Pathogenic Variability of *Puccinia coronata* f. sp. *avenae* and *P. graminis* f. sp. *avenae* on Oat in South Africa

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ABSTRACT

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Although crown rust (caused by *Puccinia coronata* f. sp. *avenae*) and stem rust (caused by *Puccinia graminis* f. sp. *avenae*) are generally considered to be the most widespread and damaging diseases of oat (*Avena* spp.) in South Africa, pathogenic variability has never been studied. During 1997 and 1998, one dominant crown rust pathotype (SBLL) was identified with virulence to resistance genes *Pc40*, *Pc45*, *Pc46*, *Pc51*, and *Pc54*. Four other pathotypes (SGLL, PBBB+*Pc35*, SDQL, and JBBM+*Pc35*), occurring at low frequencies and further rendering resistance genes *Pc35*, *Pc39*, *Pc48*, *Pc50*, *Pc52*, and *Pc64* ineffective, were also detected. Resistance gene *Pc40* was postulated in Wisconsin X1588-2; *Pc51* in Euro, Maluti, Overberg, OX88I 075-106, Perdeberg, and Swartberg; and *Pc39* was confirmed in the cultivar Fidler. During the same period, four stem rust pathotypes were identified with virulence to resistance genes *Pg1*, *Pg2*, *Pg4*, *Pg8*, *Pg9*, *Pg12*, *Pg15*, and *Pga*. Resistance gene *Pga* was postulated in Alpha, OX87 080-1, OX88I 075-106, Sederberg, and W94/4; *Pg2* and/or *Pg4* in Euro, Perdeberg, Potberg, and Swartberg; and *Pg9* in Pallinup and Victorian. Collections of wild oat species *Avena fatua*, *A. byzantina*, *A. sterilis*, and *A. barbata* were susceptible to all crown rust pathotypes, while the four stem rust pathotypes were virulent on all species except *A. barbata*.

Oat (*Avena sativa* L.) is an important cereal, used both for human consumption and as a feed grain, and ranks sixth in world cereal production after wheat, maize, rice, barley, and sorghum (22). Crown (leaf) rust, caused by *Puccinia coronata* Corda f. sp. *avenae* Eriks., has been considered the most important fungal disease of cultivated oat (30), while stem rust, caused by *Puccinia graminis* Pers.:Pers. f. sp. *avenae* Eriks. & E. Henn., occurs in most of the oat growing areas of the world, periodically causing severe crop losses (18,19,30).

P. coronata f. sp. *avenae* is highly variable in virulence and can rapidly evolve new pathotypes that overcome commonly used resistance genes (7,17,30). Early studies on pathogenic variability in *P. graminis* f. sp. *avenae* indicated that different virulence combinations occurred widely. As new genes for resistance were discovered, virulence on them was usually found concurrently, and in most cases, virulence to these genes was present in the pathogen

population before cultivars with the genes were grown over large areas (19).

In South Africa, Sawyer (28) reported in 1909 that oat crops in KwaZulu-Natal were destroyed to such an extent by crown rust that oat could no longer be grown there. Sawyer (28) also provided evidence of dramatic changes in cultivar disease rankings over time which, although speculative, could have been due to pathogenic adaptation. Evans (11) reported that oat rust was not known in South Africa before 1858 but that the disease had often restricted production during the latter part of the nineteenth century. Although records of collections of *P. graminis* (host and forma specialis unknown) in 1874 exist (4), Bailey was the first to identify a race of *P. graminis* f. sp. *avenae* in South Africa in 1925 (19), while Verwoerd (35) identified two "forms" of *P. graminis* f. sp. *avenae*, with "form 2" resembling the race identified by Bailey. According to Doidge (9) and Verwoerd (33,34), *P. graminis* f. sp. *avenae* was found on oat 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. (10) reported that no aecidial form is known for crown rust in South Africa.

Currently, oat production is completely deregulated in South Africa, and no official

records of the total area cultivated or annual production exist. Oat is used for various purposes, including oat grain for breakfast cereal, horse feed, general feed rations, health foods, and bird feed. In the Western Cape, oat 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 cooperatives and established that 8.6% of cultivated oat 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 oat were planted during 1990. This is probably a conservative estimate because many farmers retain their own seed, especially when oat is grown for grazing purposes. At present, oat has also increased in popularity as a rotation crop under irrigation and as grazing in the Highveld. W. Engelbrecht (report presented at the meeting of the South African National Seed Organisation, 12 to 14 May 1999) estimated the potential for oat production in South Africa to be $\pm 700,000$ ha.

