Occurrence and Pathogenicity of *Puccinia hordei* on Barley in South Africa

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ABSTRACT

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Although leaf rust, caused by *Puccinia hordei*, is considered an important disease of barley (*Hordeum vulgare*) and regularly reaches epidemic proportions, pathogenic variability has never been studied in South Africa. From 1994 to 1997, only one pathotype (SAPh 3231) was identified with virulence to resistance genes *Rph1*, *Rph4*, *Rph5*, *Rph10*, and *Rph11*. During 1998, a second pathotype (SAPh 7321) was identified, differing from pathotype SAPh 3231 only in virulence to *Rph12*. Pathotype SAPh 7321 increased rapidly in the area where it was first detected, resulting in localized epidemic outbreaks in 1999. The reactions of various South African cultivars and breeding lines toward these pathotypes were determined, and the presence of *Rph12* was postulated for B93/4, Krona, Optic, Prisma, and SSG 532. *Rph* genes showed varying degrees of temperature sensitivity, with none of the known genes displaying major changes in their phenotypes except *Rph8*, which was less effective at higher temperatures. Eight accessions of two wild *Hordeum* spp. occurring abundantly in the barley growing regions were found to be either weak or nonhosts for *P. hordei*.

Leaf rust, caused by the fungus *Puccinia hordei* Otth, is an important disease of barley (*Hordeum vulgare* L.) in several areas of the world, including Australia, Europe, North America, and South America (1-3). Until 1970, the disease was considered unimportant in economic terms, but more recently changes in cropping practices have resulted in an increase in the importance of leaf rust, with severe epidemics occurring more regularly (3). Lim and Gaunt (9) concluded that *P. hordei* should be regarded as a serious source of potential yield loss, while Cotterill et al. (4) recorded yield losses of 26 to 31% in Australia during a moderate to severe epidemic in 1990.

In South Africa, the disease was first reported by Doidge et al. (5) in the south Western Cape in 1953. Here, barley grown mainly for malting purposes comprises approximately 130,000 ha under dry-land conditions. 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 by the Sensako breeding program. Due to susceptibility of Clipper to foliar diseases, especially scald (caused by *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 favorable for scald, e.g., 1992, 1997, 1998, and 1999, leaf rust epidemics occurred.

The main emphasis of local breeding programs has thus far been on increased yield, brewing quality, and scald resistance. With many of these objectives reached in new cultivars and advanced breeding lines, as well as recurring *P. hordei* epidemics over the last few seasons, resistance to leaf rust has now become a priority. It is therefore considered important to identify the resistance genes that are effective against barley leaf rust in South Africa and to know what pathotypes are prevalent. To achieve this, a survey was carried out between 1994 and 1999 to determine the extent of pathogenic variation in *P. hordei* and to subsequently determine the reaction of leading cultivars and breeding lines to prevailing pathotypes. The susceptibility of possible accessory grass hosts for *P. hordei* was also investigated.

MATERIALS AND METHODS

Seedling tests. Leaf rust-infected barley leaves were either collected personally from trap nurseries, experimental plots, or commercial fields, or received by mail from coworkers. 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-

sterilized soil, were inoculated with urediniospores either directly from field samples or following a heat shock treatment (42°C for 4 min) for those stored at -70°C . Prior to inoculation, emerging Clipper seedlings were treated with 50 ml per pot of a 0.3 g/liter maleic hydrazide solution to retard plant development and stimulate sporulation on inoculated plants. 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 $16^{\circ}\text{C} \pm 2^{\circ}\text{C}$. Upon removal from the dew chamber, plants were allowed to dry for 2 h in fan-circulated air before pots were placed in separate glass compartments in a greenhouse. Greenhouse temperatures were maintained between 18 and 22°C , and natural daylight was supplemented with $120 \mu\text{E}\cdot\text{m}^{-2}\cdot\text{s}^{-1}$ photosynthetically active radiation emitted by cool-white fluorescent tubes arranged directly above plants (14 h/day).

