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PATHOGENIC VARIABILITY AND YIELD LOSSES ASSOCIATED WITH RUST DISEASES OF BARLEY AND OATS IN SOUTH AFRICA

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"I can do all things through Christ who strengthens me."

Phil. 4:13

GENERAL INTRODUCTION

Both barley (*Hordeum vulgare* L.) and oats (*Avena sativa* L.) are important cereal crops in South Africa with the potential to be grown over approximately 130 000 ha and 700 000 ha, respectively. Barley production is restricted to the south Western Cape (winter rainfall region) where the crop is grown mainly for malting purposes. Oats is grown mainly for grazing (76.4%) in the summer rainfall region with 15% of the crop being used for silage, principally in the Western Cape. Only a small proportion (8.6%) of the oat crop, grown mainly under irrigation, is produced for grain. Potentially the demand for oat grain is considerably bigger, but due to the unacceptable low hectolitre mass of the local oat harvest, grain for human consumption is largely imported.

Although the various rust diseases occurring on barley and oats are in some years major constraints to profitable production, very little research has been done on them in South Africa. It has been proposed that the low hectolitre mass of oats may be due to the detrimental effects of crown rust (*Puccinia coronata* Corda. f. sp. *avenae* Eriks.) and stem rust (*P. graminis* Pers. f. sp. *avenae* Eriks. & Henn). Furthermore, the effect of leaf rust (*Puccinia hordei* Otth.), separated from other foliar diseases, on the yield and quality of South African barley is also unknown.

Genetic resistance to crown and stem rust of oat, and leaf rust of barley, is regarded as the most feasible control measure world-wide. However, all three rusts are known for their ability to adapt and overcome existing resistance. It is also generally accepted that breeding for resistance to rusts is inefficient without knowledge of pathogenic variation, and the availability of these pathotypes for screening purposes.

The aim of this study was firstly to investigate the variation in these three rust pathogens, thus determining which resistance genes are still effective and how many pathotypes occur in which areas. Secondly, *Hordeum, Ornithogalum* and *Avena* species occur in South Africa and their possible involvement as accessory or alternate hosts, and role in the epidemiology, were also investigated.

Thirdly, this study aimed at providing representative pathotypes to enable scientifically-founded evaluation of barley and oat cultivars, as well as promising breeding lines, for their reaction to these diseases.

Finally, the influence of these rust diseases on yield and other economically important parameters of barley and oats was determined.

LITERATURE OVERVIEW OF BARLEY LEAF RUST (*PUCCINIA HORDEI* OTTH.)

INTRODUCTION AND LIFE CYCLE

Leaf rust, caused by the fungus *Puccinia hordei* Otth., is considered the most important rust disease of barley. *Puccinia hordei* is widely distributed and occurs as widespread as its primary host, *Hordeum vulgare* L. (Parlevliet, 1983). Leaf rust is considered an important disease of barley in several areas of the world including Australia, Europe, North America and South America (Alemayehu & Parlevliet, 1996; Borovkova *et al.*, 1997). Although it does not cause severe losses on a regular basis, leaf rust remains an important disease, particularly in the cool temperate regions of barley cultivation (Clifford, 1985).

Although barley leaf rust has been described as a minor disease in the United States, Griffey *et al.* (1994) concluded that races of *P. hordei* with *Rph7* virulence can cause severe damage. Most of the commercial barley cultivars grown in the United States are susceptible to *P. hordei* (Steffenson *et al.*, 1993).

Barley leaf rust is a macrocyclic, heteroecious rust. Uredia and telia occur on wild and cultivated *Hordeum* spp., and aecia on *Ornithogalum*, *Leopoldia* and *Dipcadi* spp. in the Liliaceae (Clifford, 1985). *Ornithogalum* spp. as the alternate host of *P. hordei* was first implicated by Tranzschel (according to Clifford, 1985), while d'Oliveria (according to Clifford, 1985) demonstrated that 32 species of *Ornithogalum*, together with *Dipcadi serotium* (L.) Medic., acted as hosts for *P. hordei*. *Ornithogalum* spp. have been confirmed as alternate hosts of *P. hordei* in Australia, England, France, Germany, Hungary, Israel, Portugal, Switzerland, the United States and the Soviet Union (Clifford, 1985). In Israel the *Ornithogalum* flora co-exists with wild *Hordeum* spp. and the alternate host is essential for the survival of the pathogen and for generation of pathogenic variability in the uredial stage (Anikster *et al.*, according to Clifford, 1985).

In other parts of the world, e.g. central Europe, the alternate host is unimportant since teliospore germination is not synchronised with the growth of *Ornithogalum* spp. (Clifford, 1985).

The uredial and telial stages occur widely on cultivated barley (*Hordeum vulgare* L.) and on the wild species *H. spontaneum* C. Koch and *H. bulbosum* L. in Israel (Anikster & Wahl, 1979). Ellis (according to Clifford, 1985) reported the uredial stage on *H. murinum* L. in England. Anikster *et al.* (according to Clifford, 1985) presented evidence against the classification of *P. hordei-murini* Buch. as an autonomous species on *H. murinum* and *H. bulbosum* and suggested that these forms are co-specific with *P. hordei*.

SIGNS AND SYMPTOMS

On the barley host uredial infections occur as small, orange-brown pustules mainly on the upper, but also on the lower surface of leaf blades, and on leaf sheaths. These pustules darken with age and are often associated with chlorotic haloes. With severe infections late in the season, stem, glume and awn infections may occur while general tissue chlorosis and eventual necrosis are often associated with such late infections. The blackish-brown telia are formed later during the season (Clifford, 1985).

ECONOMIC IMPORTANCE

The effect of *P. hordei* on the host depends on the duration and severity of the infection but according to the nature of biotrophy, adverse effects on photosynthesis, respiration, and transport of nutrients and water, usually result in the general debilitation of the plant (Clifford, 1985). Severe infections at early growth stages can result in a reduction in root and shoot growth, which gives rise to stunting and in turn a reduction in the number of fertile tillers and grains per ear (Udeogalanya & Clifford, 1982). In general, epidemics tend to occur later and consequently the most common effects are on grain size and quality (Udeogalanya & Clifford, 1982). Grain

characteristics of importance to the brewing industry can also be affected (Newton *et al.*, according to Clifford, 1985). Heavily infected plants tend to ripen prematurely and these general effects are exacerbated by other stress factors such as low fertility, drought and excessively high temperatures (Clifford, 1985).

Several reports on yield losses, varying from 20% (C.A. Griffey, unpublished according to Steffenson *et al.*, 1993) to 80% exists (Levine & Cherewick, 1952). In some cases total crop devastation occurred. In this regard Griffey *et al.* (1994) mentioned the total devastation of breeding nurseries in Blacksburg where leaf rust reached epidemic proportions prior to the heading stage. Calpouzos *et al.* (according to Griffey *et al.*, 1994) reported that the magnitude of yield loss is directly related to the plant stage at which rust epidemics are initiated. This was confirmed by Lim & Gaunt (1986), who found that leaf rust epidemics occurring after medium milk stage (GS75) (Zadoks *et al.*, 1974), had little effect on grain yield.

Melville *et al.* (1976) determined that each 1% increment of rust assessed on the flag leaf at GS75 (Zadoks *et al.*, 1974) resulted in yield losses of 0.77%. In a similar experiment, a yield loss of 0.6% was obtained by King & Polley (1976). However, if the disease was assessed on the penultimate leaf, a lower yield loss estimate of 0.4% was obtained. This correlates with the 0.42% (31.3 kg ha^{-1}) grain yield loss for each 1% increment of leaf rust severity on the upper two leaves at the early dough stage (GS83) of development (Griffey *et al.*, 1994). When disease on whole plants was assessed at GS75, a yield loss estimate of 0.6% for each 1% increment of rust was obtained (Udeogalanya & Clifford, 1982). However, under a low nitrogen regime, a much higher loss (1.5%) was observed. This suggests that assessments of yield loss should take into account the physiological state of plants, since the effect of rust infection appears more pronounced under stress conditions.

Griffey *et al.* (1994) reported an average yield loss of 32% in the susceptible cultivar Barsoy and an average loss of 6-16% in the other genotypes tested. They also found test weight to be reduced by as much as 105 kg ha^{-1}, stating that, whereas in the past barley leaf rust was of little economic importance, it may become a disease of greater importance regarding losses in grain yield and quality.

Yield losses as high as 40% were reported by Jenkins *et al.* (1972) and Teng & Close (1978). Dill-Macky *et al.* (according to Cotterill *et al.*, 1994) estimated crop losses of 30% for commercial crops in Australia and stated that, similar to other parts of the world, *P. hordei* has become more important. Cotterill *et al.* (1994), quoting Cotterill *et al.*, mentioned yield losses of 26-31% in Australia during the moderate to severe epidemic of 1990. According to Cotterill *et al.* (1994), Australian barley is either susceptible or at risk of becoming susceptible to pathotypes of leaf rust present in Australia, emphasising the importance of this disease.

Teng (according to Lim & Gaunt, 1986) reported yield losses of up to 45% due to *P. hordei*. Their data also suggested that green leaf area rather than disease severity is a more suitable measure for yield loss studies. They concluded that *P. hordei* must be regarded as a serious potential source of yield loss.

EPIDEMIOLOGY

Mathre (according to Clifford, 1985) stated that *Ornithogalum spp.* are unimportant in the survival and development of the pathogen in the major barley producing areas. Likewise, Reinhold & Sharp (1982) were of the opinion that *Ornithogalum umbellatum* L., and other species, are not of any importance regarding the disease cycle in the United States. In central Europe the alternate host was found unimportant because the teliospore germination is not synchronised with the growth of *Ornithogalum* spp. (Clifford, 1985). Since summer months in Mediterranean areas are dry, the fungus may be dependent on sexual reproduction on an alternate host to complete its life cycle, resulting in a higher frequency of new physiologic races (Reinhold & Sharp, 1982). The first pathotype of *P. hordei* able to overcome *Rph7* resistance was found in the vicinity of the alternate host (Golan *et al.*, 1978). This was reaffirmed by Cotterill *et al.* (1995) who derived six pathotypes from seven isolates of aeciospores taken from *O. umbellatum* in South Australia. Furthermore, *Ornithogalum* spp. were found to be essential in the survival of the fungus in Israel, and in the evolution of virulence, where it co-exists with wild *Hordeum* spp. (Anikster *et al.*, according to Clifford, 1985).

Overwintering in the uredial state, including complex combinations of virulence, occurs on autumn-sown crops and volunteer barley plants in the major barley-growing areas of Europe (Tan, 1976).

Levine & Cherewick (1952) mentioned the sensitivity of *P. hordei* to the biotic and physical environment including temperature and light sensitivity. *Puccinia hordei* needs free moisture for germination and penetration and this requirement is usually satisfied by nighttime dew (Simkin & Wheeler, 1974a). Joshi *et al.* (according to Clifford, 1985) and Simkin & Wheeler (1974a) reported that germination is optimal between 10°C and 20°C. Germination will, however, occur over a temperature range of 5°C to 25°C. Appressoria are frequently formed between 10°C and 20°C with an optimum at 15°C, but declines when temperatures exceed 25°C (B.C. Clifford, unpublished, according to Clifford, 1985).

Colonisation is limited by temperature, increasing to an optimum from 5°C to 25°C (Simkin & Wheeler, 1974b; Teng & Close, 1978). Although sporulation begins 6-8 days after infection, it may take up to 60 days at 5°C (Simkin & Wheeler, 1974a). Teng & Close (1978) found that although the sporulation (infectious) period is not significantly influenced in the temperature range 10°C-20°C, it declines as temperature and uredial density increase. Furthermore, uredial size, generation time and sporulation period are reduced with an increase in uredial density. In cloudy weather (simulated), spores can survive for 38 days, rapidly losing viability when exposed to sunlight during warm summer days (Teng & Close, 1980). Clifford (1985) used the above data to emphasise that the uredial stage can survive and develop under winter conditions prevalent in cool temperate regions, highlighting the importance of the autumn-sown crop in Europe as a "green bridge". Rapid disease development only occurs in warm, summer weather and when free moisture is available overnight. Clifford (1985) stated that day temperature is critical in the field and quoted Polley who considered that at least 9 h of surface wetness is required at 7°C.

PATHOGENIC VARIATION

As stated by Clifford (1985), variation in pathogenicity can only be measured in relation to identified resistance in the host. The main objective of such studies is the identification of isolates that are pathogenic on host resistance factors of importance to breeders and the industry. The basis of such studies is a set of barley cultivars and lines that carry the resistant factors in question, and which can be employed to differentiate among pathogenicity of isolates.

Most studies with *P. hordei* have been related to type I resistance governed by *Rph*-genes. The first differential set comprised the cultivars; Speciale, Reka 1, Sudan, Bolivia, Oderbrucker, Quinn, Egypt 4, Gold, and Lechtaler, (Levine & Cherewick, 1952). Clifford (1974) used Bolivia, Reka 1, Quinn, Sudan, Gold, Egypt 4, Estate, Batna, Peruvian, Cebada Capa, and Ricardo as differentials in his study of physiologic races in Britain. Steffenson *et al.* (1993) used the same differentials as Clifford (1974) although Reka 1, Batna and Ricardo were excluded. Quinn was replaced with Magnif and Hor2596 (*Rph9*) and Triumph (*Rph12*) was added. Cotterill *et al.* (1995) also used the same differentials as Clifford (1974), but added Magnif 104 (*Rph5*), Abyssinian (*Rph9*), Triumph (*Rph12*) and Prior (*RphP*). Jin & Steffenson (1994) used the same differentials as Steffenson *et al.* (1993), adding Clipper BC8 (*Rph10*) and Clipper BC67 (*Rph11*).

Levine & Cherewick (1952) mentioned the frequent occurrence of mutations in the laboratory, implying that variants similarly occurred under natural conditions. The strikingly large proportion of new races to the number of isolates studied, was also mentioned.

Taking into account the high number of virulence genes in the pathogen and the low frequency of *Rph* genes in commercial barley cultivars, Parlevliet (1980) stated that the many unnecessary virulence factors are difficult to explain and that the virulence patterns are probably not the result of recent developments. He also mentioned the widespread and frequent occurrence of certain virulence combinations, e.g. virulence against *Rph1*, *Rph2*, *Rph4*, *Rph5*, *Rph6* and *Rph8*, and that geographically distant isolates often were similar. Similarly, Reinhold & Sharp (1982) found that isolates

from Montana contained many genes for virulence in the absence of the resistant genes in the host population. Conversely, they found that isolates from Texas had not accumulated many genes for virulence. In Europe and elsewhere the most common pathotypes were those carrying a wide range of virulence, including virulence to *Rph1*, *Rph2*, *Rph4*, *Rph5*, *Rph6* and *Rph8*. The most effective resistance was conferred by *Rph3* and *Rph7* (Clifford, 1974).

Cromey & Viljanen-Rollinson (1995) reported that the New Zealand *P. hordei* population had virulence to all recognised *Rph* genes, except *Rph7* and the combination of *Rph3* and *Rph5*. This situation was due to the high selection pressure for certain resistance genes, as well as a stepwise increase in virulence in response to these resistance genes.

In general, North American races appear to carry few virulence genes, with race 4 carrying virulence only for *Rph8* which has dominated for 30 years (Mains, according to Clifford, 1985). This low frequency of virulence genes in the North American *P. hordei* population was confirmed by Andres *et al.* (1983) who found race 8, with virulence to only *Rph1* and *Rph4*, and a mesothetic reaction to *Rph8*, to dominate the period 1979-1982. During this period the second most common race was race 4, having virulence to only *Rph8*. Nevertheless, Steffenson *et al.* (1993) reported virulence to *Rph1*, *Rph2*, *Rph4*, *Rph6+2*, *Rph7* and *Rph8*, while according to Reinhold & Sharp (1982) virulence was detected to *Rph1*, *Rph2*, *Rph2+5*, *Rph4* and *Rph8* in Montana, but not to any of the other known resistance genes.

Virulence to *Rph3* has been detected throughout Europe (Tan, according to Reinhold & Sharp, 1982; Clifford & Udeogalanya, according to Clifford, 1985). Golan *et al.* (1978), Anikster & Wahl (1979) and Anikster *et al.* (according to Clifford, 1985) have detected virulence to what has historically been the most effective resistance factor, namely *Rph7*. Essentially none of the *Rph* genes have been used widely in the industry, the exception being *Rph7* and *Rph12*. Gene *Rph7* was widely used in Virginia (Steffenson *et al.*, 1993) and *Rph12*, which is present in the widely grown European cultivar Triumph (Trumpf), for which virulence has been detected in East Germany (Walther, according to Clifford, 1985) and the United Kingdom (B.C. Clifford, unpublished, according to Clifford 1985). No virulence has been detected

for *Rph12* in North America (B.J. Steffenson & T.G. Fetch, *unpublished data*, according to Borovkova *et al.*, 1998).

For a time *Rph7* was considered the most effective leaf rust resistance gene in barley after virulence to *Rph3* became widespread in the *P. hordei* population of Europe (Clifford, 1985). This situation changed in the late 1970's when pathotypes with virulence for the Cebada Capa resistance were identified in Israel (Golan *et al.*, 1978), later in Morocco (Parlevliet *et al.*, 1981) and also the United States in 1990 (Steffenson *et al.*, 1993). The origin of *Rph7* virulent isolates in North America is not known, but thought to be mutation. In Israel new virulence types of *P. hordei* were reported from the alternate hosts, *Ornithogalum nabonense* L., *O. montanum* Cyr. and *O. brachystachys* C. Koch (Golan *et al.*, 1978), most likely as a result of sexual recombination.

Cebada Capa resistance remained effective for 22 years in different cultivars that were widely grown in Virginia (Steffenson *et al.*, 1993). The durable resistance of the Virginian barley cultivars may have been due to more than just *Rph7*, since Parlevliet & Kuiper (according to Steffenson *et al.*, 1993) reported three to four additional genes in Cebada Capa conferring a longer latent period.

Cotterill *et al.* (1994) quoted Cotterill *et al.* regarding the identification of a pathotype in Tasmania with virulence to both *Rph9* and *Rph12*. Furthermore, Cotterill *et al.* (1995) mentioned the presence of virulence to genes *Rph1*, *Rph2*, *Rph4*, *Rph5*, *Rph6*, *Rph8*, *Rph9* and *Rph12* in Australia.

Isolate ND89-3, which is virulent to all *Rph*-genes except *Rph3*, possesses one of the widest virulence profiles known in *P. hordei* (Jin & Steffenson, 1994). This isolate is also virulent to several new sources of resistance (Jin *et al.*, in press, according to Jin & Steffenson 1994).

Several authors described physiologic specialisation to genotypes carrying type II resistance, but in most cases results could not be confirmed by other authors nor by tests. Clifford & Clothier (according to Clifford, 1985) first reported physiologic specialisation to genotypes carrying type II resistance. In glasshouse trials it was

found that field isolates from different cultivars were generally adapted to that cultivar. When circular field plots of the cultivars were inoculated with selected adapted isolates, no epidemiological advantage over nonadapted isolates could be demonstrated (Clifford, according to Clifford, 1985). Parlevliet (1977) identified a specific interaction between the moderately resistant cultivar Julia and isolate 18 of *P. hordei* that was expressed as a shortening of the latent period. From this and the observation that Julia carried a minor resistance gene not present in other cultivars, it was concluded that a specific virulence factor in isolate 18 was interacting with a specific resistance gene in Julia (Parlevliet, 1978). However, Niks (1982), in his comparative histological study of Julia infected with isolate 18, together with other genotype-isolate combinations, failed to detect any specific adaptation in terms of abortion of fungal colonies in seedling leaves.

Two Moroccan isolates of *P. hordei* have also been reported as influencing a reduced latent period on the cultivars Peruvian, Bolivia, and Vada (Parlevliet *et al.*, 1981), while dramatic interactions between pathogen isolates and German cultivars were reported (Aslam & Schwarzbach, according to Clifford, 1985). Despite the above findings, trap nurseries of type II resistant cultivars grown in the field have failed to detect adapted isolates. Furthermore, there has been no reduction in the expression of type II resistance, which has been widely deployed in cultivars grown in Britain (Clifford, according to Clifford, 1985).

DISEASE CONTROL

Fungicides

Several chemicals are available to control *P. hordei*. Melville *et al.* (1976) as well as Udeogalanya & Clifford (1982) reported that in many cases two or more fungicide applications, often being uneconomical, are needed for effective control of leaf rust. Table 1 represents fungicides registered for the control of *P. hordei* in South Africa (Nel *et al.*, 1999).

Table 1. Fungicides registered for the control of *P. hordei* in South Africa

Active ingredient	Type	Grams active ingredient	Dosage
Carbendazim/flusilazole	SC	125/250 g l^{-1}	400^a / 500^b ml ha^{-1}
Carbendazim/flutriafol	SC	150/94 g l^{-1}	1.5 l ha^{-1}
Carbendazim/tebuconazole	SC	133/167 g l^{-1}	600 ml ha^{-1}
Cyproconazole	SL	100 g l^{-1}	400^a / 500^b ml ha^{-1}
Fenbuconazole	EC	50 g l^{-1}	1.3-2.0^a / 1.6-2.5^b l ha^{-1}
Flusilazole	EC	250 g l^{-1}	400^a / 475^b ml ha^{-1}
Flusilazole	EW	250 g l^{-1}	400^a / 475^b ml ha^{-1}
Flutriafol	SC	125 g l^{-1}	1.0^a / 1.25^b l ha^{-1}
Propiconazole	EC	250 g l^{-1}	400^a / 500^b ml ha^{-1}
Propiconazole	EC	500 g l^{-1}	200^a / 250^b ml ha^{-1}
Propiconazole	Gel	625 g kg^{-1}	200^a / 240^b g ha^{-1}
Tebuconazole	EW	250 g l^{-1}	750 ml ha^{-1}
Tebuconazole	EC	250 g l^{-1}	750 ml ha^{-1}
Triadimefon	EC	250 g l^{-1}	750 ml ha^{-1}

[a] Dosage for ground application

[b] Dosage for aerial application

Host resistance

It is generally accepted that genetic resistance in the host is the best way to control this disease. Two types of resistance have been recognised. The first type (type I) is the major gene, hypersensitive type of resistance, which results in death of host cells at some stage during infestation of the tissue and is characterised by lower (resistant) infection types. The second type (type II) of resistance is the polygenic, non-hypersensitive type of resistance, characterised by fewer and smaller urediosori of a susceptible (higher) infection type (Parlevliet 1978, 1983; Clifford, 1985). In the former type several genes (*Rph1 to Rph14*) for barley leaf rust resistance have been identified, but most of these genes have been rendered ineffective by new races of the pathogen. The latter, partial resistance, provides more durable protection to leaf

rust (Parlevliet, 1983). According to Parlevliet & Van Ommeren (1975) and Parlevliet *et al.* (1985) the major component of partial resistance is a longer latent period. Parlevliet (1976a) estimated that long latent period is governed by the cumulative action of a recessive gene with large effect, and some four or five minor genes with small, additive effects.

Ethiopia is recognised as a center of diversity of cultivated barley. Most of the barley acreage of about 900 000 ha is still planted with landraces and may, according to Alemayehu & Parlevliet (1996), constitute a rich source of resistance genes. Since barley leaf rust and barley landraces have co-existed in Ethiopia for many years, resistance observed in these landraces should be of a durable nature. They argued that this durability could have arisen from the collective effect of non-durable, races-specific major genes acting in multilines, or from partial, polygenic resistance. Evidence that effective, race-specific resistance genes are virtually absent from Ethiopian barley landraces, negated the first hypothesis. A similar situation applies for many west European cultivars which have remained resistant since the 1970s and which can be considered durable (Steffenson *et al.*, 1993). Nearly all of them contain partial resistance at levels that vary from fairly low to fairly high (Parlevliet & Van Ommeren 1975; Parlevliet *et al.*, 1980), without any major resistance genes (Alemayehu & Parlevliet, 1996).

Although partial resistance may be expressed in the seedling and juvenile stages of growth (Parlevliet, 1975; Niks, 1982), the greatest expression of resistance is in the adult plant stage, particularly the young flag leaf stage (Parlevliet, 1975).

Resistance genes

To date, 14 *Rph* genes (formerly *Pa*) for resistance to *P. hordei* have been identified in barley and its wild progenitor, *H. vulgare* spp. *spontaneum* (C.Koch) Thell. (Jin *et al.*, 1993; Jin *et al.*, 1996).

According to Roane & Starling (1967) *Rph1* was first identified in Oderbrucker by Watson & Butler in 1948. Franckowiak *et al.* (1997) recommended the use of *Rph1* as a gene symbol for the gene present in Oderbrucker (CI 940). *Rph1* is situated on chromosome 2 (Tuleen & McDaniel, according to Jin *et al.* 1993).

The *Rph2* gene was first identified by Henderson (according to Roane & Starling, 1967) in Weider and other cultivars, and later by Watson & Butler (according to Roane & Starling, 1967) in "No. 22", reputedly the same as Weider. Many sources of *Rph2* have since been identified. These sources vary greatly in reaction to different *P. hordei* isolates (Roane & Starling, 1967; Reinhold & Sharp, 1982; Y. Jin & B.J. Steffenson, *unpublished data*, according to Jin *et al.*, 1996), indicating that this might be a complex locus. Therefore, Reinhold & Sharp (1982) suggested the need for further differentiation. Borovkova *et al.* (1997) also suggested *Rph2* to be a complex locus and placed the gene just distal to chromosome 7 secondary constriction. Franckowiak *et al.* (1997) recommended the use of gene symbols *Rph2.b* for the gene in Peruvian (CI935), *Rph2.j* in Batna (CI 3391), *Rph2.k* in Weider/No.22 (PI 39398), *Rph2.l* in Juliaca (PI 39151), *Rph2.m* in Kwan (PI 39367), *Rph2*.n in Chilean D (PI 48136), *Rph2.r* in Ricardo (PI 45492), *Rph2.t* in Reka 1 (CI 5051) and *Rph2.u* in Ariana (CI 14081).

Rph3, originally designated Pa_1, was detected in Estate by Henderson in 1945 (according to Roane & Starling, 1967). The gene was later renamed *Pa3* by Roane & Starling, (1967). The *Rph3* allele in Estate was placed on the long arm of chromosome 1 and more distal than that of the X_a locus (Jin *et al.*, 1993). Jin & Steffenson (1994) confirmed the resistance of Aim to be *Rph3*, as has been postulated by Brückner (according to Clifford, 1985). Jin & Steffenson (1994) found that *Rph3* was inherited as a dominant gene when tested with isolate ND8702, but was inherited recessively when inoculated with isolate ND89-3. This reversal of the inheritance pattern from dominant to recessive has not been observed previously in the barley-leaf rust pathosystem. Franckowiak *et al.* (1997) recommended the use of gene symbol *Rph3.c* for the gene in Estate (PI 57700).

Roane & Starling (1967) designated the gene in Gold and Lechtaler as *Rph4*. This gene has been placed in the chromosome 5(1H) linkage group, using the *Reg1* (*Ml-a*) powdery mildew resistance gene as a genetic marker (McDaniel & Hathcock, 1969), which was confirmed by Tan (1978) using trisomics. Franckowiak *et al.* (1997) recommended the use of gene symbol *Rph4.d* for the gene in Gold (CI 1145).

Roane & Starling (1967) designated the B locus in Quinn to be *Rph5*. Borovkova *et al.* (1997) and Jin *et al* (1996) placed this gene on chromosome 7, while Franckowiak *et al.* (1997) recommended the use of gene symbol *Rph5.e* + *Rph2.q* for the genes in Quinn (PI 39401), and *Rph5.e* for the gene in Magnif 102 (CI 13806). *Rph5* might be linked to several other *Rph* loci (Y. Jin & B.J. Steffenson, *unpublished data*, according to Jin *et al.*, 1996). The gene symbol *Rph6.f* + *Rph2.s* was recommended for the genes in Bolivia (PI 36360) (Franckowiak *et al.*, 1997).

Rph7 was found to be associated with chromosome 3 by Tuleen & McDaniel (according to Jin *et al.*, 1993) and this was confirmed by Tan (1978). Franckowiak *et al.* (1997) recommended the use of gene symbol *Rph7.g* for the gene in Cebada Capa (PI 53911). Apart from *Rph7* there are a number of minor genes in Cebada Capa which are responsible for slower colony development (Parlevliet & Kuiper, according to Clifford, 1985). Clifford & Udeogalanya (according to Clifford, 1985) showed that this gene is temperature sensitive and does not express resistance at very low (5°C) temperatures.

Franckowiak *et al.* (1997) recommended the use of gene symbol *Rph8.h* for the gene in Egypt 4 (CI 6481).

The source of the *Rph9* gene is the Ethiopian lines Hor 2596 (CI 1243), Abyssinian Schwarz, Uadera, and Ab 14 (Tan, 1977). This gene was thought to be present in the East German release Trumpf and its derived selection Triumph (Clifford, 1985). However, Jones & Clifford (according to Borovkova *et al.*, 1998) showed that Hor 2596 and Triumph exhibited different infection types in response to some *P. hordei* isolates. Jin *et al.* (1993) detected one incomplete dominant gene in Triumph against isolate ND8702 of *P. hordei*, which was confirmed by Borovkova *et al.* (1997, 1998) and Jin *et al.* (1996). Borovkova *et al.* (1998) found that *Rph9* and *Rph12* are allelic and linked to a common molecular marker ABC155, at distances of 20.6 and 24.4cM, respectively. The linkage identified with ABC155 places both *Rph9* and *Rph12* on the long arm of chromosome 7(5H). *Rph9* also showed linkage (20.1 cM) with the sequence-tagged marker ABG3 (Borovkova *et al.*, 1998). Franckowiak *et al.* (1997) recommended the use of gene symbol *Rph9.i* for the gene in Hor 2596 (CI

1243). Clifford & Udeogalanya (according to Clifford, 1985) reported the gene in CI 1243 to become less effective with an increase in temperature from 5°C to 25°C.

Rph10 is a partially dominant gene derived from an Israeli selection of *H. spontaneum* crossed with Clipper (BC-line 8) and was mapped on chromosome 3 and linked to isozyme locus *Est2* by Feuerstein *et al.* (1990). Franckowiak *et al.* (1997) recommended the use of gene symbol *Rph10.o* for the gene in Clipper BC8.

Rph11, a partially dominant gene derived from an Israeli selection of *H. spontaneum* crossed with Clipper (BC-line 67), was mapped to chromosome 6 where the gene is linked with the isozyme loci *Acp3* and *Dip2* (Feuerstein *et al.*, 1990). Franckowiak *et al.* (1997) recommended the use of gene symbol *Rph11.p* for the gene in Clipper BC67.

Jin *et al.* (1993) identified an incomplete dominant gene in Triumph and designated it *Rph12*. They found this gene to be linked with the *r* and *s* loci on chromosome 7 and indicated it to be more distal than the *r* locus on the long arm of chromosome 7. Borovkova *et al.* (1998) recently concluded that the gene of Triumph is indeed an allele at the *Rph9* locus and that the *Rph12* designation should be changed to the allele designation of *Rph9.z*, according to the proposed nomenclature of Franckowiak *et al.* (1997) for leaf rust resistance in barley.

The symbol *Rph13* was recommended for the complete dominant resistance gene present in the barley line Berac*3/HS2986 (PI 531849) since it is not allelic to any of the previously reported *Rph* loci. A linkage was detected between *Rph13* and *Rph9* with a recombination fraction of 30.4 ± 4.5% (Jin *et al.*, 1996). *Rph13* was resistant to 52% of the 90 *P. hordei* isolates tested (B.J. Steffenson and T.G. Fetch, Jr., *unpublished data,* according to Jin *et al.*, 1996). Franckowiak *et al.* (1997) recommended the use of gene symbol *Rph.x* for the gene in PI 531849.

The symbol *Rph14* was recommended for the incompletely dominant resistance gene present in barley accession PI 584760, since it is not allelic to any previously reported *Rph* loci (Jin *et al.*, 1996). *Rph14* was resistant to 96% of the 90 *P. hordei*

isolates tested (B.J. Steffenson and T.G. Fetch, Jr., *unpublished data,* according to Jin *et al.,* 1996), emphasising its value in resistance breeding.

Steffenson *et al.* (1995) and Jin & Steffenson (according to Borovkova *et al.,* 1997), identified and tentatively designated gene *RphQ* in line Q21861. Q21861 is an accession with unknown parentage and was originally selected from a barley breeding nursery at the International Maize and Wheat Improvement Center (CIMMYT) (Steffenson *et al.,* 1995). Poulsen *et al.* (1995) identified a RAPD marker ($OU02_{2700}$) linked to this gene at a distance of 12 cM. Borovkova *et al.* (1997) found this gene to be allelic or closely linked to the *Rph2* locus, while the data also indicated a linkage relationship between *RphQ* and *Rph5* with a recombination fraction of 34.5 ± 5.7%. *RphQ* can be distinguished from *Rph2* in various donors based on its infection response to several *P. hordei* isolates (Y. Jin and B.J. Steffenson, *unpublished results,* according to Borovkova *et al.,* 1997). Borovkova *et al.* (1997) mapped five RAPD markers at 8-10cM from the *RphQ* locus. *RphQ* is inherited as an incompletely dominant gene and was mapped to the centromeric region of chromosome 7, with a linkage distance of 3.5 cM from the RFLP marker CDO749. Rrn2, and RFLP clone from the ribosomal RNA intergenic spacer region, was found to be closely linked with *RphQ*, based on bulked segregant analysis. An STS marker, ITS1, derived from Rrn2 was also closely linked (1.6 cM) to *RphQ* (Borovkova *et al.,* 1997). Allelism studies showed the gene in TR306 to be the same as the one in Q21861.

Franckowiak *et al.* (1997) recommended the use of gene symbol *Rph.v* for the dominant gene in Beni Olid (PI 235186) as was identified by Jin & Steffenson (1994) in *H. vulgare.* They reported this gene to be similar to *Rph3* in its reaction to *P. hordei* but that they are distinguishable when using appropriate isolates.

Jin & Steffenson (1994) described a putative new gene in the *H. vulgare* accession PI 531849. However, according to Franckowiak *et al.* (1997), the origin of this gene was *H. spontaneum.* This gene is inherited dominantly and segregates independently from *Rph3*, while its relationship with other defined *Rph* genes are under investigation (Jin & Steffenson, 1994). Franckowiak *et al.* (1997) suggested the use of gene symbol *Rph.x* for this gene.

Jin & Steffenson (1994) described a recessive gene in addition to *Rph3* in the *H. vulgare* accession PI 531990, while Franckowiak *et al.* (1997) recommended the use of gene symbol *Rph.w* for the gene in *H. spontaneum* accession PI 466324.

An incompletely dominant gene was identified in accessions PI 531840 and PI 531841. The resistance gene in PI 531840 and PI 531841 is allelic or closely linked to *Rph2*. A linkage between *Rph5* and the gene in PI 531841 and PI 531840 and *Rph5* was found to be 33.8 ±3.8 and 17.0 ±3.5%, respectively (Jin *et al.*, 1996). Franckowiak *et al.* (1997) recommended the use of gene symbol *Rph.y* for the gene in HJ198*3/HS2310 (PI 531841). The use of gene symbol *Rph.w* was recommended for the gene in B*4/PI 466324 (PI 466324) (Franckowiak *et al.*,1997).

Yahyaoui *et al.* (1988) reported three previously unknown dominant genes in Tunisian land races Tu17, Tu27 and Tu34.

Jin & Steffenson (1994) found effective resistance in *H. vulgare* while resistance in *H. spontaneum* was fairly common, thereby confirming the data of Manisterski *et al.* (1986) and Moseman *et al.* (1990). Jin & Steffenson (1994) also stated that regarding the number of genes involved, the spectrum of resistance conferred by these genes, and the phenotypic expression, resistance in wild species was more diverse than in *H. vulgare*. Similarly, Feuerstein *et al.* (1990) confirmed that *H. spontaneum* is a rich source of resistance to *P. hordei*. They found that resistance was less frequent in *H. spontaneum* populations growing in arid regions than those growing in moist and presumably more disease prone habitats. Moseman *et al.* (1990) confirmed these findings, while Anikster *et al.* (according to Moseman *et al.*, 1990) found that many of the *H. spontaneum* accessions collected close to *Ornithogalum* spp. were resistant while fewer resistant accessions were collected from the more arid regions. In their study Moseman *et al.* (1990) found evidence to support the hypothesis that resistance genes in the host and virulence genes in the pathogen co-evolved in areas where the host and the pathogen had co-existed for millennia. Furthermore, fewer resistance genes are effective in the hosts against the virulence genes in the pathogens from the areas where they have co-evolved.

Tolerance

Tolerance was first recorded by Newton *et al.* (according to Clifford, 1985) who observed that Mensury, although heavily infected with rust, was hardly affected in terms of yield and quality compared with other cultivars. However, identification of true tolerance is possible only with precise assessment of infection and damage.

Breeding for resistance

As mentioned earlier leaf rust of barley has been controlled primarily by the use of resistant cultivars (Jin *et al.*, 1996). The continued use of single *Rph* genes in barley cultivars will probably result in ephemeral resistance, because virulence for all describe leaf rust resistance genes is known in the global population of *P. hordei* (Clifford, 1985; B.J. Steffenson & Y. Jin, *unpublished*, according to Steffenson *et al.*, 1993). Greater durability of host resistance might be achieved through the transfer of several *Rph* genes into a single pure line cultivar. However, the detection of these genes in lines might be difficult unless the appropriate "tester" cultures of *P. hordei* are available (Steffenson *et al.*, 1993). An alternative strategy is to breed for slow rusting or type II resistance as described by Clifford (1985). This type of resistance has been used in Europe since the early 1970s and remains effective (Steffenson *et al.*, 1993).

Parlevliet (1980, 1983) warned against the use of low-infection type resistance in commercial cultivars and stated that it only provided temporary protection with more serious consequences than breeders realised. In situations where there are high frequencies of low-infection type resistance in commercial cultivars, selections for partial resistance becomes increasingly difficult, if not impossible (Parlevliet, 1983). The widespread use of low-infection type resistance would prevent the selection of readily available partial resistance and this effective, durable form of resistance would ultimately be replaced by a resistance of which the effectiveness in the long run is far less certain (Parlevliet & Van Ommeren, 1975; Parlevliet, 1980).

Niks & Kuiper (1982) stated that plants combining hypersensitive resistance with a high proportion of small or aborted colonies lacking host cell necrosis, should be promising parental material, because they may carry a high level of durable resistance in their genetic background.

Partial resistance in barley to leaf rust is characterised by a reduced rate of epidemic development despite a susceptible infection type and varies greatly between cultivars (Parlevliet & Van Ommeren, 1975). Latent period, infection frequency and spore production are the important components of partial resistance. Of these, latent period appears strongly correlated with the partial resistance in the field (Parlevliet & Van Ommeren, 1975; Neervoort & Parlevliet, 1978). Partial resistance is polygenically inherited (Parlevliet, 1976a; 1978; Johnson & Wilcoxson 1978) and behaves largely in a race non-specific way, although small differential interactions occur (Parlevliet, 1976b; 1977). Parlevliet & Van Ommeren (1975), Johnson & Wilcoxson (1978) and Parlevliet et al. (1980), concluded that partial resistance is readily available, should be fairly easy to transfer, while selection for it should also be possible.

This was proven by Parlevliet & Van Ommeren (1988), when they found that mild recurrent selection against susceptibility was a powerful method of accumulating partial resistance. Best results were obtained when defined pathogen populations were used in the absence of confounding major race-specific genes. After taking inter-plot interference into account they found that sporulating leaf tissue in the S_7 generation was 300-900 fold less than that of the S_0 generation. However, little progress was made when the host population contained major race-specific genes and was exposed to racial mixtures.

Parlevliet et al. (1980) found selection for partial resistance very effective in all stages tested, namely seedling, single adult plant and small plots. They found latent period in the adult plant stage to be highly correlated with partial resistance. The most effective selection was done in small adjacent plots and this was the stage at which the breeder most often selected.

Feuerstein et al. (1990) found partial resistance can be difficult to classify correctly in segregating families. Because of the more variable genetic background, the classification of all individual seedlings was more difficult in the F_2 than was the case in the BC_3F_2.

According to Parlevliet (1975) the expression of resistance is stable to the environment, while the relative latent period has been unaffected by temperature, photoperiod, or light.

Although there are some reports of pathogen strains having adapted to type II resistance, it has nevertheless remained stable and effective in widespread agricultural use over 10 years (Clifford, 1985). On the other hand, type I resistance has a history of ephemerality. One general problem with the use of hypersensitive resistance is that when effective, it masks the degree of background resistance. Consequently its breakdown is often associated with the "vertifolia effect" (Vanderplank, according to Clifford, 1985). For these reasons it is highly desirable to combine different resistance into one genotype, thus giving a broader spectrum of resistance. Several methods to achieve this have been cited by Clifford (1985). According to Cotterill *et al.* (1994, 1995), this is the approach Australian breeders are following, incorporating *Rph7* and *Rph3* into breeding material, as well as the slow rusting or partial resistance of the European varieties.

Steffenson *et al.* (1995) emphasised the usefulness of double-haploids in the breeding of barley with multiple disease resistance. One of the many advantages is the possibility of simultaneous inoculation of plants with various races or diseases saving time. It also has the advantage that possible linkage relations among genes can be readily resolved.

REFERENCES

Alemayehu, F. & Parlevliet, J.E. 1996. Variation for resistance to *Puccinia hordei* in Ethiopian barley landraces. Euphytica 90: 365-370.

Andres, M.W., Wilcoxson, R.D., Roelfs, A.P. & Pretorius, Z.A. 1983. Races of *Puccinia hordei* in the United States from 1979 through 1982. Plant Disease 67: 1336-1337.

Anikster, Y. & Wahl, I. 1979. Coevolution of the rust fungi on Gramineace and Liliaceae and their hosts. Annual Review of Phytopathology 17: 367-403.

Borovkova, I.G., Jin, Y. & Steffenson, B.J. 1998. Chromosomal location and genetic relationship of leaf rust resistance genes *Rph9* and *Rph12* in Barley. Phytopathology 88: 76-80.

