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# Pathogenicity of the *Rhynchosporium secalis* population in the Western Cape province of South Africa

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**Summary**

The virulence spectra of 50 *Rhynchosporium secalis* isolates from a population in the Western Cape province of South Africa were determined, and 21 races were detected when evaluated against 17 differential cultivars. The virulence spectrum of the *R. secalis* population shows considerable variation, and carries unnecessary virulence genes which is quite unexpected, since chiefly susceptible barley cultivars are grown in the south Western Cape. The two most prevalent races, namely races 4 and 7 had three and four virulence genes respectively. Both race 4 and 7 were virulent on the most susceptible cultivars, West China, Steudelli, C.I.8618 and C.I.2226. Considering the resistance genes reported for the cultivars Atlas 46, Turk, and C.I.3515 which showed no susceptible cultivar-pathogen interaction, it would appear that the *Rh-Rh3-Rh4* complex is primarily involved in conferring resistance to the local *R. secalis* isolates.

## Introduction

*Rhynchosporium secalis* (Oudem.) Davis, the cause of barley leaf scald, is reported to be most severe on barley (*Hordeum vulgare* L.) in the cool, moist areas of the temperate zones. The *R. secalis* population from each of the barley producing areas in the world comprises several unique races that differ in their ability to attack different barley cultivars. Race specialisation in *R. secalis* was first demonstrated in Argentina by Sarsola & Campi (1947) and genetic variability in *R. secalis* populations has been studied in several countries since then. Disease reaction data obtained by inoculating host differentials with fungal isolates often reveals important properties of pathogen populations. From the literature it is evident that *R. secalis* is pathogenically highly variable, being made-up of populations which differ in virulence spectrum between the different barley producing countries (Table 1). High variability in the pathogen has been reported from the USA (California), Canada (Western Canada, Southern Ontario), Denmark and Italy, whereas considerably less variation has been reported from Finland, South Eastern Australia and New Zealand (Williams & Owen, 1973; Jackson & Webster, 1976; Ceolini, 1980; Cromey, 1987; Brown, 1990; Xue & Hall, 1991; Robinson et al., 1996). Although Table 1 suggests that the UK has a population with less variation, recent research has provided evidence for the presence of several more races (A.C. Newton, personal communication).

Barley scald is an economically important disease in South Africa, and yield increases of up to 37% have been reported after fungicide applications on the susceptible cultivar Clipper (Scott et al., 1992). Clipper dominates the malting barley industry in the Western Cape of South Africa, and thus not much variation in virulence would be expected within the *R. secalis* population of the south Western Cape. Despite this, barley breeders have not yet been able to develop a good malting quality, scald resistant cultivar. To date the race composition of the *R. secalis* populations has

*Table 1.* A summary of variation in pathogenicity found among *Rhynchosporium secalis* populations world-wide

| Country | References | No. of cultivars tested | No. of isolates tested | No. of races | Resistant cultivars identified |
|---|---|---|---|---|---|
| Britain | Williams & Owen, 1973 | 15 | 122 | 2 | La Mesita, Osiris & Trebi |
| Norway | Hansen & Magnus, 1973 | 15 | 72 | Not specified | Hudson |
| West Australia | Ali & Boyd, 1973 | 27 | 27 | Not specified | Atlas 46, Atlas 47, Hudson, Osiris Psaknon, Sultan, Trebi & Turk |
| California, USA | Jackson & Webster, 1976 | 14 | 175 | 75 | None |
| Italy | Ceoloni, 1980 | 13 | 100 | 17 | Atlas, Atlas 46 & Osiris |
| Victoria, Australia | Brown, 1985 | 15 | 319 | 5 | Abyssinian, Atlas 46, Hudson, La Mesita, Modoc, Nigrinudum, Osiris, Turk, Wisconsin & Winter × Glabron, C.I.3515, C.I.4364 |
| New Zealand | Cromey, 1987 | 18 | 149 | 4 | Abyssinian, Atlas 46, Hudson, Ilia, Kitchen, Modoc, Priver, Tripper & Trebi |
| South East Australia | Brown 1990 | 15 | 182 from barley grass & 94 from barley | 20 | C.I.3515 |
| Western Canada | Tekauz, 1991 | 10 | 111 from 20 different cultivars | 45 | None |
| South-Ontario, Canada | Xue & Hall, 1991 | 5 | 352 | 20 | Atlas & Atlas 46 |
| California, USA | Zhang et al., 1992 | 14 | 275<br>273 | 180 (1983)<br>183 (1984) | None |
| Denmark | Jørgensen & Smedegaard-Petersen, 1995 | 23 | 38 | 28 | Atlas & Osiris |
| Finland | Robinson et al., 1996 | 17 | 20 | –[a] | Atlas, Atlas 46, Armelle, Brier, Hudson, Jet, Magnugm, Modoc, Nigrinudum, Wisconsin Winter × Glabron, Kitchen, Osiris, C.I.2376, Bey C.I.5581, Atrada × Atlas C.I.7189 |
| Norway | Salamati & Tronsmo, 1997 | 24 | 42 | 32 | Osiris |