After 10 to 14 days, between 2 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 reinoculated on Clipper seedlings for further urediniospore multiplication. In certain instances, there was need for a second multiplication. Urediniospores obtained after multiplication were inoculated onto a differential set of barley cultivars with known genes for resistance (Table 1). Differentials were planted as four clumps of seed (± 10 seeds per clump) per 10-cm-diameter pot. Two to three days after initial eruption of uredinia, plants were assessed for their disease response using a 0 to 4 scale (16).

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 10 seeds each, were planted in clumps in 10-cm-diameter pots filled with steam-sterilized 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 postinoculation conditions were maintained as described above. As in the case of differentials, infection types were assessed 2 to 3 days after the eruption of primary uredia.

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Adult plant tests. Differential lines (Table 1) and commercial cultivars were planted in the field in 1-m-row plots spaced 20 cm apart and surrounded by two rows of Clipper. Plots were planted at the SGI (Bethlehem, Free State, artificial infection with pathotype SAPH 3231), Langgewens experimental station (Western Cape, natural infection), and Tygerhoek experimental station (south Western Cape, natural infection). An artificial epidemic at Bethlehem was created by injecting spreader rows with freshly collected urediniospores suspended in water (17). Disease assessments were recorded on flag and penultimate leaves at least two times during the season using a modified Cobb scale (13). 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 2-liter-capacity pots filled with steam-sterilized soil. Three pots per cultivar were planted at 7-day intervals to ensure synchronization of flag leaf emergence. Pre- and postinoculation 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 g/liter 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 manner to that of seedlings. After 10 days, flag leaves were rated for leaf rust severity using the modified Cobb scale (13).

During 1998, elite lines of the SGI breeding program 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 3 weeks earlier and inoculated by injecting tillers at growth stage 36 (19) with a suspension of freshly collected urediniospores (SAPH 3231) in water containing the surfactant Tween 20 (1 ml/liter). Infection of plots was encouraged by regular overhead mist irrigation, ensuring high humidity conditions.

Temperature study. The influence of temperature on the seedling reaction of *P. hordei* differential lines was also determined. Differential lines (Table 1) were grown in 10-cm plastic pots containing a sterilized soil:peat moss mixture (1:1, vol/vol) at 19/24°C night/day temperature. After 7 days (first leaf fully expanded), plants were inoculated with fresh urediniospores (SAPH 3231) and incubated overnight in a dark dew-simulation chamber before placement in growth chambers set at 11, 18, and 25°C, respectively. Fourteen hours of light was provided by cool-white fluorescent tubes and incandescent bulbs arranged 30 cm above plants, providing 200 µE·m⁻²·s⁻¹ of photosynthetically active radiation. Each temperature regime was represented by three replications of the differential set. Disease assessment was done when Clipper (susceptible control) showed a compatible (3 to 3+) reaction.

Accessory hosts. To investigate the relationship between *P. hordei* and wild *Hordeum* species occurring in the Western Cape, certain *Hordeum* accessions were collected during 1998. After identification, seeds of these accessions were harvested, planted, and infected with appropriate cultures of *P. hordei*. Pre- and postinoculation treatment of seedlings was conducted as described previously.

RESULTS

Pathogenic variation. During 1994, 15 leaf rust samples were collected from the

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

Cultivar	CI number	Gene	Octal value	Reaction types ^a	
				Pathotype SAPH 3231	Pathotype SAPH 7231
Sudan	CI 6489	<i>Rph1</i>	1	3+	3+
Peruvian	CI 935	<i>Rph2</i>	2	X2	X2
Aim	CI 3737	<i>Rph3</i>	4	0;	0;
Gold	CI 1145	<i>Rph4</i>	10	3	3
Magnif 104	CI 13806	<i>Rph5</i>	20	3	3
Bolivia	CI 1257	<i>Rph6+2</i>	40	X-	X-
Cebada Capa	CI 6193	<i>Rph7</i>	100	:N	:N
Egypt 4	CI 6481	<i>Rph8</i>	200	3C	3C
Abyssinian	CI 1243	<i>Rph9</i>	400	2C	2C
Clipper BC8	None	<i>Rph10</i>	1000	3+	3+
Clipper BC67	None	<i>Rph11</i>	2000	3	3
Triumph	PI 268180	<i>Rph12</i>	4000	:1N	3
Clipper	CI 14844	...	...	3+	3+