Borovkova, I.G., Jin, Y., Steffenson, B.J., Kilian, A., Blake, T.K. & Kleinhofs, A. 1997. Identification and mapping of a leaf rust resistant gene in barley line Q21861. Genome 40: 236-241.

Clifford. B.C. 1974. The choice of barley genotypes to differentiate races of *Puccinia hordei* Otth. Cereal Rusts Bulletin 2: 5-6.

Clifford, B.C. 1985. Barley leaf rust. Pages 173-205 in: The Cereal Rusts. Vol. 2. A.P. Roelfs and W.R. Bushnell, eds. Academic Press, New York.

Cotterill, P.J., Park, R.F. & Rees, R.G. 1995. Pathogenic specialization of *Puccinia hordei* Otth., in Australia, 1966-1990. Australian Journal of Agricultural Research 46: 127-134.

Cotterill, P.J., Rees, R.G. & Platz, G.J. 1994. Response of Australian barley cultivars to leaf rust (*Puccinia hordei*). Australian Journal of Experimental Agriculture 34: 783-788.

Cromey, M.G. & Viljanen-Rollinson, S.L.H. 1995. Virulence of *Puccinia hordei* on barley in New Zealand from 1990-1993. New Zealand Journal of Crop and Horticultural Science 23: 115-119.

Feuerstein, U., Brown, A.H.D. & Burdon, J.J. 1990. Linkage of rust resistant genes from wild barley (*Hordeum spontaneum*) with isozyme markers. Plant Breeding 104: 318-324.

Franckowiak, J.D., Jin, Y. & Steffenson, B.J. 1997. Recommended allele symbols for leaf rust resistance genes in barley. Barley Genetic Newsletter 27: 36-44.

Golan, T., Anikster, Y., Moseman, J.G. & Wahl, I. 1978. A new virulent strain of *Puccinia hordei*. Euphytica 27: 185-189.

Griffey, C.A., Das, M.K., Bladwin, R.E. & Waldenmaier, C.M. 1994. Yield losses in winter barley resulting from new races of *Puccinia hordei* in North America. Plant Disease 78: 256-260.

Jenkins, J.E.E., Melville, S.C. & Jemmett, J.L. 1972. The effect of fungicides on leaf disease and on yield in spring barley in south-west England. Plant Pathology 21: 49-58.

Jin, Y., Cui, G.H., Steffenson, B.J. & Franckowiak, J.D. 1996. New leaf rust resistance genes in barely and their allelic and linkage relationships with other *Rph* genes. Phytopathology 86: 887-890.

Jin, Y., Statler, G.D., Franckowiak, J.D. & Steffenson, B.J. 1993. Linkage between leaf rust resistant genes and morphological markers in barley. Phytopathology 83: 230-233.

Jin, Y. & Steffenson, B.J. 1994. Inheritance of resistance to *Puccinia hordei* in cultivated and wild barley. Journal of Heredity 85: 451-454.

Johnson, D.A. & Wilcoxson, R.D. 1978. Components of slow-rusting in barley infected with *Puccinia hordei*. Phytopathology 68: 1470-1474.

King, J.E. & Polley, R.W. 1976. Observations on the epidemiology and effect on grain yield of brown rust in spring barely. Plant Pathology 25: 63-73.

Levine, M.N. & Cherewick, W.J. 1952. Studies on dwarf leaf rust of barley. United States Department of Agriculture Technical Bulletin 1056: 1-17.

Lim, L.G. & Gaunt, R.E. 1986. The effect of powdery mildew (*Erysiphe graminis* f. sp. *hordei*) and leaf rust (*Puccinia hordei*) on spring barley in New Zealand. I. Epidemic development, green leaf area and yield. Plant Pathology 35: 44-53.

Manisterski, J., Treeful, L.M., Tomerlin, J.R., Anikster, Y., Moseman, J.G. Wahl, I. & Wilcoxson, R.D. 1986. Resistance of wild barley accessions from Israel to leaf rust collected in the USA and Israel. Crop Science 26: 727-730

McDaniel, M.E., & Hathcock, B.R. 1969. Linkage of the Pa_4 and Mla loci in barley. Crop Science 9: 822.

Melville, S.C., Griffin, G.W. & Jemmett, J.L. 1976. Effects of fungicide spraying on brown rust and yield in spring barley. Plant Pathology 25: 99-107.

Moseman, J.G., Nevo, E. & El-Morshidy, M.A. 1990. Reactions of *Hordeum spontaneum* to infection with two cultures of *Puccinia hordei* from Israel and United States. Euphytica 49:169-175.

Nel, A., Krause, M., Ramautar, N. & Van Zyl, K. 1999. A guide for the control of plant diseases. National Department of Agriculture, Republic of South Africa.

Niks, R.E. 1982. Early abortion of colonies of leaf rust, *Puccinia hordei* in partially resistant barley seedlings. Canadian Journal of Botany 60: 714-723.

Niks, R.E. & Kuiper, H.J. 1982. Histology of the relation between minor and major genes for resistance of barley to leaf rust. Phytopathology 73: 55-59.

Neervoort, W.J. & Parlevliet, J.E. 1978. Partial resistance of barley to leaf rust, *Puccinia hordei* V. Analysis of the components of partial resistance in eight barley cultivars. Euphytica 27: 33-39.

Parlevliet, J.E. 1975. Partial resistance of barley to leaf rust, *Puccinia hordei*. I. Effect of cultivar and development stage on latent period. Euphytica 24: 21-27.

Parlevliet, J.E. 1976a. Partial resistance of barley to leaf rust, *Puccinia hordei*. III. The inheritance of the host plant effect on latent period in four cultivars. Euphytica 25: 241-248.

Parlevliet, J.E. 1976b. Evaluation of the concept of horizontal resistance in barley, *Puccinia hordei* host pathogen relationship. Phytopathology 66: 494-497.

Parlevliet, J.E. 1977. Evidence of differential interaction in the polygenic *Hordeum vulgare–Puccinia hordei* relation during epidemic development. Phytopathology 67: 776-778.

Parlevliet, J.E. 1978. Further evidence of polygenic inheritance of partial resistance in barley to leaf rust, *Puccinia hordei*. Euphytica 27: 369-379.

Parlevliet, J.E. 1980. Race-specific resistance and cultivar specific virulence in the barley-leaf rust pathosystem and their consequences for the breeding of leaf rust resistant barley. Euphytica 32: 367-375.

Parlevliet, J.E. 1983. Race-specific resistance and cultivar-specific virulence in the barley-leaf rust pathosystem and their consequences for the breeding of leaf rust resistant barley. Euphytica 32: 367-375.

Parlevliet, J.E., Leijn, M. & Van Ommeren, A. 1985. Accumulating polygenes for partial resistance in barley to barley leaf rust, *Puccinia hordei*. II. Field evaluation. Euphytica 334: 15-20.

Parlevliet, J.E., Lindhout, W.H., Van Ommeren, A. & Kuiper, H.J. 1980. Level of partial resistance to leaf rust, *Puccinia hordei*, in West-European barley and how to select for it. Euphytica 29: 1-8.

Parlevliet, J.E., van der Beek, J.G. & Pieters, R. 1981. Presence in Morocco of brown rust, *Puccinia hordei*, with a wide range of virulence to barley. Cereal Rusts Bulletin 9: 3-8.

Parlevliet, J.E. & Van Ommeren, A. 1975. Partial resistance of barley to leaf rust, *Puccinia hordei*. II. Relationships between field trials, micro plot tests and latent period. Euphytica 24: 293-303.

Parlevliet, J.E. & Van Ommeren, A. 1988. Accumulation of partial resistance in barley to barley leaf rust and powdery mildew through recurrent selection against susceptibility. Euphytica 37: 261-274.

Poulsen, D.M.E., Henry, R.J., Johnston, R.P., Irwin, J.A.G. & Rees, R.G. 1995. The use of bulk segregant analysis to identify a RAPD marker linked to leaf rust resistance in barley. Theoretical and Applied Genetics 91: 270-273.

Reinhold, M. & Sharp, E.L. 1982. Virulence types of *Puccinia hordei* from North America, and the Middle East. Plant Disease 66: 1009-1011.

Roane, C.W. & Starling, T.M. 1967. Inheritance of reaction to *Puccinia hordei* in barley. II. Gene symbols for loci in differential cultivars. Phytopathology 57: 66-68.

Simkin, M.B. & Wheeler, B.E.J. 1974a. The development of *Puccinia hordei* on barley cultivar Zephyr. Annals of Applied Biology 78: 225-235.

Simkin, M.B. & Wheeler, B.E.J. 1974b. Overwintering of *Puccinia hordei* in England. Cereal Rusts Bulletin 2: 2-4.

Steffenson, B.J., Jin, Y. & Griffey, C.A. 1993. Pathotypes of *Puccinia hordei* with virulence for barley leaf rust resistance gene *Rph7* in the United States. Plant Disease 77: 867-869.

Steffenson, B.J., Jin, Y., Rossnagel, B.G., Rasmussen, J.B. & Kao, K. 1995. Genetics of multiple disease resistance in a double-haploid population of barley. Plant Breeding 114: 50-54.

Teng, P.S. & Close, R.C. 1978. Effect of temperature and uredinium density on urediniospore production, latent period, and infectious period of *Puccinia hordei* Otth. New Zealand Journal of Agricultural Research 21: 287-296.

Teng, P.S. & Close, R.C. 1980. Effects of solar radiation on survival of *Puccinia hordei* uredospores in New Zealand. Cereal Rusts Bulletin 8: 23-29.

Tan, B.H. 1976. Recovery and identification of physiologic races of *Puccinia hordei* from winter barley. Cereal Rusts Bulletin 4: 36-39.

Tan, B.H. 1977. A new gene for resistance to *Puccinia hordei* in certain Ethiopian barleys. Cereal Rusts Bulletin 5: 39-43.

Tan, B.H. 1978. Verifying the genetic relationships between three leaf rust resistance genes in barley. Euphytica 27: 317-323.

Udeogalanya, A.C.C. & Clifford, B.C. 1982. Control of barley brown rust, *Puccinia hordei* Otth., by benodanil and oxycarboxin in the field and the effect on yield. Crop Protection 1: 299-308.

Yahyaoui, A.H., Sharp, E.L. & Reinhold, M. 1988. New sources of resistance to *Puccinia hordei* in barley land race cultivars. Phytopathology 78: 905-908.

Zadoks, J.C., Chang, T.T. & Konzak, C. F. 1974. A decimal code for the growth stages of cereals. Weed Research 14: 415-421.

LITERATURE OVERVIEW OF OAT CROWN RUST (*PUCCINIA CORONATA CORDA F. SP. AVENAE* ERIKS.)

INTRODUCTION AND LIFE CYCLE

Crown (leaf) rust of oat (*Avena sativa* L.), which is caused by *Puccinia coronata* Corda f. sp. *avenae* Eriks., was described more than two centuries ago when Tozzetti (according to Simons, 1985) recognised the disease as distinct from stem rust in 1767. Crown rust is generally considered to be the most widespread and damaging disease of oat (Simons, 1985; Wise & Gobelman-Werner, 1993; Wise *et al.*, 1996) and is therefore of global importance (Brière & Kushalappa, 1995). Cultivated oat, which ranks sixth in world cereal production (Murphy & Hoffman, according to O'Donoughue *et al.*, 1995), is an important cereal crop used for both animal feed and human consumption. *Puccinia coronata* f. sp. *avenae* is highly variable in virulence and can rapidly evolve new pathotypes that overcome commonly used resistance genotypes leading to an almost innumerable array of pathogenic variants. It has repeatedly demonstrated its ability to adapt to constraints imposed by man as control measures (Simons, 1985).

Puccinia coronata f. sp. *avenae* is a typical heteroecious, long-cycle rust, with its repeating dikaryotic uredial stage occurring on oats more or less throughout their active growing period (Simons, according to Simons, 1985). As the season advances and the plants start to mature, telia are formed around the uredia, and these serve to overwinter the fungus. Meiotic reduction occurs in the teliospores, and germination of the teliospores results in haploid basidiospores. These infect young leaves of susceptible species of *Rhamnus*. In climates where the winters are mild, the fungus may live indefinitely in the uredial stage on cultivated, volunteer or wild oats (Simons, 1985).

TAXONOMY

Species level

The fungus responsible for crown rust of oat and other grasses was first described in the aecial stage as *Aecidium rhamni* by Persoon (Gmelin according to Simons, 1985). The telial stage was described by Corda (according to Simons, 1985), who listed the rush *Luzula albida* as host. Corda named the fungus *P. coronata* due to the crown-like projections on the apical end of the teliospore. Castagne (according to Simons, 1985) was the first to recognise the fungus as a grass rust in 1845. He observed the disease on *A. sativa*, *A. fatua* and *Festuca arundinacea*, and named it *Solenodonta graminis*. Towards the end of the 19th century Klebahn found, as had Nielsen earlier, that crown rust occurred in two forms, i.e. one that parasitised *Rhamnus frangula* and certain grasses, and one that parasitised *R. cathartica*, oats, and certain other grasses (Simons, 1985). He regarded the form on *R. cathartica* and oats as a different species and designated it as *P. coronifera*, while retaining the name *P. coronata* for the form on *R. frangula*.

Later others divided the crown rust complex into several species according to the reaction of various species of *Rhamnus* and genera of grasses, resulting in some controversy regarding the number of species (Simons, according to Eshed & Dinoor, 1980). The controversy has continued and the status of the specific names *P. coronifera* and *P. lolii* is still not settled. Azbukina (according to Simons, 1985) contended that *P. coronifera* should be maintained as a species separate from *P. coronata*, noting that they differed somewhat in morphology and markedly in aecial and uredial hosts, and that *P. coronata* and *P. coronifera* do not cross. Nevertheless, most researchers followed Cummins (according to Simons, 1985) and Cunningham (according to Simons, 1985) who regarded all species differentiated on the basis of pathogenicity as synonyms of *P. coronata*. They recognised however, that forms of the fungus show considerable diversity, which might indicate a need for subspecific taxa based on morphology (Simons, 1985).

Sub-species level

About sixteen *formae speciales* are recognised in *P. coronata* and these are named after the hosts from which they were isolated (Eshed & Dinoor, 1980). Erikson

(according to Simons, 1985) showed in 1894 that urediospores from one grass genus did not infect species of other grass genera. The term *formae speciales* (*f. sp.*) was introduced to describe such pathogen strains, and the term *P. coronata* f. sp. *avenae* became generally accepted for isolates of the fungus that parasitised wild and cultivated oats. Since then many forms were found not to be specific to the host of origin and some overlapping in host range occurs (Eshed & Dinoor, 1980). This led Simons (according to Eshed & Dinoor, 1980) to the conclusion that the use of "forms" is more a matter of habit or convenience than adherence to reliable taxonomy. Eshed & Dinoor (1980) stated that there is no essential difference between a race and a form and it is only a matter of whether they were classified on oat cultivars or alternatively on grass species. Simons (1985) therefore questioned the use of Latin trinomials such as *P. coronata avenae* following the suggestion of Eshed & Dinoor (1980) that the sub-specific division of *P. coronata* be abandoned.

Consistent with recent literature, however, the name *P. coronata* f. sp. *avenae* was used throughout this study.

GEOGRAPHIC DISTRIBUTION

Global

The crown rust fungus occurs nearly worldwide on oats. Its distribution even includes islands far from any landmass (Simons, 1985). The aecial stage has been reported from all major oat-producing areas of the northern hemisphere where Rhamnaceous hosts occur in proximity to oats, including the Middle East where both hosts and the fungus presumably originated (Wahl, 1970). Susceptible species of *Rhamnus* are rare or nonexistent in South America and Australia, and therefore the aecial stage does not occur there. It is also rare in some areas of relatively mild climate, such as the southern United States, even where susceptible hosts exist. This is due to the presumed requirement of low temperatures to break teliospore dormancy, although there are some unanswered questions regarding this aspect (Simons, 1985). Simons & Michel (1964) reported *P. coronata* f. sp. *avenae* from Argentina, Israel, Brazil, England, United States, Australia, Brazil, New Zealand, Canada and Sweden,

South Africa

Sawer (1909) reported that, initially, oats were grown under irrigation in vlei areas in Natal as a principal green feed for stock during winter. After early cutting or grazing the crop was allowed to regrow and produce seed. Oat was grown in this way until approximately1895 to1899 when crown rust destroyed it to such an extent that oat production in those regions was abandoned. The varieties grown up to then were known as Cape and Free State. The search for a new rust resistant variety was one of the main arguments used to support the farmers' proposal for establishing an experimental farm. In 1904, 1905 and 1906 the first screening of cultivars was done. According to Sawer (1909), two varieties coming third and fifth during the 1904-1905 evaluations dropped to the 16th and 21st positions respectively during the next season, while rest of the varieties maintained their positions. Although speculative, this might have been due to a new race. Sawer (1909) also noted that with the exception of one cultivar Giant Yellow, all the resistant varieties had a spreading growth habit while young, becoming bushy with numerous tillers as it matured. Stems were thin, tough and wiry while leaf blades were narrow. Susceptible plants were more erect with taller, thicker and succulent stems and broad leaf blades.

Doidge (1927) reported the presence of *P. coronata* f. sp. *avenae* on *Av. sativa* in Pretoria, Standerton (Feb. 1906), Cedara, Natal, Zoutpansberg District, Potchefstroom (Nov. 1910) and Salisbury, Rhodesia. Doidge *et al.* (1953) also mentioned the occurrence of *R. prinoides* and *R. zeyheri,* which are closely related to *R. frangula* and stated that no aecidial form is known on either of these. Furthermore, *P. coronata* f. sp. *avenae* on oat was reported in Stellenbosch and Moorreesburg (Verwoerd, 1929) and in the Bloemfontein district (Verwoerd, 1931).

SIGNS AND SYMPTOMS

The uredial stage of *P. coronata* f. sp. *avenae* occurs mainly on the leaf blades of the oat plant, but to some extent on the sheaths and floral structures. On susceptible cultivars, the uredia appears as bright orange-yellow, round-to-oblong pustules that may be up to 5 mm or more in length. Telia usually appear after uredia are well

established and sometimes form in rings around the uredia. Telia are black or dark-brown and remain covered for some time by the epidermis (Simons, 1985).

ECONOMIC IMPORTANCE

Puccinia coronata f. sp. *avenae* has caused serious losses in oat ever since the crop was first cultivated and evidence has existed for more than two centuries (Tozzetti, according to Simons, 1985). Ohm & Shaner (according to Bush *et al.*, 1994) as well as Gregory & Wise (1994) reported reduction in yield and quality of grain and forage as well as an increase in lodging due to this disease.

Losses in yield

In Uruguay, winter oat was frequently killed by crown rust before the crop reached heading (Gassner, according to Simons, 1985). In 1940 an epidemic of crown rust in Portugal was so severe that barely enough seed was saved for the next year's sowing (d'Oliveira, according to Simons, 1985).

In the mild rust year of 1956, crown rust destroyed over 43,000 metric tons of grain in Illinois, showing that significant losses can result from low disease incidence. Losses of 26% and 31% were reported in fungicide control trials, and in other cases losses of up to 40% have been recorded (Simons, 1985). Stevenson, according to Simons (1985), found that for each 1% increase in disease, the yield was reduced by 11.95 kg ha^{-1}.

More recently Rooney *et al.* (1994) reported extensive crop losses due to crown rust throughout the north central USA, attributing it to weather conditions being favorable for rust development and a shift in rust races rendering several cultivars susceptible. Yield losses were estimated at 10-20% in the upper Midwestern states with heavier localised losses. According to Gregory & Wise (1994), crown rust causes an average yield loss of 3.7% in the United States, but severe epidemics have reduced total yields in some years by 30% or more.

Losses in quality

In some years severe crown rust infections in the southern parts of Ontario and Quebec can reduce groat yield with 15-30% in susceptible cultivars (Fleischmann & McKenzie, 1965; Martens *et al.*, according to Chong & Seaman, 1991).

Fleischmann & McKenzie (1965) reported average groat production losses of 28% in early-seeded plots, and 50% in late-seeded plots under artificial inoculation situations during 1963 and 1964. In a naturally infected trial they recorded a 15% reduction in groat mass over both seeding dates and years. This 15% loss was considered to be conservative since the early seeding date was earlier than that of farmers and the late seeding date was unusually late for that area. Furthermore, both years were relatively mild for crown rust incidence. The importance of the stage of development of the host as related to the severity is clearly demonstrated in this study where groat mass losses of less than 10% occurred in early seeded plots while losses of 25% occurred in late seeded plots.

Loss assessment

Murphy *et al.* (1940) found that later maturing oats were more severely damaged by *P. coronata* f. sp. *avenae*. Studies of the severe epidemic of 1938 showed that the coefficient of infection (an index that combines percentage of leaf area covered by uredia with the size and other characteristics of the uredia), was more significantly correlated with damage from *P. coronata* f. sp. *avenae* than percentage leaf area covered alone (Simons, 1985).

Fleischmann & McKenzie (1965) preferred using groat yield per acre, which reflects both quantitative and qualitative losses. This was based on the fact that oat hulls are relatively low in nutritional value. Yield in ton ha^{-1} expresses only the quantitative factor, whereas kernel weight alone provides only the qualitative factors, presumably because the weight of the hulls remains constant while groat itself is reduced in size. Kernel weight is usually reliable although late-seeded oat or early crown rust epidemics (as early as flowering) might cause some kernels not to developed far enough to be threshed or they might not develop at all.

Other losses

Puccinia coronata f. sp. *avenae* can have other adverse effects, e.g. reductions in forage yield and quality, in addition to reducing grain yield and quality (Gregory & Wise, 1994). Severe damage to oats being grown for forage has been reported from South Africa (Sawer, 1909), Germany (Strabe, according to Simons, 1985), and Australia (Miles & Rosser, according to Simons, 1985). At two New Zealand locations, *P. coronata* f. sp. *avenae* reduced the dry-matter yield of forage oats during late winter and early spring from 13.6 to 11.4 t ha^{-1}and from 6.66 to 2.85 t ha^{-1} (Eagles & Taylor , according to Simons, 1985). In Texas the grazing season was shortened by as much as 4-6 weeks by crown rust infection (Reyes & Futrell, according to Simons, 1985). Straw yield can be as much as halved by severe epidemics (Simons, 1980). Winter hardiness is a concern in many areas where winter oats are grown. *Puccinia coronata* f. sp. *avenae* reduces the ability of young oat plants to harden at low temperatures, with the loss of cold resistance proportional to the severity and length of exposure to the disease (Murphy, according to Simons, 1985). In the field, greater winter injury was observed in sections where the disease had been severe, and resistant oats had less winter injury than susceptible varieties (Rosen *et al.*, according to Simons, 1985).

Ohm & Shaner (according to Bush *et al.*, 1994) reported reductions in yield and quality of grain and forage, as well as an increase in lodging which indirectly resulted in a reduction in yield.

PATHOGENIC VARIATION

The high variability of the fungus is evident from annual crown rust surveys in the eastern prairie regions of Canada where as many as 112, 107, 134, and 101 virulence phenotypes were identified from 212, 167, 202, and 189 isolates respectively, in 1991 (Chong & Seaman, 1993), 1993 (Chong & Seaman, 1994), 1994 (Chong & Seaman, 1996), and 1995 (Chong & Seaman, 1997). In 1990, Chong & Seaman (1991) identified 102 virulence combinations from 174 isolates using a differential set containing 19 single-gene backcross lines. This was the largest number of virulence combinations ever identified in Manitoba and eastern

Saskatchewan during an annual survey. The eastern and prairie regions of Canada were dominated by avirulent phenotypes and phenotypes virulent to single genes derived from *A. sterilis* before the release of cultivars with *Pc39* and the *Pc38/Pc39* combination. This imposed strong directional selection to *Pc39* in the eastern population and to *Pc38* and *Pc39* in the prairie population. A stepwise progression of virulence occurred thereafter (Chong & Kolmer, 1993). They also reported a fluctuation in virulence of between 9 and 53% for *Pc35* and *Pc56* in an area where these genes are not known to be present in the cultivars grown.

Eshed & Dinoor (1980) reported that Israeli isolates of *P. coronata* f. sp. *avenae* are virulent on certain grass species in the Agrostideae, Aveneae, Phalarideae and Festuceae families. They found differences among isolates of the same race regarding their capability to attack other grass species and reported that this phenomenon was more pronounced in Israel than the rest of the world, probably since the pathogen and different hosts had co-evolved there. Eshed & Dinoor (1980) found that isolates from different host species do not necessarily contain reciprocal virulence, i.e. if an isolate from host species A is able to attack host species B, it does not necessarily mean that isolates from B are able to attack A.

Race surveys in northern Europe have generally indicated that *P. coronata* f. sp. *avenae* in that area were highly specialised (Simons, 1985). This was exemplified by Hermansen (according to Simons, 1985), who isolated five races from six isolates.

Role of *Rhamnus*

The origin of pathogenic diversity in *P. coronata* f. sp. *avenae* has important practical implications in the development of methods to control the disease. One way through which new combinations of pathogenicity, or races, arise is by recombination of existing genes for virulence during the sexual cycle (Al-Kherb *et al.*, 1987; Brière *et al.*, 1994).

The sexual phase provides a mechanism for recombining existing genes resulting in larger diversity for virulence than does asexual reproduction (Fleischmann, according to Simons, 1985; Simons *et al.*, 1979; Groth & Roelfs, 1982; Simons,

1985; Brière *et al.,* 1994). These researchers showed that more races (including rare or new races) were obtained from *Rhamnus*, oat near *Rhamnus,* or in regions where the sexual cycle occurred. However, little difference was found between the populations regarding numbers of virulence genes. This emphasises that *Rhamnus* serves only to segregate virulence genes, and plays no role in their origin. Such segregation conceivably could expedite the development of "super races" having a wide virulence. The data however failed to support that possibility. Simons (1985) concluded that *P. coronata* f. sp. *avenae* on *Rhamnus* follows, rather than leads, the evolution of virulence in the fungus. It is important to note that sexual reproduction generates more phenotypes, but that their numbers and frequencies are altered in subsequent asexual generations owing to selection pressure exerted by the host population and other environmental factors (Al-Kherb *et al.,* 1987).

Schwartz (according to Al-Kherb *et al.,* 1987) found that 54 isolates of a 1972 sexual population from the same nursery were all different when evaluated on 60 differential hosts.

Oard & Simons (1983) found that uredial dimensions and urediospore production exhibited significantly higher mean values in the sexual population than in the asexual population. Uredial latent period and time to the formation of telia were also significantly shorter in the sexual population. In contrast, there were no significant differences between the sexual and asexual populations regarding the amount of genotypic or phenotypic variation, suggesting that sexual reproduction conferred no advantage in producing a greater range of variability for traits of aggressiveness.

Genetics of *Puccinia coronata* f. sp. *avenae*
According to Simons (1985) mutation is the only source of basic new virulence in the fungus but there are suggestions of selection forces other than host resistance.

Nof & Dinoor (1981) crossed two isolates of *P. coronata* f. sp. *avenae* and two oat cultivars, and studied segregating populations for virulence and resistance, respectively. They found that virulence in the one isolate was controlled by a recessive gene. Virulence in the other was controlled by two loci, one being recessive for virulence and the other being an inhibitory gene, which will, when

homozygous recessive, inhibit virulence determined by the first gene. They found that the oat-*P. coronata* f. sp. *avenae* system is under gene-for-gene control, as has been demonstrated for other rust fungi earlier (Flor, 1956). Biali & Dinoor (according to Simons, 1985), however, showed that pathogenicity is not always recessive and that some loci are dominant.

Since virulence is often recessive the frequency of virulence alleles can be moderately high in an asexual population, and yet be unexpressed initially in heterozygotes. However, with selection operating during repeating cycles of asexual reproduction, isolates with the necessary genes for fitness (including virulence) constitute the majority of the final population (Wahl, 1970).

Bartoš *et al.* (1967) showed that hyphal anastomosis of *P. coronata* f. sp. *avenae* may be a possible source of pathogenic variation. Martens *et al.* (according to Simons, 1985) found that most isolates of *P. coronata* f. sp. *avenae* carried apparently unnecessary genes for virulence that seemed to confer no competitive disadvantage. Such data do not support stabilising selection as a force in *P. coronata* f. sp. *avenae*.

Chong & Seaman (1991) as well as Chong & Kolmer (1993) reported that virulence to *Pc63* and *Pc38* is associated, since all isolates virulent to *Pc63* are also virulent to *Pc38*. They found the same association for *Pc39* and *Pc55*, where isolates virulent on *Pc39* were also virulent on *Pc55*, suggesting that this might be due to virulence genes that are allelic or linked in a coupling phase.

Diversity assessment
The primary objective of virulence surveys is to help in developing resistant oat cultivars. Information needed for this major objective includes (1) year-to-year prevalence of various races and (2) early detection of new and potentially dangerous races. Unbiased data on the relative prevalence of common races can best be obtained from material collected on universally susceptible cultivars. This prevents the sampling bias resulting when material is collected from cultivars that have resistance, screening out part of the fungus population. On the other hand, the appearance of new and rare forms can best be detected in collections made from

oats with the important resistance genes (Simons, according to Simons, 1985). Browder (according to Simons, 1985) suggested that it would be more meaningful to present survey data on the basis of virulence to single host cultivars or, even better, as virulence corresponding to single genes of resistance.

The first standard differential set was proposed in 1935 (Simons, according to Chong & Kolmer, 1993). During 1935–1950, 13 standard differentials were used while descriptions of 112 races were given by Simons & Murphy (according to Simons, 1985). This set had lost it usefulness by 1950 due to the prevalence of new or formerly unimportant races. In 1950 and 1951 Simons & Murphy (according to Simons, 1985) tested isolates on additional cultivars leading to a second set of ten cultivars as a standard for identification of races. This new standard set of 10 oat cultivars was subsequently established in 1952, of which six were of interest as potential sources of resistance (Simons & Murphy, according to Chong & Kolmer, 1993). This set was widely used for 20 years and a total of 294 races had been described by 1976 (Michel & Simons, according to Simons, 1985). By 1970 most of these differentials were highly susceptible to the prevalent races (Fleischmann & Baker, according to Chong & Kolmer, 1993).

Fleischmann & Baker (according to Chong & Kolmer, 1993) proposed the use of single-gene lines using the newly derived *A. sterilis* resistance. This led to the development of the standard set of 19 single-gene lines containing the *A. sterilis*-derived resistance genes *Pc35*, *Pc38*, *Pc39*, *Pc40*, *Pc45*, *Pc46*, *Pc47*, *Pc48*, *Pc50*, *Pc54*, *Pc55* and *Pc56* in a Pendek background in 1974 (Harder, according to Chong & Kolmer, 1993). Other *A. sterilis* derived single-gene lines with *Pc58*, *Pc59*, *Pc60*, *Pc61*, *Pc62*, *Pc63*, *Pc64*, and *Pc67* in various backgrounds were added to the set in 1979 and 1982 (Harder 1980; Harder & Chong, 1983). Chong & Seaman (1997) used a set of 22 lines as differentials namely *Pc35*, *Pc38*, *Pc39*, *Pc40*, *Pc45*, *Pc46*, *Pc48*, *Pc50*, *Pc51*, *Pc52*, *Pc54*, *Pc56*, *Pc58*, *Pc59*, *Pc60*, *Pc61*, *Pc62*, *Pc63*, *Pc64*, *Pc68*, *Pc94*, and *Pc96*.

According to Simons (1985) the trend in the study of pathogenic specialisation of *P. coronata* f. sp. *avenae* at host cultivar level is away from the use of standard differential sets of different cultivars. Individual investigators in different areas now

commonly survey and evaluate the virulence of *P. coronata* f. sp. *avenae* in their area in relation to available sources of resistance or resistance found in commercial cultivars grown locally. Examples are the studies by Martens *et al.* (1977) in New Zealand and Harder (1980) in Canada.

EPIDEMIOLOGY

Role of *Rhamnus*

The relative importance of *Rhamnus* in areas of the world where it functions in the epidemiology of the disease varies greatly. *Puccinia coronata* f. sp. *avenae* on oats is invariably associated with *Rhamnus* in Siberia (Wahl *et al.*, 1960). Since the fungus cannot overwinter in the uredial stage in severe climate and there is no source of wind-borne urediospores, the disease is dependent on the presence of *Rhamnus*. In northern Europe, *P. coronata* f. sp. *avenae* does not overwinter in the uredial stage, and *Rhamnus* is the primary source of inoculum (Straib, according to Simons, 1985). Simons (1985) mentioned that *R. frangula* and *R. lanceolata* are occasionally susceptible to forms that parasitise oat but they are of little or no importance to the epidemiology of *P. coronata* f. sp. *avenae*. He also stated that of the four *Rhamnus* species occurring in Israel, only two are susceptible to *P. coronata* f. sp. *avenae* while only *R. palaestina* is of epidemiological importance. *R. cathartica* is the most important species involved in the infection of oats.

Chong & Kolmer (1993) reported that populations are more diverse in areas where the alternate host occurs. The sexual cycle is epidemiologically important in eastern Canada, as infected buckthorn plants are often sources of primary inoculum for *P. coronata* f. sp. *avenae* in these regions (Fleischmann, 1964; Chong, 1988; Chong & Seaman, 1991).

Rhamnus generally does not function in the epidemiology of crown rust in areas where winters are relatively mild and where summers are long and hot. Oats is produced under such conditions in many areas of the world, including the southern United States, South America, Australia and South Africa. It is known that oats are rusted during at least part of the cool or winter season. It is not always clear how the

fungus survives the long, hot summers in the uredial stage to provide initial inoculum in the fall or winter. Generally the fungus survives on volunteer or wild oats that grow through the summer and autumn (Simons, 1985).

DISEASE CONTROL

The primary strategy for controlling crown rust is the use of cultivars resistant to or tolerant of the pathogen (Browning & Frey, 1969; Simons, 1985).

Resistance
Complete resistance to all races of the pathogen is a desired method of controlling crown rust, but this increases selection pressure on the pathogen, resulting in the development of new, virulent races. This "boom and bust" cycle of rust resistance frequently occurs with newly released cultivars (Brière & Kushalappa, 1995).

Quantitative disease resistance components have been shown to be quite valuable in selecting for partial resistance under both field and controlled environment conditions, with the exception of yield parameters, which have been proven to be unreliable (Politowski & Browning, 1978; Singleton *et al.*, 1982).

Specific resistance
The failure of oat cultivars with single genes for resistance to give lasting control to crown rust prompted combining of resistance genes in a cultivar. The majority of studies of inheritance or resistance to *P. coronata* f. sp. *avenae* have reported resistance to be dominant or partially dominant. The degree of dominance exhibited seems dependent on the genetic background involved. Dominance also varies with different races (Sebesta, according to Simons, 1985).

Allelism of genes for resistance is of importance because the resistance conferred by allelic genes cannot be combined in the same plant. The majority of studies have shown that genes for resistance in unrelated oats are generally at different loci. Exceptions, however, have been found (Upadhyaya & Baker, according to Simons, 1985).

Partial resistance

An alternative to race-specific or vertical resistance is partial or horizontal resistance. Partial resistance is characterised by a reduced rate of epidemic buildup despite a susceptible infection type, absence of large race-specific effects, and durability (Parlevliet, 1988).

The manipulation of partial resistance in breeding programmes will require large populations in spite of the high heritability values (Harder & McKenzie, 1984; Simons, 1985). Kiehn *et al.* (1976) showed that resistance in two strains of *A. sterilis* was controlled by a number of minor recessive genes having additive effects, a type of inheritance commonly associated with partial resistance.

Partial resistance to *P. coronata* f. sp. *avenae* is thought to be less subjected to the vagaries of pathogenic specialisation than is specific resistance. Simons (according to Simons, 1985) found that cultivars having useful sources of general resistance are clearly susceptible to certain races. It now appears unlikely that a source of general resistance will be discovered that will be effective against all forms of *P. coronata* f. sp. *avenae* (Simons, 1985). General resistance is of potential value in the control of *P. coronata* f. sp. *avenae*, but the complexity of its inheritance coupled with difficulties associated with its evaluation delay practical application (Simons, 1985).

Harder & McKenzie (1984) concluded that resistance in *A. sterilis* accessions CAV1358 and CAV1376 was based on several additive host genes. Since only one virulent isolate out of 1500 over a five year period was found they concluded that this resistance is valuable to breeders although its complex inheritance requires that large populations should be used in breeding programmes.

Peturson (according to Simons, 1985) found that five oat cultivars, susceptible in the seedling stage, differed in resistance in the adult stage. Brière *et al.* (1994) identified a few cultivars and lines with partial resistance as well as a screening method that can be used in a breeding programme. Among the cultivars they identified were Woodstock and Fidler having gene *Pc39* (Chong & Kolmer, 1993; Kolmer & Chong, 1993) which is a race-specific gene. However, Woodstock was withdrawn from

production due to crown rust susceptibility (Chong & Kolmer, 1993), raising the question whether it contained true partial resistance.

Pyramiding of resistance

The combination of two or more resistance genes in the same cultivar to control *P. coronata* f. sp. *avenae,* sometimes referred to as stacking or pyramiding resistance genes, is theoretically attractive. An examination of races that have appeared in the past, however, suggests that pyramiding genes for resistance in the same cultivar may not guarantee long-lasting protection (Simons *et al.,* according to Simons, 1985). In the 1950s two resistance genes carried by the cultivars Landhafer and Santa Fe, respectively, were widely used. Although no virulence was known for these genes, a race appeared in the mid 1950s that was virulent on the combination. There were, however, no races with virulence to only one gene, thus showing that a combination of genes would not have prevented the breakdown in resistance (Simons, 1985).

According to Chong & Seaman (1994) the breeding strategy for crown rust resistance is the pyramiding of effective resistance genes such as *Pc48* and *Pc68* into well adapted cultivars that have *Pc38* and *Pc39*.

Bush *et al.* (1994) identified five RFLP markers closely linked to three crown rust resistance loci present in six near-isogenic lines. They were, however, unable to link these loci to *Pc51*, *Pc52* and *Pc71*, which are genes previously associated with the germplasm they were using.

Telia development

Murphy (according to Simons, 1985) showed that early development of telia was positively correlated with host resistance. Races showing restricted virulence generally developed telia more readily. Zimmer & Schafer (according to Simons, 1985), however, found that the rate of telia formation was not correlated with virulence or with maturity of the host. They believed that telia formation was a manifestation of a specific relationship between a cultivar and an isolate of the fungus (Simons, 1985).

Tolerance

Caldwell *et al.* (according to Simons, 1985) defined tolerance as the capacity of a susceptible plant enabling it to endure severe attack without sustaining severe losses in yield or quality. A clear distinction was made between tolerance and intermediate or lesser degrees of resistance. Theoretically tolerance should be more stable than resistance. Simons (1966a) discussed the difficulties involved in the use of tolerance in breeding programmes and concluded that the reduction in seed weight attributable to *P. coronata* f. sp. *avenae* was the best measure of tolerance, with reduction in yield a useful adjunct. Simons (1972) estimated the heritability of tolerance on 76% for the three strains of *A. sterilis* he used. Singleton *et al.* (1982) found that the yields of some moderately resistant cultivars equaled or exceeded the yields of highly resistant cultivars under severe epidemic conditions, even though moderately resistant cultivars were more heavily infected. Simons (1969), through genetic studies, showed tolerance to be inherited as a complex quantitative trait in two crosses.

Singleton *et al.* (1982) reported, in accordance with Murphy *et al.* (1940), that crown rust generally reduced yield in proportion to the severity of the disease. They also found that yield ratios and kernel weight ratios did not differentiate cultivars clearly, since some of the moderately resistant cultivars were less affected by crown rust than highly resistant cultivars.

Mutation

Some host genes for resistance to *P. coronata* f. sp. *avenae* presumably originated as mutations (Simons, 1985). Rosen (1955) found a single disease-free plant in a field of oats that was otherwise heavily infected with *P. coronata* f. sp. *avenae.* Circumstances and the characteristics of the resistance of this plant were such that mutation appeared to be its likely source. Simons (1985) mentioned various authors obtained resistance by the use of artificial mutagenic agents on susceptible oat cultivars.

Resistance genes

Resistance to crown rust is typically conferred by single, dominant genes (Simons *et al.* 1978; Nof & Dinoor, 1981). However, recessive genes and incompletely dominant genes for resistance have been identified in hexaploid and diploid oat, as well as genes that inhibit resistance, complementary genes, adult-plant resistance genes, and high-temperature resistance genes (Simons *et al.*, 1978; Wise *et al.*, 1996). Resistance can also be explained by the interaction of two or more genes acting together in either a dominant or additive fashion (Simons *et al.*, 1978; Harder & McKenzie, 1984). According to Bush & Wise (1996) resistance genes to crown rust are located throughout the hexaploid genome. Ninety-five genes for crown rust resistance have been designated with permanent symbols (Simons *et al.,* 1978).

Dominant suppressors of rust resistance genes have been identified in hexaploid oat (Simons *et al.*, 1978). Dietz & Murphy (1930) identified a dominant inhibitor derived from Fulghum that inhibited a *Pc* gene present in Sunrise 23. Cochran *et al.* (1945) identified two complementary inhibitors present in Richlanf-Flunghum that interacted with two complementary crown rust resistance genes present in Anthony-Bond. Finker (according to Gregory & Wise, 1994) reported a dominant suppressor in Clinton inhibiting *Pc10*. Chang (according to Gregory & Wise, 1994) identified two dominant suppressor genes in Gopher and other lines that inhibit *Pc13* and *Pc14*, respectively. That same year, Upadhyaya & Baker (according to Gregory & Wise, 1994) reported a dominant suppressor in Garry inhibiting *Pc26*. Suppression of crown rust resistance has been reported among crosses of diploid oat species as well (Rayapati *et al.*, 1993; Wise *et al.*, 1996). Wilson & McMullen (1997) identified a dominant suppressor of *Pc62* and suggested that this suppressor may be linked to *Pc38,* or may be *Pc38* itself, and that background seemed to be unimportant in the suppressive relationship.