[a] No significant differences in specific pathogenicity.

not been determined, and neither have possible resistance sources been identified. The objectives of this study were therefore to characterise the current virulence spectrum of the *R. secalis* population in the south Western Cape of South Africa and evaluate the local *R. secalis* isolates against most of the existing resistance sources in barley using standard differential cultivars. These data will lay the foundation for strategies for breeding resistance against *R. secalis* in South Africa.

## Materials and methods

### *Collection and isolation of the fungus*

Barley leaves with leaf scald symptoms were collected from 29 different locations (24 different farms) within the south Western Cape. Fifty isolates were collected during the 1993–1995 growing seasons. To isolate the fungus a leaf segment with a scald lesion was cut and surface sterilised (30 sec in alcohol, 120 sec in 1% aqueous NaOCl, 30 sec in alcohol), rinsed in sterile distilled water and placed on moist filter paper in a Petri dish sealed with Parafilm. After two days lesions were scraped with a scalpel, rinsed in a drop of water on a 1.2% water agar (WA) plate, spores distributed over the surface of the WA plate, the plate inverted and incubated overnight. Germinated spores were located

under a stereo microscope and transferred to lima bean agar (LBA, 124 g lima beans, 12 g agar 1 l water) plates amended with 0.05 g/l streptomycin. Resulting single spore colonies were subsequently stored on lima bean agar slants under sterile mineral oil at 3°C.

*Differential cultivars*

Each isolate collected was tested against five plants of each of 17 cultivars (Abyssinian, Atlas 46, C.I.4364, C.I.2226, C.I.3515, C.I.8618, Clipper, Jet, La Mesita, Modoc, Nigrinudum, Osiris, Psaknon, Steudelli, Turk, West China, Wisconsin Winter × Glabron) and breeding line B87/14 (Small Grain Institute, South Africa). Differential cultivars were kindly provided by the Victorian Institute for Dryland Agriculture (Australia) and the National Small Grain Centre (USA). Genes for resistance against *R. secalis* have been reported for all the cultivars except C.I.2226. Each cultivar was grown as groups of five plants in 15 cm diameter plastic pots in a glasshouse where the temperature ranged from 17–20°C. The susceptible cultivar Clipper was planted in the middle of each pot to check the viability of the inoculum. The experimental design was a randomised block with two replications of each cultivar-isolate combination. The whole experiment was repeated.

*Preparation of inoculum, inoculation and rating*

*Rhynchosporium secalis* cultures were incubated on LBA plates at 17 ± 1°C in the dark for 2 weeks. Conidal suspensions were obtained from these cultures by adding sterile distilled water to the plates and scraping the conidia from the surface. The resulting conidial suspensions were adjusted to approximately $3 \times 10^5$ spores/ml.

Barley seedlings at 2–3 leaf stage (approximately two-wk-old), were first sprayed with a 0.1% aqueous solution of Tween 20 in order to wet the leaves, after which a inoculum was sprayed until run-off by applying approximately 125 ml of inoculum to a set of test plants. Inoculated seedlings were transferred to a dew chamber where the plants were kept in the dark at approximately 17°C and a relative humidity of 95–98% for 48 hours. Afterwards the seedlings were transferred to the glasshouse.

After 14 days in the glasshouse the symptoms were classified according to the scale described by Jackson & Webster (1976). No visible symptoms = 0; very small lesions confined to leaf margins = 1; small lesions not confined to leaf margins = 2; large coalescing lesions, involving the majority of the leaf area = 3; total collapse of the leaf with no distinct lesions = 4. Ratings of 0–2 were indicative of a resistant reaction, whereas ratings of 3–4 were considered as indicating a susceptible reaction. Reactions between some cultivar-isolate combinations could not be uniformly categorised in either the susceptible or resistant group, in which case the reaction type was classified according to the most prevalent rating.