^a Infection types (16): 0 = no uredia, ; = fleck, 1 = small uredia with necrotic border, 2 = small to medium uredia with chlorosis or necrosis, 3 = medium-sized uredia, 4 = large uredia without chlorosis or necrosis, N = more necrosis than normal, C = more chlorosis than normal, + = uredia larger than normal, - = uredia smaller than normal, X = random distribution of variable-sized uredia. Infection type of 3 and higher considered susceptible.

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	CI number	<i>Rph</i> gene	Field				Greenhouse Bethlehem (SAPH 3231)
			Tygerhoek 1997 (SAPH 3231)	Tygerhoek 1999 (SAPH 3231 and SAPH 7231)	Napier 1999 ^a (SAPH 3231 and SAPH 7231)	Bethlehem 1998 (SAPH 3231)	
Sudan	CI 6489	<i>Rph1</i>	60MS-S ^b (Y)	70MS-S	80MS	80S	60MS-S
Peruvian	CI 935	<i>Rph2</i>	60MS-S (Y)	20MS	0	60MR-MS	40R-MR
Aim	CI 3737	<i>Rph3</i>	30MS	-	-	20R	10R
Gold	CI 1145	<i>Rph4</i>	80S	80S	90S	80S	80S
Magnif 104	CI 13806	<i>Rph5</i>	60MS-S	-	100S	80MS	60MS-S
Bolivia	CI 1257	<i>Rph6+2</i>	20MR-MS (Y)	0	0	30MR-MS	40R-MS
Cebada Capa	CI 6193	<i>Rph7</i>	0	0	0	20R	10R
Egypt 4	CI 6481	<i>Rph8</i>	40MS-MR	-	30MR	40MR-MS	40MR-MS
Abyssinian	CI 1243	<i>Rph9</i>	20R-MR	-	50MS	30MR-MS	40R-MR
Clipper BC8	None	<i>Rph10</i>	60MS-S	60MS	50MS	60S	60S
Clipper BC67	None	<i>Rph11</i>	80S	70S	60S	60S	80S
Triumph	PI 268180	<i>Rph12</i>	20MR-MS (Y)	80S	60MS	30R	40R-MR
Clipper	CI 14844	...	80S	100S	100S	80S	90S
B94/2	...	...	10MR-R	90S	-	20MR-R	-
SSG 532	...	...	10MR-R	100S	100S	20MR-R	-

^a Ratings were done on field plots at the SENSAGO Research Station.

^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; -, reaction types not determined.

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). 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 (6).

During the 1999 season, it was established that the formerly resistant cultivar SSG 532 and line B94/2 were susceptible to leaf rust. Isolates were sampled from these two lines 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 nurseries (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-pustule isolates, 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 intermediate 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 an intermediate seedling response reacted in a resistant manner in the adult plant stage, with *Rph2* being the only exception, exhibiting a tendency toward moderate susceptibility at Tygerhoek during 1997 and Bethlehem in 1998 (Table 2).

Host response. The seedling response of 1998 and 1999 elite SGI barley lines and crossing parents, as well as other cultivars, to both pathotypes is shown in Table 3.

Results of adult plant tests of the 1998 entries to pathotype SAPH 3231 are also given. In general, seedling reactions corresponded with those of adult plants, although a few exceptions occurred, such as B97/3, displaying a moderately susceptible reaction (infection type = 23) in the seedling stage and a resistant reaction (30R-MR) in the adult plant stage. Based on their reactions to the two pathotypes, it was possible to postulate resistance gene *Rph12*

in B93/4, Krona, Optic, Prisma, and SSG 532.

