungicide = 0.3707

cv% replication = 2.3

cv% replication/treatment = 3.6

cv% replication/treatment/fungicide = 7.2

Analysis of variance for hectolitre mass obtained after the application of different fungicide treatments to control leaf rust at Langgewens during 1999

Source	d.f.	s.s.	m.s.	F-value	Pr> F
Replication	3	3.3536	1.1179	2.75	
Treatment	2	79.5939	39.7969	97.88	0.001
Residual	6	2.4394	0.4066	0.59	
Fungicide	8	40.0867	5.0108	7.27	0.001
Treatment/Fungicide	16	20.0944	1.2559	1.82	0.044
Residual	72	49.6144	0.6891		
Total	107	195.1825			

l.s.d. treatment = 0.3677; l.s.d. fungicide = 0.6756; l.s.d. treatment/fungicide = 1.1417

cv% replication = 0.3

cv% replication/treatment = 0.3

cv% replication/treatment/fungicide = 1.1

Appendix 9 (cont.). Analysis of variance for grain protein content measured after the application of different fungicide treatments to control leaf rust at Langgewens during 1999

Source	d.f.	s.s.	m.s.	F-value	Pr> F
Replication	3	0.70272	0.23424	2.38	
Treatment	2	1.34374	0.67187	6.82	0.028
Residual	6	0.59079	0.09846	1.28	
Fungicide	8	0.322	0.04025	0.52	0.834
Treatment/Fungicide	16	1.61468	0.10092	1.31	0.212
Residual	72	5.52577	0.07675		
Total	107	10.09969			

l.s.d. treatment = 0.1810

cv% replication = 0.7

cv% replication/treatment = 0.8

cv% replication/treatment/fungicide = 2.2

Appendix 10. Analysis of variance performed for AUDPC calculated for different seed and foliar fungicide treatments applied to control stripe rust at SGI during 1999

Source	d.f.	s.s.	m.s.	F-value	Pr> F
Replication	3	25965	8655	3.12	
Treatment	7	6950396	9929148	358.03	0.001
Residual	21	58238	2773		
Total	31	7034599			

l.s.d. treatment = 77.44

cv% replication = 7.0

cv% replication/treatment = 11.1

Analysis of variance for yield obtained after the application of different seed and foliar fungicide treatments to control stripe rust at SGI during 1999

Source	d.f.	s.s.	m.s.	F-value	Pr> F
Replication	3	101635	33878	2.62	
Treatment	7	4420850	631550	48.84	0.001
Residual	21	271525	12930		
Total	31	4794011			

l.s.d. treatment = 167.2

cv% replication = 2.4

cv% replication/treatment = 4.1

Appendix 10 (cont.). Analysis of variance for hectolitre mass obtained after the application of different seed and foliar fungicide treatments to control stripe rust at SGI during 1999

Source	d.f.	s.s.	m.s.	F-value	Pr> F
Replication	3	1.1059	0.3686	1.17	
Treatment	7	31.2022	4.4575	14.1	0.001
Residual	21	6.6366	0.3160		
Total	31	38.9447			

l.s.d. treatment = 0.8267

cv% replication = 0.3

cv% replication/treatment = 0.8

Analysis of variance for grain protein content measured after the application of different seed and foliar fungicide treatments to control stripe rust at SGI during 1999

Source	d.f.	s.s.	m.s.	F-value	Pr> F
Replication	3	0.1756	0.0585	0.2	
Treatment	7	2.9432	0.4205	1.47	0.233
Residual	21	6.0227	0.2868		
Total	31	9.1416			

cv% replication = 0.5

cv% replication/treatment = 3.2

Appendix 11. Analysis of variance for AUDPC calculated for different fungicide seed treatments applied to control stripe rust at SGI during 1999

Source	d.f.	s.s.	m.s.	F-value	Pr> F
Replication	3	59007	19669	4.43	
Treatment	5	705049	141010	31.74	<0.001
Residual	15	66648	4443		
Total	23	830704			

l.s.d. treatment = 100.5

cv% replication = 8.8

cv% replication/treatment = 10.3