*Cluster analysis*

Statistical analysis was performed using SAS (Statistical Analysis System, Cary, NC). Cluster analysis was performed using PROC CLUSTER with Density Linkage and the k=. (kth-nearest neighbour method) option. The number of clusters were assessed graphically with a dendrogram. The dendrogram was bisected when the level of similarity (R-squared value) reached 75%.

## Results

*Races, variability and cluster analysis*

Twenty-one races were identified from 50 isolates when evaluated against 17 differential cultivars (Table 2). On seven different instances when more than one isolate were collected from a lesion, these isolates were found to differ in pathotype. Races varied from having a simple (one virulence gene) to a complex (12 virulence genes) virulence spectrum.

More than half of the *R. secalis* isolates tested had four or less virulence genes, which were also the highest association of virulence genes (>59%) among the isolates tested. Races 4 and 7 were the two most prevalent races, being found over 5 districts within the barley producing area of the south Western Cape (Figure 1). These two races were found at 18 of the 29 fields (15 of the 24 farms) where samples were taken, but were virulent to only four cultivars, namely Steudelli, West China, C.I.8618 and C.I.2226. The frequency in association of virulence genes found in races 4 and 7 were 88% and 60% respectively. Races with 6 and more virulence genes were characterised by the occurrence of one isolate per race, and a low association (<13%) of virulence genes among the isolates tested.

Twelve clusters were resolved among 21 races (Figure 2). The first cluster contained 10 races. Races included in this cluster were pathogenic on a mean of

*Table 2.* Disease ratings (– = resistant, + = susceptible) of 17 barley cultivars and B87/14 inoculated with South African isolates of *Rhynchosporium secalis*

| Races | B87/14 | At.46[a] | Turk | Osiris | Psak.[b] | C.I.3515[c] | Aby[d] | LaM.[e] | Brier | Modoc | W.W.G.[f] | Jet | Nig.[g] | Steud.[h] | C.I.4364 | C.I.2226 | W.C.[i] | C.I.8618 | No. of isolates |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | – | – | – | – | – | – | – | – | – | – | – | – | – | + | – | – | – | – | 2 |
| 2 | – | – | – | – | – | – | – | – | – | – | – | – | – | + | – | – | + | – | 2 |
| 3 | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | + | + | + | 1 |
| 4 | – | – | – | – | – | – | – | – | – | – | – | – | – | + | – | – | + | + | 11 |
| 5 | – | – | – | – | – | – | – | – | – | – | – | – | – | + | + | – | + | + | 1 |
| 6 | – | – | – | – | – | – | – | – | + | – | – | – | – | + | – | – | + | + | 1 |
| 7 | – | – | – | – | – | – | – | – | – | – | – | – | – | + | – | + | + | + | 11 |
| 8 | – | – | – | – | – | – | – | – | – | – | + | – | – | + | – | – | + | + | 1 |
| 9 | – | – | – | – | – | – | + | – | – | – | – | – | – | + | – | + | + | + | 3 |
| 10 | – | – | – | – | – | – | – | – | – | – | – | – | – | + | + | + | + | + | 6 |
| 11 | – | – | – | – | – | – | – | – | – | – | – | + | + | + | + | + | + | – | 1 |
| 12 | – | – | – | – | – | – | – | – | – | + | – | – | – | + | + | + | + | + | 1 |
| 13 | – | – | – | – | – | – | – | – | – | – | – | + | + | + | – | + | + | + | 1 |
| 14 | – | – | – | + | – | – | + | – | – | – | – | – | + | + | + | + | + | + | 1 |
| 15 | – | – | – | – | – | – | – | – | – | – | – | + | + | + | + | + | + | + | 1 |
| 16 | – | – | – | – | – | – | + | – | – | + | – | + | – | + | + | + | + | + | 1 |
| 17 | – | – | – | – | – | – | + | + | – | + | – | – | – | + | + | + | + | + | 1 |
| 18 | – | – | – | – | – | – | + | – | + | – | + | + | – | + | + | + | + | + | 1 |
| 19 | – | – | – | – | – | – | + | – | – | + | + | + | + | + | + | + | + | + | 1 |
| 20 | – | – | – | – | – | – | + | + | – | + | + | + | + | + | + | + | + | + | 1 |
| 21 | – | – | – | – | + | – | + | + | – | + | + | + | + | + | + | + | + | + | 1 |