Differential sets maintained at different temperatures revealed minor changes in their reaction to an isolate of pathotype SAPH 3231. No dramatic change from resistance to susceptibility or vice versa could be demonstrated. However, *Rph8*, *Rph9*, and *Rph12* tended to be more susceptible at 25°C, whereas the opposite was true for *Rph5*, *Rph10*, and *Rph11* (Table 4).

Table 3. Seedling and adult plant reaction of a collection of breeding lines and control genotypes to pathotypes SAPH 3231 and SAPH 7231 of *Puccinia hordei*

Line	Seedling reaction ^a		Adult plant reaction ^b
	SAPH 3231	SAPH 7231	SAPH 3231
Alexis	;12	;1N	—
B93/2	22+	22+	TMR
B93/4	;CN	3—	0
B94/2	2+CN	3—	30R-MR
B94/5	X	X—	—
B94/7	X—	X—	—
B95/10	3+	3	50MS-S
B96/1	;CN	;	10R
B97/10	23	3—	40MS
B97/11	X	—	0-40S
B97/12	3—	3—	30MS
B97/13	223	—	60S
B97/14	2+2	2+	40MS-MR
B97/15	22+	2+2	20MS
B97/16	22+C	—	30MS
B97/3	23	X	30R-MR
B97/6	23	—	60MS
B97/7	X—	—	5MS
B97/8	23	—	20MS
B97/9	3—	3—	20MS
B98/1	2C	—	10R
B98/2	3—	3—	20S
B98/3	23	3—	30S
B98/4	X+	3	50S
B98/5	3—	3—	50S
B98/6	2+C	3—	40MR-MS
B99/1	3—	3—	—
B99/2	3	3—	—
B99/3	3+	3	—
B99/4	X	3	—
B99/5	3—	3—	—
B99/6	3	3—	—
B99/7	3—	3—	—
B99/8	3—	3—	—
B99/9	3—	3—	—
B99/10	2—	3—	—
B99/11	3—	3—	—
B99/12	3—	X+	—
Barke	;N	;	—
Blenheim	2	3—	10-20MS
Chariot	X	—	—
Clipper	3	3	80S
Krona	;N1N	3—	—
Maresi	2	3	—
Optic	;N	2+	—
Prisma	;N	3—	—
Scarlett	0;	;	—
SSG 532	3P3- 6P;CN	3	30MR-MS
SSG 525	3	3	80MS
Stirling	3+	3	40MS
Triumph	;N	3—	—

^a Infection types (16): 0 = no uredia, ; = fleck, 1 = small uredia with necrotic border, 2 = small to medium uredia with chlorosis or necrosis, 3 = medium-sized uredia, 4 = large uredia without chlorosis or necrosis, N = more necrosis than normal, C = more chlorosis than normal, + = uredia larger than normal, — = uredia smaller than normal, X = random distribution of variable-sized uredia. Infection type of 3 and higher considered susceptible.

^b Artificial inoculation at Small Grain Institute, Bethlehem during 1998. 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; —, reaction types not determined.

During the 1998 survey, six accessions of *Hordeum murinum* L. and two of *H. capense* Thunb. were tested for their reaction to SAPH 3231. Four of the *H. murinum* accessions were weak hosts with reactions varying from ;2C to 2, while the other two accessions and *H. capense* were resistant to *P. hordei*.

DISCUSSION

Based on the differential set, it is clear that during 1994 to 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 southern parts of the Western Cape, where it was first identified, suggesting that significant shifts in distribution had 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. Due to the number and distribution of the samples over the fairly small barley production area, isolates were considered to be representative of the pathogen population.

Reinhold and Sharp (15) 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 [3]) reported on the co-existence of *Ornithogalum* flora with wild *Hordeum* spp. and mentioned that the alternate host is essential for survival and the generation of pathogenic variability in Israel. Taking into account that local barley producing areas have a Mediterranean climate and that several *Hordeum* spp., as well as 54 species of the alternate host *Ornithogalum*

spp., occur in South Africa, many of which may be found in the barley producing area (10), more than one pathotype would have been expected. In contrast to the diversity that may be expected, only two pathotypes, one being a recent addition, were found. Although the role of the alternate host has not been studied in detail, aecial infections of *Ornithogalum* spp. are not known to occur in South Africa, therefore making sexual recombination unlikely.