Harder & McKenzie (1984) described temperature sensitivity in *A. sterilis* accessions CAV1358 and CAV1376 with additive resistance, where breakdown occurred at 20°C while little resistance was expressed at 26°C. This temperature sensitivity, however, appeared to have no effect in the field. Straib (according to Simons, 1985) and Al-Kherb *et al.* (1987) mentioned that greenhouse temperature fluctuations can result in variation in pathogen phenotype.

Gregory & Wise (1994) noted complementary action for two linked genes while Simons *et al.* (1978) reported complementary action between *Pc3* and *Pc4*, *Pc4c* and *Pc3b*, *Pc7* and *Pc8* as well as between *Pc24* and *Pc25*. The following genes were summarised by Simons *et al.* (1978): *Pc1*, *I-Pc1*, *Pc2*, *Pc2b*, *Pc3*, *Pc3c*, *I-Pc3*, *Pc4*, *Pc4c*, *I-Pc4*, *Pc5*, *Pc6*, *Pc6c*, *Pc6d*, *Pc7*, *Pc8*, *Pc9*, *Pc9c*, *Pc10*, *I-Pc10*, *Pc11*. *Pc12*, *Pc13*, *Pc13c*, *Pc13d*, *I-Pc13*, *Pc14*, *I-Pc14*, *Pc15*, *Pc16*, *Pc17*, *Pc18*, *Pc19*, *Pc20*, *Pc21*, *Pc22*, *Pc23*, *Pc24*, *Pc25*, *Pc26*, *I-Pc26*, *Pc27*, *Pc28*, *Pc29*, *Pc30*, *Pc31*, *Pc32*, *Pc33*, *Pc34*, *Pc35*, *Pc36*, *Pc37*, *Pc38*, *Pc39*, *Pc40*, *Pc41*, *Pc42*, *Pc43*, *Pc44*, *Pc45*, *Pc46*, *Pc47*, *Pc48*, *Pc49*, *Pc50*, *Pc51*, *Pc52*, *Pc53*, *Pc54*, *Pc55*, *Pc56*, *Pc57*, *Pc59*, *Pc60* and *Pc61*.

Pc15 was transferred from *A. strigosa* (Sharma & Forsberg, 1977). *Pc35* is derived from *A. sterilis* (McKenzie & Fleischmann, 1964), while *Pc38* was derived from *A. sterilis* (McKenzie *et al.*, 1984) and is allelic or tightly linked with genes *Pc62* and *Pc63* (Harder *et al.*, 1980)

Chong & Seaman (1991, 1993) reported that virulence to *Pc63* and *Pc38* were associated as all isolates virulent to *Pc63* were also virulent on *Pc38*. Chong & Kolmer (1993), Chong (1988) and Chong & Seaman (1989, 1990, 1991, 1993) reported that all the phenotypes with virulence to *Pc39* were also virulent to *Pc55* and suggested that this might be due to virulence genes that are allelic or linked in a coupling phase. It can also be explained by the fact that *Pc39* is allelic or closely linked to *Pc55* (Kiehn *et al.*, 1976). *Pc38* is allelic or tightly linked to *Pc62* and *Pc63* (Harder *et al.*, 1980) and for virulence to a genotype with one of these genes, virulence to the other is a prerequisite.

Pc39 is derived from *A. sterilis* (Fox *et al.*, 1997) and is tightly linked to *Pc55* (Kiehn *et al.*, 1976). *Pc39* in the eastern Canadian cultivar Woodstock broke down within three years after being released (Chong & Kolmer, 1993).

Pc40 is derived from *A. sterilis* (Fox *et al.*, 1997), while *Pc45* is a dominant gene also derived from *A. sterilis* (Fox *et al.*, 1997). The dominance of this gene has been described to vary with host background (McDaniel, according to, Harder *et al.*, 1980).

Pc46 derived from *A. sterilis* (Fox *et al.*, 1997) is allelic with or linked to *Pc50* (Fleischmann *et al.*, 1971). Both *Pc48* and *Pc54* were derived from *A. sterilis* (Martens *et al.*,1980; Fox *et al.*, 1997). The latter gene is tightly linked or allelic with *Pc35* (Harder *et al.*, 1980). *Pc50* is derived from *A. sterilis* accession CAV2643 collected from Zana, Tunisia (Harder, 1980) and is linked or allelic to *Pc46* (Fleischmann *et al.*, 1971). Also derived from *A. sterilis* are *Pc55* and *Pc60* (Fox *et al.*, 1997).

Pc62 is a dominant gene derived from the *A. sterilis* accession CAV4274 which were collected at Fes, Morocco. *Pc62* is allelic to *Pc63* and linked or allelic to *Pc38* (Harder *et al.*, 1980). *Pc63* is a dominant gene derived from *A. sterilis* accession CA4540 which were collected from Blida, Algeria. *Pc63* is possibly allelic with *Pc62* and linked to or allelic with *Pc38* (Harder *et al.*, 1980). *Pc64*, *Pc65* and *Pc66* were derived from *A. sterilis* accession CAV 4248 originating from Tit-Mellil, Morocco (Wong *et al.*, 1983).

Pc67 is a temperature sensitive gene (J. Chong, *unpublished data*, according to Chong & Kolmer, 1993) derived from *A. sterilis* accession CAV 4656 originating from Tizi Ouzov, Algeria (Wong *et al.*, 1983). *Pc68*, a dominant gene derived from *A. sterilis* accession CAV 4904, originated from Algiers, Algeria. *Pc68* is either allelic or closely linked to *Pci* (Wong *et al.*, 1983) which in turn is closely linked to *Pc50* (Fleischmann *et al.*, 1971). *Pg3* and *Pg9,* which are linked with *Pc44*, are also near this locus (Wong *et al.*, 1983). Penner *et al.* (1993) reported that *Pc68* is still highly effective against the Canadian crown rust population. They also identified a RAPD marker that was tightly linked, in repulsion, to *Pc68*, while mentioning a protein marker identified by Howes & Chong.

The *Pc71* resistance gene was also introgressed from *A. sterilis*. Twelve RFLP markers have been identified within ca. 6 cM of *Pc71* (Bush & Wise, 1998). *Pc91* was derived from Amagalon (PI 497742) which is a colchicine-derived hexaploid from a interspecific cross between tetraploid *A. magna* and a diploid *A. longiglumis*, with *A. magna* the probable source of resistance (Rothman, 1984). Rooney *et al.* (1994) reported this gene to be dominant and identified a RFLP marker for this gene

and localised it on chromosome 18. *Pc92* was derived from Obee/Midsouth (PI 497874) which has a diverse pedigree including *A. sativa* and several *A. strigosa* accessions, with the resistance believed to be from any one (or all) of the lines composing the autotetraploid *A. strigosa* line (Rothman, 1984). Rooney *et al.* (1994) identified a RFLP marker for this gene.

Pc94 was derived from a diploid *A. strigosa* accession (T. Aung & J. Chong, *unpublished data*, according to Chong & Seaman, 1997). *Pc96* (RL1730) is linked with *Pc35* and was derived from an *A. sativa* accession obtained from the National Research Council Germplasm Institute, Bari, Italy (Chong & Brown, 1996). *PcS42* was derived from the diploid *A. strigosa* accession RL1697 (Chong & Seaman, 1994). Fox *et al.* (1997) identified a previously unknown resistance gene in *A. sterilis* accession IB3432.

Other measures of control

Besides genetic resistance there are several other measures of control including eradication of *Rhamnus,* which sometimes is unpractical and often not economically viable. Wild and volunteer oats and certain grass species susceptible to *P. coronata* f. sp. *avenae* may serve as reservoirs of inoculum and may be a primary source of resistance but are generally regarded as unimportant in the start of epidemics (Simons, 1985).

Several agronomical practices are available to help reduce the effect of *P. coronata* f. sp. *avenae* on oats. One of these measures is to avoid or escape epidemics by altering planting dates. Fleischmann & McKenzie (1965), as well as Simons (1966b), showed that losses were less in earlier seeded than later seeded oats. Early-maturing oat cultivars tend to be less damaged by crown rust than do later cultivars probably because they escape damage during sensitive growth stages (Simons & Michel, 1968). Fertilisation can also influence the disease and although other studies showed that nitrogen favours crown rust and potassium reduces the disease, it is generally accepted that any fertilisation promoting vigorous growth and high yield will enhance disease if other conditions are favourable. Although fungicide treatment is highly effective it is often not economically viable (Simons, 1985).

REFERENCES

Al-Kherb, S.M., Roelfs, A.P. & Groth, J.V. 1987. Diversity for virulence in a sexually reproducing population of *Puccinia coronata*. Canadian Journal of Botany 65: 994-998.

Bartoš, P., Fleischmann, G., Green, G.J. & Samborski, D.J. 1967. Nuclear reassociation in *Puccinia coronata* f. sp. *avenae*. Phytopathology 57: 803 (abstr.).

Brière, S.C. & Kushalappa, A.C. 1995. Evaluation of components of resistance in oat breeding lines and cultivars to crown rust (*Puccinia coronata* f. sp. *avenae*) under controlled environmental conditions. Canadian Journal of Plant Pathology 17: 319-324.

Brière, S.C., Kushalappa, A.C. & Mather, D.E. 1994. Screening for partial resistance to an isolate of crown rust (*Puccinia coronata* f. sp. *avenae*) race 264 in oat cultivars and breeding lines. Canadian Journal of Plant Pathology 16: 49-55.

Browning, J.A. & Frey, K.J. 1969. Multiline cultivars as a means of disease control. Annual Review of Phytopathology 7: 355-381.

Bush A.L. & Wise R.P. 1996. Crown rust resistance loci on linkage groups 4 and 13 in cultivated oat. Journal of Heredity 87: 427-432.

Bush A.L. & Wise R.P. 1998. High-resolution mapping adjacent to the *Pc71* crown-rust resistance locus in hexaploid oat. Molecular Breeding 4: 13-21.

Bush, A.L., Wise, R.P., Rayapati, P.J. & Lee, M. 1994. Restriction fragment length polymorphisms linked to genes for resistance to crown rust (*Puccinia coronata*) in near-isogenic lines of hexaploid oat (*Avena sativa*). Genome 37: 823-831.

Chong, J. 1988. Virulence and distribution of *Puccinia coronata* in Canada in 1987. Canadian Journal of Plant Pathology 10: 348-353.

Chong J. & Brown P.D. 1996. Genetic resistance to *Puccinia coronata* f. sp. *avenae* in two *Avena sativa* accessions. Canadian Journal of Plant Pathology 18: 286-292.

Chong, J. & Kolmer, J.A. 1993. Virulence dynamics and phenotypic diversity of *Puccinia coronata* f. sp. *avenae* in Canada from 1974 to 1990. Canadian Journal of Botany 71: 248-255.

Chong, J. & Seaman, W.L. 1989. Virulence and distribution of *Puccinia coronata* in Canada in 1988. Canadian Journal of Plant Pathology 11: 439-442.

Chong, J. & Seaman, W.L. 1990. Distribution and virulence of *Puccinia coronata* in Canada in 1989. Canadian Journal of Plant Pathology 12: 431-435.

Chong, J. & Seaman, W.L. 1991. Distribution and virulence of *Puccinia coronata* in Canada in 1990. Canadian Journal of Plant Pathology 13: 365-370.

Chong, J. & Seaman, W.L. 1993. Distribution and virulence of *Puccinia coronata* f. sp. *avenae* in Canada in 1990. Canadian Journal of Plant Pathology 15: 41-45.

Chong, J. & Seaman, W.L. 1994. Incidence and virulence of *Puccinia coronata* f. sp. *avenae* in Canada in 1993. Canadian Journal of Plant Pathology 16: 335-340.

Chong, J. & Seaman, W.L. 1996. Incidence and virulence of *Puccinia coronata* f. sp. *avenae* in Canada in 1994. Canadian Journal of Plant Pathology 18: 293-299.

Chong, J. & Seaman, W.L. 1997. Incidence and virulence of *Puccinia coronata* f. sp. *avenae* in Canada in 1995. Canadian Journal of Plant Pathology 19: 176-180.

Cochran, G.W., Johnson, C.O., Heyne, E.G. & Hansing, E.D. 1945. Inheritance of reaction to smut, stem rust, and crown rust in four oat crosses. Journal of Agricultural Research 70: 43-61.

Dietz, S.M. & Murphy, H.C. 1930. Inheritance of resistance to *Puccinia coronata avenae*, p.f. III. Phytopathology 20: 120.

Doidge, E.M. 1927. Preliminary study of South African rust fungi. Bothalia 2: 1-228.

Doidge, E.M., Bottomley, A.M., Van der Plank, J.E. & Pauer, G.D. 1953. A revised list of plant diseases in South Africa. Union of South Africa Department of Agriculture, Science Bulletin 346: 62.

Eshed, N. & Dinoor, A. 1980. Genetics of pathogenicity in *Puccinia coronata*: Pathogenic specialization at the host genus level. Phytopathology 70: 1042-1046.

Fleischmann, G. 1964. Physiologic races of oat crown rust isolated from aecia on buckthorn and their relation to the racial population on oat in southeastern Ontario and Manitoba. Canadian Journal of Botany 42: 1151-1157.

Fleischmann, G. & McKenzie, R.I.H. 1965. Yield losses in Garry oats infected with crown rust. Phytopathology 55: 767-770.

Fleischmann, G., McKenzie, R.I.H. & Shipton, W.A. 1971. Inheritance of crown rust resistance in *Avena sterilis* L. from Israel. Crop Science 11: 451-453.

Flor, H.H. 1956. The complementary genic systems in flax and flax rust. Advances in Genetics 8: 29-54.

Fox, S.L., Brown, P.D. & Chong, J. 1997. Inheritance of crown rust resistance in four accessions of *Avena sterilis* L. Crop Science 37: 342-345.

Gregory, J.W. & Wise, R.P. 1994. Linkage of genes conferring specific resistance to oat crown rust in diploid *Avena*. Genome 37: 92-96.

Groth, J.V. & Roelfs, A.P. 1982. Effects of sexual and asexual reproduction on race abundance in cereal fungus populations. Phytopathology 72: 1503-1507.

Harder, D.E. 1980. Virulence and distribution of *Puccinia coronata avenae* in Canada in 1979. Canadian Journal of Plant Pathology 2: 249-252.

Harder, D.E. & Chong, J. 1983. Virulence and distribution of *Puccinia coronata* in Canada in 1982. Canadian Journal of Plant Pathology 5: 185-188.

Harder, D.E. & McKenzie, R.I.H. 1984. Complex additive-type resistance to *Puccinia coronata* in *Avena sterilis*. Canadian Journal of Plant Pathology 6: 135-138.

Harder, D.E., McKenzie, R.I.H. & Martens, J.W. 1980. Inheritance of crown rust resistance in three accessions of *Avena sterilis*. Canadian Journal of Genetics and Cytology 22: 27-33.

Kiehn, F.A., McKenzie, R.I.H. & Harder, D.E. 1976. Inheritance of resistance to *Puccinia coronata avenae* and its association with seed characteristic in four accessions of *Avena sterilis*. Canadian Journal of Genetics and Cytology 12: 230-236

Kolmer, J.A. & Chong, J. 1993. Distribution of virulence in two populations of *Puccinia coronata* f. sp. *avenae* in Canada. Canadian Journal of Botany 71: 946-950.

Martens, J.W., Burnett, P.A. & Wright, G.M. 1977. Virulence in *Puccinia coronata* f. sp. *avenae* and *P. graminis* f. sp. *avenae* in New Zealand. Phytopathology 67: 1519-1521.

Martens, J.W., McKenzie, R.I.H. & Harder, D.E. 1980. Resistance to *Puccinia graminis avenae* and *P. coronata avenae* in the wild and cultivated *Avena* populations of Iran, Iraq, and Turkey. Canadian Journal of Genetics and Cytology 22: 641-649.

McKenzie, R.I.H., Brown, P.D., Martens, J.W., Harder, J., Nielsen, J., Gill, C.C & Boughton, G.R. 1984. Registration of Dumont oats. Crop Science 24: 207.

McKenzie, R.I.H. & Fleischmann, G. 1964. The inheritance of crown rust resistance in selections from two Israeli collections of *Avena sterilis*. Canadian Journal of Genetics and Cytology 6: 232-236.

Murphy, H.C., Burnett, L.C., Kingsolver, C.H., Stanton, T.R., & Coffman, F.A. 1940. Relation of crown-rust infection to yield, test weight, and lodging of oats. Phytopathology 30: 808-819.

Nof, E. & Dinoor, A. 1981. The manifestation of gene-for-gene relationships in oat and crown rust. Phytoparasitica 9: 240

O'Donoughue, L.S., Kianian, S.F., Rayapati, P.J., Penner, G.A., Sorrels, M.E., Tanksley, S.D., Phillips, R.L., Rines, H.W., Lee, M., Fedak, G., Molnar, S.J., Hoffman, D., Salas, C.A., Wu, B., Autrique, E., & Van Deynze, A. 1995. A molecular linkage map of cultivated oat. Genome 38: 368-380.

Oard, J.H. & Simons, M.D. 1983. A growth chamber comparison of traits of aggressiveness in sexual and asexual populations of *Puccinia coronata*. Phytopathology 73: 1226-1229.

Parlevliet, J.E. 1988. Strategies for the utilization of partial resistance for the control of cereal rusts. Breeding strategies for resistance to the rusts of wheat. CIMMYT. Mexico.

Penner, G.A., Chong, J., Wight, C.P., Molnar, S.J. & Fedak, G. 1993. Identification of an RAPD marker, for the crown rust resistance gene *Pc68* in oats. Genome 36: 818-820.

Politowski, K. & Browning, J.A. 1978. Tolerance and resistance to plant disease: An epidemiological study. Phytopathology 68: 1177-1185.

Rayapati, P.J., Gregory, M., Lee, M. & Wise, R.P. 1993. A linkage map of diploid *Avena* based on RFLP loci and a locus conferring resistance to isolates of *Puccinia coronata* var *avenae*. Theoretical and Applied Genetics 89: 831-837.

Rooney, W.L., Rines, H.W. & Phillips, R.L. 1994. Identification of RFLP markers linked to crown rust resistance genes *Pc91* and *Pc92* in oat. Crop Science 34: 940-944.

Rosen, H.R. 1955. New germ plasm for combined resistance to *Helminthosporium* blight and crown rust of oats. Phytopathology 45: 219-221.

Rothman, P.G. 1984. Registration of four stem rust and crown rust resistant oat germplasm lines. Crop Science 24: 1217-1218.

Sawer, E.R. 1909. The cereals in South Africa. Cedara Memoirs of South African Agriculture. 1: 264-272.

Sharma, D.C. & Forsberg, R.A. 1977. Spontaneous and induced interspecific gene transfer for crown rust resistance in *Avena*. Crop Science 17: 855-860.

Simons, M.D. 1966a. Relative tolerance of oat varieties to the crown rust fungus. Phytopathology 56: 36-40.

Simons, M.D. 1966b. Relationship of date of planting of oats to the crown rust damage. Phytopathology 56: 41-45.

Simons, M.D. 1969. Heritability of crown rust tolerance in oats. Phytopathology 59: 1329-1333.

Simons, M.D. 1972. Crown rust resistance of *Avena sativa*-type oats derived from wild *Avena sterilis*. Phytopathology 662: 1444-1446.

Simons, M.D. 1980. Effect of *Puccinia coronata* on straw yield and harvest index of oats. Phytopathology 70: 604-607.

Simons, M.D. 1985. Crown rust. Pages 131-172 in: The Cereal Rusts. Vol. 2. A.P. Roelfs and W.R. Bushnell, eds. Academic Press, New York.

Simons, M.D., Martens, J.W., McKenzie, R.I.H., Nishiyama, I., Sadanaga, K, Sebesta, J. & Thomas, H. 1978. Oats: A standardized system of nomenclature for genes and chromosomes a catalogue of genes governing characters. United States Department of Agriculture Handbook. 509.

Simons, M.D. & Michel, L.J. 1964. International register of pathogenic races of *Puccinia coronata* var. *avenae*. Plant Disease Reporter 48: 763-766.

Simons, M.D. & Michel, L.J. 1968. Oat maturity and crown rust response. Crop Science 8: 254-256.

Simons, M.D., Rothman, P.G. & Michel. L.J. 1979. Pathogenicity of *Puccinia coronata* from buckthorn and from oats adjacent to and distant from buckthorn. Phytopathology 69: 156-158.

Singleton, L.L., Moore, M.B., Wilcoxson, R.D. & Kernkamp, M.F. 1982. Evaluation of crown rust disease parameters and yield in moderately resistant cultivars. Phytopathology 72: 538-540.

Verwoerd, L. 1929. A preliminary checklist of diseases of cultivated plants in the winter rainfall area of the Cape Province. Union of South Africa Department of Agriculture Science Bulletin 88: 1-28.

Verwoerd, L. 1931. A preliminary list of fungi from the Bloemfontein district of the Orange Free State. South African Journal of Science, Vol. XVIII: 298-301.

Wahl, I. 1970. Prevalence and geographic distribution of resistance to crown rust in *Avena sterilis*. Phytopathology, 60: 746-749.

Wahl, I., Dinoor, A., Halperin, J. & Schreiter, S. 1960. The effect of *Rhamnus palaestina* on the origin and persistence of oat crown rust races. Phytopathology 50: 562-567.

Wilson W.A. & McMullen M.S. 1997. Dosage dependent genetic suppression of oat crown rust resistance gene *Pc62*. Crop Science 37: 1699-1705.

Wise, R.P. & Gobelman-Werner, K.S. 1993. Resistance to oat crown rust in diploid and hexaploid *Avena*. Plant Disease 77: 335-358.

Wise, R.P., Lee, M. & Rayapati, P.J. 1996. Recombination within a 5-centimorgan region in diploid *Avena* reveals multiple specificities conferring resistance to *Puccinia coronata*. Phytopathology 86: 340-346.

Wong, L.S.L., McKenzie, R. I. H., Harder, D.E. & Martens, J.W. 1983. The inheritance of resistance to *Puccinia coronata* and of floret characters in *Avena sterilis*. Canadian Journal of Genetics and Cytology 25: 329-335.

LITERATURE OVERVIEW OF OAT STEM RUST (*PUCCINIA GRAMINIS* PERS. F. SP. *AVENAE* ERIKS AND E. HENN.)

INTRODUCTION

Stem rust of oat (*Avena* spp. L.) is caused by the fungus *Puccinia graminis* Pers. f. sp. *avenae* Eriks. and E. Henn. The disease has been known to man for more than 2000 years, but according to Martens (1985) it was first described by Tozzetti & Fontana in 1767. Persoon was the first to name the causative fungus *Puccinia graminis* in 1797 (Arthur, according to Martens, 1985). Until 1894 it was regarded as a single species capable of attacking all the common cereals and grasses, when Eriksson recognised the pathogen as a distinct biological taxon and named it *Puccinia graminis* f. *avenae* (Eriksson according to Martens, 1985).

Puccinia graminis f. sp. *avenae* is an obligate parasite of which the repeating uredinial cycle occurs on cultivated (*Avena sativa* L.) and wild oats (*A. fatua, A. sterilis, A. barbata, A. byzantina* etc.). The sexual phase occurs on the alternate host *Berberis* L. The disease occurs in most of the oat growing areas of the world, periodically causing severe crop losses (Martens, 1985). During 1953, yield losses of 25% were reported by Roelfs & Long (1980) in Minnesota, whereas Martens (1985) reported losses of approximately 35% in Manitoba and Eastern Saskatchewan during a severe epidemic in 1977. More recently, McVey *et al.* (1996) reported a severe oat rust epidemic in experimental plots in Louisiana, USA.

PATHOGENIC VARIATION

Although there are relatively few known genes that condition resistance to *P. graminis* f. sp. *avenae*, which in turn could imply only a few avirulence genes, pathogenic variation occurs frequently. This was exemplified by Martens (1981), who identified 21 races.

Specialisation within *P. graminis* f. sp. *avenae* was first reported in 1923 by Stakman and his co-workers, when they described four variants from more than 100 collections from five countries, using two differentials that carried genes *Pg1* and *Pg2*, and the susceptible cultivar Victory (Martens, 1985).

In 1950 differential oat lines with the genes *Pg3* and *Pg4* were added, followed by *Pg8* in 1961, *Pg9* in 1964, *Pg13* in 1969 and *Pg15*, *Pg16* and *Pga* in 1978. These genes still comprise the current set used to differentiate pathogenic variability in *P. graminis* f. sp. *avenae* (Harder, 1994a).

According to Martens (1985), early (1925-1930) studies on specialisation within the oat stem rust pathogen indicated that avirulence-virulence combinations occurred widely in different parts of the world. As new genes for resistance were discovered, virulence on them in various combinations, was usually also found. In most cases, virulence (e.g. to *Pg4*, *Pg9*, *Pg13* and *Pga*) was present in the pathogen population before cultivars carrying these genes were grown over large areas (Martens, 1985).

North America

Virulence to all of the *Pg* resistance genes has occurred at some time in the North American *P. graminis* populations, regardless of the exposure of the population to these resistance genes (Harder, 1994a). Reasons for the lack of stem rust epidemics in the United States could be the small number of overwintering uredinia, the late onset of the disease (Roelfs & Long, 1980; Roelfs *et al.*, 1993), the small area of oats grown, or unfavourable environmental conditions for development of regional epidemics (McVey *et al.*, 1997). Compared to other cereal rust fungi in North America, common pathotypes of *P. graminis* have tended to dominate populations for 25 years, while some races have been isolated for as long as 40 years (Harder, 1994a).

Between 1965 and 1981 virulence on *Pg13*, *Pg16* and *Pga* occurred infrequently or was non-existent in North America. In the eastern United States virulence occurred on all the resistance genes except *Pg8*, *Pg13*, *Pg16* and *Pga*, whereas in the Great-plains virulence was detected on all genes except *Pg9*, *Pg13*, *Pg16* and *Pga*. The most common race during this period was 2,4,9,13/1,3,8. As most cultivars are

resistant to this race, it is obvious that other factors also influence population composition (Martens, 1985). In the southern United States and Mexico only races with virulence to *Pg15* and/or *Pg3* occur (Roelfs *et al.*, 1980).

In Canada, all oat cultivars recommended for Manitoba and eastern Saskatchewan carry combinations of either *Pg2* and *Pg13*, or genes *Pg2*, *Pg9* and *Pg13* (Chong *et al.*, according to Harder, 1997). In these areas genes *Pg9* and *Pg13* have provided resistance to *P. graminis* since their initial release in cultivars in the early 1980s (Harder, 1997). Race NA26 is virulent to gene *Pg13*, and isolates with virulence to this gene have been common in Ontario in recent years (Harder, 1997).

Virulence in regional *P. graminis* populations in Canada appeared to be stable until 1940. A period of diversity followed, where after populations seemed to stabilise from the mid-1960s onwards. The widespread use of genes *Pg1*, *Pg2*, and *Pg4* as resistance sources in North American oat cultivars has been implicated as the main cause for race shifts in *P. graminis* occurring during this period (Harder, 1994a).

Eurasia

In general, the *P. graminis* population in Eurasia has been characterised by considerable variability in the population with many races having been identified. Typical is also the high frequency of virulence genes in the pathogen population that is apparently not related to the resistance genes in the host population (Martens, 1985). Šebesta *et al.* (1998) stated that the effectiveness of *Pg* resistance has changed dramatically in Europe during the period 1988 to 1996. They found the cultivar Saia (with resistance from *A. strigosa*) to be the most resistant followed by *Pg13* and *Pg16*. The *A. sterilis* accessions VIR 343-1 and VIR 343-2 also proved to be valuable sources of resistance although virulence occurred to all known resistance genes.

The Middle East and East Africa

Literature describing the variability in the oat stem rust pathogen in the Middle East and East Africa is limited to that of Martens (1985), indicating that the population is characterised by considerable variation with apparent unnecessary virulence.

Australia

Stem rust is a major constraint in oat production thoughout Australia (Adhikari *et al.*, 1999). The resistance genes *Pg8*, *Pg13* and *Pga* conferred resistance to stem rust in Australia until the late 1980s when they where rendered ineffective by new pathotypes. (Oates according to Adhikari *et al.*, 1999). The lack of genetic variability for stem rust resistance among important sources of germplasm used by Australian breeders is currently a major cause for concern (Adhikari *et al.*, 1999).

South America

Martens (1985) reported that although only limited data are available, the South American rust population appears genetically variable with many genes for virulence.

In Colombia stem rust occurs wherever oat is grown and is a limiting factor to its production. Orjuela *et al.* (according to Martens, 1985) reported 22 different races including some races not found elsewhere, and some virulent to all of the resistance genes used. *Berberis* spp. are known to occur in Colombia but their role in the life cycle of the rust fungus is not clear. Coelho (according to Martens, 1985) reported 10 races from Brazil and noted that none of them were virulent on *Pg8* resistance.

South Africa

Berkeley (1876) mentioned the collection of *P. graminis* in 1874 from the foot of Table Mountain, but did not state from which host. According to Doidge (1927), *P. graminis* was found on oats in Standerton (Nov. 1905 and Feb. 1906), Pretoria district (Jan. 1907), and Potchefstroom. Verwoerd (1929) mentioned the occurrence of *P. graminis* on oats in the districts of Moorreesburg, Malmesbury, Hopefield, Stellenbosch, Philadelphia, Knysna, George and Swellendam. In 1925, Bailey identified race 2,3/1 in South Africa (Martens, 1985), while Verwoerd (1931) mentions the occurrence of two pathotypes in South Africa. "Form 2" virulent to White Tartar (C.I. 551) and Joanette (C.I. 2660) (*Pg3*), with an intermediate reaction to Richland (C.I. 787), indicating that it could be the same pathotype as was described by Bailey. The second pathotype, "form 3", was virulent to White Tartar, avirulent to Joanette and produced the same intermediate reaction on Richland.

Virulence and competitive ability

It is not known why *P. graminis* f. sp. *avenae* carries so many genes for virulence or how they arise. Martens (1985) suggested that in some cases it is possible for resistance genes to be more widely distributed in the host population than is recognised e.g. *Pg3* and *Pg9*. He stated, however, that it is most unlikely that the genes from *A. sterilis* L. *Pg13* and *Pg15* ever occurred in North America before they where intentionally introduced. The corresponding virulence genes were already present before the pathogen had the opportunity to 'recognise' these genes for resistance. Perhaps these genes impart a selective advantage other than virulence on *Avena* spp. The pathogen possibly parasites hosts other than *Avena* spp., where these 'unnecessary' genes are in fact useful, if not necessary (Martens, 1985).

The theory of genetic polymorphism in parasitic systems (Person, 1966) indicates that unnecessary alleles will eventually decline when the corresponding host genes are no longer available. As discussed by Martens *et al.* (1970), this should have resulted in the predominance of races virulent to genes *Pg2* and *Pg4*, but not to others, in the 1960s. However, this did not occur. The *P. graminis* population has shown much greater buffering capacity in the retention of virulence or avirulence. Some selection for virulence has occurred as a result of resistance gene deployment, but the increase or decrease in frequencies of other unrelated virulence alleles, at least in the asexual populations, appeared to be influenced more by the virulent clusters in which they occurred and how these are selected by resistance genotypes and possibly other factors related to survival (Harder, 1994a).

Stability in the population can be illustrated by the persistence of avirulence to genes *Pg1* and *Pg4*, despite selection against this avirulence, and virulence to *Pg3* despite no selective advantage from at least 1921 to the present. Similarly, virulence to genes *Pg9* and *Pg15* has continued at high frequencies in the eastern Canadian population for over 35 years despite the apparent absence of these genes in this region (Harder, 1994a).

ENVIRONMENTAL FACTORS AFFECTING THE HOST-PARASITE INTERACTION

There are several factors other than the presence or absence of resistance genes in the host, or virulence or avirulence in the pathogen, that influence the host-parasite interaction. Harder (1994a) pointed out that *P. graminis* carries virulence factors unrelated to selection and unnecessary for survival under natural conditions and that other more complex environmental or physiological (Groth, 1984) factors probably have more significant effects on host-parasite interaction.

The majority of stem rust resistance genes are temperature sensitive and several authors have demonstrated the thermostability or sensitivity of different host genes. Gordon (according to Martens, 1985) demonstrated the thermostability of *Pg1* and *Pg2* and the sensitivity of *Pg3*, while Roberts (according to Martens, 1985) and Martens *et al.* (1976) demonstrated the thermosensitivity of *Pg4*. According to Martens *et al.* (1967, 1979) *Pg1* and *Pg2* are temperature-insensitive, whereas the genes *Pg9*, *Pg8*, *Pg13*, *Pg15*, *Pg16*, *Pg4* and *Pg3* are temperature-sensitive, in order of increasing sensitivity.

In some cases complicated temperature regimes influence the expression of resistance or virulence. Roberts (according to Martens, 1985) demonstrated the thermosensitivity of *Pg4* and found that the expression of gene *Pg4* was dependent on a certain critical temperature during the latent period. Plants kept at 22°C after inoculation, did not express resistance if grown at 30°C for at least 3 days prior to the flecking stage. Plants kept at 30°C after inoculation, required at least 4 days at 22°C before flecking, to completely express the characteristic resistance phenotype. Pre-inoculation temperatures had no effect. Factors such as race effects further complicated matters, while Martens *et al.* (1976) showed that temperature sensitivity is independent of host genotype.

Gordon (according to Martens, 1985) noted that telia were formed more rapidly at 24-28°C than at 12-16°C, while Martens *et al.* (1976) found the optimum temperature for rust development to be 20-25°C. Germination of urediniospores occurred at 10-30°C but optimum conditions for germtube growth and formation of appressoria, was

at 20°C in the dark (Kochman & Brown 1976a). Maximal penetration was achieved with a dew period of 16h (Kochman & Brown, 1976b). According to Martens & Dyck (1988) light sensitivity has also been documented as an environmental factor influencing the host-parasite interaction.

DISEASE CONTROL USING *Pg* GENES

Origin and nature of *Pg* genes

Although resistance genes effective to *P. graminis* have been difficult to find, Martens & Dyck (1988) were of the opinion that those available are adequate in controlling oat stem rust. All the historically important *Pg* genes that influenced pathogen dynamics, or which are presently important in terms of breeding for resistance, are included in the international differential set. The genes *Pg1*, *Pg2*, *Pg3*, *Pg4*, *Pg8*, *Pg9*, *Pg13*, *Pg15*, *Pg16*, and *Pga*, all of which are available as single genes in a Rodney background, constitute the basis of the set used internationally to differentiate pathotypes of *P. graminis* f. sp. *avenae* (Martens *et al.,* 1979).

Gene *Pg1* is a dominant (Simons *et al.*, 1978; Martens, 1985; Martens & Dyck, 1988), thermostable gene(Gordon, according to Martens 1985; Martens *et al.*, 1967) that was introduced into the United States in approximately 1880. It originates from northern or central Russia in the cultivar White Russian (Simons *et al.*, 1978; Martens, 1985). The gene has been used extensively and is still present in North American cultivars (Martens & Dyck, 1988).

The *Pg2*-gene is a dominant (Simons *et al.*, 1978; Martens, 1985), thermostable (Gordon, according to Martens, 1985; Martens *et al.*, 1967) gene which was introduced into North America in 1873. It has been used extensively in breeding programmes (Martens & Dyck, 1988) and occurs in more recently released cultivars e.g. Dumont (McKenzie *et al.*, 1984). The origin of *Pg2* seems to be the Ukraine in the form of the cultivars Green Russian (Simons *et al.*, 1978) and Sixty-Day, or possibly the Black Sea in the form of the cultivar Kherson (Martens, 1985).

Gene *Pg3* is a dominant, thermolabile gene with a mesothetic reaction to some races, and a highly resistant reaction to avirulent races at low temperatures (Martens *et al.,* 1967). The gene is either closely linked to a gene conferring crown rust resistance, or may confer resistance to both rusts (McKenzie *et al.,* 1968). The crown rust resistance gene linked to *Pg3* in the cultivar Jostrain and selection X1588-2 appears to be *Pc*95 (Harder *et al.,* 1995). *Pg3* is also either allelic or tightly linked in repulsion to *Pg9* (McKenzie & Green, 1965; O'Donoughue *et al.,* 1996). Penner *et al.* (1993) identified a RAPD marker for this gene. *Pg3* was introduced into Canada from France in the cultivar Joanette in 1888-1889 (Simons *et al.,* 1978; Martens, 1985).

Pg4 is a dominant gene (Simons *et al.,* 1978; Martens, 1985) introduced to the USA via South Africa in the cultivar Hajira in 1919 (Welsh & Johnson, 1951; Simons *et al.,* 1978), but was not used until the early 1950s. Together with *Pg1* and *Pg2*, this gene formed the basis of stem rust resistance breeding in North America (Martens, 1985). Martens & Dyck (1988) reported this gene to be allelic with *Pg13*. According to Welsh & Johnson (1951) *Pg5* is probably the same gene as *Pg4*.

Genes *Pg6* and *Pg7* may be the same gene (Dyck, 1966) and are dominant, occur in the diploid species *A. strigosa,* and conditions resistance to a wide range of races (Murphy *et al.,* 1958; Dyck & Zillinsky, 1962). However, it has not yet been possible to transfer this resistance into hexaploid genotypes.

Pg8 is a recessive (Simons *et al.,* 1978; Martens, 1985), thermolabile gene(Martens *et al.,* 1967) which could be difficult to detect (Martens, 1985). The origin of *Pg8* seems to be North Africa, amongst other possibilities (Martens, 1985). According to Simons *et al.* (1978) *Pg8* may be allelic or closely linked to *Pg1* and *Pg2*.

Pg9 is a thermolabile, recessive gene (Martens, 1985; Martens *et al.,* 1967) closely associated with several crown rust resistance genes, e.g. *PcX* (Chong *et al.,* 1994; O'Donoughue *et al.,* 1996). Six markers are available for *Pg9* (O'Donoughue *et al.,* 1996). *Pg9* is either allelic to, or tightly linked in repulsion to *Pg3* (McKenzie & Green, 1965; O'Donoughue *et al.,* 1996). *Pg9* is linked to *Pc68* (O'Donoughue *et al.,* 1996; Chong *et al.,* 1994) and *Pc68* in turn, has been shown to be linked or allelic to

*Pc*46 and *Pc*50 (Wong *et al.*, 1983). Gene *Pg9* has also been shown to be linked in repulsion to *Pc*44 (Martens *et al.*, 1968). According to Harder (1994a) the expression of resistance by *Pg9* may also vary considerably under similar environmental conditions with different isolates of single phenotype, such as NA27. Harder (1994a) states that interpretations of reactions by *Pg3* and *Pg9* differentials at different times by different workers have been inconsistent.

Pg10 is a dominant or partially dominant gene (Martens, 1985) conferring a moderate but broad range of resistance which may be useful in resistance breeding (Harder *et al.*, 1995). Because of its susceptible reaction *Pg10* is not included in differential sets. Thus far there has been no evidence of race specificity involving *Pg10* resistance. The extended dark pigmented necrosis, reduced infection, and possible race-nonspecificity associated with *Pg10* are unique features and have not been observed for any other known stem rust resistance genotype (Harder *et al.*, 1995).

Pg11 is an incompletely recessive gene and, apart from *Pg17*, it is the only stem rust resistant gene that is exclusively effective in the adult plant stage to all races tested. *Pg11* is associated with a chlorophyll deficiency expressed at the adult plant stage which leads to a yellow plant colour, weak straw and somewhat reduced yield in the absence of rust (Martens, 1985; Harder *et al.*, 1971). It is probable that the resistance associated with gene *Pg11* is due to a physiological deficiency and may not represent a resistance gene in the usual sense (Martens, 1985; Harder *et al.*, 1990).

Martens *et al.* (1981) reported the *Pga* complex to consist of recessive gene *Pg12* and a complementary or interacting gene(s). Resistance to *Pg12* diminishes as the plant develops while some resistance can still be observed at more mature growth stages as adult plants appear moderately susceptible (Martens *et al.*, 1968). Infection types range from (;) to (2) and are associated with severe necrosis, although no chlorosis of surrounding tissue occurs. In interaction with other resistance genes, it provides highly effective resistance against all but two of the races that occur in North America (Martens *et al.*, 1981). An unknown Finnish

cultivar from the Kuopia area has been postulated as the most likely origin of *Pg12* (Martens, 1985).

More recently, Adhikari *et al.* (1999) demonstrated that *Pg12* is not involved in the *Pga* complex and that the resistance is conferred by two complementary recessive genes. Adhikari, according to Adhikari *et al.* (1999), also showed that the resistance conferred by *Pga* is influenced by temperature and is ineffective at temperatures higher than 26°C. According to Martens *et al.* (1981) the cultivar Omega is the reference source of *Pga*.

The recessive gene *Pg13* from a Tunisian collection of *A. sterilis* was discovered in the late 1960s (Martens, 1985; Howes *et al.*, 1992) and is one of the most effective genes available to breeders (Roelfs *et al.*, 1982; Martens, 1981; Martens & Dyck, 1988). Although races with virulence have been observed, the gene has been found effective in most of the countries where it was tested (Martens *et al.*, 1976). Nine RFLP markers and 2 RAPD markers have been identified by O'Donoughue *et al.* (1996). According to Harder (1994a) *Pg13* is currently the most important component of stem rust resistance in oat cultivars released for the rust area of the Canadian Prairies. McKenzie *et al.* (1970) reported *Pg13* to be associated with a chromosome translocation which was also associated with reduced transmission of the gene through pollen, while Martens & Dyck (1988) reported this gene to be allelic with *Pg4*.

The origin of the partially dominant gene *Pg14* is not clear. According to Martens (1985) it was isolated by MacKey & Mattsson from the cultivars Milford (C.I. 5039), Winter Turf (C.I. 1570) and other lines. Winter Turf is an old cultivar introduced to the United States from England in about 1764, and apparently it was noted for its stem rust resistance (Coffman according to Martens, 1985).