[a] Atlas 46
[b] Psaknon
[c] Cereal Inventory Number, Agricultural Research Service, United States Department of Agriculture
[d] Abyssinian
[e] La Mesita
[f] Wisconsin Winter × Glabron
[g] Nigrinudum
[h] Steudelli
[i] West China

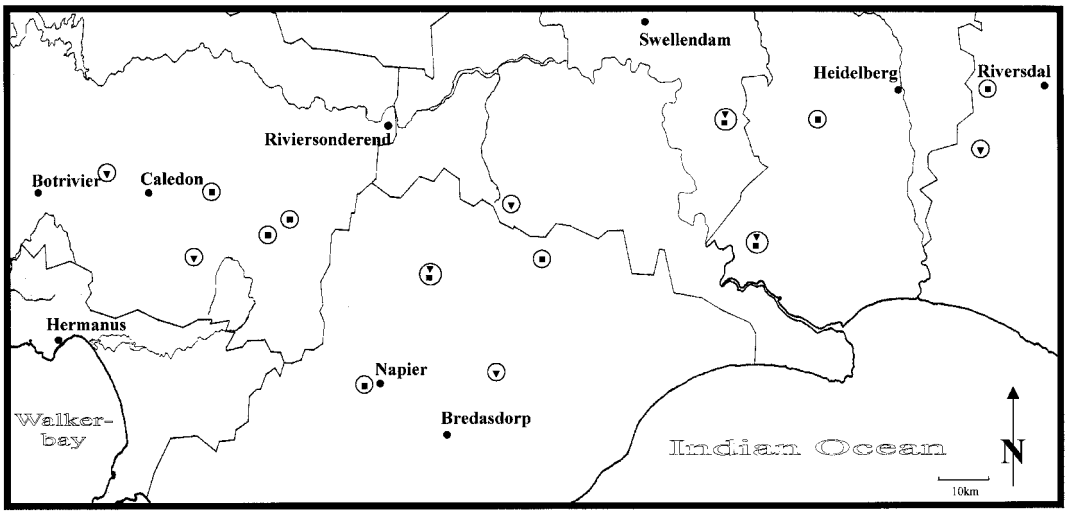
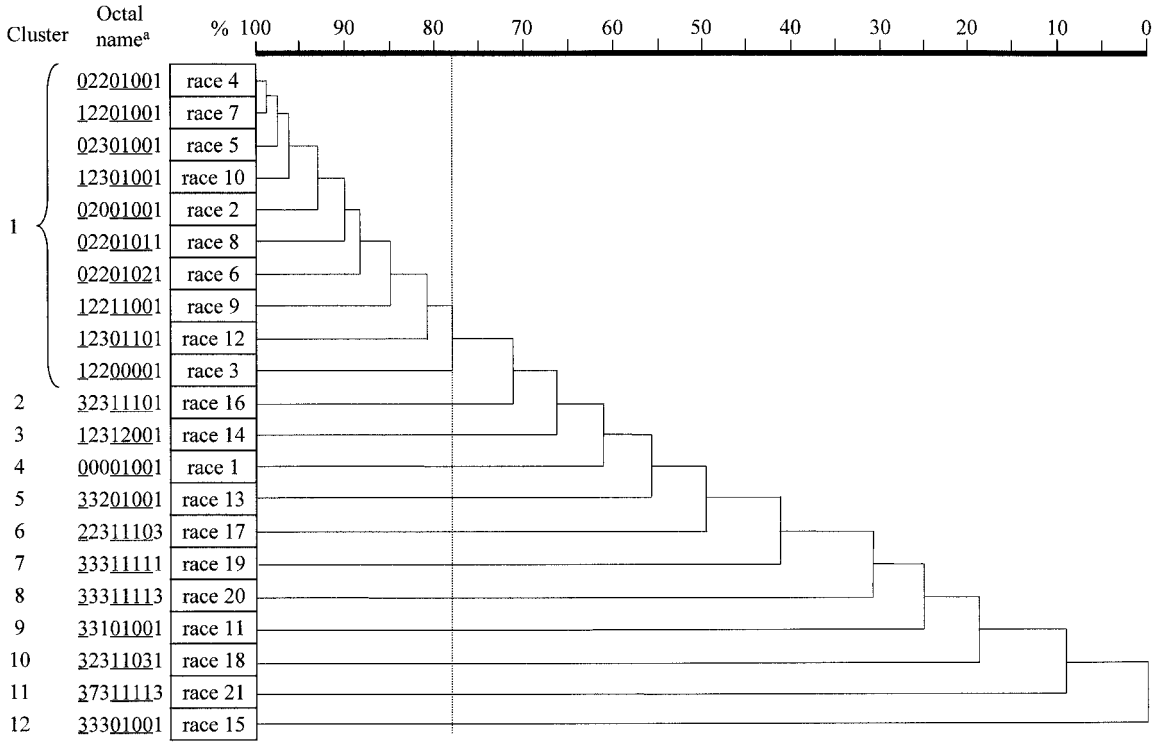