Levine and Cherewick (8) and Steffenson et al. (18) mentioned mutation as a likely cause for the origin of new pathotypes. This would make a single-step mutation the most obvious explanation for the appearance of pathotype SAPH 7231. Since the leaf rust-susceptible cultivar Clipper historically constituted approximately 95% of all barley planted, no direct selective advantage existed for virulence in new pathotypes. The release of SSG 532 in 1997, and its large-scale production in 1998, provided this advantage to pathotypes with virulence to *Rph12*. In addition to Triumph, low and high reactions to pathotypes SAPH 3231 and SAPH 7231, respectively, were observed in B93/4, B94/2, B98/4, B99/4, B99/10, Blenheim, Maresi, Optic, Prisma, and SSG 532, suggesting the presence of *Rph12*. However, several of the low infection types, e.g., B94/2, B98/4, B99/4, B99/10, Blenheim, and Maresi, were higher than the typical *Rph12* response of Triumph and thus cannot be attributed to this gene without further testing.

Varying degrees of temperature sensitivity were expressed by *Rph* genes. With the exception of *Rph8*, which was less effective at higher temperatures, none of the genes displayed major changes in their phenotype. Clifford and Udegalanya (according to Clifford [3]) reported that the gene in CI 1243 (*Rph9*) became less effective with an increase in temperature from 5 to 25°C. In the present study, some indica-

tions were found that *Rph9* tended to be more susceptible at 25°C (2C) than at 11°C (;1=). Based on the authors' observations made over several years of working with *P. hordei*, slight variation in infection type occurs over time, and it seems likely that the interaction between the barley host and leaf rust pathogen may also be influenced by other factors. Further studies on interactions among temperature, light intensity, and duration, and plant nutrition during pre- and postinoculation periods, are necessary to clarify this variation.

As the use of resistant cultivars is globally accepted as the primary strategy to control leaf rust of barley (7), it is important to ensure that resistance is durable. Parlevliet (11,12) warned against the use of hypersensitive (low infection type) resistance in commercial cultivars and stated that it only provided temporary protection. 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* (3,18). Alternatively, Steffenson et al. (18) 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 several 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 (3). According to Parlevliet (12), this type of resistance is more durable and has remained effective in Europe since the early 1970s (18). According to Qi et al. (14), progress has been made in this regard. Six quantitative trait loci (QTL) for partial resistance in the barley cultivar Vada were identified. It was also significant that these QTL mapped to different positions than those for hypersensitive resistance, suggesting different gene families.

The appearance of a new barley leaf rust pathotype in 1998, and an ensuing epidemic in 1999, emphasize the importance of virulence surveys and the application of this information in breeding programs. Although resistance to *P. hordei* is a selection criterion of breeders, screening in South Africa has relied mostly on natural infection. In addition to inconsistent results, these selections were made without any knowledge of which genes are effective in this region. This study has now provided a basis for more directed leaf rust

Table 4. Infection types of differential barley lines to pathotype SAPH 3231 of *Puccinia hordei* at different temperatures and in different controlled environments

Cultivar	CI number	Resistance gene	Growth chamber ^a		
			25°C	18°C	11°C
Sudan	CI 6489	<i>Rph1</i>	33+	33+	3+
Peruvian	CI 935	<i>Rph2</i>	2	X+	X
Aim	CI 3737	<i>Rph3</i>	;;1	0	;
Gold	CI 1145	<i>Rph4</i>	3	3	3-3
Magnif 104	CI 13806	<i>Rph5</i>	2	3=	3
Bolivia	CI 1257	<i>Rph6+2</i>	2-	X= (Y)	X= (Y)
Cebada Capa	CI 6193	<i>Rph7</i>	;N	;N	;CN
Egypt 4	CI 6481	<i>Rph8</i>	3++	23	22+C
Abyssinian	CI 1243	<i>Rph9</i>	2C	;1	;;1=
Clipper BC8	None	<i>Rph10</i>	23	3	3
Clipper BC67	None	<i>Rph11</i>	23	23	3
Triumph	PI 268180	<i>Rph12</i>	2-C	;;1+	;;1N
Clipper	CI 14844	-	3+	3	3= (2+3)