Pg15 is a partially dominant gene introduced from *A. sterilis* (Martens, 1985). Races collected from the Grain Plains region, which are avirulent on *Pg9,* are usually also avirulent on *Pg15* (Roelfs *et al.*, 1980; Martens, 1981). Lines with this gene have been shown to be highly effective in trap nurseries for detecting variability in the

pathogen population (Martens & McKenzie, 1979). According to Martens (1985) this gene has not been used in commercial cultivars.

Pg16 is a highly effective gene from tetraploid *A. barbata* that has been transferred and stabilised into advanced breeding lines of *A. sativa* (Brown *et al*. according to Martens & Dyck, 1988). There was only one occurrence of virulence to *Pg16* in Canada in 1984 (Harder, 1994a). Although highly effective, *Pg16* is not used in North America (Harder, 1994b).

Pg17 is a single dominant gene derived from *A. sterilis* accession IB3056, originally collected from the Iberian Peninsula. The gene confers a moderate level of resistance in the adult plant stage (Harder *et al*., 1990).

Thus available evidence suggests that most of the genes for stem rust resistance in hexaploid oats originated from two relatively small geographic areas; the region around the western Black Sea (Russia, Turkey) yielded *Pg1, Pg2, Pg9, Pg15* and possibly *Pg11*, while North Africa contributed *Pg4, Pg8, Pg13* and possibly *Pg3* via France. Finland and England are putative centres of origin for the genes *Pg12* and *Pg14* (Martens, 1985).

Although additional, unexploited sources of resistance exists in diploid and tetraploid accessions like *A. strigosa* Schreb. (Murphy *et al*., 1958; Dyck according to Martens, 1985), *A. longiglumis* Durieu. (Martens *et al*., *unpublished data*, according to Martens, 1985) and *A. barbata* Pott ex Link. (Dinoor & Wahl according to Martens 1985; Martens *et al*., 1980), it is difficult to transfer them to the hexaploid level (Martens & Dyck, 1988).

In oat stem rust a few genes display almost the entire range of genetic expression that has been observed in cereal rust pathosystems; dominance, recessiveness, allelism, adult plant resistance, seedling resistance only, cytoplasmically inherited resistance, complementary gene action, and thermal and light sensitivity, have all been documented (Martens & Dyck, 1988).

Breeding for durable stem rust resistance

In oats, resistance genes effective against present day populations of *P. graminis* have been difficult to find, but appear to be adequate to control oat stem rust in certain areas (Martens & Dyck, 1988).

The limited number of host resistance genes and wide virulence range of the pathogen throughout the world, make breeding for enduring disease control a major challenge. According to Martens (1985) the best intermediate-term prospect for efficient control of stem rust in oats lies in the synthesis of cultivars carrying several effective genes for resistance. Confirmation of this statement was given by Harder (1997) who reported that cultivars with the gene combination *Pg9* and *Pg13* have exhibited resistance since their release in the early 1980s. Martens (1985) further stated that the genes presently available offer considerable opportunities for breeding complex resistance. This is despite the problem of allelism, illustrated by the fact that seven of the seedling genes in the hexaploid host occur in three independent linkage groups: genes *Pg1*, *Pg2*, and *Pg8* in one group; genes *Pg4* and *Pg13* in another; and genes *Pg3* and *Pg9* in a third group. Genes *Pg12* and *Pg15* occur independently of these groups and of each other (McKenzie *et al.*, 1970; Martens *et al.*, 1980). Genes *Pga* and *Pg8* have not been used in resistance breeding in North America, and genes *Pg13*, *Pg15* and *Pg16*, derived from exotic sources, are recent introductions (Harder, 1994a). Martens (1985) stated that the combination of *Pg13* and *Pga* looks promising.

Several techniques, including marker-assisted selection, are available to combine different resistance genes. Penner *et al.* (1993) identified a random amplified polymorphic DNA (RAPD) marker linked to the *Pg3* gene. *Pg13* has been linked to a 56.6-kDA polypeptide locus resolved using sodium dodecyl sulfate-polyacryl-amide gel electrophoresis (SDS-PAGE) (Howes *et al.*, 1992; Chong *et al.*, 1994), and *Pg9* has been linked with an avenin band resolved by acide-PAGE (Chong *et al.*, 1994). Since cultivated oat is highly polymorphic at the DNA level (O'Donoughue *et al.*, 1996), the occurrence of these markers is not surprising.

Adhikari & McIntosh (1998) showed that *Pga* can be detected in the seedling stage by inoculating seedlings with a virulent crown rust race and an avirulent *P. graminis*

race, leading to induced *P. graminis* susceptibility. This provides a valuable bioassay for screening and positive identification of *Pga*.

REFERENCES

Adhikari, K.N. & McIntosh, R.A. 1998. Susceptibility in oats to stem rust induced by co-infection with leaf rust. Plant Pathology 47: 420-426.

Adhikari, K.N., McIntosh, R.A. & Oates, J.D. 1999. Inheritance of the stem rust resistance phenotype *Pg*-a in oats. Euphytica 105: 143-154.

Berkeley, M.J. 1876. An enumeration of the fungi collected at the Cape of Good Hope during the stay of the English transit of Venus expedition in 1874. Journal of Botany, London, Vol XIV (new series Vol. V): 173-175.

Chong, J., Howes, N.K., Brown, P.D. & Harder, D.E. 1994. Identification of the stem rust resistance gene *Pg9* and its association with crown rust resistance and endosperm proteins in "Dumont" oat. Genome 37: 440-447.

Doidge, E.M. 1927. A preliminary study of the South African rust fungi. Bothalia 2: 1-228.

Dyck, P.L. 1966. Inheritance of stem rust resistance and other characteristics in diploid oats, *Avena strignosa*. Canadian Journal of Genetics and Cytology 8: 444-450.

Dyck, P.L. & Zillinsky, F.J. 1962. Segregation for crown rust and stem rust resistance in diploid and autotretraploid *Avena strignosa*. Canadian Journal of Genetics and Cytology 4: 469-474.

Groth, J.V. 1984. Virulence frequency dynamics of rust fungi. Pages 231-252 in: The Cereal Rusts. Vol. 1. W.R. Bushnell and A.P. Roelfs, eds. Academic Press, Orlando, FL.

Harder, D.E. 1994(a). Virulence dynamics of *Puccinia graminis* f. sp. *avenae* in Canada, 1921-1993. Phytopathology 84: 739-746.

Harder, D.E. 1994(b). Identification of new races of *Puccinia graminis* f. sp. *avenae*. Plant Disease 78: 367-368.

Harder, D.E. 1997. Stem rust on wheat, barley, and oat in Canada in 1995. Canadian Journal of Plant Pathology 19:171-175.

Harder, D.E., Chong, J. & Brown, P.D. 1995. Stem and crown rust resistance in the Winsconsin oat selection X1588-2. Crop Science 35: 1011-1015.

Harder, D.E., Chong, J., Brown, P.D. & Martens, J.W. 1990. Inheritance of resistance to *Puccinia coronata avenae* and *P. graminis avenae* in an accession of *Avena sterilis* from Spain. Genome 33: 198-202.

Harder, D.E., Martens, J.W. & McKenzie, R.I.H. 1971. Changes in chlorophyll and carotenoid content in oats associated with the expression of adult plant resistance to stem rust conferred by gene *Pg11*. Canadian Journal of Botany 49: 1783-1785.

Howes, N.K., Chong, J. & Brown, P.D. 1992. Oat endosperm proteins associated with resistance to stem rust of oats. Genome 35: 120-125.

Kochman, J. K. & Brown, J. F. 1976(a). Effect of temperature, light and host on pre-penetration development of *Puccinia graminis avenae* and *Puccinia coronata avenae*. Annals of Applied Biology 82: 241-249.

Kochman, J. K. & Brown, J. F. 1976(b). Host and environmental effects on the penetration of oats by *Puccinia graminis avenae* and *Puccinia coronata avenae*. Annals of Applied Biology 82: 251-258.

Martens, J.W. 1981. Incidence and virulence of *Puccinia graminis* f. sp. *avenae* in Canada in 1980. Canadian Journal of Plant Pathology 3: 231-234.

Martens, J.W. 1985. Oat stem rust. Pages 103-129 in: The Cereal Rusts. Vol 2. A.P. Roelfs and W.R. Bushnell, eds. Academic Press, New York.

Martens, J.W. & Dyck, P.L. 1988. Genetics of resistance to rust in cereals from a Canadian perspective. Canadian Journal of Pathology 11: 78-85.

Martens, J.W., Green, G.J. & McKenzie, R.I.H. 1976. International oat stem rust virulence survey. Plant Disease Reporter 60: 25-528.

Martens, J.W. & McKenzie, R.I.H. 1979. Virulence dynamics of *Puccinia graminis* f. sp. *avenae* in Canada. Canadian Journal of Botany 57: 952-957.

Martens, J.W., McKenzie, R.I.H. & Fleischmann, G. 1968. The inheritance of resistance to stem and crown rust in Kyto oats. Canadian Journal of Genetics and Cytology 10: 808-812.

Martens, J.W., McKenzie, R.I.H. & Green, G.J. 1967. Thermal stability of stem rust resistance in oat seedlings. Canadian Journal of Botany 45: 451-458.

Martens, J.W., McKenzie, R.I.H. & Green, G.J. 1970. Gene-for-gene relationships in the *Avena: Puccinia graminis* host-parasite system in Canada. Canadian Journal of Botany 48: 969-975.

Martens, J.W., McKenzie, R.I.H. & Harder, D.E. 1980. Resistance to *Puccinia graminis avenae* and *P. coronata avenae* in the wild and cultivated *Avena* populations of Iran, Iraq, and Turkey. Canadian Journal of Genetics and Cytology 22: 641-649.

Martens, J.W., Roelfs, A.P., McKenzie, R.I.H., Rothman, P.G., Stuthman, D.D. & Brown, P.D. 1979. System of nomenclature for races of *Puccinia graminis* f. sp. *avenae*. Phytopathology 69: 293-294.

Martens, J.W., Rothman, P.G., McKenzie, R.I.H. & Brown, P.D. 1981. Evidence for complementary gene action conferring resistance to *Puccinia graminis avenae* in *Avena sativa*. Canadian Journal of Genetics and Cytology 23: 591-595.

McKenzie, R.I.H., Brown, P.D., Martens, J.W., Harder, D.E., Nielsen, J., Gill, G.G. & Boughten, G.R. 1984. Registration of Dumont oats. Crop Science 24: 207.

McKenzie, R.I.H. & Green, G.J. 1965. Stem rust resistance in oats. I. The inheritance of resistance to race 6AF in six varieties of oats. Canadian Journal of Genetics and Cytology 7: 268-274.

McKenzie, R.I.H., Martens, J W. & Ajhathy, T. 1970. Inheritance of oat stem rust resistance in Tunisian strain of *Avena sterilis.* Canadian Journal of Genetics and Cytology 12: 501-505.

McKenzie, R.I.H., Martens, J.W., Fleischmann, G. & Sambroski, D.J. 1968. An association of stem rust and crown rust resistance in Jostrain oats. Canadian Journal of Genetics and Cytology 10: 190-195.

McVey, D.V., Long, D.L. & Roberts, J.J. 1996. Races of *Puccinia graminis* in the United States during 1994. Plant Disease 80: 85-89.

McVey, D.V., Long, D.L. & Roberts, J.J. 1997. Races of *Puccinia graminis* in the United States during 1995. Plant Disease 81: 306-310.

Murphy, H.C., Zillinsky, F.J., Simons, M.D. & Grindeland, R. 1958. Inheritance of seed color and resistance to race of stem and crown rust in *Avena strigosa.* Agronomy Journal 50: 539-541.

O'Donoughue, L.S., Chong, J., Wight, C.P., Fedak, G. & Molnar, S.J. 1996. Localisation of stem rust resistance genes and associated molecular markers in cultivated oat. Phytopathology 86: 719-727.

Penner, G.A., Chong, J., Lévesque-Lemay, M., Molnar, S.J. & Fedak, G. 1993. Identification of RADP marker linked to the stem rust resistance gene *Pg3*. Theoretical and Applied Genetics 85: 702-705.

Person, C. 1966. Genetic polymorphism in parasitic systems. Nature 212: 266-267.

Roelfs, A.P. & Long, D.L. 1980. Analysis of recent oat stem rust epidemics. Phytopathology 70: 436-440.

Roelfs, A.P., Long, D.L. & Casper, D.L. 1980. Races of *Puccinia graminis* f. sp. *avenae* in the United States during 1979. Plant Disease 64: 947-949.

Roelfs, A.P., Long, D.L. & Casper, D.L. 1982. Races of *Puccinia graminis* f. sp. *avenae* in the United States and Mexico during 1980. Plant Disease 66: 208-209.

Roelfs, A.P., Long, D.L. & Roberts, J.J. 1993. Races of *Puccinia graminis* in the United States during 1991. Plant Disease 77: 129-132.

Šebesta, J., Zwartz, B., Roderick, H.W., Harder, D.E., Corazza, L. & Stojanovic, S. 1998. Incidence of oat stem rust and virulence of *Puccinia graminis* Pers. f. sp. *avenae* Erikss. et Henn. On oat and the effectiveness of resistance in Europe during 1988 to 1996. Archives of Phytopathology and Plant Protection 31: 393-413.

Simons, M.D., Martens, J.W., McKenzie, R.I.H., Nishiyama, I., Sadanaga, K., Sebesta, J. & Thomas, H. 1978. Oats: A standardised system of nomenclature for genes and chromosomes and catalog of genes governing characters. United States Department of Agriculture, Agriculture Handbook 509: 1-40.

Verwoerd, L. 1929. A preliminary checklist of diseases of cultivated plants in the winter rainfall area of the Cape Province. Union of South Africa, Department of. Agricultural Science Bulletin 88: 1-28.

Verwoerd, L. 1931. Die fisiologiese vorms van *Puccinia graminis* Pers. wat in

Suid-Afrika voorkom. South African Journal of Science 28: 274-279.

Welsh, J.N. & Johnson, T. 1951. The source of resistance and the inheritance of reaction to 12 physiologic races of stem rust, *Puccinia graminis avenae* (Erikss. and Henn.). Canadian Journal of Botany 29: 189-205.

Wong, L.S.L., McKenzie, R.I.H., Harder, D.E. & Martens, J.W. 1983. The inheritance of resistance to *Puccinia coronata* and floret characters in *Avena sterilis*. Canadian Journal of Genetics and Cytology 25: 329-335.

OCCURRENCE AND PATHOGENICITY OF *PUCCINIA HORDEI* ON BARLEY IN SOUTH AFRICA

INTRODUCTION

Leaf rust, caused by the fungus *Puccinia hordei* Otth., is considered an important disease of barley (*Hordeum vulgare* L.) in several areas of the world including Australia, Europe, North America and South America (Alemayehu & Parlevliet, 1996; Borovkova *et al.*, 1997). Until 1970 the disease was considered unimportant in economic terms, but more recently changes in cropping practises have resulted in an increase in the importance of leaf rust with severe epidemics occurring more regularly (Clifford, 1985). Teng (according to Lim & Gaunt, 1986) concluded that *P. hordei* should be regarded as a serious source of potential yield loss, while Cotterill *et al.* (according to Cotterill *et al.*, 1994) recorded yield losses of 26-31% in Australia during a moderate to severe epidemic in 1990.

In South Africa the disease was first reported by Doidge *et al.* (1953) in the south Western Cape. Here, barley production, grown mainly for malting purposes, comprises approximately 130 000 ha under dry-land conditions (Fig. 1) (Engelbrecht, 1999). The possibility of barley production under irrigation is currently being investigated although cultivars that can compete economically with wheat are not yet available. Up to 1997, the only cultivars planted were Clipper, Stirling and Schooner, with Clipper comprising about 95% of the total production. The cultivar SSG 532 was released in 1997. Due to susceptibility of Clipper to other foliar diseases, especially scald (*Rhynchosporium secalis* Oud.), extensive and repeated fungicide applications are generally required. This has led to the "unintentional" control of leaf rust, but in seasons not favourable for scald, e.g. 1992, 1997, 1998 and 1999, leaf rust epidemics occurred.

The main emphasis of local breeding programmes has so far been on increased yield, good brewing quality, as well as resistance to scald. With many of these objectives reached in new cultivars and advanced breeding lines, as well as the recurring *P. hordei*

Figure 1. Map of South Africa indicating barley producing areas.

epidemics over the last few seasons, resistance to leaf rust has become more important.

It is important to identify the resistance genes still effective against leaf rust in South Africa and to know how many pathotypes are prevalent. To achieve this, a virulence survey was carried out between 1994 and 1999 to determine the extent of pathogenic variation in the *P. hordei* population as well as to determine the reaction of leading cultivars and breeding lines to the prevalent pathotypes. To investigate consistency in leaf rust phenotypes, the influence of temperature and light intensity on infection types controlled by different resistance genes was investigated. The susceptibility of possible accessory hosts for *P. hordei*, and the role of a uredinial pathogen of *Ornithogalum* spp., was also investigated.

MATERIALS AND METHODS

Seedling tests

Leaf rust-infected barley leaves were either collected personally from trap nurseries, experimental trials, and commercial fields, or received by mail from co-workers during annual surveys. Urediniospores were then collected using a cyclone spore collector and stored at $-70°C$ when Clipper seedlings were not available for immediate inoculation. Six-day-old (first leaf fully extended) Clipper seedlings grown in 10-cm-diameter plastic pots, filled with steam-sterilised soil, were inoculated with urediniospores either directly from field samples, or following a heat shock treatment (42°C for 4 minutes) had they been stored at $-70°C$. Prior to inoculation, emerging Clipper seedlings were treated with 50 ml/pot of a 0.3 gl^{-1} maleic hydrazide solution to retard plant development and stimulate sporulation. Seedlings were spray-inoculated with urediniospores suspended in light mineral oil (Soltrol 170), allowed to dry for 1 h in fan-circulated air at room temperature, and then placed in the dark in a dew-simulation chamber for 24 h at 16°C (± 2°C). Upon removal from the dew chamber, plants were allowed to dry for 2 h in fan-circulated air before being placed in separate glass compartments in a greenhouse. Greenhouse temperatures were maintained at between 18°C and 22°C while natural

day light was supplemented with 120 $\mu Em^{-2}s^{-1}$ photosynthetically active radiation emitted by cool-white fluorescent tubes arranged directly above plants (14 h/day).

After 10 to 14 days, between two and 10 single pustule isolates were collected from each sample depending on the locality and genotype from which the sample was originally collected. Isolates were then re-inoculated on Clipper seedlings for further urediniospore multiplication. In certain instances there was a need for a second multiplication. Urediniospores obtained after multiplication were inoculated on to a differential set of barley cultivars with known genes for resistance (Table 1). Two to three days after initial eruption of uredinia, plants were assessed for their disease response using a 0-4 scale (Roelfs, 1988).

The reaction of cultivars, crossing parents and elite entries of Small Grain Institute (SGI) breeding lines to *P. hordei* infection was also determined in the seedling stage. Four entries, consisting of ten seeds each, were planted in clumps in 10-cm-diameter pots filled with steam-sterilised soil. Lines were inoculated with freshly collected urediniospores of pathotype SAPh 3231 in 1997 and with both pathotypes SAPh 3231 and SAPh 7231 in 1998. Pre- and post-inoculation conditions were maintained as described above. As in the case of differentials, infection types were assessed two to three days after the eruption of primary uredia.

Adult plant tests

Differential lines (Table 1) and commercial cultivars were planted in 1-m-row field plots spaced 20 cm apart and surrounded by two rows of Clipper. Plots were planted at the SGI (Free State, artificial infection with pathotype SAPh 3231), Langgewens experimental station (Western Cape, natural infection) and Tygerhoek experimental station (south Western Cape, natural infection). Disease assessments were recorded on flag and penultimate leaves at least two times during the season using a modified Cobb scale (Peterson *et al.*, 1948). The adult plant reactions of differential cultivars to *P. hordei* infections were also studied under greenhouse conditions. Three seeds of each cultivar were planted in a 2-litre-capacity pot filled with steam-sterilised soil. Three pots per cultivar were planted at seven day intervals to ensure synchronisation of flag

Table 1. Infection types of differential barley lines to two South African pathotypes of *Puccinia hordei* detected during annual surveys from 1994 to 1999

Cultivar	Gene	Octal value	Pathotype SAPh 3231		Pathotype SAPh 7231
			Typical reaction	Variation	
Sudan	*Rph1*	1	3+[a]	Consistent	3+
Peruvian	*Rph2*	2	X2	0; to 2+C	X2
Aim	*Rph3*	4	0;	0; to 2	0;
Gold	*Rph4*	10	3	Consistent	3
Magnif 104	*Rph5*	20	3	2++C to 3	3
Bolivia	*Rph6+2*	40	X-	;1 to 2+	X-
Cebada Capa	*Rph7*	100	;N	Consistent	;N
Egypt 4	*Rph8*	200	33C	2+C to 3+	33C
Abyssinian	*Rph9*	400	2C	;12 to 2+C	2C
Clipper BC8	*Rph10*	1000	3+	Consistent	3+
Clipper BC67	*Rph11*	2000	3	Consistent	3
Triumph	*Rph12*	4000	;1N	;N to 2N	3
Clipper	-		3+	Consistent	3+

[a] Infection types (Roelfs, 1988).

leaf emergence. Pre- and post-inoculation greenhouse conditions were the same as those for seedling tests. Plant nutrition was supplied in three weekly applications of 50 ml per pot of a 3 gl^{-1} hydroponic solution (6.5:2.7:13 N:P:K, plus micro-elements). Flag leaves were inoculated with freshly collected urediniospores of pathotype SAPh 3231 in a similar fashion to that of seedlings. After 10 days flag leaves were rated using the modified Cobb scale (Peterson *et al.*, 1948).

During 1998, elite lines of the SGI breeding programme were evaluated in the field for adult plant resistance. Entries were planted at SGI in hill plots each consisting of 10 seeds and bordered by Clipper spreader rows. Spreader rows were planted three weeks earlier and inoculated by injecting tillers at growth stage 36 (Zadoks *et al.*, 1974) with a suspension of freshly collected urediniospores (SAPh 3231) in water containing the surfactant Tween 20 (1 ml per litre). Infection of plots was encouraged by regular overhead mist irrigation ensuring high humidity conditions.

Temperature study

Due to variable responses of certain differential lines to *P. hordei* infection the influence of temperature on these reactions was determined. Differential lines (Table 1) were grown in 10 cm plastic pots containing a sterilised soil : peatmoss mixture (1:1 v/v) at 19°C/ 24°C night/day temperature. After seven days (first leaf fully expanded) plants were inoculated with fresh urediniospores (SAPh 3231) and incubated as described previously. After allowing the oil to evaporate from leaves, plants were incubated overnight in a dark dew-simulation chamber before placement in growth chambers set at 11°C, 18°C and 25°C, respectively . These treatments were compared with differential sets maintained at 18-24°C in the regular greenhouse environment. Fourteen hours of light was provided by cool-white fluorescent tubes and incandescent bulbs arranged 30 cm above plants, providing 200 $\mu Em^{-2}s^{-1}$ of photosynthetically active radiation. Each temperature regime was represented by three replications of differential sets. Disease assessment was done when Clipper (susceptible control) showed a compatible (3 to 3+) reaction.

Light intensity study

To determine whether light intensity influenced the expression of infection types, differential sets were inoculated with pathotypes SAPh 3231 and SAPh 7231 and exposed to two light regimes. Inoculation and pre-inoculation procedures were as described previously. Upon removal from the dew chamber plants were placed in two greenhouse cubicles where a 22°C/18°C day/night cycle was maintained. Conditions in both cubicles were similar except that in one treatment all direct sunlight was screened out using 80% shade cloth, while in the other treatment additional lighting was provided by Mercury-Tungsten bulbs emitting 400 $\mu Em^{-2}s^{-1}$ photosynthetically active radiation. In both cubicles natural day light was supplemented with 14 h of 120 $\mu Em^{-2}s^{-1}$ photosynthetically active radiation emitted by cool-white fluorescent tubes arranged directly above plants. Ten days after inoculation infection types were assessed using the 0-4 scale (Roelfs, 1988).

Accessory hosts

The relationship between *P. hordei* and wild *Hordeum* species occurring in the Western Cape was investigated. Various *Hordeum* accessions were collected during annual surveys. After identification, seed of *Hordeum* spp. were harvested, planted and infected with appropriate cultures of *P. hordei*. Pre- and post-inoculation treatment of seedlings was conducted as described previously. Urediniospores from rusted *Ornithogalum* spp. were also collected and inoculated onto Clipper seedlings.

RESULTS

Pathogenic variation

During 1994, 15 leaf rust samples were collected from the south Western Cape, and one each from the Western and Eastern Cape. In 1995, 48 samples were collected from the south Western Cape and two from Bergville in KwaZulu-Natal. Twenty six samples were collected in the south Western Cape during the 1996 season, while 35, 4 and 3 samples were collected during the 1997 survey from the south Western, Western and Eastern Cape, respectively. During 1998, 20 samples were collected from

the south Western Cape while the Western Cape produced five and the Eastern Cape three samples. Since it was the first study of this nature in South Africa, 211 single pustule isolates were established during 1994. During 1995 a similar approach was followed resulting in 316 isolates, while the 1996, 1997 and 1998 collections yielded 97, 84, and 102 isolates, respectively.

All except one of the 810 isolates gave the same reaction on the differential set (Table 1). Isolate 98-18-2 was collected from Clipper on the farm Voorstekop in the Swellendam-district in the south Western Cape during 1998 and differed from the other isolates only in terms of its virulence to the resistance gene *Rph12* in the cultivar Triumph (Table 1 and Fig. 2). The common pathotype was designated as SAPh 3231, while isolate 98-18-2 was identified as pathotype SAPh 7231 using the octal notation described by Gilmour (1973).

During the 1999 season it was established that the formerly resistant cultivar SSG 532 and line B94/2 were susceptible to leaf rust. Isolates from these two lines were sampled in 1999 and inoculated onto a differential set which confirmed that susceptibility was due to pathotype SAPh 7231. This was substantiated by the susceptible reaction of Triumph in field traps (Table 2). In total, 28 samples were collected from the south Western Cape and 12 from the Western Cape during 1999. The 28 south Western Cape samples produced 62 single pustules of which 95% were SAPh 7231 and 5% were SAPh 3231. All 31 single pustule isolates from the Western Cape represented pathotype SAPh 3231.

Due to mesothetic seedling reactions obtained with pathotype SAPh 3231 on some of the differentials, reactions were tested in the adult plant stage to determine whether these genes were still effective. This was done under greenhouse conditions as well as under artificial and naturally infected field conditions. It was found that differentials with a mesothetic seedling reaction reacted in a resistant manner in the adult plant stage with *Rph2* being the only exception, exhibiting a tendency towards moderate susceptibility at Tygerhoek during 1997 and Bethlehem in 1998 (Table 2).

Figure 2. Infection types produced by pathotypes SAPh 3231 and SAPh 7231 of *Puccinia hordei* on seedling leaves of Clipper and Triumph (*Rph12*). From left to right: Clipper/SAPh 7231, Triumph/SAPh 3231 and Triumph/SAPh 7231.

Table 2. Leaf rust severity and reaction type produced in the field and greenhouse on adult plants of differential barley lines to pathotypes SAPh 3231 and SAPh 7231 of *Puccinia hordei*

Cultivar	*Rph* gene	Tygerhoek 1997 (SAPh 3231)	Tygerhoek 1999 (SAPh 3231 and SAPh 7231)	Napier 1999 [a] (SAPh 3231 and SAPh 7231)	Bethlehem Greenhouse (SAPh 3231)	Bethlehem Field 1998 (SAPh 3231)
Sudan	*Rph1*	60MS-S [b] (Y)[c]	70MS-S	80MS	60S-MS	80S
Peruvian	*Rph2*	60MS-S (Y)	20MS	0	40R-MR	60MS-MR
Aim	*Rph3*	30MS	-	-	10R	20R
Gold	*Rph4*	80S	80S	90S	80S	80S
Magnif 104	*Rph5*	60MS-S	-	100S	60MS-S	80MS
Bolivia	*Rph6+2*	20MS-MR (Y)	0	0	40R-MS	30MS-MR
Cebada Capa	*Rph7*	0	0	0	10R	20R
Egypt 4	*Rph8*	40MS-MR	-	30MR	40MR-MS	40MS-MR
Abyssinian	*Rph9*	20MR-R	-	50MS	40R-MR	30MS-MR
Clipper BC8	*Rph10*	60MS-S	60MS	50MS	60S	60S
Clipper BC67	*Rph11*	80S	70S	60S	80S	60S
Triumph	*Rph12*	20MR-MS (Y)	80S	60MS	40R-MR	30R
Clipper	_	80S	100S	100S	90S	80S

[a] Ratings were done on field plots at SENSAKO.

[b] S, susceptible; MS, moderately susceptible; R, resistant; MR, moderately resistant; 0, no visible signs of disease; Y, more and/or bigger pustules at leaf tip; -, infection types not determined.

Temperature study

Differential sets maintained at different temperatures revealed only minor changes in their reaction to an isolate of pathotype SAPh 3231. No dramatic change from resistant to susceptible or *vice versa* could be demonstrated although *Rph8* tended to be more susceptible at 25°C (Table 3 & Figures 3 & 4). There were, however, noticeable differences between growth chamber and greenhouse environments in the reactions of the differentials inoculated at the same time with the same isolate (Table 3 & Figures 5 & 6).

Light intensity study

Although differences in light intensity between cubicles were much more pronounced than that normally observed in the greenhouse, light intensity did not explain the variation in reaction types observed in routine tests over time. The only notable difference was the onset of sporulation approximately 12 h earlier in the high light intensity environment. Magnif 104 tended to produce a lower reaction (2) at lower light intensity than at the higher light intensity (23).

Cultivar evaluation

Elite barley lines of the SGI were evaluated for resistance to pathotype SAPh 3231 in both the seedling and adult plant stage in 1998 (Table 4). The 1999 elite lines as well as crossing parents and cultivars were screened for both pathotypes SAPh 3231 and SAPh 7231 in the seedling stage (Table 5 & 6). Seedling reactions corresponded in general with those of adult plants, although a few exceptions such as B97/3, displaying a moderately susceptible (23) reaction in the seedling stage and a resistant (30R-MR) reaction in the adult plant stage occurred. Based on their reactions to the two pathotypes it was possible to postulate resistance gene *Rph12* in SSG532, B94/2, B98/4, B98/6, B99/4, Prisma, Optic, Krona, and Maresi.

Accessory hosts

During the 1998 survey six different accessions of *Hordeum murinum* L. and two of *H. capense* Thunb. were collected. These were tested for their reaction to *P. hordei* race SAPh 3231. Four of the *H. murinum* accessions were weak hosts with reactions varying

Table 3. Infection types of differential barley lines to South African pathotype SAPh 3231 of *Puccinia hordei* at different temperatures and in different controlled environments

Cultivar	Resistance gene	Growth Chamber			Greenhouse UOFS	Greenhouse SGI
		25°C	18°C	11°C	18°-24°C	18-22°C
Sudan	*Rph1*	33+	33+	3+	3	33+
Peruvian	*Rph2*	2	X+	X	X2	X2
Aim	*Rph3*	;;1	0	;	0;	0
Gold	*Rph4*	3	3	3-3	3	3
Magnif 104	*Rph5*	2	3=	3	3-	3-
Bolivia	*Rph6+2*	2-	X= (Y)	X= (Y)	2-N	X-
Cebada Capa	*Rph7*	;N	;N	;CN	;N	;CN
Egypt 4	*Rph8*	3++	23	22+C	2-C	3
Abyssinian	*Rph9*	2C	;1	;;1=	1N	2C
Clipper BC8	*Rph10*	23	3	3	3	3+
Clipper BC67	*Rph11*	23	23	3	2+3	3
Triumph	*Rph12*	2-C	;;1+	;;1N	;1N	;N
Clipper		3+	3	3= (2+3)	2+3	3+
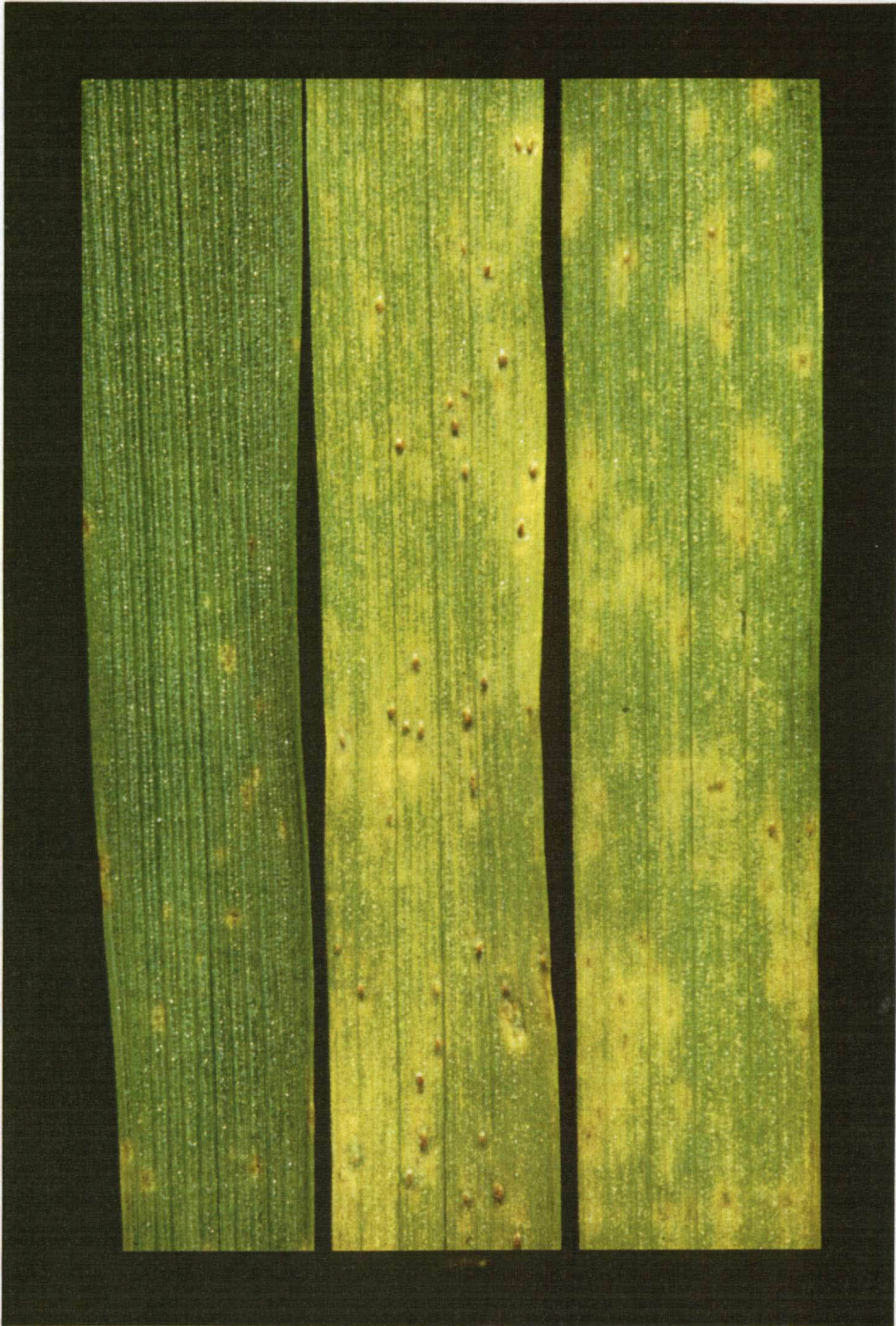
Figure 3. Reaction type of seedling leaves of Cebada Capa (*Rph7*) (left), Egypt 4 (*Rph8*) (middle) and Abyssinian (*Rph9*) at 11°C to pathotype SAPh 3231 of *Puccinia hordei*.

Figure 4. Reaction type of seedling leaves of Cebada Capa (*Rph7*) (left), Egypt 4
(*Rph8*) (middle) and Abyssinian (*Rph9*) 25°C (B) to pathotype SAPh 3231 of
Puccinia hordei.
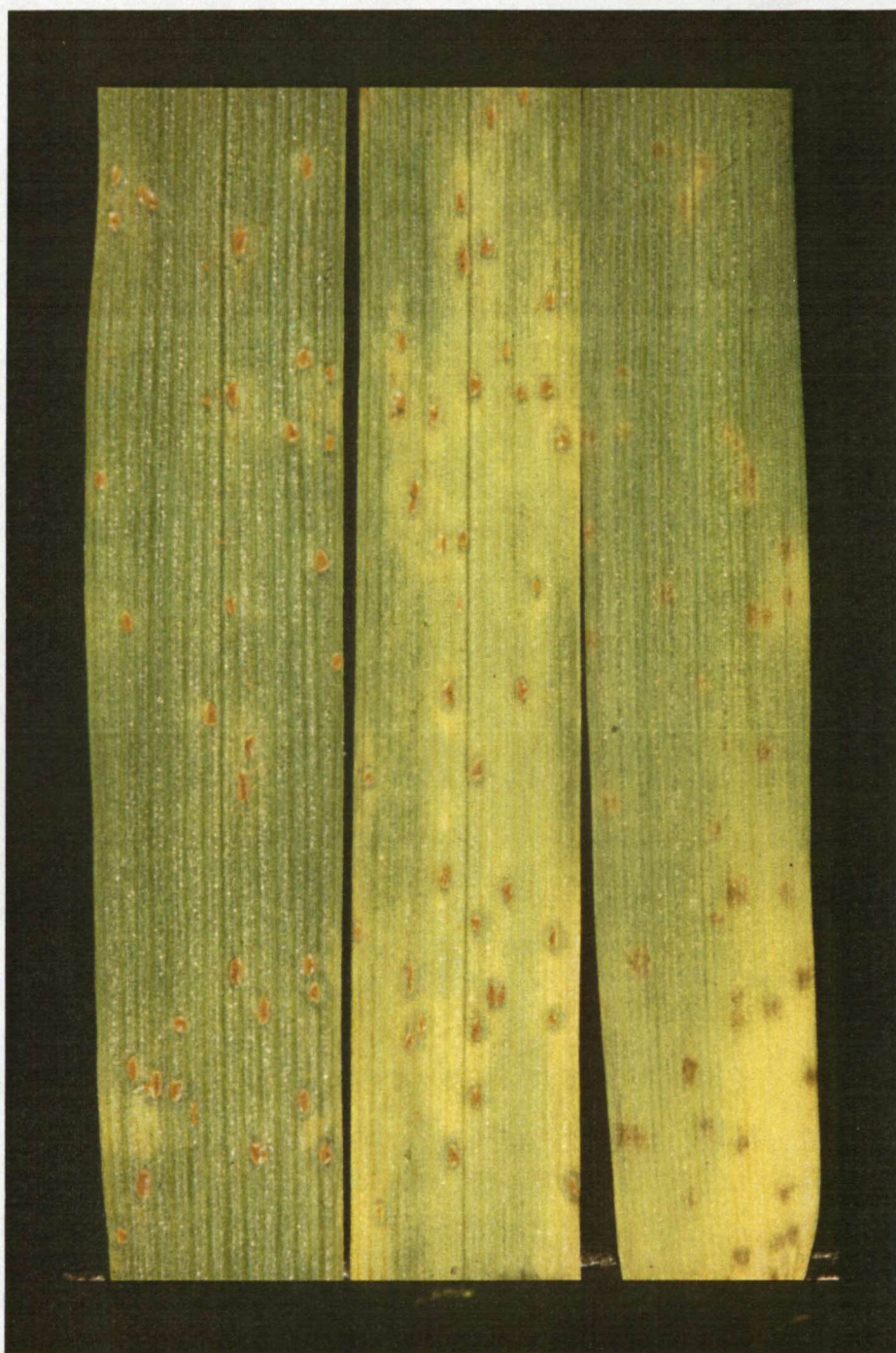
Figure 5. Infection types of seedling leaves of Clipper BC8 (*Rph10*) (left), Clipper BC67 (*Rph11*) (middle) and Triumph (*Rph12*) (right) at greenhouse conditions when infected with pathotype SAPh 3231 of *Puccinia hordei*.
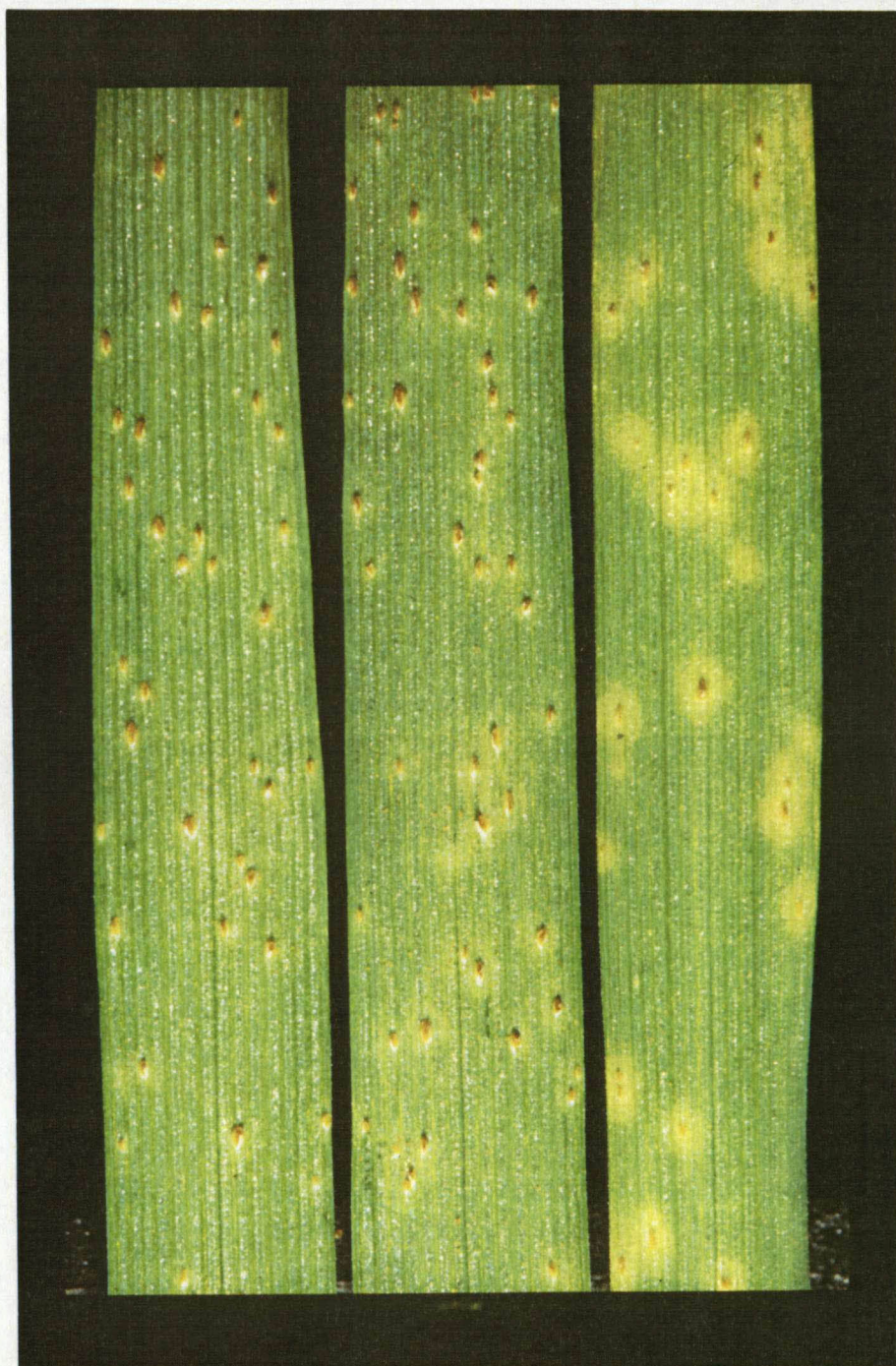
Figure 6. Infection types of seedling leaves of Clipper BC8 (*Rph10*) (left), Clipper BC67 (*Rph11*) (middle) and Triumph (*Rph12*) (right) at growth chamber conditions (25°C) when infected with pathotype SAPh 3231 of *Puccinia hordei*.