*Figure 1.* A map showing the main districts and towns (●) of the barley producing area in the south Western Cape. Farms are also indicated (○) where the two most prominent races (race 4 ■ and race 7 ▼) of the local *Rhynchosporium secalis* population were found.




[a] According to the octal nomenclature proposed by Goodwin et al. (1990) (Abysinian was used as the source of the Rh9 resistance instead of Kithchin and cultivars proposed for digit 8 were not evaluated in this study, instead Jet and C.I.2226 were incorporated).

*Figure 2.* A dendrogram of homology (%) between *Rhynchosporium secalis* isolates according to their virulence to a set of differential cultivars using the kth-nearest neighbour method of cluster analysis (dendrogram bisected at 78% similarity as indicated by the dotted line).

*Table 3.* Genes reported to condition resistance to *Rhynchosporium secalis* in barley cultivars used in this study[a]

| Cultivars | Genes | References |
|---|---|---|
| B87/14 | not characterised | |
| Atlas 46 | Rh2 and Rh3 | Dyck & Schaller, 1961 |
| | Rh | Habgood & Hayes, 1971 |
| | One dominant gene at the Rh-Rh3-Rh4 complex | Starling et al., 1971 |
| Turk | Rh3 and Rh5 | Dyck & Schaller, 1961 |
| | Rh and rh6 | Habgood & Hayes, 1971 |
| | One dominant gene at the Rh-Rh3-Rh4 complex | Starling et al., 1971 |
| Osiris | Rh4 | Dyck & Schaller, 1961 |
| | Rh4, Rh10 & rh6 | Habgood & Hayes, 1971 |
| Psaknon | 1 to 3 dominant genes | Ali, 1975 |
| C.I.3515 | Rh4 & Rh10 | Habgood & Hayes, 1971 |
| | One dominant gene at the Rh-Rh3-Rh4 complex | Starling et al., 1971 |
| Abyssinian | Rh9 | Baker & Larter, 1963 |
| La Mesita | Rh4 | Dyck & Schaller, 1961 |
| | Rh4 & Rh10 | Habgood & Hayes, 1971 |
| | One dominant gene at the Rh-Rh3-Rh4 complex | Starling et al., 1971 |
| Brier | Rh & rh6 | Habgood & Hayes, 1971 |
| | One dominant gene at the Rh-Rh3-Rh4 complex | Starling et al., 1971 |
| Modoc | Rh4 | Dyck & Schaller, 1961 |
| | Rh2 & rh6 | Habgood & Hayes, 1971 |
| | One dominant gene at the Rh-Rh3-Rh4 complex | Starling et al., 1971 |
| Wisconsin Winter × Glabron | Rh3 | Habgood & Hayes, 1971 |
| Jet | rh6 & rh7 | Baker & Larter, 1963 |
| | rh5 | Habgood & Hayes, 1971 |
| Nigrinudum | rh8 | Habgood & Hayes, 1971 |
| Steudelli | rh6 & rh7 | Baker & Larter, 1963 |
| C.I.4364 | rh11 | Habgood & Hayes, 1971 |
| C.I.2226 | not characterised | |
| West China | Two dominant genes | Ali, 1975 |
| C.I.8618 | One dominant gene | Starling et al., 1971 |

[a] Table accepted from Goodwin et al. (1990).

four differentials. The remaining eleven clusters each consisted of a single race.

*Cultivars and cluster analysis*

Genes reported to condition resistance to *R. secalis* in barley cultivars are summarised in Table 3. All isolates were virulent to the susceptible cultivar Clipper, and 98%, 96%, 90% and 64% were virulent towards barley cultivars Steudelli, West China, C.I.8618 and C.I.2226. The remainder of the cultivars (Abyssinian, La Mesita, Modoc, Wisconsin Winter × Glabron, Jet, Nigrinudum and C.I.4364) were susceptible to 6–34% of the isolates tested, and 14–57% of the races were virulent towards these cultivars. La Mesita was susceptible to 6% of the isolates and 14% of the races while C.I.4364 was susceptible to 34% of the isolates and 57% of the races. No susceptible cultivar-pathogen interactions were observed for Atlas 46, Turk, C.I.3515 and line B87/14. One isolate was found to be virulent on Osiris and Psaknon and two isolates were virulent on Brier.