^a Infection types (16): 0 = no uredia, ; = fleck, 1 = small uredia with necrotic border, 2 = small to medium uredia with chlorosis or necrosis, 3 = medium-sized uredia, 4 = large uredia without chlorosis or necrosis, N = more necrosis than normal, C = more chlorosis than normal, + = uredia larger than normal, - = uredia smaller than normal, X = random distribution of variable-sized uredia. Infection type of 3 and higher considered susceptible.

research and resistance breeding in South Africa.

LITERATURE CITED

1. Alemayehu, F., and Parlevliet, J. E. 1996. Variation for resistance to *Puccinia hordei* in Ethiopian barley landraces. *Euphytica* 90:365-370.
2. Borovkova, I. G., Jin, Y., Steffenson, B. J., Kilian, A., Blake, T. K., and Kleinhofs, A. 1997. Identification and mapping of a leaf rust resistant gene in barley line Q21861. *Genome* 40:236-241.
3. Clifford, B. C. 1985. Barley leaf rust. Pages 173-205 in: *The Cereal Rusts*. Vol. II. Diseases, Distribution, Epidemiology, and Control. A. P. Roelfs and W. R. Bushnell, eds. Academic Press, New York.
4. Cotterill, P. J., Rees, R. G., Platz, G. J., and Dill-Macky, R. 1992. Effects of leaf rust on selected Australian barleys. *Aust. J. Exp. Agric.* 32:747-751.
5. Doidge, E. M., Bottemley, A. M., Van der Plank, J. E., and Pauer, G. D. 1953. A revised list of plant diseases in S. A. Dep. Agric. Sci. Bull. No. 346.
6. Gilmour, J. 1973. Octal notation for designating physiological races of plant pathogens. *Nature* 242:260.
7. Jin, Y., Cui, G. H., Steffenson, B. J., and Franckowiak, J. D. 1996. New leaf rust resistance genes in barley and their allelic and linkage relationships with other *Rph* genes. *Phytopathology* 86:887-890.
8. Levine, M. N., and Cherewick, W. J. 1952. Studies on dwarf leaf rust of barley. U.S. Dep. Agric. Tech. Bull. 1056:1-17.
9. Lim, L. G., and 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 Pathol.* 35:44-53.
10. Obermeyer, A. A. 1978. *Ornithogalum*: A revision of the southern African species. *Bothalia* 12:377-393.
11. 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.
12. 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.
13. Peterson, R. F., Campbell, A. B., and Hannah, A. E. 1948. A diagrammatic scale for estimating rust intensity of leaves and stems of cereals. *Can. J. Res.* 26:496-500.
14. Qi, X., Niks, R. E., Stam, P., and Lindhout, P. 1998. Identification of QTLs for partial resistance to leaf rust (*Puccinia hordei*) in barley. (Abstr.) *Theor. Appl. Genet.* 96:1205.
15. Reinhold, M., and Sharp, E. L. 1982. Virulence types of *Puccinia hordei* from North America, North Africa, and the Middle East. *Plant Dis.* 66:1009-1011.
16. Roelfs, A. P. 1988. Genetic control of phenotypes in wheat stem rust. *Annu. Rev. Phytopathol.* 26:351-367.
17. Roelfs, A. P., Singh, R. P., and Saari, E. E. 1992. Rust Diseases of Wheat: Concepts and methods of disease management. CIMMYT, Mexico, D.F.
18. Steffenson, B. J., Jin, Y., and Griffey, C. A. 1993. Pathotypes of *Puccinia hordei* with virulence for the barley leaf rust resistance gene *Rph7* in the United States. *Plant Dis.* 77:867-869.
19. Zadoks, J. C., Chang, T. T., and Konzak, C. F. 1974. A decimal code for the growth stages of cereals. *Weed Res.* 14:415-421.