Table 4. Seedling and adult plant reaction of a collection of breeding lines[a] and control genotypes to pathotype SAPh 3231 of *Puccinia hordei*

Barley elite lines (Dry-land)			Barley elite lines (Irrigation)		
Line	Seedlingreaction	Adult plant reaction[b]	Line	Seedling reaction	Adult plant reaction[b]
Clipper	3	80S	Blenheim	2	10-20MS
Stirling	3+	40MS	B93/2	22+	tMR
SSG525	3	80MS	B93/4	;CN	0
SSG532	2CN	30MR-MS	B94/2	2+CN	40R
B94/2	2+CN	30R-MR	B95/10	3+	50MS-S
B95/10	3+	50MS-S	B97/12	2	30MS
B97/3	23	30R-MR	B97/13	223	60S
B97/6	23	60MS	B97/16	X+	30MS
B97/7	X-	5MS	B98/2	X+	20S
B97/8	23	20MS	B98/3	23	30S
B97/9	2+	20MS	B98/4	X+	50S
B97/10	23	40MS	B98/5	X+	50S
B97/11	X	0-40S	B98/6	2+C	40MR-MS
B97/12	2	30MS			
B97/13	223	60S			
B97/14	2+2	40MS-MR			
B97/15	X+	20MS			
B97/16	X+	30MS			
B98/1	2C	10R			
B98/2	X+	20S			
B98/3	23	30S			
B98/4	X+	50S			
B98/5	X+	50S			
B98/6	2+C	40MR-MS			
B96/1	;CN	10R			

[a] Small Grain Institute 1998 elite lines for dry-land and irrigation areas respectively.
[b] Artificial inoculation at Small Grain Institute, Bethlehem during 1998.

Table 5. Seedling infection types of the 1999 Small Grain Institute barley crossing block and elite entries to pathotypes SAPh 3231 and SAPh 7231 of *Puccinia hordei*

1999 Crossing block			1999 Elite lines (Dry-land)			1999 Elite lines(Irrigation)		
Line	Pathotype SAPh 3231	Pathotype SAPh 7231	Line	Pathotype SAPh 3231	Pathotype SAPh 7231	Line	Pathotype SAPh 3231	Pathotype SAPh 7231
Clipper	3	3	Clipper	3	3	Blenheim	-	3-
Stirling	3	3	Stirling	3	3	B95/10	3-	3-
SSG 532	5P;1N 3P3	3-	B95/10	3	3	B93/4	0;	3-
B94/2	2C	2++	SSG 532	3P3- 6P;CN	3	B94/2	;N	3-
B94/5	X	X-	B94/2	2N	3-	B98/2	X	X+
B94/7	X-	X-	B97/15	22+	2+2	B98/3	3-	3-
B95/10	3	3	B97/16	22+C	-	B98/5	3-	3-
B96/1	;1	;	B98/1	3P ;N 3P 3	-	B98/6	2N	3-
B97/3	3	X	B98/2	2+2	3	B93/2	-	22+
B97/9	3-	3-	B98/3	23	23	B99/1	X+	X
B97/10	3-	3-	B98/4	X-	3	B99/3	3-	3-
B97/12	3-	3-	B98/5	2C	2+	B99/6	3	3-
B97/14	2+2	2+	B98/6	2+	-	B99/8	3-	3-
B97/15	3-	2+2	B99/1	3-	3-	B99/9	X	X
B97/16	X	-	B99/2	3	3-			
B98/2	3-	3-	B99/3	3+	3			
B98/6	3-	-	B99/4	X	3			
Chariot	X	-	B99/5	3-	3-			
Prisma	;N	3-	B99/6	3	3⁻			
Scarlett	0;	;	B99/7	3-	3⁻			
Optic	;N	2+	B99/8	X+	X+			
Barke	;N	;	B99/9	3-	3-			
Alexis	;12	;1N	B99/10	2-	3-			
Krona	;N1N	3-	B99/11	3-	3-			
Maresi	2	3	B99/12	3-	X+			

Table 6. Seedling infection types of South African barley cultivars to pathotypes SAPh
3231 and SAPh 7231 of *Puccinia hordei*

Cultivar	Pathotype SAPh 3231	Pathotype SAPh 7231
Clipper	3	3
B94/2	2N	3-
B95/10	3	3
SSG 532	;N,2N and 3	3
SSG 525	3	3
Stirling	3	3
Triumph	;N	3-

from ;2C to 2, while the other *H. murinum* accessions and *H. capense* were resistant to *P. hordei*, indicating between- and within-species variation for response to this pathogen. Urediniospores collected from *Ornithogalum* spp. were inoculated onto Clipper seedlings but no susceptible reactions were observed.

DISCUSSION

The appearance of the new barley leaf rust pathotype SAPh 7231 in 1998, and the ensuing epidemic in 1999, emphasised the importance of virulence surveys. Although resistance to *P. hordei* is used as selection criteria in breeding programmes, screening has relied mostly on natural infection. In addition to inconsistent results, these selections were made without any knowledge of which genes are still effective in the South African environment.

From the present data it is clear that during the period 1994–1997 only one *P. hordei* pathotype (SAPh 3231), with virulence to the genes *Rph1*, *Rph4*, *Rph5*, *Rph10*, and *Rph11*, occurred in South Africa. In 1998 a second pathotype (SAPh 7231), adding virulence to *Rph12*, appeared. During 1999 pathotype SAPh 7231 was detected only in the south Western Cape where it was first identified in 1998, suggesting that significant shifts in distribution have not yet occurred. Future surveys should indicate whether factors such as fitness, survival, and the availability of selective hosts will influence the occurrence of the new pathotype on barley in South Africa.

Reinhold & Sharp (1982) stated that in Mediterranean areas, where summer months are dry, the fungus may be dependent on sexual recombination to complete its life cycle, thus resulting in a higher frequency of new physiologic races. Anikster *et al.* (according to Clifford, 1985) reported on the co-existence of *Ornithogalum* flora with wild *Hordeum* spp. and mentioned that the alternate host is essential for the survival of the pathogen and for the generation of pathogenic variability in the uredinial stage in Israel. Taking into account that local barley producing areas have a Mediterranean climate and that many *Hordeum* spp., as well as 54 species of the alternate host

Ornithogalum spp., occur in South Africa, many of which in the barley producing area (Obermeyer, 1978), more than one pathotype could be expected. Contradictory, only two pathotypes, one being a recent addition, were found. This phenomenon could probably best be explained by the cultivars grown during this period. Approximately 95% of barley planted up to 1997 was Clipper indicating that there was no selective advantage for virulence in new pathotypes. The release of SSG 532 in 1997, and large scale production in 1998, provided this advantage to pathotypes with virulence to *Rph12*.

Furthermore, the teleomorph of *P. hordei* is not known to occur in South Africa, making sexual recombination unlikely. Levine & Cherewick (1952) and Steffenson *et al.* (1993) mentioned mutations as a likely cause for the origin of new virulence genes. This would make a single step mutation the most obvious explanation for the formation of pathotype SAPh 7231. In addition to SSG 532, *Rph12* resides in many breeding lines. Line B94/2 which is currently being released, is also susceptible to pathotype SAPh 7231 but resistant to SAPh 3231. Several other lines including B98/4, B98/6, B99/4, Prisma, Optic, Krona and Maresi seem to have *Rph12* (Table 5) since they are resistant to SAPh 3231 but susceptible to SAPh 7231.

Leaf rust samples from Clipper, included in a wheat rust trap nursery in KwaZulu-Natal during 1995, were unique in the sense that they were collected approximately 1000 km from the nearest barley producing area. This may indicate that although leaf rust was not found in the other irrigation areas, favourable weather conditions and large areas of susceptible cultivars could lead to barley leaf rust becoming a problem in these areas.

Levine & Cherewick (1952) reported the sensitivity of barley leaf rust to temperature and light effects. In the present study, the fact that the same isolate produced different reactions on differential sets kept in the greenhouse and growth chambers, supported the sensitivity of *P. hordei* to the biotic and physical environments. Likewise, it was found that reaction types of breeding lines in successive tests may vary considerably in their reaction to the same isolate. For example, line B97/16 displayed X+ reactions

in some ratings and 22+C reaction in others, while B98/2 varied from 3- to X+ (Tables 4 & 5). It was also noticed that in certain cases the reaction was associated with chlorosis, while in others the same isolate on the same line produced a more necrotic infection type. Furthermore, the time after inoculation had a considerable influence on the infection type as exemplified by *Rph8* present in Egypt 4. If scored two to three days after eruption of the first pustules the reaction tended to be compatible (3) but if scored two to three days later chlorosis (3C) and necrosis (3N) became evident.

Not much evidence was obtained for temperature sensitivity of *Rph* genes, with the exception of *Rph8* which varied from 22+C at 11°C to 23 at 18°C, and 3++ at 25°C (Table 3 and Fig. 3). Clifford & Udeogalanya (according to Clifford, 1985) reported that the gene in CI 1243 (*Rph9*) became less effective with an increase in temperature from 5°C-25°C. In the present study some indications were found that *Rph9* tended to be more susceptible at 25°C (2C) than at 11°C (;;1=) (Fig. 3). It should also be taken into account that the pathotype used is considered to be avirulent to *Rph8* and *Rph9*. Apart from the reaction of Magnif 104 there was no indication that light intensity could be responsible for the variation observed. It is likely that the infection type is not only influenced by host and pathogen genotypes, but by complicated interactions between temperature, light intensity, light duration and plant nutrition, possibly during pre- and post-inoculation periods.

As the use of resistant cultivars is globally accepted as the primary strategy to control leaf rust of barley (Jin *et al.*, 1996), it is important to ensure that resistance is durable. Parlevliet (1980, 1983) warned against the use of low-infection type resistance in commercial cultivars and stated that it only provided temporary protection with more serious consequences than breeders realised. The continued use of single *Rph* genes in barley cultivars will probably result in ephemeral resistance, because virulence for all described leaf rust resistance genes is known in the global population of *P. hordei* (Clifford, 1985; Steffenson *et al.*, 1993). Alternatively, Steffenson *et al.* (1993) stated that greater durability of host resistance might be achieved through the transfer of several *Rph* genes into a single pure line cultivar. This is an attractive option in the South African situation where many genes remain effective, with *Rph3* and *Rph7*

probably the best candidate genes to combine. However, the detection of these genes in breeding lines is difficult without appropriate markers or "tester" cultures of *P. hordei*. For this approach to succeed in industry, it is imperative that neither of the resistance genes be exposed singly to the rust population. This would allow stepwise pathogenic adaptations eventually rendering the combination ineffective. An alternative strategy is to breed for slow-rusting or type II resistance as described by Clifford (1985). According to Parlevliet (1983) partial resistance is more durable and has remained effective in Europe since the early 1970s (Steffenson *et al.*, 1993). According to Qi *et al.* (1998) progress has been made in this regard. Six QTLs for partial resistance in the barley cultivar Vada were identified. It was also significant that these QTLs mapped to different positions than those for hypersensitive resistance, suggesting different gene families.

REFERENCES

Alemayehu, F. & Parlevliet, J.E. 1996. Variation for resistance to *Puccinia hordei* in Ethiopian barley landraces. Euphytica 90: 365-370.

Borovkova, I.G., Jin, Y., Steffenson, B.J., Kilian, A., Blake, T.K. & Kleinhofs, A. 1997. Identification and mapping of a leaf rust resistant gene in barley line Q21861. Genome 40: 236-241.

Clifford, B.C. 1985. Barley leaf rust. Pages 173-205 in: The Cereal Rusts. Vol. 2. A.P. Roelfs and W.R. Bushnell, eds. Academic Press, New York.

Cotterill, P.J., Rees, R.G. & Platz, G.J. 1994. Response of Australian barley cultivars to leaf rust (*Puccinia hordei*). Australian Journal of Experimental Agriculture 34: 783-788.

Doidge, E.M., Bottemley, A.M., Van der Plank, J.E. & Pauer, G.D. 1953. A revised list of plant diseases in South Africa. Science Bulletin No. 346, Department of Agriculture.

Engelbrecht, W. 1999. Report presented at SANSOR (South African National Seed Organisation) meeting 12-14 May 1999.

Gilmour, J. 1973. Octal notation for designating physiological races of plant pathogens. Nature 242: 260.

Jin, Y., Cui, G.H., Steffenson, B.J. & Franckowiak, J.D. 1996. New leaf rust resistance genes in barely and their allelic and linkage relationships with other *Rph* genes. Phytopathology 86: 887-890.

Lim, L.G. & Gaunt, R.E. 1986. The effect of powdery mildew (*Erysiphe graminis* f. sp. *hordei*) and leaf rust (*Puccinia hordei*) on spring barley in New Zealand. I. Epidemic development, green leaf area and yield. Plant Pathology 35: 44-53.

Levine, M.N. & Cherewick, W.J. 1952. Studies on dwarf leaf rust of barley. United States Department of Agriculture, Technical Bulletin 1056: 1-17.

Obermeyer, A.A. 1978. *Ornithogalum*: a revision of the southern African species. Bothalia 12: 377-393.

Parlevliet, J.E. 1980. Level of partial resistance to leaf rust, *Puccinia hordei* in West-European barley and how to select for it. Euphytica 29: 1-8.

Parlevliet, J.E. 1983. Race-specific resistance and cultivar-specific virulence in the barley-leaf rust pathosystem and their consequences for breeding of leaf rust resistant barley. Euphytica 32: 367-375.

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

Qi, X., Niks, R.E., Stam, P. & Lindhout, P. 1998. Identification of QTLs for partial resistance to leaf rust (*Puccinia hordei*) in barley. Theoretical and Applied Genetics 96: 1205 (abstr.).

Roelfs, A.P. 1988. Genetic control of phenotypes in wheat stem rust. Annual Review of Phytopathology 26: 351-367.

Reinhold, M. & Sharp, E.L. 1982. Virulence types of *Puccinia hordei* from North America, North Africa, and the Middle East. Plant Disease 66: 1009-1011.

Steffenson, B.J., Jin, Y. & Griffey, C.A. 1993. Pathotypes of *Puccinia hordei* with virulence for barley leaf rust resistance gene *Rph7* in the United States. Plant Disease 77: 867-869.

Zadoks, J.C., Chang, T.T. & Konzak, C. F. 1974. A decimal code for the growth stages of cereals. Weed Research 14: 415-421.

OCCURRENCE AND PATHOGENICITY OF *PUCCINIA CORONATA* F. SP. *AVENAE* AND *P. GRAMINIS* F. SP. *AVENAE* ON OAT IN SOUTH AFRICA

INTRODUCTION

Oat (*Avena sativa* L.) is an important cereal, used either for human consumption or as a feed grain, and ranks sixth in the world cereal production after wheat, maize, rice, barley and sorghum (Murphy & Hoffman according to O'Donoughue *et al.*, 1995). Crown (leaf) rust, caused by the fungus *Puccinia coronata* Corda f. sp. *avenae* Eriks., is considered the most important fungal disease of cultivated oat (Simons, 1985), while stem rust, caused by *Puccinia graminis* Pers. f. sp. *avenae* Eriks. and E. Henn., occurs in most of the oat growing areas of the world, periodically causing severe crop losses (Martens, 1978; 1985). Šebesta *et al.* (1998) reported oat stem rust to be potentially a more destructive disease than crown rust.

Puccinia coronata f. sp. *avenae* is highly variable in virulence and can rapidly evolve new pathotypes that overcome commonly used resistance genotypes leading to an almost innumerable array of pathogenic variants. It has repeatedly demonstrated its ability to adapt to constraints imposed by man as control measures (Simons, 1985; Chong & Kolmer, 1993; Kolmer & Chong, 1993). Early specialisation studies on *P. graminis* f. sp. *avenae* indicated that virulence combinations occurred widely in different parts of the world. As new genes for resistance were discovered, virulence on them was usually found concurrently and, in most cases, virulence was present in the pathogen population before cultivars carrying these genes were grown over large areas (Martens, 1985).

The first reference of stem rust in South Africa was by Berkeley (1876) who mentioned the collection of *P. graminis* in 1874 from the foot of Table Mountain but did not state from which host it was isolated. Bailey identified the race 2,3/1 in South Africa in 1925 (Martens, 1985).

Sawer (1909) observed that oat crops in KwaZulu Natal were destroyed to such an extent by crown rust that it could no longer be grown there. Sawer (1909) also provided evidence of dramatic changes in cultivar rankings over time which, although speculative, could have been due to rust infections or even pathogenic adaptation. Evans (1911) reported that oat rust was not known in South Africa before 1858 but that rust had often restricted oat production since then. According to Doidge (1927) and Verwoerd (1929; 1931a) *P. graminis* f. sp. *avenae* was found on oats throughout South Africa, and reference was made to the occurrence of *P. coronata* f. sp. *avenae* from Stellenbosch in the south to as far north as Salisbury, Rhodesia (now Harare, Zimbabwe). Doidge *et al.* (1953) reported that no aecidial form is known for crown rust in South Africa.

At present oat production is completely deregulated in South Africa and no official records of the total area cultivated and annual production exist. Apart from this oats is also used for various purposes including grain oats for breakfast cereal, horse feed (especially race horses), general feed rations, health foods as well as bird food. In the Western Cape oats is planted primarily for silage whereas it is grown as green feed during the dry winter months in the summer rainfall areas. In 1991 the Small Grain Institute conducted a survey among 27 agricultural co-operatives and established that 8.6% of the cultivated oats was used for grain production, 76.4% for grazing and 15% for silage. According to this survey, seed sales suggested that 59 620 ha of oats was planted during 1990. This is probably a conservative estimate taking into account that many farmers retain their own seed, especially when oats is grown for grazing purposes. At present oats has also increased in popularity as a rotation crop under irrigation and as grazing in the Highveld. Engelbrecht (1999) estimated the potential for oat production in South Africa to be ±700 000 ha.

Historically, most oat varieties cultivated in South Africa have been susceptible to crown rust. In association with stem rust, crown rust is often responsible for significant losses in oat seed quantity and quality. Although both pathogens occur annually in most oat growing areas of South Africa, rust diseases are especially severe in the Western Cape. Occasionally severe epidemics will occur in the summer rainfall area, e.g. the

widespread crown rust infections experienced in the Free State during 1998. The objectives of this study were to determine the pathogenic variability in *P. coronata* f. sp. *avenae* and *P. graminis* f. sp. *avenae* in South Africa. The availability of representative pathotypes will be useful in the screening of oat germplasm for resistance to these diseases.

In addition oat cultivars and breeding lines were evaluated for seedling and adult plant resistance towards pathotypes of both oat rust pathogens. The role of wild *Avena* spp. as possible accessory hosts for both crown and stem rust was also investigated.

MATERIALS AND METHODS

Survey

Oat leaves and stems infected with either *P. coronata* f. sp. *avenae* or *P. graminis* f. sp. *avenae* were collected from trap nurseries, commercial fields and wild oats during the 1997 and 1998 annual rust surveys conducted by the Small Grain Institute. Urediniospores were removed from infected leaves and stems using a cyclone spore collector and stored in gelatine capsules at $-70°C$. Upon retrieval from low-temperature storage prior to inoculation, spores were given a heat treatment of 6 min at $47°C$. Oat seedlings for purifying field samples were raised at a continuous temperature of $22°C$ and 14 h of light, provided by cool-white fluorescent tubes and incandescent bulbs arranged 30cm above plants and emitting 200 $\mu Em^{-2}s^{-1}$ of photosynthetically active radiation, in a disease free growth chamber. Seedlings were grown in 10-cm-diameter plastic pots filled with steam- sterilised soil and treated with 50 ml/pot of a 0.3 gl^{-1} maleic hydrazide solution to retard plant development and stimulate sporulation.

Using light mineral oil (Soltrol 170) as carrier medium, urediniospores of *P. coronata* f. sp. *avenae* and *P. graminis* f. sp. *avenae* were inoculated onto seven-day-old (first leaf fully extended) Overberg or Makuru seedlings, respectively. Seedlings were allowed to dry for 1 h in fan-circulated air at room temperature before being placed in the dark in a dew simulation chamber at $18°C$ ($\pm2°C$) for 18 h. After removal from the dew

chamber plants were allowed to dry for 2 h before placement in a greenhouse. Different isolates were separated in glass compartments to avoid contamination. Greenhouse temperatures were maintained at 22°C for crown rust and 19°C for stem rust studies, with natural day light supplemented by 14 h of 120 $\mu Em^{-2}s^{-1}$ photosynthetically active radiation emitted by cool-white fluorescent tubes arranged directly above plants. After 10 to14 days, two to four single-pustules per sample were collected, increased on either Overberg or Makuru, and inoculated onto the respective differential sets (Tables 1 & 2). Three to four days after the initial eruption of uredia, infection types were assessed using a 0 to 4 scale (Roelfs, 1988). *Puccinia coronata* f. sp. *avenae* infection types were assessed on the adaxial surface of leaves whereas those of *P. graminis* f. sp. *avenae* were rated on abaxial surfaces.

The adult plant reactions of the various differentials were also determined. Crown and stem rust differential lines (Tables 3 & 4) were planted in a screening nursery at Bethlehem during the 1998 season. Each entry was sown in a 1-m row planted 17 cm apart and bordered by two rust spreader rows consisting of a mixture of Makuru, Maluti, Pallinup, Euro and Overberg. When the first spreader plants reached growth stage 36 (Zadoks *et al.*, 1974), an equal amount of urediniospores of crown rust pathotype SAPc 1 and stem rust pathotypes SAPga 1, SAPga 2, SAPga 3 and SAPga 4 suspended in water with the aid of Tween 20, was injected into tillers. When infected flag leaves appeared in spreader rows, disease progress was assisted by overhead mist irrigation at night. During the course of the epidemic disease reactions were assessed using a modified Cobb scale (Peterson *et al.*, 1948).

Cultivar reaction

The reaction of all oat cultivars grown in South Africa as well as promising breeding lines was determined in the seedling stage to crown rust pathotypes SAPc 1, SAPc 39M, SAPc 50M, SAPc 52M and SAPc 64M as well as stem rust pathotypes SAPga 1, SAPga 2, SAPga 3 and SAPga 4. Four cultivars were planted in clumps of ±10 seeds each in a 10-cm plastic pot filled with steam-sterilised soil. When first leaves were fully expanded, plants were inoculated with freshly collected urediniospores of the different pathotypes. Inoculation and incubation procedures were similar to those described

Table 1. Infection types[a] produced on seedlings of differential lines of *Avena sativa* containing single *Pc* genes when infected with pathotypes SAPc 1, SAPc 39M, SAPc 50M, SAPc 52M and SAPc 64M of *Puccinia coronata* f. sp. *avenae*

Pc genes	*P. coronata* f. sp. *avenae* pathotypes[b]				
	SAPc 1	SAPc 39M	SAPc 50M	SAPc 52M	SAPc 64M
Pc35	;1CN	;N	2+3	;C	2+3
Pc38	0;	;	;C	0;	;C
Pc39	;	3	;	;	;C
Pc40	2+3	3	3	2+3	;C
PcS42	;	0	0;	0	0
Pc45	2+3	2+3	;	2+3	3
Pc46	2+3	2+3	2+3	2+3	2+3
Pc48	;C	;	;	2+3	;C
Pc50	;	;	2+3	0;	0;
Pc51	2+3	3	;	2+3	;
Pc52	;C	;	0;	2+3	;
Pc54	2+3	2+3	;C	2+3	2+3
Pc56	;1=C	;C	;C	;C	X(;, 2)
Pc58	;	;	X(;C, 2)	;C	X(;, 2)
Pc59	;	;	;	;	;
Pc60	2C	2C	X(;, 2C)	;C	X(;, 2)
Pc61	2C	2C	;	;	X(;, 2)
Pc62	;1CN	;1	;C	;C	X(;, 2)
Pc63	;	;	;	0	;C
Pc64	;	;	;	;	3
Pc68	0	0;	0;	0;	0;
Pc96	0;	;	0;	0;	X

[a] Roelfs (1988).

[b] Pathotype SAPc 1 was isolated from field collections whereas SAPc 39M, SAPc 50M, SAPc 52M and SAPc 64M originated as greenhouse mutants.

Table 2. Infection types[a] produced on seedlings of differential lines of *Avena sativa* containing single *Pg* genes as well as gene combinations by pathotypes SAPga 1, SAPga 2, SAPga 3 and SAPga 4 of *Puccinia graminis* f. sp. *avenae*

Pg gene	CI number	*P. graminis* f. sp. *avenae* pathotypes			
		SAPga 1	SAPga 2	SAPga 3	SAPga 4
Pga	-	;	;	;	3
Pg1	CI9318	2+3	2+3	2+3	3
Pg2	CI 9319	3	3	;	3
Pg3	CI 9320	X	2	X-	2
Pg4	CI 6661	3	3	0	3
Pg8	CI 9321	3	3	3	3
Pg9	CI 9322	;2	2+3	;1	3
Pg10	-	0	X-	0	;C
Pg13	CI 9212	;	;1	;C	;1C
Pg15	CI 9351	2+3	2+3	3	3
Pg16	CI 9352	;C	;	X-	;C
Pg17[b]	-	2+3	2+3	2+3	3
Pg1+2[c]	-	3	4	;	3
Pg2+4[c]	-	3	4	;	3
Pg4+8[c]	-	2+3	2+3	;	3
Pg2+4+9[c]	-	X	2+3	;	3

[a] Roelfs (1988).

[b] Adult plant resistance.

[c] Gene combinations and not considered as part of differential set.

Table 3. Adult plant reactions[a] of differential oat lines to artificial infection by pathotypes SAPc 1 of *Puccinia coronata* f. sp. *avenae* at Bethlehem in 1998

Resistance gene	Adult plant reaction
Pc35	0
Pc38	0
Pc39	0
Pc40	100S
PcS42	0
Pc45	100S
Pc46	80S
Pc48	0
Pc50	0
Pc51	80S
Pc52	0
Pc54	100S
Pc56	0
Pc58	0
Pc59	0
Pc60	20MS-MR
Pc61	15MS-MR
Pc62	0
Pc63	0
Pc64	0
Pc68	0
Pc96	0

[a] S, susceptible; MS, moderately susceptible; R, resistant; MR, moderately resistant; 0, no visible signs of disease; (modified Cobb scale, Peterson *et al.*, 1948).

Table 4.   Adult plant reactions[a] of differential oat lines to artificial infection of a
mixture of pathotypes SAPga 1, SAPga 2, SAPga 3 and SAPga 4 of
Puccinia graminis f. sp. *avenae* at Bethlehem in 1998

Resistance gene	Adult plant reaction
Pga	80MS-MR
Pg1	80S
Pg2	30MS
Pg3	-
Pg4	100S
Pg8	-
Pg9	80MS
Pg10	10MR
Pg13	0
Pg15	50S
Pg16	-
Pg17	0
Pg1+2	80S
Pg2+4	50S
Pg4+5	60S
Pg2+4+9	tMS
Swan	100S

[a]   S, susceptible; MS, moderately susceptible; R, resistant; MR, moderately resistant;
t, trace; 0, no visible signs of disease; -, not determined (modified Cobb scale,
Peterson *et al.*, 1948).

previously.

Disease assessments (modified Cobb scale) were done on entries in the annual oat cultivar adaptation trials conducted in the main production areas by the Agronomy department of the Small Grain Institute. These replicated trials consisted of commercial cultivars as well as the most promising breeding lines (Table 5).

Accessory hosts

Several *Avena* spp., including *A. fatua*, *A. byzantina*, *A. sterilis* and *A. barbata,* occur in South Africa with *A. fatua* being the most common (Smit, 1993). Collections of these species were evaluated for seedling resistance to all the pathotypes of *P. coronata* f. sp. *avenae* and *P. graminis* f. sp. *avenae* detected during the study period. Procedures for growing of plants as well as inoculation and disease assessment were as described above.

RESULTS

Crown rust

During 1997, 11 samples were collected from the south Western Cape, 14 from the Western Cape, six from KwaZulu Natal and 17 from the Free State. During 1998, 15 samples were received from each of the south Western- and Western Cape regions respectively, five from KwaZulu Natal and 13 from the Free State. In total 269 single-pustule isolates were tested on differential sets, revealing one dominant pathotype (SAPc 1, Table 1) and four mutant isolates detected in the greenhouse (Table 1, Fig. 1-5).

Stem rust

During 1997, 59 samples were collected, six from the Western Cape, 16 from the south Western Cape and 37 from the Free State. During 1998, 18 samples were collected from the south Western Cape, seven from the Western Cape, four from the KwaZulu Natal, 13 from the Free State and two from Lesotho. This resulted in the establishment and differentiation of 242 single pustule isolates. From these, four pathotypes were

Table 5. Adult plant reactions[a] of various oat cultivars and lines to natural infection of
Puccinia coronata f. sp. *avenae* and *P. graminis* f. sp. *avenae* at Tygerhoek
experimental station and Piketberg during 1999

Cultivar/Line	Crown rust reaction		Stem rust reaction	
	Tygerhoek	Piketberg	Tygerhoek	Piketberg
Overberg	60S	100S	10S	10S
OX90:123-11-17C	0	0	60S	60S
Potberg	50S	70S	tS	10S
Perdeberg	60S	100S	10MR-MS	10S
Sederberg	0	0	80S	40S
Pallinup	80S	100S	10S	30S
Euro	80S	100S	40S	40S
OX91:066-7RL	5MS	0	30S	40S
Heros	30MS-S	70S	tS	10S
OX87:073-10-14	0	0	0	0
Kompasberg	0	0	10S	10-20S
Tafelberg	0	0	10MS-S	20S
OX87:072-13-3C	tMS	0	tS	0
OX90:109-49CL	0	0	20S	50S

[a] S, susceptible; MS, moderately susceptible; R, resistant; MR, moderately resistant;
t, trace; 0, no visible signs of disease (modified Cobb scale, Peterson *et al.*, 1948).

Figure 1. Reaction types produced by the pathotypes, from left to right: SAPc 1, SAPc 39M, SAPc 50M, SAPc 52M and SAPc 64M of *P. coronata* f. sp. *avenae* on resistance gene *Pc35*.

Figure 2. Reaction types produced by the pathotypes, from left to right: SAPc 1, SAPc 39M, SAPc 50M, SAPc 52M and SAPc 64M of *P. coronata* f. sp. *avenae* on resistance gene *Pc48*.

Figure 3. Reaction types produced by the pathotypes, from left to right: SAPc 1, SAPc 39M, SAPc 50M, SAPc 52M and SAPc 64M of *P. coronata* f. sp. *avenae* on resistance gene *Pc50*.

Figure 4. Reaction types produced by the pathotypes, from left to right: SAPc 1, SAPc 39M, SAPc 50M, SAPc 52M and SAPc 64M of *P. coronata* f sp. *avenae* on resistance gene *Pc51*.

Figure 5. Reaction types produced by the pathotypes, from left to right: SAPc 1, SAPc 39M, SAPc 50M, SAPc 52M and SAPc 64M of *P. coronata* f sp. *avenae* on resistance gene *Pc54*.

identified namely, SAPga 1 (3,9,10,13,16,a/1,2,4,8,15), SAPga 2 (3,10,13,16,a/1,2,4,8,9,15), SAPga 3 (2,3,4,9,10,13,16,a/1,8,15) and SAPga 4 (3,10,13,16/1,2,4,8,9,15,a) (Table 2 & Fig. 6-9) (Martens et al., 1979). During 1997, 43.1% of the isolates belonged to pathotype SAPga 1 and 30.4% to SAPga 4. Pathotypes SAPga 3 and SAPga 2 represented 22.4% and 4.1% of the isolates, respectively. In 1998 pathotype SAPga 4 was isolated most frequently (67.7%), followed by SAPga 1 (17.1%), SAPga 3 (9.2%) and SAPga 2 (6.1%) (Table 6).

Culitvar reaction

Commercial cultivars as well as promising breeding lines were evaluated in the seedling stage for their reaction to crown rust pathotypes SAPc 1, SAPc 39M, SAPc 50M, SAPc 52M and SAPc 64M (Table 7) and stem rust pathotypes SAPga 1, SAPga 2, SAPga 3 and SAPga 4 (Table 8). Adult plant reactions of oat elite lines to natural infections of crown and stem rust during the 1999 season were also assessed at two field localities (Table 5).

Accessory hosts

Collections of *A. fatua*, *A. byzantina*, *A. sterilis* and *A. barbata* were susceptible to all five pathotypes of *P. coronata* f. sp. *avenae,* whereas all pathotypes of *P. graminis* f. sp. *avenae* were virulent on *A. fatua*, *A. byzantina* and *A. sterilis*. Accessions of *A. barbata* were resistant to all stem rust pathotypes.

DISCUSSION

This survey clearly showed the presence of only one crown rust pathotype, SAPc 1, with virulence to the resistance genes *Pc40, Pc45, Pc46, Pc51* and *Pc54*, on cultivated and wild oat in South Africa during 1997 - 1998. This confirms the preliminary data of Pretorius et al. (1998) who described the common occurrence of the same pathotype. This domination by a single pathotype is unusual if one takes into account that *P. coronata* f. sp. *avenae* is characterised world-wide by a countless number of variants (Simons, 1985; Chong & Kolmer, 1993; Kolmer & Chong, 1993). What makes

Figure 6. Reaction of resistance gene *Pga* to South African *P. graminis* f. sp. *avenae* pathotypes (from left to right) SAPga 1, SAPga 2, SAPga 3 and SAPga 4.

Figure 7. Reaction of resistance gene *Pg2* to South African *P. graminis* f. sp. *avenae* pathotypes (from left to right) SAPga 1, SAPga 2, SAPga 3 and SAPga 4.

Figure 8. Reaction of resistance gene *Pg4* to South African *P. graminis* f. sp. *avenae* pathotypes (from left to right) SAPga 1, SAPga 2, SAPga 3 and SAPga 4.
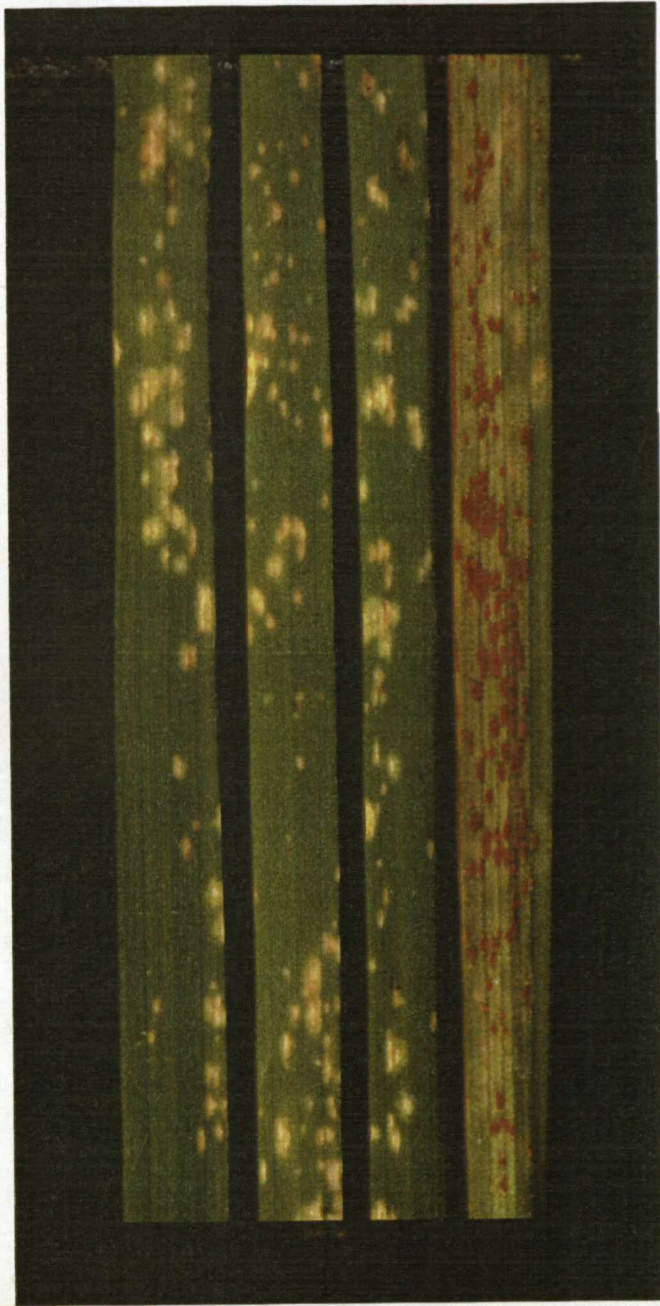
Figure 9. Reaction of resistance gene *Pg9* to South African *P. graminis* f. sp. *avenae* pathotypes (from left to right) SAPga 1, SAPga 2, SAPga 3 and SAPga 4.

Table 6. Distribution of *Puccinia graminis* f. sp *avenae* pathotypes in South Africa during 1997 and 1998

Region	1997				1998			
	SAPga 1	SAPga 2	SAPga3	SAPga 4	SAPga 1	SAPga 2	SAPga 3	SAPga 4
Western Cape	48.4[a]	7.9	23.8	19.9	9.8	1.2	6.2	82.8
South Western Cape	40.1	4.2	21.3	34.4	7.3	1.3	5.6	85.8
Free State	43.5	3.4	22.7	30.4	30.5	2.3	15.6	51.6
KwaZulu Natal	-	-	-	-	38.4	1.2	14.2	46.2
Lesotho	-	-	-	-	0	100	0	0
Total[b]	43.08	4.08	22.43	30.42	17.05	6.05	9.18	67.72

[a] Percentage isolates identified per region.

[b] Total percentage of isolates identified in survey.

Table 7. Infection types[a] produced on seedlings of South African cultivars as well as additional lines of *Avena sativa* to infection with *Puccinia coronata* f. sp. avenae pathotypes SAPc 1, SAPc 39M, SAPc 50M, SAPc 52M and SAPc 64M

Cultivars	*P. coronata* f. sp. *avenae* pathotypes				
	SAPc 1	SAPc 39M	SAPc 50M	SAPc 52M	SAPc 64M
Alpha	02+	2+3	2+3	;2	2
Drakensberg	0	0	0;	0	0
Dumont	0;	0	0;	0	0
Echidna	2+3	2+3	2+3	2+3	-
Euro	03 (Y)	2+3	;C	2+3	;CN
Fidler	;C	2+3	;C	;C	0;
Heros	2+3	3	2+3	2	22+
HJA81462	2+3	2+3	2+3	22++	22+
JO1298	2+3	2+3	2+3	2+3	2+3
Kompasberg	;	0	;	0	0
Kyto	2+3	22+3	2+3	2+3	22+
Langeberg	22+3	-	-	-	22+
Makura	2++3	2+3	2+3	22++	2
Maluti	2+3	3	2	3	;1C
Overberg	2+3	3	;	3	;
OX87 080-1	2+3	2+3	;	0;	0
OX88 123-104	;C	;1c	;	0;	0;
OX88I 075-106	22+3	2++C	;CN	22+3 (Y)	0;CN
Pallinup	;22+3 (Y)	2++3	;C	;	;CN
Perdeberg	2+3	2++3	;	2+3	;CN
Potberg	2+3	2++3	2+3	2+3	22+3
Sederberg	;	0;	;	0;	0;
Sisko	2+3	22+3	2+3	2	2++
SSH421	22+	2	;C 2	; 2+3 (Y)	;12
SSH423	2++3	2+3	2+3	2+	2+3
Swan	2+3	2+3	3	3	2+
Swartberg	2+3	3	;	2 3(Y)	;
Tafelberg	0;	0;	;	0;	0;
Texan	2+3	3	3	2+3	2+3
Veli	22+3	3	3	22+	22+
Victorian	22+3	3	2+3	2+3	-
Virma	22+3	2+3	2+3	22+	2++
Wisconsin1588	22+3 (Y)	2++3	2+3	22+3	;12
Witteberg	22+3	2+3	2+3	22+3	2+3
YTY	2+3	2+3	3	2++3	2++3
W 94/4	0	;12	;C	;C	0
SWK001	2+3	2+3	3	2+3	2+3

[a] Roelfs (1988).