## Discussion

The virulence spectrum of the *R. secalis* population studied shows considerable variation and carries unnecessary virulence genes which is quite unexpected, since susceptible barley cultivars are grown throughout the south Western Cape. Most of the isolates from the same field and even same lesion were different

in their virulence spectrum, which confirms a similar observation made by Brown (1990). Unnecessary virulence in *R. secalis* populations has previously been recorded in Norway and Western Australia (Ali, 1981; Salamati & Tronsmo, 1997). Variation in Norway has been explained by a foreign seed source which may have been contaminated, whereas in Australia it has been explained by the extensive hectares of *Hordeum* species. However, in South Africa neither of these explanations are applicable, since barley seed is multiplied locally for commercial use, and seed treatments are standard practice. Furthermore, barley grass is not the predominant grass weed and does not occupy vast areas in the barley producing area of the Western Cape.

With the number of cultivars (17) used in this study it should be possible to detect 131 072 ($=2^{17}$) races if they were present in the population. Twenty-one races, from which more than half were represented by only one isolate, were revealed when 50 isolates were tested. It is, however, more than likely that a greater sample size would reveal more races. Despite this variation, less virulent races dominated, with races 4 and 7 being the most prominent. These two races showed a high (99%) homology in regard to their virulence spectra, with only cultivar C.I.2226 distinguishing the two races from each other. The three most common races (56% of isolates tested) were fixed for virulence on three differentials, fixed for avirulence on 13, and varied for only two differentials. Furthermore, races in cluster 1 represented 76% of the isolates tested in the study and were virulent to a mean of only four differentials (Figure 2). Although *R. secalis* populations are potentially variable (Table 1) it seems that under conditions which do not demand a variety of virulence genes, the population tends to be dominated by simple races. There is also an indication that the local *R. secalis* population is dominated by an association of virulence genes characteristic of races with less virulence, compared to the more complex races which were less prevalent. These observations support the stabilising theory (races with unnecessary virulence are less fit) proposed by Van der Plank (1968). Previously, due to evidence for (Williams & Owen, 1973; Jackson & Webster, 1976b) and against (Hansen & Magnus, 1973; Ali, 1976; Jackson & Webster, 1976; Jørgensen & Smedegaard-Petersen, 1995) the stabilising theory, conflicting statements were made regarding this theory for *R. secalis*. Selection pressure differs from country to country, and factors such as commercially grown cultivars, seed sources, environment and alternative hosts influence the level of variability in *R. secalis* populations. However, it is evident from the conflicting statements that stabilising selection is not the only element shaping the virulence spectra of *R. secalis* populations worldwide. Hansen & Magnus (1973) were the first to suggest that the variation may be explained in terms of segregation of virulence genes which may implicate parasexual or sexual recombination.

Cultivars Atlas 46, Turk, C.I.3515 and line B87/14 proved to be useful sources of resistance. Although Osiris, Psaknon and Brier were also promising, they were marginally susceptible. Steudelli, which was highly susceptible in this study was also not found to be promising in other studies (Table 1). Considering the resistance genes reported for the cultivars Atlas 46, Turk, and C.I.3515, which showed no susceptible cultivar-pathogen interaction, it seems that the *Rh-Rh3-Rh4* complex is primarily involved in conferring resistance to the local *R. secalis* isolates (Table 3). Cultivars that proved to be resistant in this study, also showed resistance against *R. secalis* populations of other countries in the world such as Australia (West Australia, South Australia, Victoria); Italy; New Zealand; Canada (South-Ontario) and Finland (Table 1). Stacking of race specific genes will be a useful strategy, but the capacity of the *R. secalis* population for pathogenic variation within a small area (lesion, plant, field) may still reduce the effective period of such a combination of race specific resistance. The importance of quantitative resistance for scald control has been frequently stated (Brown, 1985; Cromey, 1987; Tekauz, 1991) and although practically more difficult to identify and incorporate, levels of quantitative resistance should ultimately be introduced in breeding programmes for durable resistance.

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