Table 8. Infection types[a] produced on seedlings of South African cultivars and lines of *Avena sativa* following infection by *Puccinia graminis* f. sp. *avenae* pathotypes SAPga 1, SAPga 2, SAPga 3 and SAPga 4

Cultivar	*P. graminis* f. sp. *avenae* pathotypes			
	SAPga 1	SAPga 2	SAPga 3	SAPga 4
Alpha	;	;C	;C	2+3
Drakensberg	;	;C1C	;	;1
Dumont	;	;2-	;1	;1
Echidna	6p2+3 12p0	2+3	;1C	2+3
Euro	2+3	3	;1C	2+3
Fidler	;1	2	;C	2
Heros	22+3	22+3	22+3	2++
HJA81462	3	2+3	2+3	22+3
JO1298	2+3	2+3	2+3	22+3
Kompasberg	;1	7p2- 10p22+3	2-C	9p2-C 6p2+3
Kyto	8p2+3 8p;	22+	22+3	22+3
Langeberg	2+3	-	;1	-
Makura	3	3	2+3	2+2+3
Maluti	2+3	2+3	22+3	2+3
Overberg	2+3	2+3	2+3	2+3
OX87 080-1	;1	2	;1	2+3
OX88 123-104	2	2+3	22+3	2+3
OX88I 075-106	;	;C	0;	2+3
Pallinup	;1	22+	;	22+3
Perdeberg	3	22+3	0	22+
Potberg	2+3	3	0;	2+3
Sederberg	;C	;C	;	2+3
Sisko	2+3	2+3	2+3	2+3
SSH421	2+3	2+3	2+3	22+3
SSH423	3	2+3	22+3	-
Swan	3	2+3	2+3	22+3
Swartberg	3	2+3	;1	2+3
Tafelberg	;	2	;1-C	2+
Texan	2+3	22+3	2++	22+3
Veli	2+3	22+	22+3	22+3
Victorian	;	2+3	0;	22+3
Virma	2+3	3	3	3
Wisconsin1588	2	3	22+3	2++3
Witteberg	3	22+	2+3	22+3
YTY	22+3	22+3	22+3	22+3
W 94/4	;	;1	;1	3
SWK001	2+3	2+3	2+3	22+3

[a] Roelfs (1988).

this even more extraordinary, is the fact that four mutant pustules, giving rise to pathotypes SAPc 39M, SAPc 50M, SAPc 52M and SAPc 64M, were detected during pathotype differentiation. This undoubtedly demonstrates the ability of the South African *P. coronata* f. sp. *avenae* population to adapt and overcome existing resistance.

Pathotype SAPc 39M was differentiated by a single step mutation adding virulence to *Pc39* (Table 1 & Fig. 2). Considering SAPc 52M, virulence to two genes (*Pc48* and *Pc52*) was gained simultaneously (Table 1 & Fig. 2). Similarly, pathotype SAPc 50M gained virulence to resistance genes *Pc35* and *Pc50*, but also lost virulence to *Pc45* and *Pc54* (Table 1 & Fig. 1, 3 and 5). The mutant pathotype SAPc 64M showed the same pattern, gaining virulence to two genes (*Pc35* and *Pc64*) and losing virulence to *Pc40* and *Pc51* (Table 1 & Fig. 1 and 10). The fact that virulence is not necessarily gained in stepwise adaptations, suggests that the combination of two effective genes may not necessarily provide durable resistance as was proposed by Simons *et al.* (according to Simons, 1985), who found a race that gained virulence to two genes without detecting virulence to the individual genes. The mutant pathotypes made it possible to postulate the following genes: *Pc39* was confirmed in Fidler (Chong & Kolmer, 1993), *Pc40* in Maluti and Wisconsin 1588, and *Pc51* in Euro, Overberg, OX88I 075-106, Perdeberg, and Swartberg (Table 7).

One of the reasons for the lack of pathogenic specialisation in the local crown rust population might be the historic cultivation of highly susceptible cultivars, as well as susceptibility in most, if not all, wild oat populations. Very little selection pressure for adaptation is thus exerted onto the oat crown rust population. The cultivars Drakensberg and Sederberg, both which have been cultivated for several years, are resistant to all variants detected, indicating that they contain resistance genes or combinations of genes other than those for which virulence was described. Continued monitoring of these cultivars as well as the newly released cultivars Kompasberg and Tafelberg is essential should shifts in virulence occur (Tables 7 & 8).

It is also possible that samples were not collected from a geographically representative area, that other variants occurring at low frequencies were not detected due to sampling

procedures, or that these pathotypes have low fitness in nature. Evidence in this regard was obtained in 1999 when a severe crown rust infection occurred on the *Pc64* line included in a disease nursery at Tygerhoek near Riviersonderend in the south Western Cape. Greenhouse trials confirmed virulence towards the *Pc64* gene while none of the other collections sampled in the immediate vicinity had virulence to this gene. It is also important to note that the differential set used in this study did not include all the designated resistance genes and that a larger set would possibly differentiate the crown rust population further. According to Al-Kherb *et al.* (1987), Schwartz identified 54 different pathotypes from 54 isolates using 60 differential lines. Consideration should thus be given to extending the current differential set to a manageable collection of resistance genes providing maximum information on pathogenic variation in *P. coronata* f. sp. *avenae* in South Africa.

During 1997 and 1998, four pathotypes of *P. graminis* f. sp. *avenae* were identified. Virulence was identified to the resistance genes *Pg1*, *Pg2*, *Pg4*, *Pg8*, *Pg9*, *Pg12*, *Pg15* and *Pga*, which indicated that only *Pg3*, *Pg10*, *Pg13*, and *Pg16* remain effective (Table 2). *Pg12* was not included in the differential set but is represented in the cultivar Kyto (Table 8) (Adhikari *et al.*, 1999). In 1925 Bailey identified race 2,3/1 in South Africa (Martens, 1985) while Verwoerd (1931b) identified to "froms" of *P. graminis* f. sp. *avenae* with "form 2" resembling the race identified by Bailey.

The 1997 survey was dominated by SAPga 1 (43.1%) and SAPga 4 (30.4%), whereas in 1998 SAPga 4 prevailed (67.7%). The common isolation of pathotype SAPga 4 can be explained partly by the widespread cultivation of the cultivar Sederberg (±80% of oat production) in the Western and south Western Cape. Sederberg carries the resistance gene *Pga* and thus acts as a selective host for this pathotype, leading to biased sampling. This was confirmed by the fact that 37% of the samples producing pathotype SAPga 4 were originally collected from Sederberg. Significant amounts of inoculum occur annually on this autumn-sown variety. These spores are then carried to the interior of the country during late winter by frontal weather systems moving in a north-easterly direction. Young oat plants in the summer rainfall area are subsequently infected, adding to the widespread distribution of SAPga 4. However, further studies

providing long term data are necessary to explain and understand the occurrence and distribution of oat stem rust pathotypes in South Africa.

According to the seedling reaction of cultivars to pathotypes SAPga 1, SAPga 2, SAPga 3 and SAPga 4, the following resistance genes were postulated: *Pga* in Alpha, OX87 080-1, OX88I 075-106, Sederberg and W94/4; *Pg2* and/or *Pg4* in Echidna, Euro, Langeberg, Perdeberg, Potberg and Swartberg; and *Pg9* in OX86 048-246, OX87 072-13, Pallinup and Victorian. According to Harder *et al.* (1995) and Rothman (1984), Wisconsin 1588 possesses resistance genes *Pg2*, *Pg4* and *Pg10*. The data presented in this study show *Pg10* to be highly effective (Table 2) while Wisconsin 1588 is susceptible to pathotypes SAPga 2, SAPga 3 and SAPga 4 but resistant to SAPga 1 (Table 8). At present this discrepancy cannot be explained and more tests with different seed stocks are necessary to interpret the *Pg10* and Wisconsin 1588 responses.

The information and oat rust pathotypes provided in this study should enable cereal pathologists and breeders to screen breeding material more comprehensively for resistance to *P. coronata* f. sp. *avenae* and *P. graminis* f. sp. *avenae*. It is anticipated that this study will form the basis of selecting and releasing rust resistant oat cultivars in South Africa, and that the severe yield and quality losses currently experienced will be minimised. It should be emphasised, however, that continual monitoring of these pathogens are necessary to provide meaningful contributions to oat rust control.

REFERENCES

Adhikari, K.N., McIntosh, R.A. & Oates, J.D. 1999. Inheritance of stem rust resistant phenotype *Pg-a* in oats. Euphytica 105: 143-154.

Al-Kherb, S.M., Roelfs, A.P. & Groth, J.V. 1987. Diversity for virulence in a sexually reproducing population of *Puccinia coronata*. Canadian Journal of Botany 65: 994-998.

Berkeley, M.J. 1876. An enumeration of the fungi collected at the Cape of Good Hope

during the stay of the English transit of Venus expedition in 1874. Journal of Botany, London, Vol XIV (new series Vol. V): 173-175.

Chong, J. & Kolmer, J.A. 1993. Distribution and virulence of *Puccinia coronata* f. sp. *avenae* in Canada in 1992. Canadian Journal of Plant Pathology 16: 64-67.

Doidge, E.M. 1927. Preliminary study of South African rust fungi. Bothalia 2: 1-228.

Doidge, E.M., Bottomley, A.M., Van der Plank, J.E. & Pauer, G.D. 1953. A revised list of plant diseases in South Africa. Union of South Africa Department of Agriculture, Science Bulletin 346: 62.

Engelbrecht, W. 1999. Report presented at SANSOR (South African National Seed Organisation) meeting 12-14 May 1999.

Evans, I.B.P. 1911. South African cereal rusts, with observations on the problem of breeding rust-resistant wheat. Journal of Agricultural Science 4: 95-104.

Harder, D.E., Chong, J. & Brown, P.D. 1995. Stem and crown rust resistance in the Wisconsin oat selection X1588-2. Crop Science 35: 1011-1015.

Kolmer, J.A. & Chong, J. 1993. Distribution of virulence in two populations of *Puccinia coronata* f. sp. *avenae* in Canada. Canadian Journal of Botany 71: 946-950.

Martens, J.W. 1978. Stem rust of oats in Canada in 1977. Canadian Plant Disease Survey 58: 51-52.

Martens, J.W. 1985. Oat stem rust. Pages 103-129 in: The Cereal Rusts. Vol 2. A.P. Roelfs and W.R. Bushnell, eds. Academic Press, New York.

Martens, J.W., Roelfs, A.P., McKenzie, R.I.H., Rothman, P.G., Stuthman, D.D. & Brown, P.D. 1979. System of nomenclature of races of *Puccinia graminis* f. sp.

avenae. Phytopathology 69: 293-294.

O'Donoughue, L.S., Kianian, S.F., Rayapati, P.J., Penner, G.A., Sorrels, M.E., Tanksley, S.D., Phillips, R.L., Rines, H.W., Lee, M., Fedak, G., Molnar, S.J., Hoffman, D., Salas, C.A., Wu, B., Autrique, E. & Van Deynze, A. 1995. A molecular linkage map of cultivated oat. Genome 38: 368-380.

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

Pretorius, Z.A., Van Niekerk, B.D. & Boshoff, W.H.P. 1998. Pathogenic variation in *Puccinia coronata* f. sp. *avenae* in South Africa. Abstracts of the 36th Annual Congress of the Southern African Society for Plant Pathology, Champagne Castle Sports Resort. South African Journal of Science 94, 11: xx.

Roelfs, A.P. 1988. Genetic control of phenotypes in wheat stem rust. Annual Review of Phytopathology 26: 351-367.

Rothman, P.G. 1984. Registration of four stem rust and crown rust resistant oat germplasm lines. Crop Science 24: 1217-1218.

Sawer, E.R. 1909. The cereals in South Africa. Cedara Memoirs of South African Agriculture 1: 264-272.

Šebesta, J., Zwartz, B., Roderick, H.W., Harder, D.E., Corazza, L. & Stojanovic, S. 1998. Incidence of oat stem rust and virulence of *Puccinia graminis* Pers. f. sp. *avenae* Erikss. et Henn. on oat and the effectiveness of resistance in Europe during 1988 to 1996. Archives of Phytopathology and Plant Protection 31: 393-413.

Simons, M.D. 1985. Crown rust. Pages 131-172 in: The Cereal Rusts. Vol 2. A.P. Roelfs and W.R. Bushnell, eds. Academic Press, New York.

Smit, H.A. 1993. Die verdraagsaamheid van wildehawer vir onkruiddoders (The tolerance of wild oat to herbicides). Ph.D. Thesis. University of the Orange Free State, South Africa.

Verwoerd, L. 1929. A preliminary checklist of diseases of cultivated plants in the winter rainfall area of the Cape province. Union of South Africa Department of Agriculture Science Bulletin 88: 1-28.

Verwoerd, L. 1931 (a). A preliminary list of fungi from the Bloemfontein district of the Orange Free State. South African Journal of Science XVIII: 298-301.

Verwoerd, L. 1931 (b). Die fisiologiese vorms van *Puccinia graminis* Pers. wat in Suid-Afrika voorkom. South African Journal of Science 28: 274-279.

Zadoks, J.C., Chang, T.T. & Konzak, C. F. 1974. A decimal code for the growth stages of cereals. Weed Research 14: 415-421.

YIELD LOSSES CAUSED BY BARLEY LEAF RUST AND OAT LEAF AND STEM RUST IN SOUTH AFRICA

INTRODUCTION

Both barley (*Hordeum vulgare* L.) and oats (*Avena sativa* L.) are important cereal crops in South Africa with the potential of being grown over approximately 130 000 ha and 700 000 ha, respectively (Engelbrecht, 1999). Barley production is restricted to the south Western Cape (winter rainfall region) where the crop is grown mainly for malting purposes.

According to a survey conducted by the Small Grain Institute amongst agricultural co-operatives, only 8.6% of the annual oat crop is produced for grain whereas 76.4% is used as grazing and 15% as silage. In the Western Cape oats is grown almost exclusively for silage and occasionally for grain production. In irrigation areas oats is planted as a rotation crop with wheat to reduce the incidence of take-all (*Gaeumannomyces graminis* Sacc. Arx & Oliv. Var *tritici* Walker.). Oat production for grazing occurs mainly on the South African highveld where summer rainfall prevails. In the winter rainfall area a mixture of barley and oats are planted occasionally for silage.

Until 1970, leaf rust of barley, caused by the fungus *Puccinia hordei* Otth., was considered economically unimportant internationally (Clifford, 1985). More recently it has increased in importance with severe epidemics occurring more regularly (Clifford, 1985) have qualified leaf rust as an important disease of barley in several areas of the world (Alemayehu & Parlevliet, 1996; Borovkova *et al.*, 1997). Teng (according to Lim & Gaunt, 1986) concluded that *P. hordei* must be regarded as a serious potential source of yield loss and Cotterill *et al.* (according to Cotterill *et al.*, 1994) recorded yield losses of 26 to 31% in Australia during a moderate to severe epidemic in 1990. Yield losses as high as 40% have been reported by Jenkins *et al.* (1972), whereas Griffey *et al.* (1994) reported total devastation of barley nurseries in Virginia when *P. hordei* reached epidemic proportions prior to heading stage.

Crown (leaf) rust, caused by the fungus *Puccinia coronata* Corda f. sp. *avenae* Eriks., is considered the most important fungal disease of cultivated oat (Simons, 1985) while stem rust, caused by *Puccinia graminis* Pers. f. sp. *avenae* Eriks. and E. Henn., occurs in most of the oat growing areas of the world, periodically causing severe crop losses (Martens, 1985). Šebesta *et al.* (1998) considered oat stem rust to be a potentially more destructive disease than crown rust. Potentially the South African market for oat grain could be increased signifcantly if test weight could be improved. Since all South African oat cultivars, except Drakensberg, are susceptible to either crown or stem rust, it is thought that low test weights may partly be due to these diseases.

In South Africa the barley cultivar Clipper dominated production since the 1960s. Before the release of SSG 532 in 1997 Clipper contributed approximately 95% of the total production. Fleischmann & McKenzie (1965) noticed that losses caused by certain diseases are often obscured by those with more striking symptoms and impact. Such minor diseases will receive attention only after the major diseases have been controlled. A similar situation occurred in South Africa where the susceptibility of Clipper to scald (*Rhynchosporium secalis* Oud.) resulted in most control measures being focused on this disease. Furthermore, Fleischmann & McKenzie (1965) stressed the importance of loss assessment due to plant diseases since it is vital in emphasising the practical need for research.

Due to the lack of scientific data on losses caused by rust diseases of barley and oat in South Africa, the influence of these diseases on economically important parameters was investigated.

MATERIALS AND METHODS

During 1998 six oat cultivars (Table 1), four barley cultivars and two advanced lines (Table 2) were planted in yield loss experiments at the Small Grain Institute, Bethlehem. A plot seeder was used to sow oats at a seeding rate of 90 kg ha^{-1} and barley at 100

Table 1. The mean percentage decrease in yield, hectolitre mass, and thousand kernel mass of oat infected with *Puccinia coronata* and *Puccinia graminis* f. sp. *avenae*

Culitvar	Percentage decrease in			AUDPC for		
	Yield	Hecolitre mass	Thousand kernel mass	Stem rust	Crown rust	Crown & Stem rust
Drakensberg	25.24 [c]	6.49 [c]	23.66 [b]	0.00 [e]	0.00 [e]	0.00 [e]
Sederberg	47.24 [c]	15.53 [b]	24.74 [b]	682.50 [c]	19.00 [d]	701.33 [d]
Overberg	77.22 [b]	38.27 [a]	32.29 [b]	1207.25 [a]	1600.52 [b]	2807.77 [b]
Euro	85.02 [a]	39.75 [a]	42.03 [a]	920.75 [b]	2415.25 [a]	3336.00 [a]
Echidna	84.76 [a]	38.26 [a]	44.83 [a]	356.00 [d]	2356.75 [a]	2712.75 [b]
Witteberg	83.05 [a]	45.01 [a]	35.37 [a]	581.25 [c]	1165.25 [c]	1746.50 [c]

[a] Means in column with different letters differ significantly at p=0.05.

Table 2. The mean percentage decrease in yield, kernel plumpness, thousand kernel mass, nitrogen content and increase in percentage siftings of barley due to *Puccinia hordei* infection

Cultivar	Percentage decrease in				% Siftings increase	AUDPC
	Yield	Kernel plumpness	Nitrogen content	Thousand kernel mass		
Clipper	38.82 [b]	43.95 [b]	8.27 [a]	4.95 [b]	188.27 [c]	1220.25 [a]
Stirling	49.44 [a]	45.91 [b]	7.97 [a]	9.26 [a]	589.98 [a]	1232.75 [a]
SSG 525	58.34 [a]	61.00 [a]	10.65 [a]	8.05 [a]	350.00 [b]	1255.75 [a]
SSG 532	4.79 [d]	21.02 [c]	-2.09 [b]	4.29 [b]	123.13 [c]	85.00 [b]
B94/2	23.33 [c]	7.89 [d]	-3.02 [b]	2.41 [b]	-17.16 [d]	112.75 [b]
B95/10	20.20 [c]	25.21 [e]	-0.59 [b]	6.60 [a]	195.65 [c]	257.43 [b]

[a] Means in column with different letters differ significantly at p=0.05.

kg ha^{-1}. Each plot consisted of two 5-m rows spaced 17 cm apart. Both the oat and barley trials were arranged according to a split-plot design with rust-infected and fungicide-sprayed treatments as main plots per block, and cultivars as subplots. Blocks were replicated four times. Each trial was bordered by four rows of appropriate rust susceptible spreaders planted three weeks earlier. Prior to planting fertiliser was placed in the soil below the designated rows at the respective rates of 50 kg N, 33 kg P and 33 kg K per hectare. The broad-leaf herbicide bromoxynil (450 g a.i ha^{-1}) and demeton-S-methyl (125 g a.i. ha-^{1}) aphicide were applied when necessary. Grass weeds were removed manually when necessary.

When spreaders reached growth stage 36 (Zadoks *et al.*, 1974), tillers were injected with freshly collected urediniospores suspended in water (Roelfs *et al.*, 1992). In the case of oats, inoculum consisted of equal amounts of crown and stem rust urediniospores. Crown rust was represented by pathotype SAPc 1 while the stem rust inoculum consisted of a mixture of SAPga 1, SAPga 2, SAPga 3 and SAPga 4 in equal amounts. Barley spreaders were inoculated in a similar fashion with pathotype SAPh 3231. On the day of inoculation all control blocks were sprayed with Folicur™ (tebuconazole) at 187.5 g a.i ha^{-1}. To ensure disease free control plants, this treatement was repeated every three weeks. After the appearance of the first infected leaves, spreading of infection to plots was encouraged by regular mist irrigation at dusk. Disease was assessed every five days according to the description of Stubbs *et al.* (1986) until foliage was decimated by rust or had reached senescence naturally. Area under the disease progress curve (AUDPC) was then calculated for each cultivar (Campbell & Madden, 1990).

Plots were harvested using a small-plot combine. Grain from each plot was dried to 12% moisture content before being weighed. Thousand-kernel mass and test weight were determined for oats while thousand kernel mass, kernel plumpness and percentage siftings as well as percentage nitrogen were determined for barley. Barley kernel size and percentage siftings were determined by sieving 100 g of rubbed seed through two screens with 2.5 mm and 2.2 mm perforations, respectively. Seed remaining in the 2.5 mm sieve were weighed and expressed as the percentage plump

kernels, whereas those passing through the 2.2 mm sieve were regarded as siftings. Thousand kernel mass was determined on plump barley kernels only. The percentage nitrogen content was determined at the quality laboratory of the Small Grain Institute. Statistical analyses were done using Agrobase 98 with the exception of regression analyses which were conducted using Microsoft PowerPoint.

RESULTS

Barley

Barley leaf rust infection of experimental entries started at flag leaf emergence (Zadoks 37) and increased rapidly to reach epidemic levels of up to 100S severity at flowering. Sprayed plots were completely free of leaf rust and no diseases other than *P. hordei* were observed. AUDPC varied significantly among cultivars and lines, with Clipper, Stirling and SSG 525 being classified as highly susceptible, B95/10 and B94/2 as moderately resistant, and SSG 532 as resistant (Fig. 1 & Table 1).

The high disease incidence resulted in a significant reduction in yield in all cultivars except SSG 532 (Fig. 2, Table 1 & App. A), with losses as high as 58% in the cultivar SSG 525. Regression analysis indicated the percentage yield loss to be relatively well correlated ($R^2 = 0.7216$) with disease severity (Fig. 3). With the exception of B94/2, leaf rust significantly reduced kernel plumpness and significantly increased the percentage siftings in all cultivars (Figs. 4,5, Table 1, & App. A). Losses amounting to 61% were recorded in kernel plumpness in the cultivar SSG 525 with an increase of as high as 590% in the percentage siftings in Stirling (Table 1). Decrease in kernel plumpness was well correlated ($R^2 = 0.7392$) with the AUDPC while increase in percentage siftings was not ($R^2 = 0.427$) (Figs. 6 & 7). Similar to kernel plumpness, thousand kernel mass was significantly reduced in all cultivars except B94/2. Reductions varied between 2 and 9% (Fig. 8 & App. A). Percentage thousand kernel mass reduction was poorly correlated ($R^2 = 0.3028$) with disease severity (Fig. 9). Nitrogen content, on the other hand, was significantly reduced in all susceptible cultivars whereas it was slightly higher in the case of moderately resistant and resistant cultivars, although not

Figure 1. Area under the disease progress curve (AUDPC) of barley cultivars Clipper, Stirling, SSG 525, SSG 532 and advanced lines B94/2 and B95/10 infected with *P. hordei* .
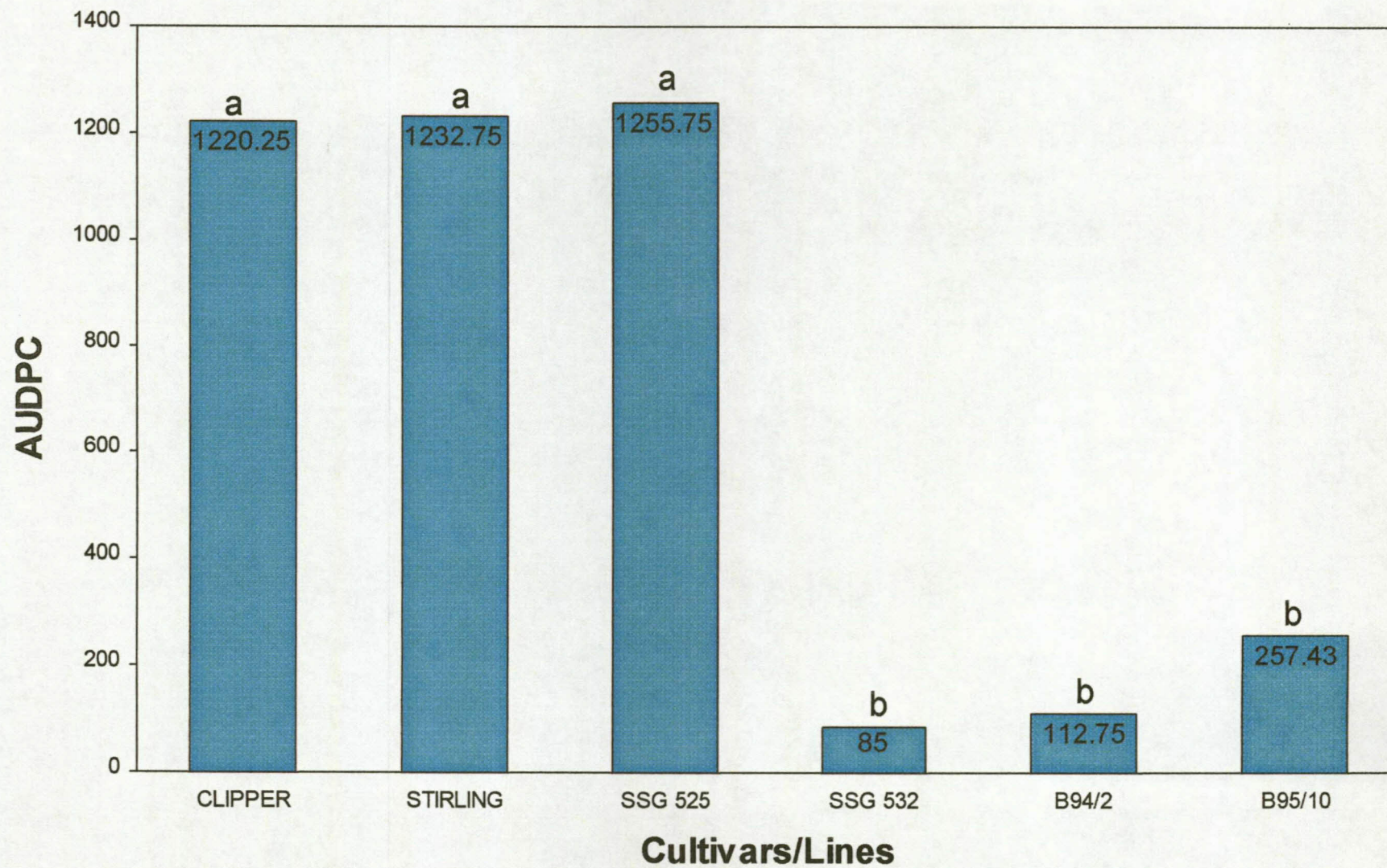
Figure 2. Mean yield of barley cultivars Clipper, Stirling, SSG 525, SSG 532 and advanced lines B94/2 and B95/10 in sprayed and unsprayed plots exposed to leaf rust infection.
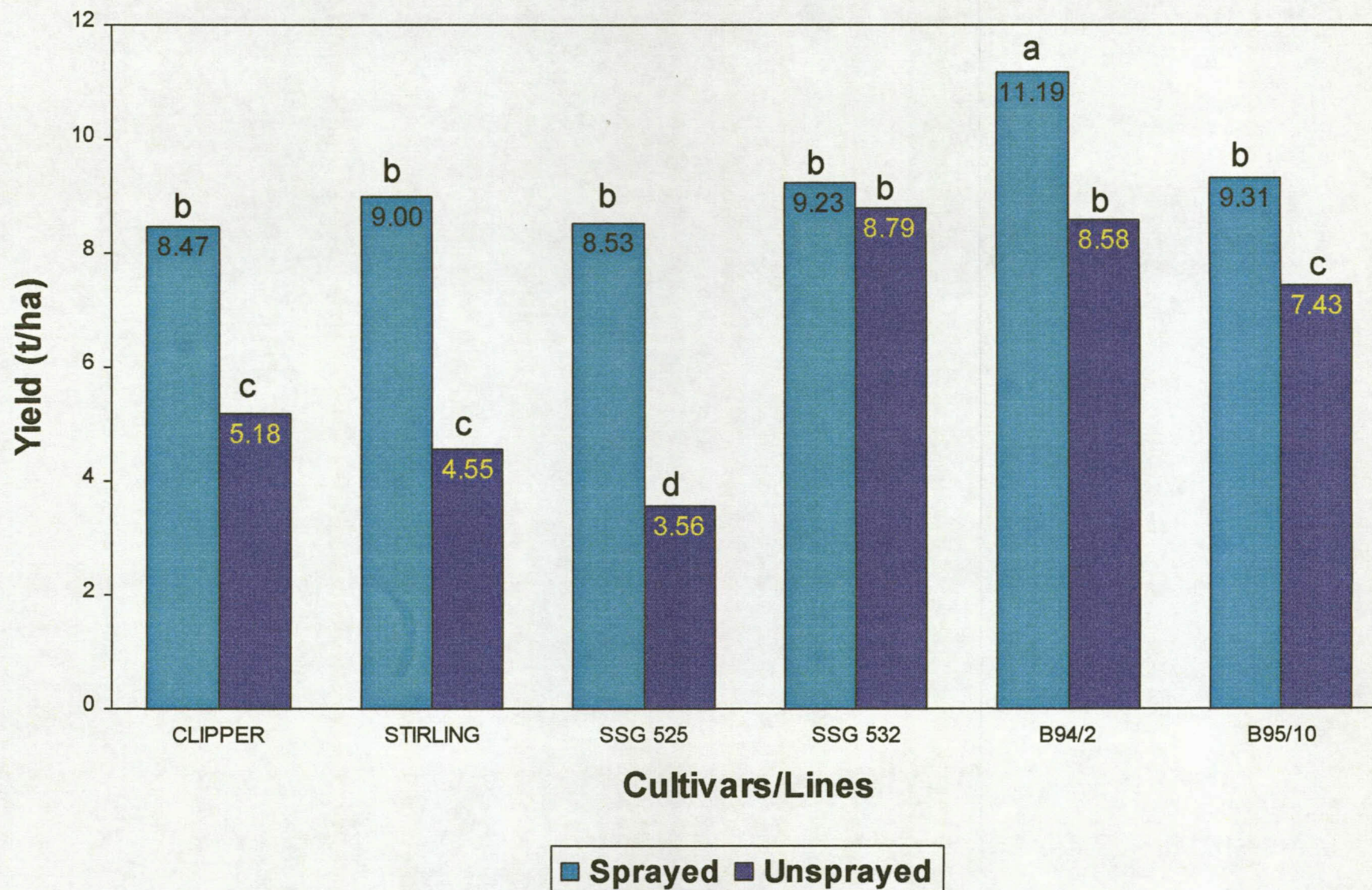
Figure 3. Regression analysis of percentage yield reduction in barley due to *P. hordei* infection, expressed as area under the disease progress curve (AUDPC).

$y = 0.0297x + 11.537$
$R^2 = 0.7216$

Figure 4. Mean kernel plumpness of barley cultivars Clipper, Stirling, SSG 525, SSG 532 and advanced lines B94/2 and B95/10 in sprayed and unsprayed plots exposed to leaf rust infection.

Figure 5. Mean percentage siftings in barley cultivars Clipper, Stirling, SSG 525, SSG 532 and advanced lines B94/2 and B95/10 in sprayed and unsprayed plots exposed to leaf rust infection.

Figure 6. Regression analysis of the percentage decrease in barley kernel plumpness due to *P. hordei* infection, expressed as area under the disease progress curve (AUDPC).

Figure 7. Regression analysis of the percentage increase in siftings in barley due to *P. hordei* infection, expressed as area under the disease progress curve (AUDPC).

The chart shows:
- Y-axis: **Percentage increase in siftings**, ranging 0 to 900
- X-axis: **AUDPC**, ranging 0 to 1600

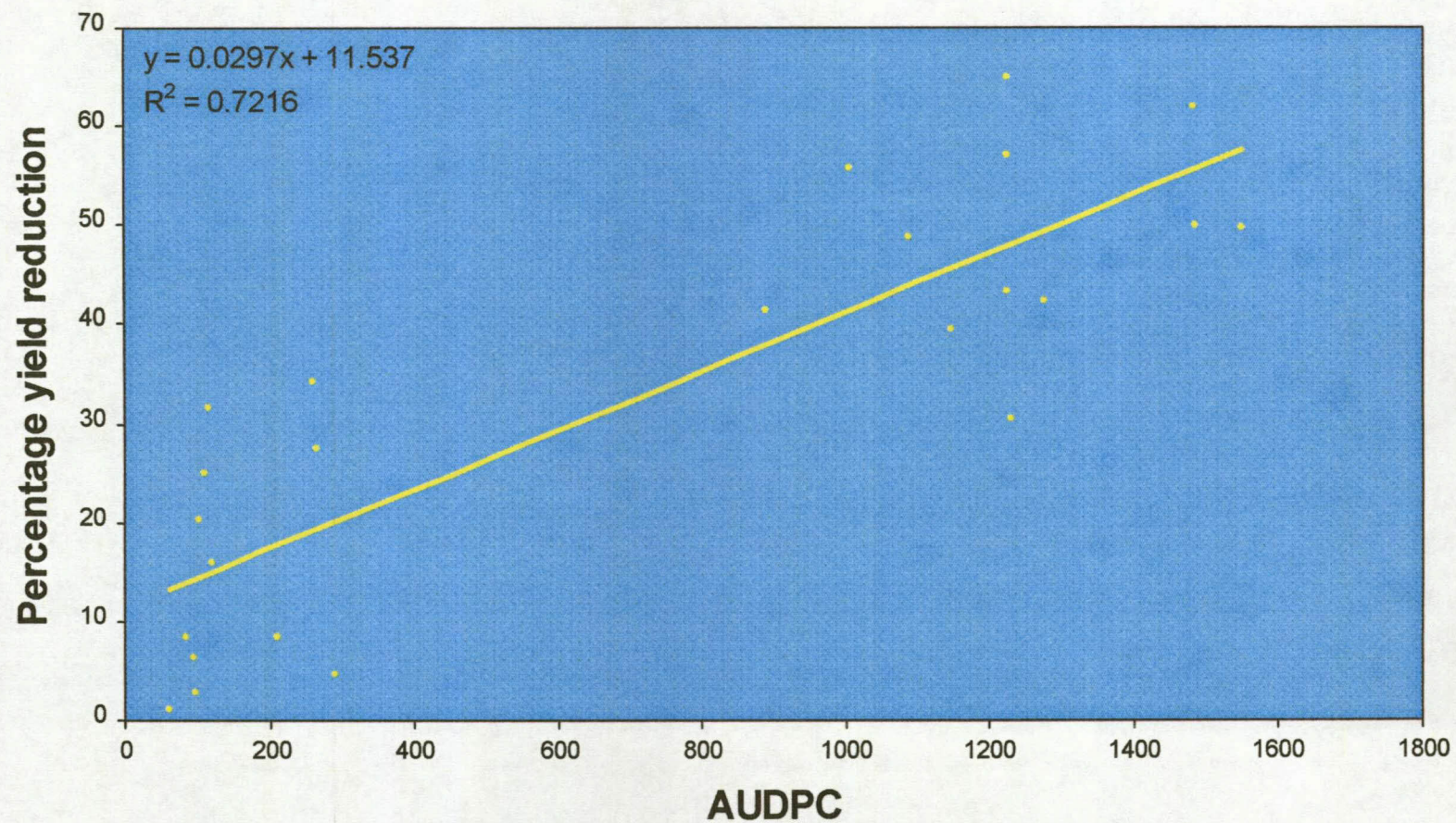$$y = -0.0004x^2 + 0.8883x - 8.5208$$
$$R^2 = 0.427$$

Figure 8. Mean thousand kernel mass of barley cultivars Clipper, Stirling, SSG 525, SSSG 532 and advanced lines B94/2 and B95/10 in sprayed and unsprayed plots exposed to leaf rust infection.
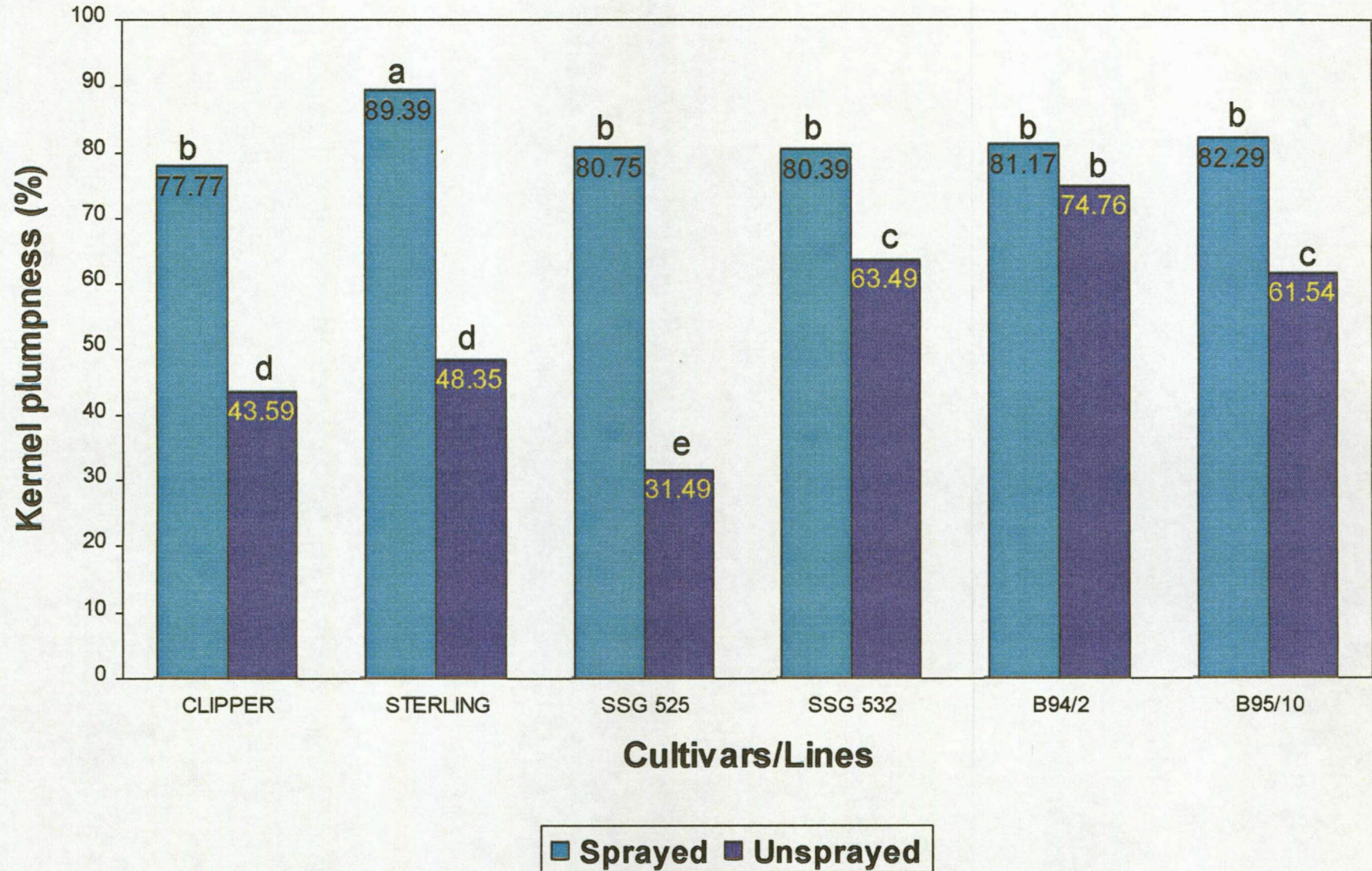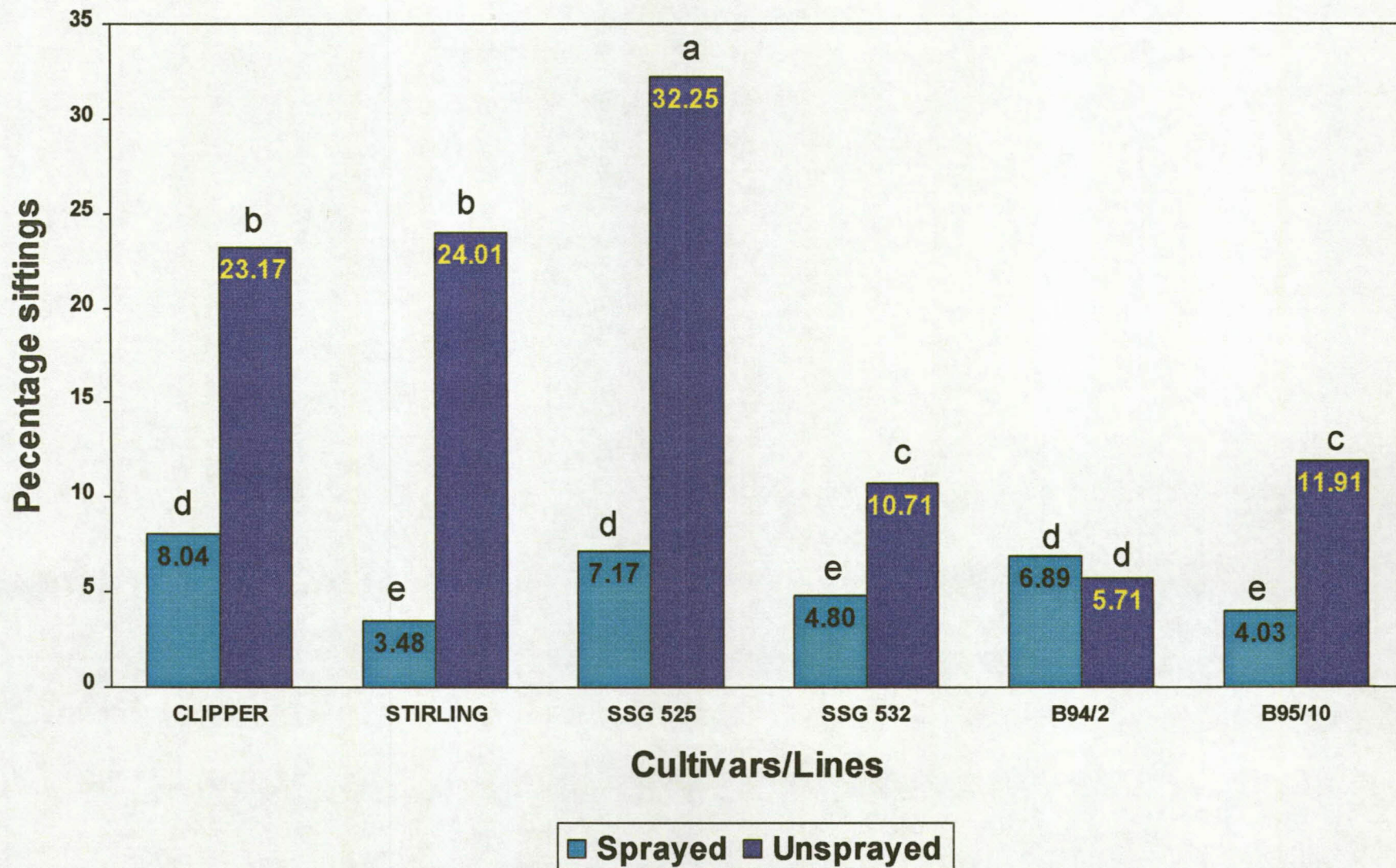
Figure 9. Regression analysis of percentage decrease in thousand kernel mass in barley due to *P. hordei* infection, expressed as area under the disease progress curve (AUDPC).
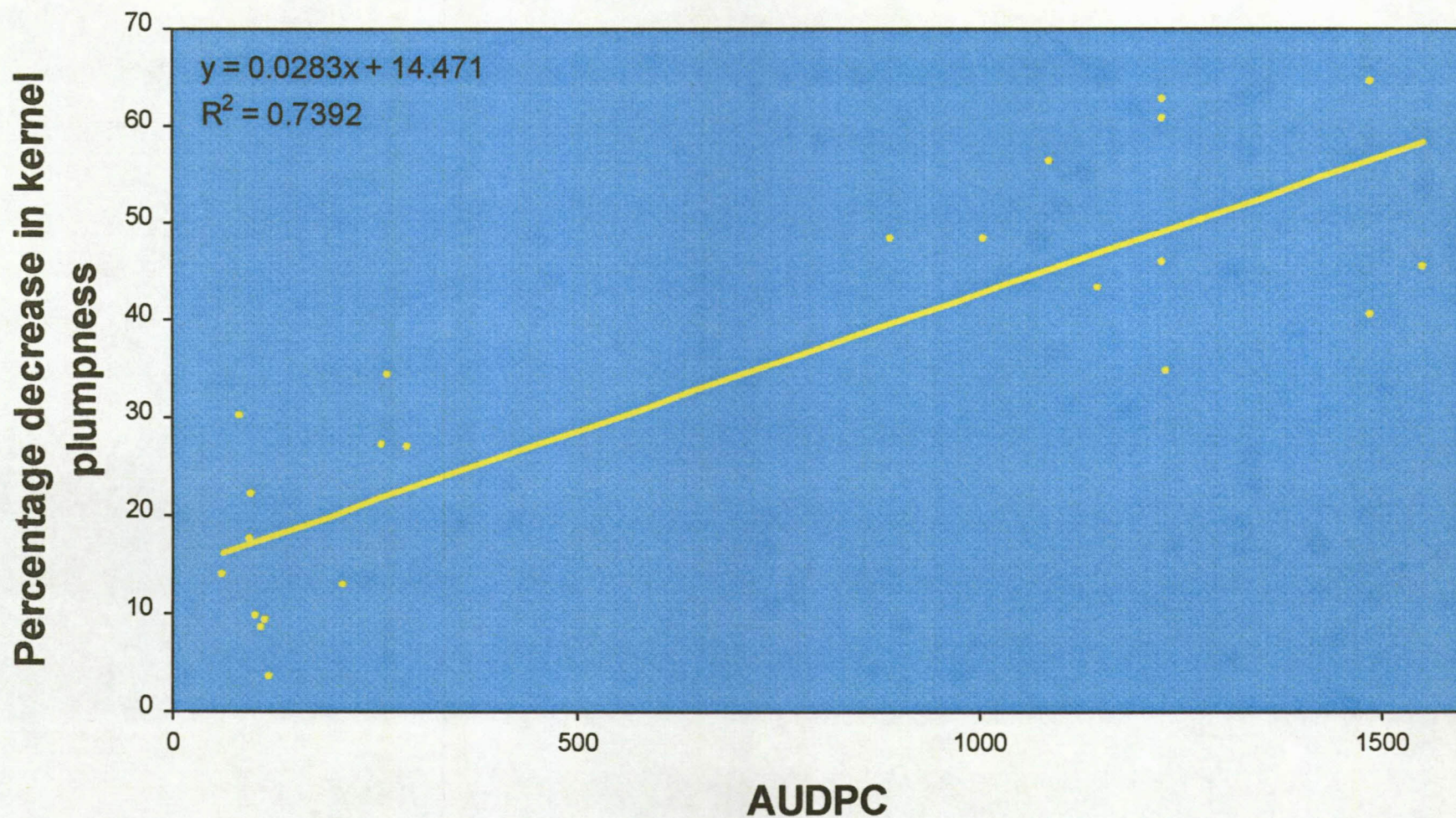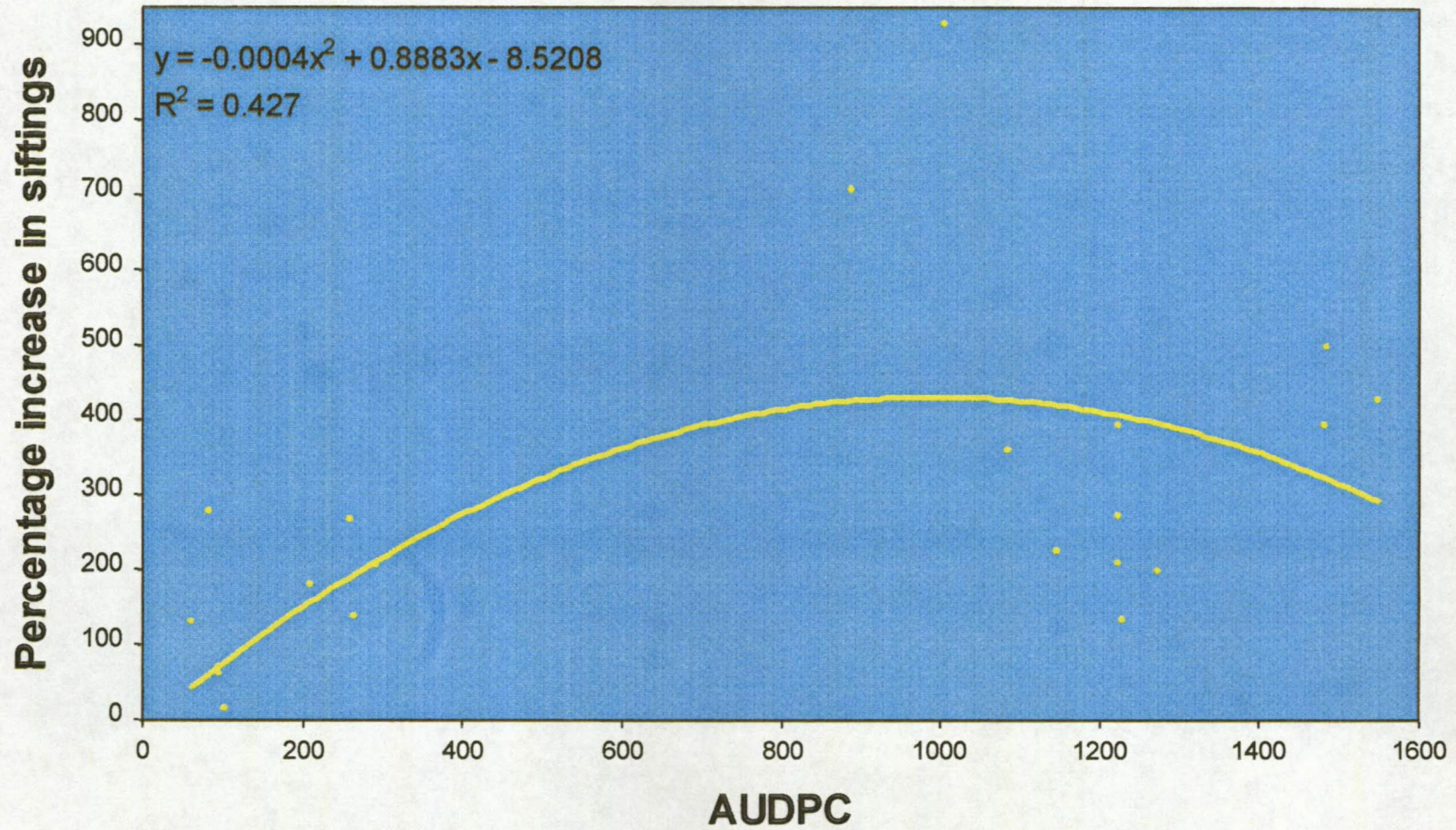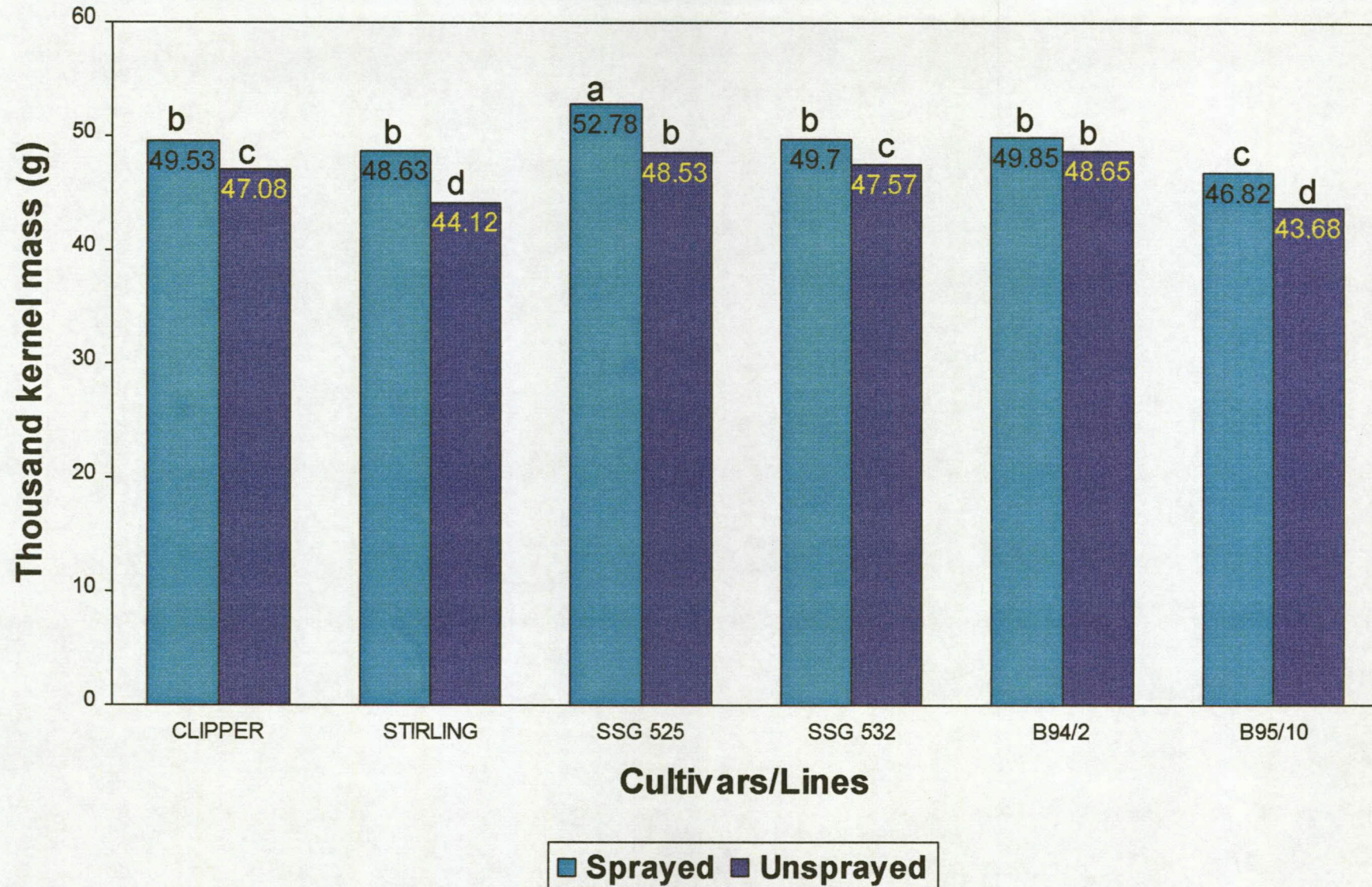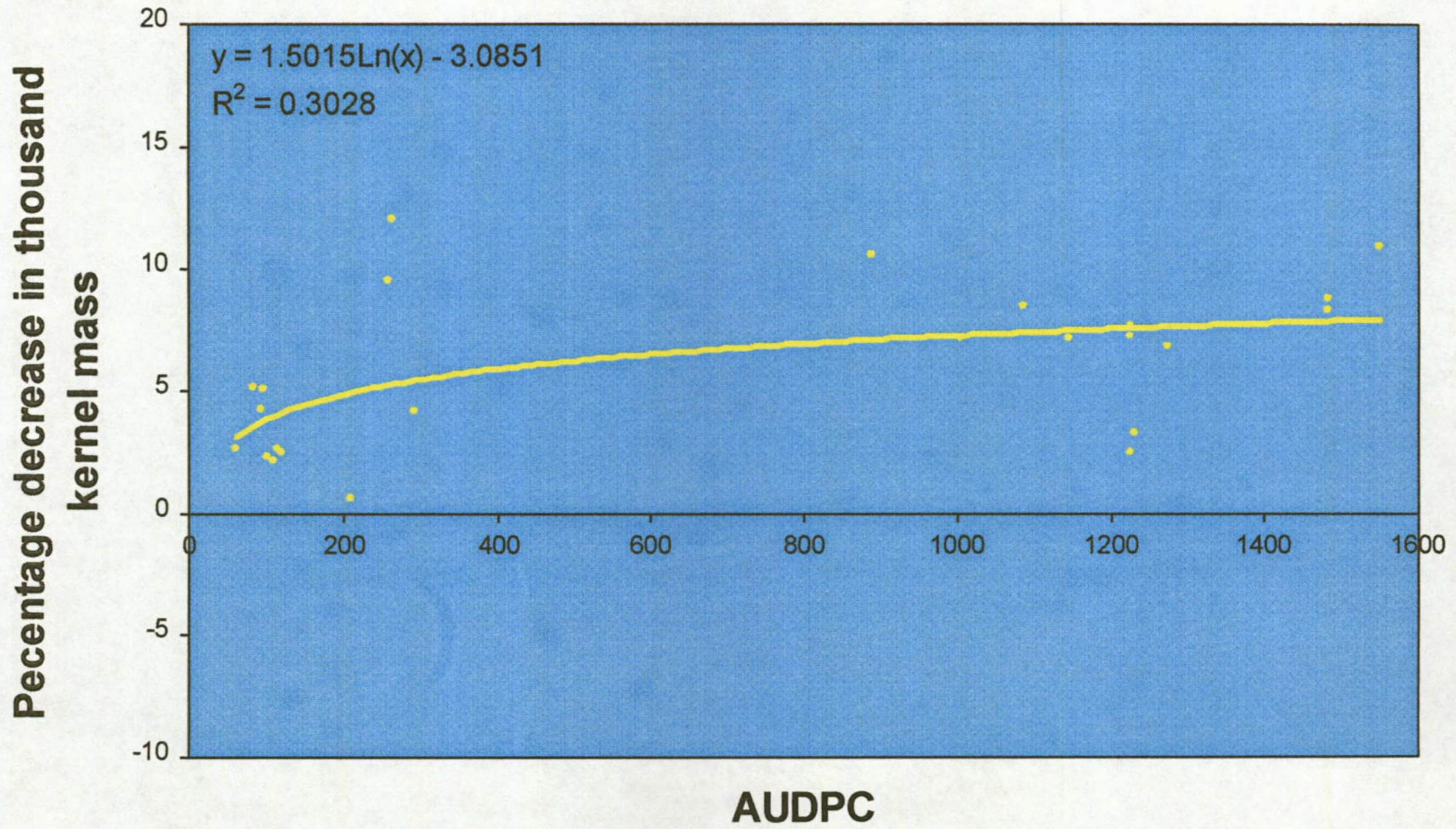
significantly so (Fig. 10 and App A). Regression analysis showed the percentage reduction in nitrogen content to be poorly correlated (R^2 = 0.5006) with disease incidence (Fig. 11). Correlation matrix analysis showed that yield was positively correlated with kernel plumpness (CC = 0.87) and unexpectedly also percentage nitrogen (CC = 0.69) and negatively correlated with percentage siftings (App. C).

Oats

The onset of crown rust infection was early, reaching epidemic proportions at plume emergence (Zadoks 50) while stem rust infection and progress were not as rapid, only reaching epidemic proportions later in the season (Zadoks 86). The fungicide treated plots were disease-free and no diseases other than crown and stem rust occurred on the unsprayed plots. For both crown and stem rust highly significant differences were found between treatments, cultivars and their interactions (App. B). The cultivars Euro and Echidna were extremely susceptible to crown rust, whereas Overberg and Euro were most affected by stem rust (Fig. 12). Sederberg was resistant to crown rust but susceptible to stem rust while Drakensberg was resistant to both crown and stem rust. In all analyses the combined (total) AUDPC value for stem and crown rust was used unless indicated otherwise.

The high disease pressure of especially crown rust led to a significant decrease in yield of all cultivars. Yield loss ranged between 25% in Drakensberg and 85% in Euro and Echidna (Fig. 13 & Table 2). Regression analysis showed percentage yield loss to be well correlated (R^2 = 0.8085) with disease severity (Fig. 14). Likewise, all cultivars showed a significantly lower hectolitre mass when crown and stem rust were not controlled (Fig. 15, Table 2 & App. B). The cultivar Witteberg was most affected with a reduction of 45% in hectolitre mass (Fig. 15 & Table 2). As with yield, the percentage reduction in hectolitre mass was also well correlated (R^2 = 0.816) with disease severity (Fig. 16). Thousand kernel mass were reduced with as much as 45% in the Echidna due to crown and stem rust infection. Similar as for yield and hectolitre mass, thousand kernel mass was significantly lower in all rusted plots (Fig. 17, Table 2 & App. B). The relationship between percentage thousand kernel decrease and disease severity was not as well correlate (R^2 = 0.5462) as the other two parameters (Fig. 18). Correlation

Figure 10. Mean percentage nitrogen content of barley cultivars Clipper, Stirling, SSG 525, SSG 532 and advanced lines B94/2 and B95/10 in sprayed and unsprayed plots exposed to leaf rust infection.
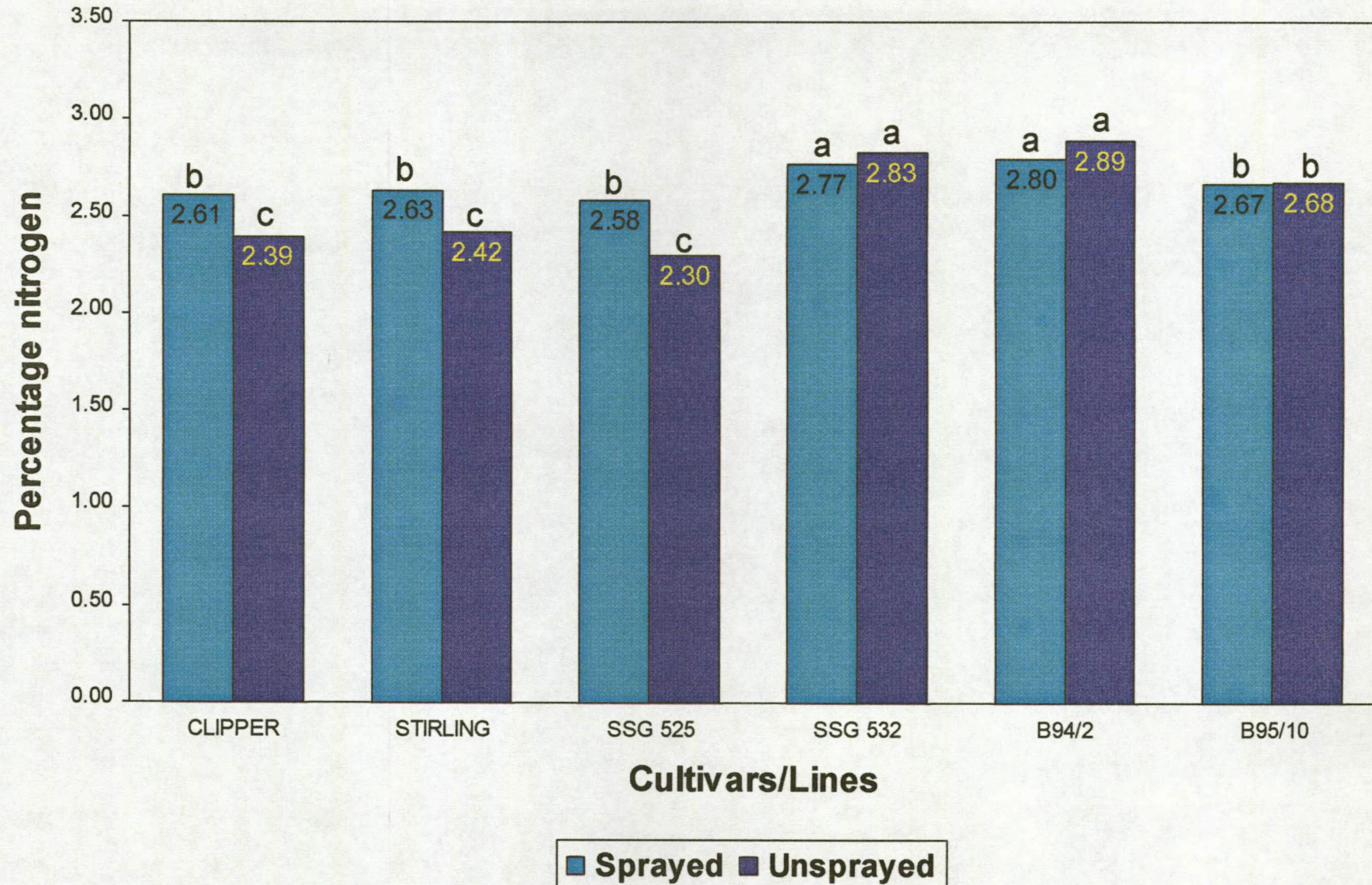
Figure 11. Regression analysis of percentage decrease in nitrogen content in barley due to *P. hordei* infection, expressed as area under the disease progress curve (AUDPC).

$$y = -1E\text{-}05x^2 + 0.0244x - 5.1262$$
$$R^2 = 0.5006$$
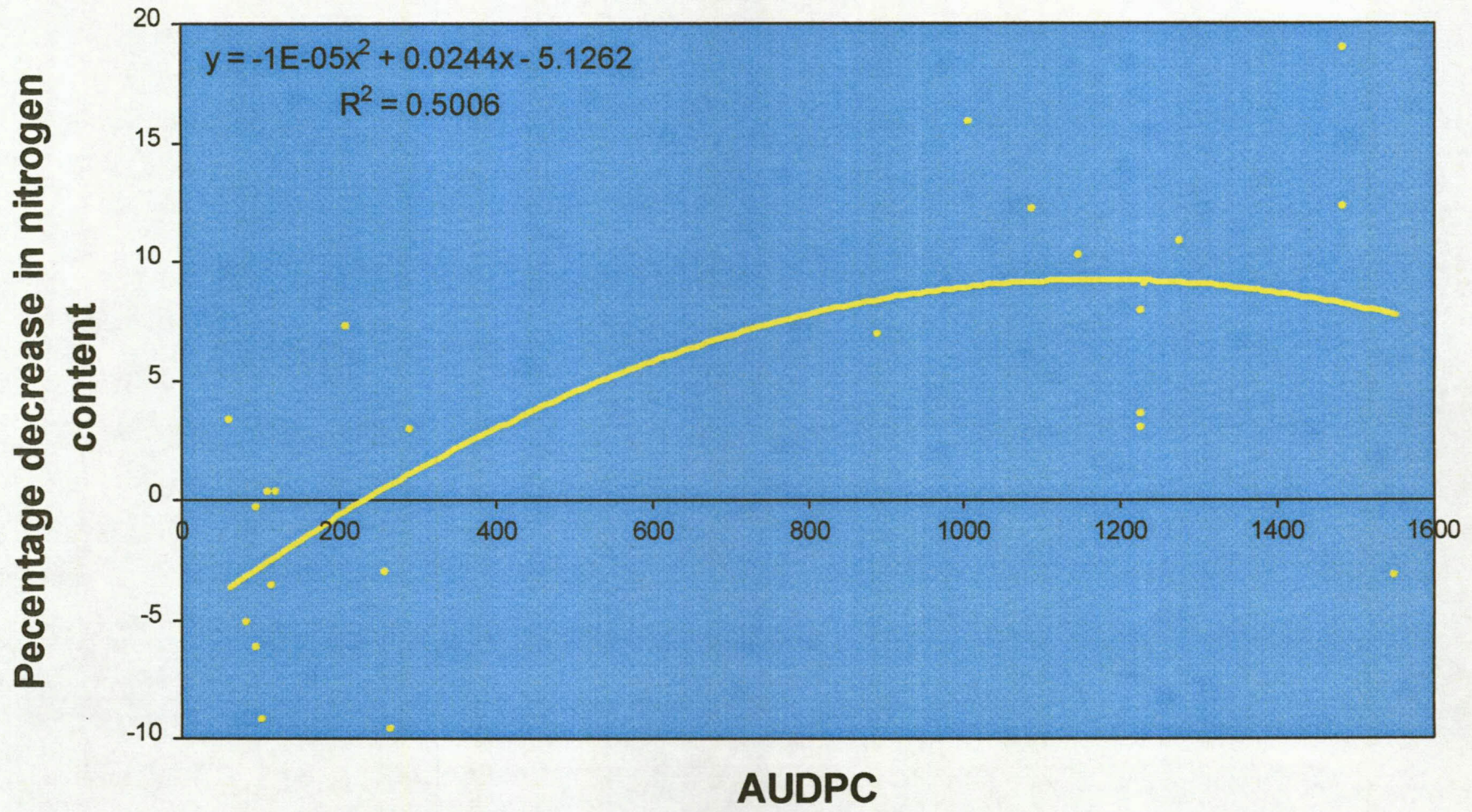
Figure 12. Area under the disease progress curve (AUDPC) of oat cultivars Drakensberg, Sederberg, Overberg, Euro, Echidna and Witteberg infected with *P. coronata* f. sp. *avenae* and *P. graminis* f.sp. *avenae*.
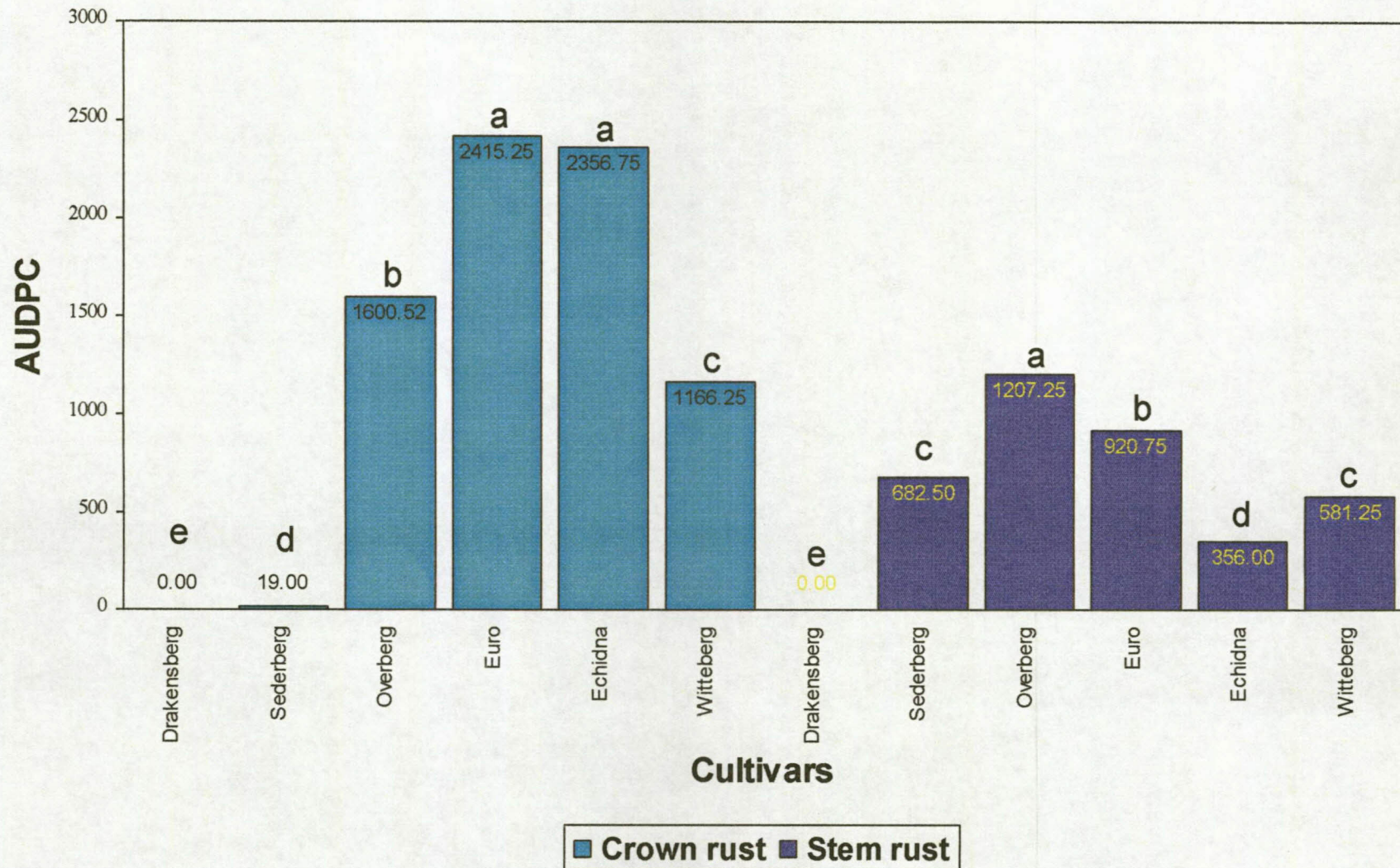
Figure 13. Mean yield of oat cultivars Drakensberg, Sederberg, Overberg, Euro, Echidna and Witteberg in sprayed and unsprayed plots exposed to leaf and stem rust infection.
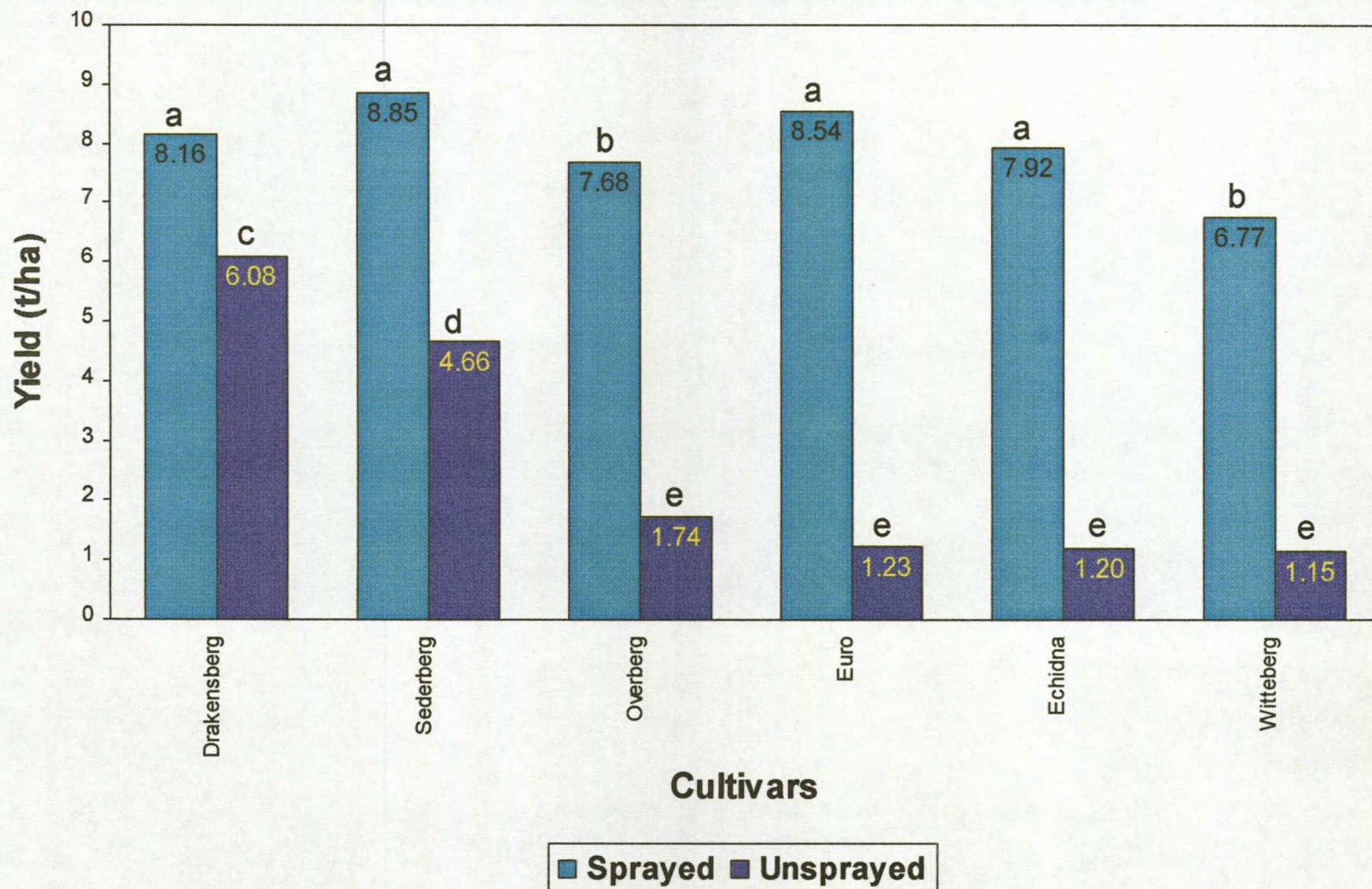
Figure 14. Regression analysis of percentage yield reduction in oats due to *P. coronata* f. sp. *avenae* and *P. graminis* f. sp. *avenae* infections, expressed as combined area under the disease progress curves (AUDPC).

$y = -8E\text{-}06x^2 + 0.0439x + 24.432$

$R^2 = 0.9381$
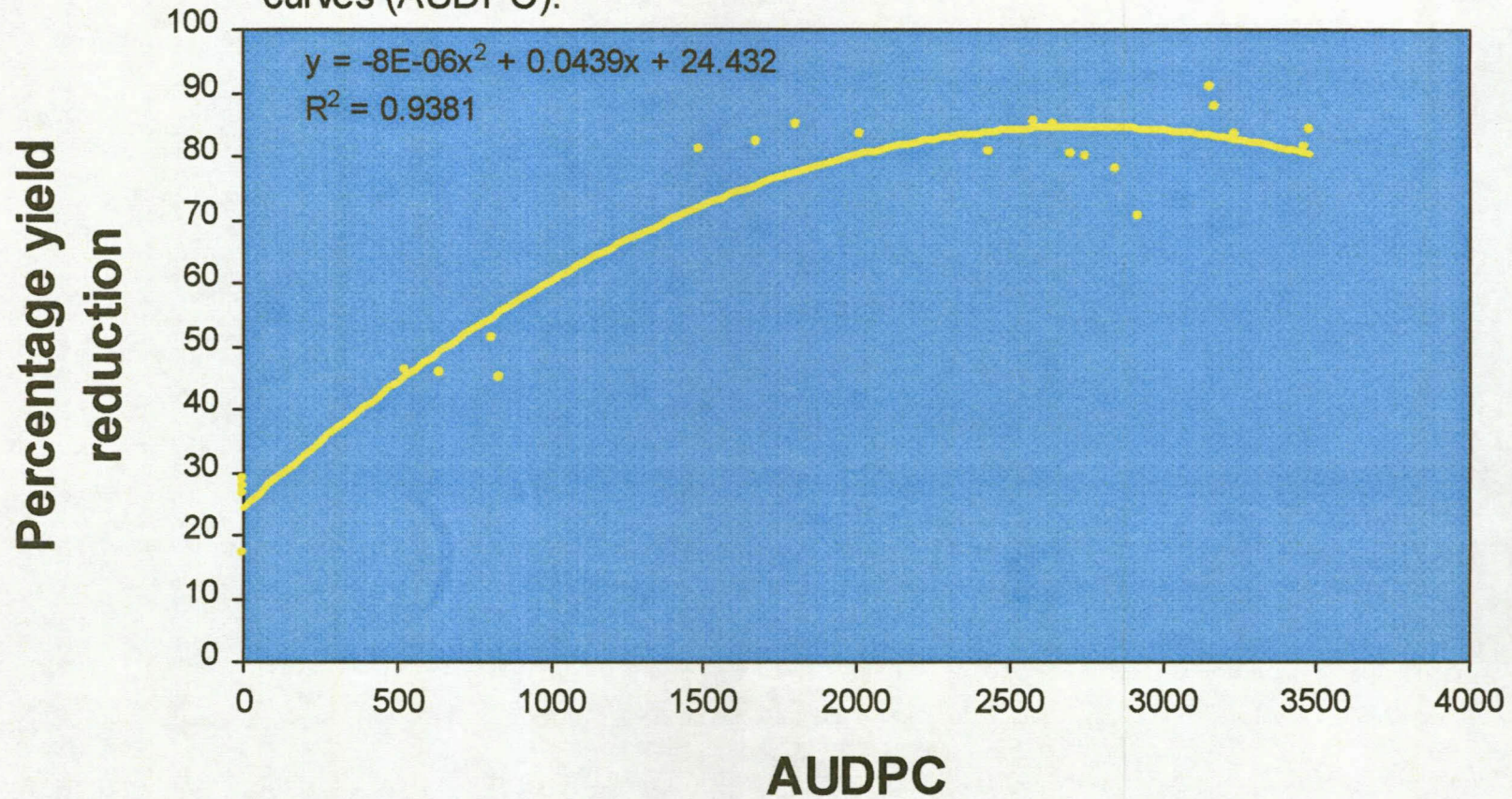
Figure 15. Mean hectolitre mass of oat cultivars Drakensberg, Sederberg, Overberg, Euro, Echidna and Witteberg in sprayed and unsprayed plots exposed to leaf and stem rust infection.
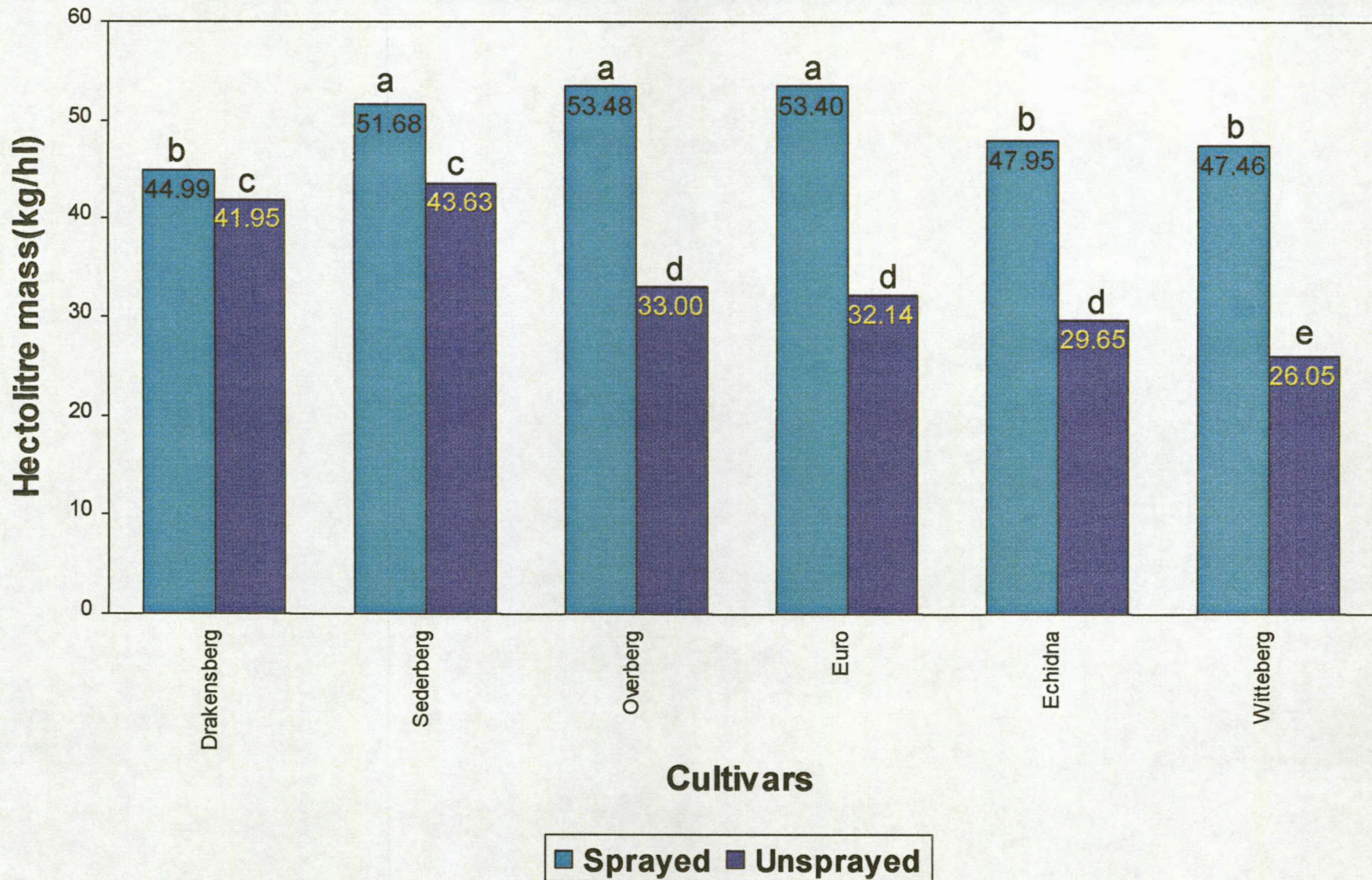
Figure 16. Regresion analysis of percentage decrease in oat hectolitre mass due to *P. coronata* f. sp. *avenae* and *P. graminis* f. sp. *avenae* infections, expressed as combined area under the disease progress curves (AUDPC).

$y = -5E-06x^2 + 0.0269x + 4.7591$

$R^2 = 0.816$
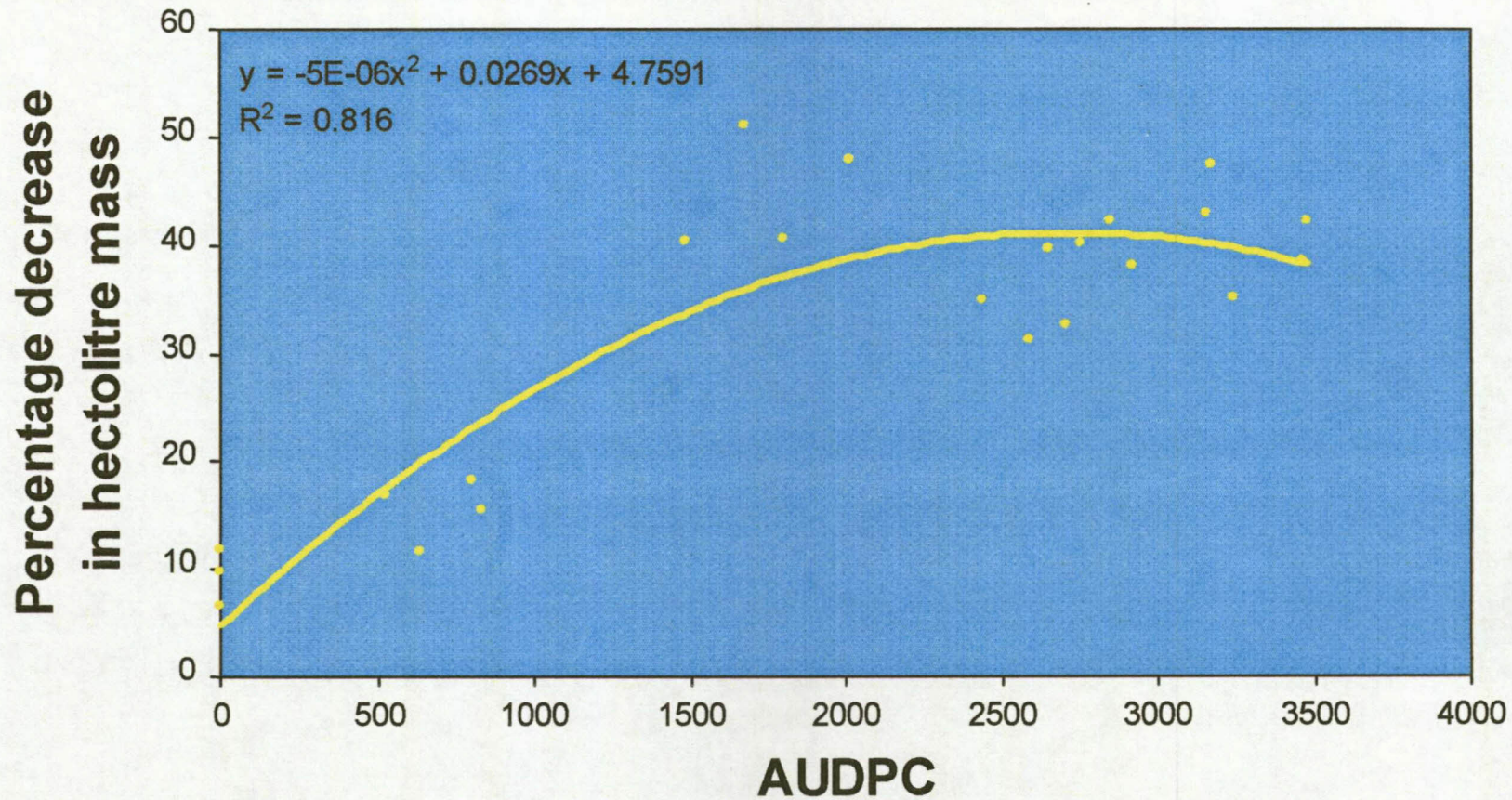
Figure 17. Mean thousand kernel mass of oat cultivars Drakensberg, Sederberg, Overberg, Euro, Echidna, and Witteberg in sprayed and unsprayed plots exposed to leaf and stem rust infection.
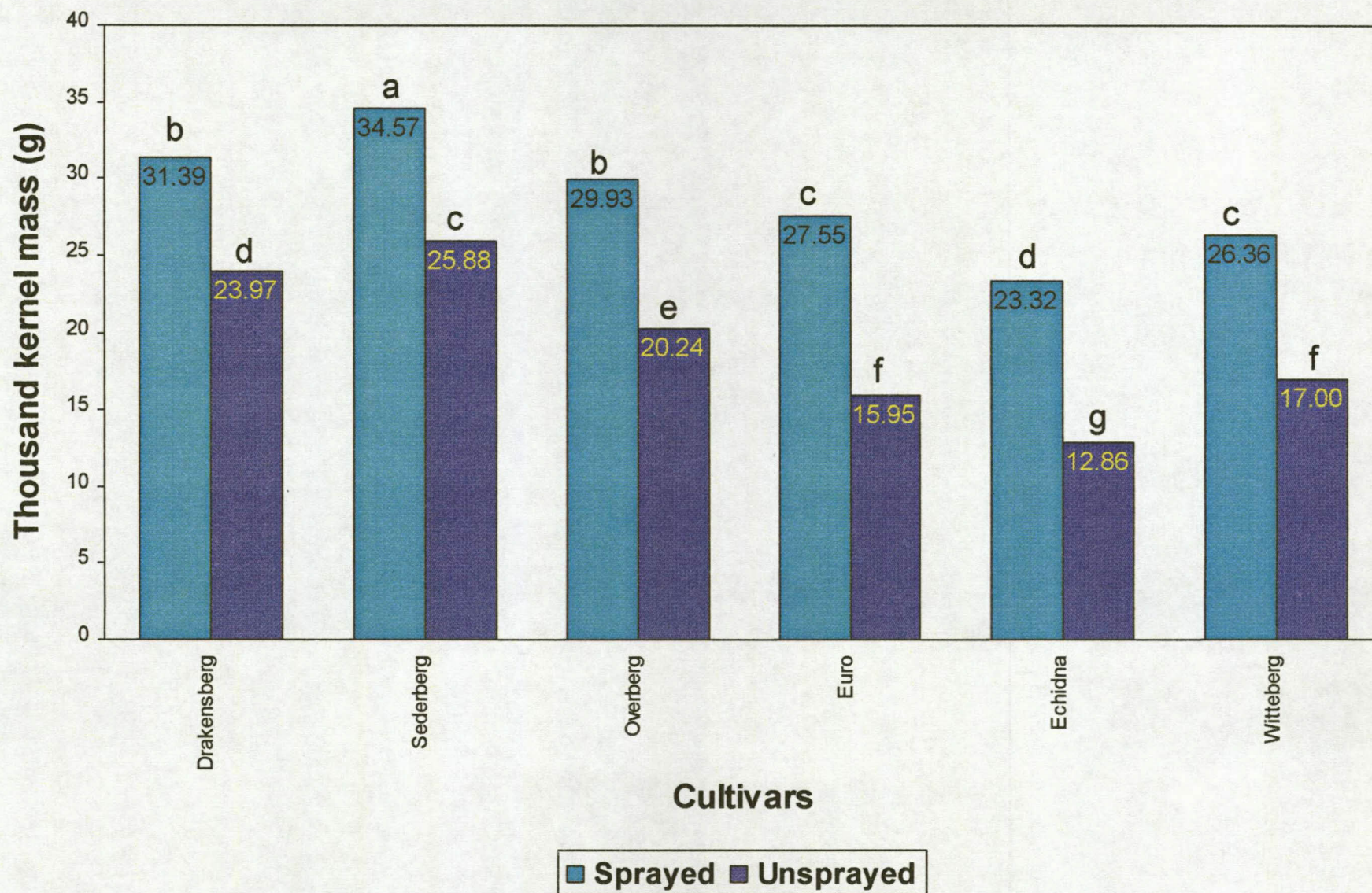
Figure 18. Regression analysis of percentage decrease in thousand kernel mass of oats due to *P. coronata* f. sp. *avenae* and *P. graminis* f. sp. *avenae* infections, expressed as combined area under the disease progress curves (AUDPC).
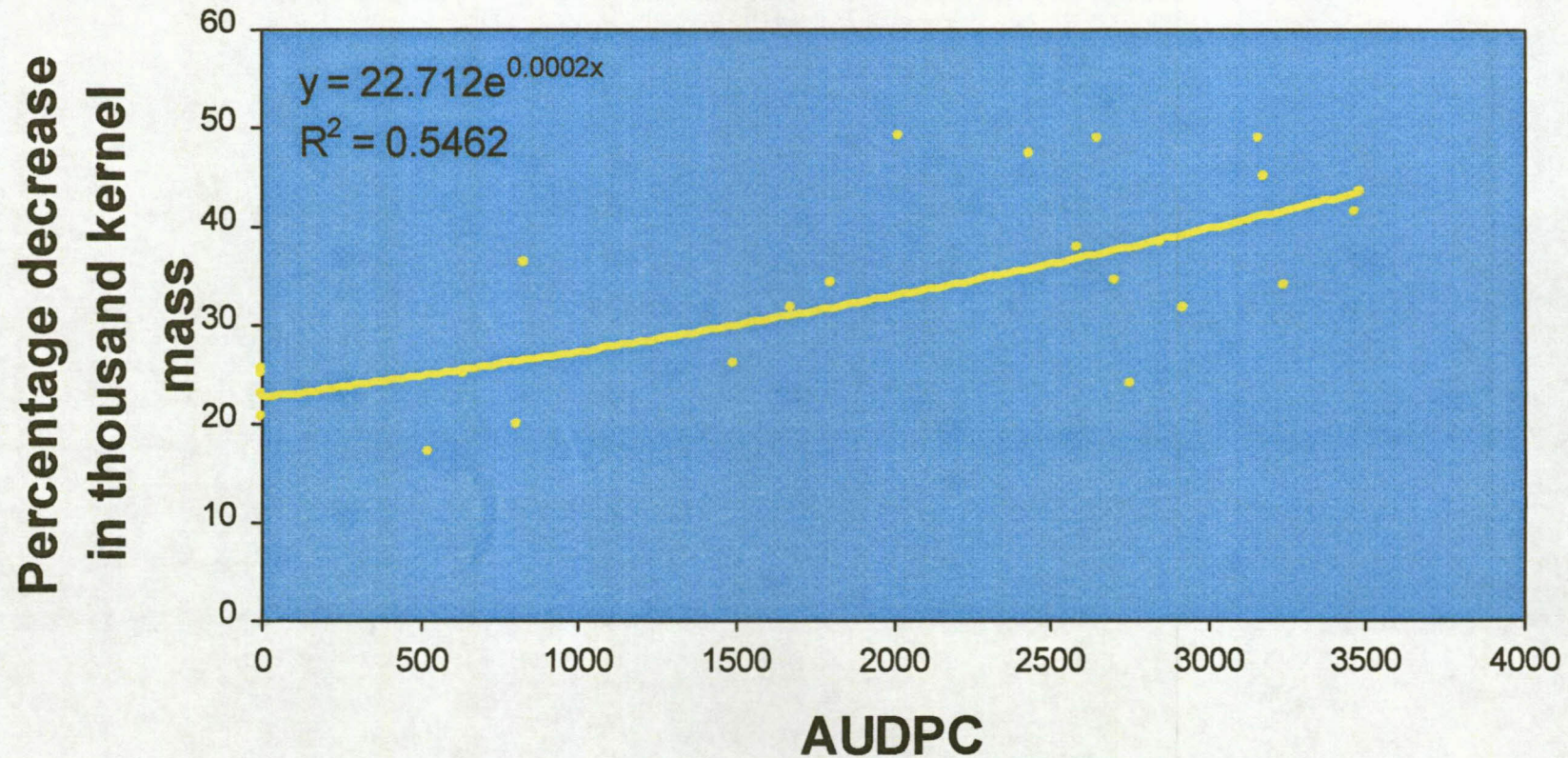
matrix analysis showed yield to be positively correlated with thousand kernel mass (CC = 0.87) and hectolitre mass (CC = 0.90) while thousand kernel mass was positively correlated with hectolitre mass (0.84) (App. D).

DISCUSSION

Barley

Puccinia hordei is known to cause severe crop losses in barley production (Griffey *et al.*, 1994). In this study yield losses of 58%, 49% and 39% were recorded on the susceptible cultivars SSG 525, Stirling and Clipper, respectively, with the moderately susceptible cultivar B95/10 recording a loss of 20%. The resistant cultivars SSG 532 and B94/2 had losses of 5% and 23%, respectively. These losses are unusually high if the yield loss data of 30% cited by Clifford (1985) and 45% reported by Teng (according to Cromey & Viljanen-Rollinson, 1995) are considered but can be attributed to the early onset of the epidemic, high disease severities and the length of the growing season. The fact that the initial infections occurred at flag leaf emergence supports the statement by Calpouzos *et al.* (according to Griffey *et al.*, 1994) that the magnitude of yield loss is directly correlated to the plant stage at which rust epidemics are initiated. Furthermore, the regular misting provided an environment highly conducive to rust development and led to a delay of senescence in resistant and sprayed plots.

The pronounced effect of leaf rust infection was clearly reflected in the regression analysis (Fig. 3) which indicated that 72% of the yield loss could be explained by disease severity. This was considerably higher than the 31-48% reported by Griffey *et al.* (1994). The significant yield difference in the resistant B94/2 line might have been due to the phytotonic influence (Griffiths & Scott, and Griffiths, according to Lim & Gaunt, 1986) of the triazole compound on the control treatment, rather than a reduction of yield in plots exposed to rust. According to Kuck *et al.* (1995) typical side effects of demethylation inhibiting fungicides on plants, e.g. triazole compounds, include shorter shoots and internodes, darker green leaves, reduced transpiration, increased resistance

to abiotic stress, and delayed senescence. In the present study the higher yield of the B94/2 control plots (Fig. 2) suggested that the fungicide applications influenced yield of control plots and that yield losses may possibly be inflated. However, the possibility of losses in resistant cultivars due to high disease pressure should also not be discounted (Lim & Gaunt, 1986). A significant amount of leaf damage, and thus photosynthetic tissue loss, is possible due to the hypersensitive reponse.

In barley it is important that losses be determined in terms of quantity and quality. Two important factors in the South African grading system are kernel plumpness and percentage siftings. For both these criteria all leaf rust-infected cultivars except B94/2 had significantly lower values. On the one hand this emphasises the possible influence of triazole treatments on increasing yield in the absence of disease, whereas it is also possible that B94/2 has an ability to maintain kernel plumpness and thus reduce its percentage siftings in spite of disease.

As far as nitrogen content, another important commercial characteristic, is concerned, it was found that all the susceptible cultivars had a significantly lower nitrogen content in the presence of *P. hordei* while that of the resistant cultivars was slightly higher, although this increase was not significant (Fig. 10). Considering susceptible cultivars, the reduction in the nitrogen content may be attributed to the loss of nutrients and photosynthetic capacity whereas in resistant cultivars it could be atrributed to a negative correlation between yield and nitrogen content. Thousand kernel mass was also significantly reduced in all cultivars except B94/2. This component was poorly correlated with disease severity, with only 32% of the reduction being attributed to leaf rust (Fig. 9). Udeogalanya & Clifford (1982) reported kernel weight to be negatively correlated with disease severity while King & Polley (1976) and Melville *et al*. (1976) found reductions in kernel weight due to leaf rust infections. It is important, however, to note that in this study only kernels larger than 2.5 mm, thus excluding siftings, were weighed. This reduced the amount of variation and it could be expected that unsifted samples would have shown a stronger relationship with disease severity.

It was also noted that disease severity had an influence on the date of maturity as well

as lodging, thus influencing various parameters indirectly. In the case of the susceptible cultivars Clipper, Stirling and SSG 525, unsprayed plots matured three weeks earlier than the sprayed plots while in the case of the more resistant cultivars SSG 532, B94/2 and B95/10 the fungicide delayed senescence by one week. It was also noticed that heavily infected plots were more prone to lodging and some of the loss in yield, especially kernel plumpness, may have been influenced by this factor.

Oats

The oat experiment confirmed the destructive nature of the oat rusts. According to Simons (1985) many reports of oats killed by crown rust prior to heading exist. In fungicide control and crown rust-infected plots, similar to the present study, losses of 26%, 31% and 50% were reported by Simons (1985) and Fleischmann & McKenzie (1965).

In this study yields were reduced significantly in all cultivars with losses of 77%, 85%, 85% and 83% respectively in the crown and stem rust susceptible cultivars Overberg, Euro, Echidna and Witteberg. The stem rust susceptible cultivar Sederberg and crown and stem rust resistant cultivar Drakensberg sustained yield losses of 47% and 25%, respectively. Hectolitre mass and thousand kernel mass confirmed plot yield data with all cultivars having significantly lower values than those determined for the control treatments. Singleton *et al.* (1982) also found a negative but highly significant correlation between disease severity and yield and kernel weight reduction.

Although Drakensberg is highly resistant to both crown and stem rust, with an immune reaction to both these diseases, yield, hectolitre and thousand kernel mass losses differed significantly between rusted and control treatments. This discrepancy can be attributed to the growth stimulating influence of the triazole containing fungicide. Sprayed Drakensberg plots matured two weeks later than unsprayed plots. Notwithstanding the fact that Drakensberg is a dual purpose cultivar with grain yield normally lower than that of other cultivars, it did not yield significantly lower than the other cultivars confirming the possible influence of the triazole. Singleton *et al.* (1982), however, found that in some highly resistant cultivars losses were higher than in certain

moderately susceptible cultivars, stating that some cultivars can support significant amounts of crown rust without sustaining losses.

Crown rust susceptible cultivars matured approximately four weeks earlier in unsprayed than in sprayed plots. This demonstrated the damaging influence of crown and stem rust on oat production, and was supported by regression analyses indicating that 94% of the yield loss and 82% of the reduction in hectolitre mass could be attributed to rust (Fig. 14 & 16). Reduction in thousand kernel mass was not as well correlated with disease severity with only 55% of the variation being explained by crown and stem rust infection. Fleischmann & McKenzie (1965) stated that thousand kernel mass is usually reliable although late-seeded oat or early crown rust epidemics (as early as flowering) may cause some kernels to not develop far enough to be threshed out or they might not develop at all. Since the epidemic started early in the present study, it is possible that the lack of correlation between thousand kernel mass, and to a lesser extent for hectolitre mass and disease, may be explained by the observation of Fleischmann & McKenzie (1965).

Similar to the reports of Gregory & Wise (1994) and Ohm & Shaner (according to Bush *et al.*, 1994), lodging occurred commonly, especially in rusted plots. Although the crown rust resistant but stem rust susceptible Sederberg yielded significantly higher than those cultivars susceptible to both diseases, it was not possible to separate losses caused by either rust. Fleischmann & McKenzie (1965) also found it difficult to distinguish between the effects of the two diseases.

It is clear that barley leaf rust, as well as crown and stem rust of oat can cause severe losses in South Africa. This is especially true when epidemics are initiated during early growth stages and when the environment remains favourable for rust infection. Although Bethlehem is not representative of the barley production area, this environment provided the opportunity to study the effects of leaf rust only. In the Western Cape, where several barley diseases occur, it is not possible to separate them in terms of yield losses caused. This study provided evidence that considerable improvement in yield and quality of barley and oat should be possible by developing or

introducing rust resistant cultivars.

REFERENCES

Alemayehu, F. & Parlevliet, J.E. 1996. Variation for resistance to *Puccinia hordei* in Ethiopian barley landraces. Euphytica 90: 365-370.

Borovkova, I.G., Jin, Y, Steffenson, B.J., Kilian, A., Blake, T.K. & Kleinhofs, A. 1997. Identification and mapping of a leaf rust resistance gene in barley line Q21861. Genome 40: 236-241.

Bush, A.L., Wise, R.P., Rayapati, P.J. & Lee, M. 1994. Restriction fragment length polymorphisms linked to genes for resistance to crown rust (*Puccinia coronata*) in near-isogenic lines of hexaploid oat (*Avena sativa*). Genome 37: 823-831.

Campbell, C.L. & Madden, L.V. 1990. Introduction to plant disease epidemiology. John Wiley & Sons, New York. 532 pp.

Clifford, B.C. 1985. Barley leaf rust. Pages 173-205 in: The Cereal Rusts. Vol. 2. A.P. Roelfs and W.R. Bushnell, eds. Academic Press, New York.

Cotterill, P.J., Rees, R.G. & Platz, G.J. 1994. Response of Australian barley cultivars to leaf rust (*Puccinia hordei*). Australian Journal of Experimental Agriculture 34: 783-788.

Cromey, M.G. & Viljanen-Rollinson, S.L.H. 1995. Virulence of *Puccinia hordei* on barley in New Zealand from 1990-1993. New Zealand Journal of Crop and Horticultural Science 23: 115-119.

Engelbrecht, W. 1999. Report presented at SANSOR (South African National Seed Orginisation) meeting 12-14 May 1999.

Fleischmann, G. & McKenzie, R.I.H. 1965. Yield losses in Garry oats infected with crown rust. Phytopathology 55: 767-770.

Gregory, J.W. & Wise, R.P. 1994. Linkage of genes conferring specific resistance to oat crown rust in diploid *Avena.* Genome 37:92-96.

Griffey, C.A., Das, M.K., Baldwin, R.E. & Waldenmaier, C.M. 1994. Yield losses in winter barley resulting from new races of *Puccinia hordei* in North America. Plant Disease 78: 256-260.

Jenkins, J.E.E., Melville, S.C. & Jemmett, J.L. 1972. The effect of fungicides on leaf diseases and on yield in spring barley in south-west England. Plant Pathology 21: 49-58.

King, J.E. & Polley, R.W. 1976. Observations on the epidemiology and effect on grain yield of brown rust in spring barley. Plant Pathology 25: 63-73.

Kuck, K.H., Scheinpflug, H. & Pontzen, R. 1995. DMI fungicides. Pages 205-258 in: Modern selective fungicides: properties, applications, mechanisms of action. 2 nd ed. H. Lyr ed. Gustav Fischer, New York. 595 pp.

Lim, L.G. & Gaunt, R.E. 1986. The effect of powdery mildew (*Erysiphe graminis* f. sp. *hordei*) and leaf rust (*Puccinia hordei*) on spring barley in New Zealand. I. Epidemic development, green leaf area and yield. Plant Pathology 35: 44-53.

Martens, J.W. 1985. Oat stem rust. Pages 103-129 in: The Cereal Rusts Vol 2. A.P. Roelfs and W.R. Bushnell, eds. Academic Press, New York.

Melville, S.C., Griffin, G.W. & Jemmett, J.L. 1976. Effects of fungicide spraying on brown rust and yield in spring barley. Plant Pathology 25: 99-107.

Roelfs, A.P., Singh, R.P. & Saari, E.E. 1992. Rust Diseases of Wheat: Concepts and

methodes of disease management. Mexico, D.F.: CIMMYT.

Šebesta, J., Zwartz, B., Roderick, H.W., Harder, D.E., Corazza, L. & Stojanovic, S. 1998. Incidence of oat stem rust and virulence of *Puccinia graminis* Pers. f. sp. *avenae* Erikss. et Henn. on oat and the effectiveness of resistance in Europe during 1988 to 1996. Archives of Phytopathology and Plant Protection 31: 393-413.

Simons, M.D. 1985. Crown rust. Pages 131-172 in: The Cereal Rusts Vol 2. A.P. Roelfs and W.R. Bushnell, eds. Academic Press, New York.

Singleton, L.L., Moore, M.B., Wilcoxson, R.D. & Kernkamp, M.F. 1982. Evaluation of oat crown rust parameters and yield in moderately resistant cultivars. Phytopathology 72: 538-540.

Stubbs, R.W., Prescott, J.M., Saari, E.E. & Dubin, H.J. 1986. Cereal Disease Methodology Manual. Centro Internacional de Mejoramiento de Maiz y Trigo (CIMMYT), Mexico.

Udeogalanya, A.C.C. & Clifford, B.C. 1982. Control of barley brown rust *Puccinia hordei* Otth, by benodanil and oxycarboxin in the field and the effects on yield. Crop Protection 1: 299-308

Zadoks, J.C., Chang, T.T. & Konzak, C. F. 1974. A decimal code for the growth stages of cereals. Weed Research 14: 415-421.

SUMMARY

This study showed less variation in the South African populations of *P. hordei, P. graminis* f. sp. *avenae* and especially *P. coronata* f. sp. *avenae* than was expected. In the case of *P. hordei* only two pathotypes were identified from a total of 810 isolates sampled over a six-year period. Pathotype SAPh 3231 was virulent to resistance genes *Rph1, Rph4, Rph5, Rph10* and *Rph11*, while pathotype SAPh 7231 had added virulence to *Rph12*. The occurrence of only one pathotype up to 1998 and the gaining of virulence to *Rph12* can be explained by the cultivars planted during this period. The cultivar Clipper dominated production until 1998 when the release of SSG 532, containing *Rph12,* supplied sufficient selection pressure to allow a mutation to establish. It was found that the reaction types of barley differential sets were not only influenced by the host and pathogen genotypes, but by a complicated interaction of environmental factors. The presence of the resistant gene *Rph12* was postulated in various cultivars. The study of accessory hosts identified some wild *Hordeum* accessions to be weak hosts for *P. hordei* while no evidence of the teleomorph of *P. hordei* could be found in South Africa.

The 269 single-pustule isolates of *P. coronata* f. sp. *avenae* revealed only one pathotype (SAPc 1) with virulence to the resistance genes *Pc40, Pc45, Pc46, Pc51* and *Pc54*. Four greenhouse mutants were also detected. These isolates overcame Pc35, *Pc48, Pc50, Pc52* and *Pc64,* indicating the vulnerability of these genes should they be used commercially. Although more pathotypes were expected, the presence of only one race could be explained by the cultivars grown and the differential set used. The wild oat species *Avena fatua, A. byzantina, A. sterilis* and *A. barbata* were susceptible to crown rust. The most variation in population composition was found in *P. graminis* f. sp. *avenae* with four pathotypes identified out of the 242 single-pustule isolates tested. Pathotype SAPga 1 had virulence to resistance genes *Pg1, Pg2, Pg4, Pg8* and *Pg15*, while pathotype SAPga 2 was similar except for added virulence to *Pg9*. Pathotype SAPga 4, on the other hand, had the same virulence profile as SAPga 2, except for added virulence to *Pga*. Pathotype SAPga 3 was virulent to *Pg1, Pg8* and *Pg 15*. All four pathotypes were virulent on *A. fatua, A. byzantina* and *A. sterilis,* while *A. barbata* was resistant to all four pathotypes.

Losses due to these three rust species in South African barley and oat cultivars were higher than expected. The barley cultivar SSG 525 sustained losses of 58% in yield and 61% in kernel plumpness, while the percentage siftings in Sterling was increased by as much as 590%. In resistant cultivars, differences between sprayed and unsprayed plots were in most cases not significant. In oats, yield was reduced by as much as 85% in the cultivars Euro and Echidna, and hectolitre mass with 45% in Witteberg.

This study clearly showed the destructive nature of these rust diseases and their impact on economically important parameters. It laid the foundation for further investigations and it should be emphasised that continual monitoring of these pathogens is necessary to provide meaningful contributions to rust control of barley and oats in South Africa.

Key words: *Puccinia hordei*, *Puccinia coronata* f. sp. *avenae*, *Puccinia graminis* f. sp. *avenae*, Pathogenic variability, Yield loss, Barley, Oats.

OPSOMMING

Die studie toon minder variasie in die Suid-Afrikaanse *Puccinia hordei*, *P. graminis* f. sp. *avenae* en veral *P. coronata* f. sp. *avenae* populasies as wat aanvanklik verwag is. Oor 'n sesjaar-periode en 'n totaal van 810 isolate kon daar slegs twee patotipes in *P. hordei* geïdentifiseer word. Patotipe SAPh 3231 was virulent op die weerstandsgene *Rph1*, *Rph4*, *Rph5*, *Rph10* en *Rph11*, terwyl patotipe SAPh 7231 bykomende virulensie het tot *Rph12*. Die voorkoms van slegs een patotipe tot en met 1998 kan verklaar word deur die garskultivars verbou gedurende die studieperiode. Tot met die vrystelling van SSG 532 in 1998 het die kultivar Clipper garsaanplantings oorheers. SSG 532, wat oor die weerstandsgeen *Rph12* beskik, het 'n genoegsame seleksievoordeel gestel vir die vestiging van die nuwe mutante patotipe. Die teenwoordigheid van *Rph12* is ook in verskeie cultivars gepostuleer. Dit is gevind dat reaksietipes van garsblaarroes differensiërende lyne nie alleen deur gasheer en patogeen genotipes beïnvloed word nie, maar ook deur 'n komplekse interaksie van omgewingsfaktore. Sekere *Hordeum* spesies kan voorts as swak gashere vir *P. hordei* dien terwyl die geslagtelike fase van die swam nie in Suid Afrika opgespoor kon word nie.

Uit die 269 *P. coronata* f. sp. *avenae* enkelpuisie-isolate kon daar sleg een patotipe (SAPc 1) geïdentifiseer word met virulensie tot die weerstandsgene *Pc40*, *Pc45*, *Pc46*, *Pc51* en *Pc54*. Vier glashuismutante is ook opgemerk. Hierdie isolate het *Pc35*, *Pc48*, *Pc50*, *Pc52* en *Pc64* oorkom wat dui op die kwesbaarheid van hierdie weerstandgene sou hulle in kommersiële kultivars gebruik word. Alhoewel meer patotipes verwag was kan die voorkoms van slegs een patotipe verklaar word deur die kultivars verbou, asook moontlik die differensiële stel wat gebruik is. Die hawerspesies *Avena fatua*, *A. byzantina*, *A.sterilis* en *A. barbata* was vatbaar vir kroonroes. Die meeste variasie in populasie-samesteling is gevind in *P. graminis* f. sp. *avenae* waar vier patotipes geïdentifseer is uit 242 enkelpuisie isolate. Patotipe SAPga 1 besit virulensie tot die weerstandsgene *Pg1*, *Pg2*, *Pg4*, *Pg8*, en *Pg15*, tewyl patotipe SAPga 2 soortgelyk was, maar met bykomende virulensie tot *Pg9*. Patotipe SAPga 4 het dieselfde virulensieprofiel as dié van SAPga 2, met die uitsondering van addisionele virulensie tot *Pga*. Patotipe SAPga 3 was virulent op

weerstandsgene *Pg1*, *Pg8* en *Pg15*. Al vier hierdie patotipes was virulent op *A. fatua*, *A. byzantina* en *A. sterilis*, maar avirulent op *A. barbata*.

Verliese veroorsaak deur hierdie drie roespatogene in Suid-Afrikaanse gars- en hawerkultivars was hoër as wat verwag is. Die garskultivar SSG 525 het verliese van 58% in opbrengs en 61% in vetkorrelpersentasie ervaar terwyl blaarroes die persentasie sifsels in Stirling met soveel as 590% verhoog het. In die geval van weerstandbiedende kultivars was die verskil tussen fungisied-bespuite en ongespuite behandelings meestal nie betekenisvol nie. In hawer het blaar- en stamroesinfeksie die opbrengs met soveel as 85% verlaag in die kultivars Euro en Echidna, terwyl hektolitermassa met 45% gedaal het in Witteberg.

Die studie toon duidelik die destruktiewe aard van hierdie roessiektes en hul betekenisvolle invloed op ekonomies-belangrike eienskappe. Hierdie studie vorm ook die basis vir verdere navorsing en beklemtoon dat die volgehoue monitering van hierdie patogene noodsaaklik is vir die doeltreffende beheer van roessiektes van gars en hawer in Suid Afrika.

APPENDIX A

Analysis of variance for barley yield

Source	df	MS	F-value	Pr>F
Total	47			
TREAT	1	103.811	508.99	0.0002
ENTRY	5	17.914	32.45	0.0000
BLOC	3	0.086	0.16	0.9249
BLOC by TREAT	3	0.204	0.37	0.7756
TREAT by ENTRY	5	5.609	10.16	0.0000
Residual	30	0.552		

Grand mean = 7.816	R-squared = 0.9307	C.V. = 9.51%

LSD for TREAT = 0.4149
LSD for ENTRY = 0.7587
LSD for TREAT*ENTRY = 1.0729

Analysis of variance for barley kernel plumpness

Source	df	MS	F-value	Pr> F
Total	47			
TREAT	1	9467.454	2722.41	0.0000
ENTRY	5	520.920	22.80	0.0000
BLOC	3	79.361	3.47	0.0282
BLOC by TREAT	3	3.478	0.15	0.9275
TREAT by ENTRY	5	520.938	22.80	0.0000
Residual	30	22.848		

Grand mean = 67.915	R-squared = 0.9561	C.V. = 7.04%

LSD for TREAT = 1.7132
LSD for ENTRY = 4.8810
LSD for TREAT*ENTRY = 6.9028

Analysis of variance for barley percentage siftings

Source	df	MS	F-value	Pr> F
Total	47			
TREAT	1	1793.408	394.14	0.0003
ENTRY	5	227.347	49.98	0.0000
BLOC	3	4.242	0.93	0.4371
BLOC by TREAT	3	4.550	1.00	0.4063
TREAT by ENTRY	5	192.441	42.30	0.0000
Residual	30	4.549		

Grand mean = 11.847   R-squared = 0.9663   C.V. = 18.00%

LSD for TREAT = 1.9597
LSD for ENTRY = 2.1779
LSD for TREAT*ENTRY = 3.0801

Analysis of variance for barley percentage nitrogen

Source	df	MS	F-value	Pr> F
Total	47			
TREAT	1	0.105	4.42	0.1263
ENTRY	5	0.231	19.87	0.0000
BLOC	3	0.064	5.48	0.0040
BLOC by TREAT	3	0.024	2.04	0.1298
TREAT by ENTRY	5	0.260	4.48	0.0036
Residual	30	0.012		

Grand mean = 2.630   R-squared = 0.8363   C.V. = 4.10%

LSD for TREAT = 0.1413
LSD for ENTRY = 0.1100
LSD for TREAT*ENTRY = 0.1556

Analysis of variance for barley thousand kernel mass

Source	df	MS	F-value	Pr> F
Total	47			
TREAT	1	104.342	378.54	0.0003
ENTRY	5	30.882	36.89	0.0000
BLOC	3	2.633	3.14	0.0396
BLOC by TREAT	3	0.276	0.33	0.8042
TREAT by ENTRY	5	3.261	3.90	0.0077
Residual	30	0.837		

Grand mean = 48.078   R-squared = 0.9187   C.V. = 1.90%

LSD for TREAT = 0.4823
LSD for ENTRY = 0.9343
LSD for TREAT*ENTRY = 1.3213

Analysis of variance for barley AUDPC

Source	df	MS	F-value	Pr> F
Total	47			
TREAT	1	5764374.083	506.38	0.0002
ENTRY	5	712569.383	59.36	0.0000
BLOC	3	11383.417	0.95	0.4297
BLOC by TREAT	3	1383.417	0.95	0.4297
TREAT by ENTRY	5	712569.383	59.36	0.0000
Residual	30	12003.517		

Grand mean = 347.542   R-squared = 0.9730   C.V. = 31.52%

LSD for TREAT = 98.0182
LSD for ENTRY = 111.8762
LSD for TREAT*ENTRY = 158.2169

APPENDIX B

Analysis of variance for oat yield

Source	df	MS	F-value	Pr> F
Total	47			
TREAT	1	338.672	1022.83	0.0001
ENTRY	5	13.281	31.08	0.0000
BLOC	3	0.567	1.33	0.2842
BLOC by TREAT	3	0.331	0.77	0.5173
TREAT by ENTRY	5	7.260	16.99	0.0000
Residual	30	0.427		

Grand mean = 5.331	R-squared = 0.9719	C.V. = 12.26%

LSD for TREAT = 0.5286
LSD for ENTRY = 0.6675
LSD for TREAT*ENTRY = 0.9440

Analysis of variance for oat thousand kernel mass

Source	df	MS	F-value	Pr> F
Total	47			
TREAT	1	1091.376	1302.95	0.0000
ENTRY	5	158.016	56.73	0.0000
BLOC	3	0.484	0.17	0.9134
BLOC by TREAT	3	0.838	0.30	0.8246
TREAT by ENTRY	5	4.149	1.49	0.2227
Residual	30	2.785		

Grand mean = 24.083	R-squared = 0.9580	C.V. = 6.93%

LSD for TREAT = 0.8408
LSD for ENTRY = 1.7042
LSD for TREAT*ENTRY = 2.4102

Analysis of variance for oat hectolitre mass

Source	df	MS	F-value	Pr> F
Total	47			
TREAT	1	2854.396	386.92	0.0003
ENTRY	5	118.179	20.08	0.0000
BLOC	3	4.941	0.84	0.4829
BLOC by TREAT	3	7.377	1.25	0.3079
TREAT by ENTRY	5	124.614	21.17	0.0000
Residual	30	5.885		

Grand mean = 42.114	R-squared = 0.9588	C.V. = 5.76%

LSD for TREAT = 2.4953
LSD for ENTRY = 2.4772
LSD for TREAT*ENTRY = 3.5033

Analysis of variance for oat stem rust AUDPC

Source	df	MS	F-value	Pr> F
Total	47			
TREAT	1	4669392.521	1340.77	0.0000
ENTRY	5	357376.071	76.37	0.0000
BLOC	3	3482.632	0.74	0.5342
BLOC by TREAT	3	3482.632	0.74	0.5342
TREAT by ENTRY	5	357376.071	76.37	0.0000
Residual	30	4679.249		

Grand mean = 312.896	R-squared = 0.9833	C.V. = 21.86%

LSD for TREAT = 54.2156
LSD for ENTRY = 69.8508
LSD for TREAT*ENTRY = 98.7840

Analysis of variance for oat crown rust AUDPC

Source	df	MS	F-value	Pr> F
Total	47			
TREAT	1	19014677.521	1091.02	0.0001
ENTRY	5	2314941.671	209.48	0.0000
BLOC	3	17428.410	1.58	0.2155
BLOC by TREAT	3	17428.410	1.58	0.2155
TREAT by ENTRY	5	2314941.671	209.48	0.0000
Residual	30	11051.093		

Grand mean = 630.396   R-squared = 0.9922   C.V. = 16.68%

LSD for TREAT = 121.2828
LSD for ENTRY = 107.3461
LSD for TREAT*ENTRY = 151.8103

Analysis of variance for oat crown and stem rust combined AUDPC

Source	df	MS	F-value	Pr> F
Total	47			
TREAT	1	42525675.000	3157.73	0.0000
ENTRY	5	3442976.950	189.96	0.0000
BLOC	3	13467.167	0.74	0.5348
BLOC by TREAT	3	13467.167	0.74	0.5348
TREAT by ENTRY	5	3442976.950	189.96	0.0000
Residual	30	18124.317		

Grand mean = 943.250   R-squared = 0.9930   C.V. = 14.27%

LSD for TREAT = 106.6126
LSD for ENTRY = 137.4720
LSD for TREAT*ENTRY = 194.4148

APPENDIX C

Barley correlation matrix

	Yield	Kernel plumpness	% Siftings	TKW	%
Nitrogen					
Kernel plumpness	0.8664				
	0.0000				
% Siftings	-0.8975	-0.9513			
	0.0000	0.0000			
TKW	0.3991	0.4351	-0.3349		
	0.0060	0.0025	0.0229		
%Nitrogen	0.6867	0.5884	-0.6856	0.0566	
	0.0000	0.0000	0.0000	0.7089	
AUDPC	-0.8926	-0.8909	0.9249	-0.4160	-0.6923
	0.0000	0.0000	0.0000	0.0040	0.0000

APPENDIX D

Oat correlation matrix

	Yield	TKW	Hectolitre mass	AUDPC-SR	AUDPC-CR
TKW	0.8665				
	0.0000				
Hectolitre mass	0.9025	0.8425			
	0.0000	0.0000			
AUDPC-SR	-0.8032	-0.5945	-0.6966		
	0.0000	0.0000	0.0000		
AUDPC-CR	-0.8682	-0.8341	-0.8204	0.7023	
	0.0000	0.0000	0.0000	0.0000	
AUDPC CR + SR	-0.9077	-0.8137	-0.8372	0.8496	0.9722
	0.0000	0.0000	0.0000	0.0000	0.0000