Historically, most oat cultivars 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 and to establish representative pathotypes for further testing of germ plasm. Oat cultivars and breeding lines were also evaluated for seedling and adult plant resistance toward both diseases, whereas the role of wild *Avena* spp. as possible accessory hosts for both crown and stem rust was also investigated.

MATERIALS AND METHODS

Survey and pathotype differentiation. Oat leaves and stems infected with either *P. coronata* f. sp. *avenae* or *P. graminis* f. sp. *avenae* were collected from trap nurser-

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ies, commercial fields, and wild oat 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 gelatin 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 in a disease-free growth chamber at a continuous temperature of 22°C and 14 h of light provided by cool-white fluorescent tubes and incandescent bulbs arranged 30 cm above plants and emitting $200\ \mu\text{E}\cdot\text{m}^{-2}\cdot\text{s}^{-1}$ of photosynthetically active radiation. Seedlings were grown in 10-cm-diameter plastic pots filled with steam-sterilized soil and treated with 50 ml per pot of a $0.3\ \text{g liter}^{-1}$ 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 7-day-old (first leaf fully extended) Overberg or Swan seedlings, respectively. Overberg was selected for this purpose due to its historic susceptibility to crown rust. Likewise, Swan has been reported to carry no genes for resistance to *P. graminis* f. sp. *avenae* (2). 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 ($\pm 2^{\circ}\text{C}$)

for 18 h. After removal from the dew chamber, plants were allowed to dry for 2 h before placement in a greenhouse. Throughout this study, the collection of spores and inoculation of seedlings were done as described by Browder (5). Plants inoculated with different isolates were placed in separate glass compartments to avoid contamination. Greenhouse temperatures were maintained at 22°C for crown rust and 19°C for stem rust studies, with natural daylight supplemented by 14 h of $120\ \mu\text{E}\cdot\text{m}^{-2}\cdot\text{s}^{-1}$ photosynthetically active radiation emitted by cool-white fluorescent tubes arranged directly above the plants. After 10 to 14 days, two to four single pustules per sample were collected, increased on either Overberg or Swan, and inoculated onto the respective differential sets (entries listed in Tables 1 and 2). Three to four days after the initial eruption of uredia, infection types were assessed using a 0 to 4 scale (26). *P. coronata* f. sp. *avenae* infection types were assessed on the adaxial surfaces of leaves, whereas those of *P. graminis* f. sp. *avenae* were rated on abaxial surfaces. Pathotype designations were allocated according to Chong et al. (8).

The adult plant reactions of the various differentials were also determined. Crown and stem rust differential lines were planted in an unreplicated screening nursery at Bethlehem during the 1998 season. Entries were sown in 1-m rows spaced 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 (36), equal amounts of urediniospores of crown rust pathotype SBLL and stem rust pathotypes SAPga 1, SAPga 2, SAPga 3, and SAPga 4 suspended in water with the aid of Tween 20 were 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 (24).

Cultivar reaction. The disease reactions of all oat cultivars grown in South Africa, as well as advanced breeding lines, were determined in the seedling stage to crown rust pathotypes SBLL, SGLL, PBBB+Pc35, SDQL, and JBBM+Pc35, as well as stem rust pathotypes SAPga 1, SAPga 2, SAPga 3, and SAPga 4. Four cultivars—lines were planted in clumps of approximately 10 seeds each in a 10-cm plastic pot filled with steam-sterilized 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 previously. Reaction patterns were interpreted for postulation of resistance genes.

Crown and stem rust severity were assessed (modified Cobb scale) on entries in the annual oat cultivar adaptation trials conducted in the main production areas by

Table 1. Infection types^a produced on seedlings of differential lines of *Avena sativa* containing single *Pc* genes when infected with pathotypes SBLL, SGLL, PBBB+Pc35, SDQL, and JBBM+Pc35 of *Puccinia coronata* f. sp. *avenae*

<i>Pc</i> gene	Tester line	<i>P. coronata</i> f. sp. <i>avenae</i> pathotypes				
		SBLL	SGLL	PBBB+Pc35	SDQL	JBBM+Pc35
<i>Pc35</i>	D137/2*Pendek ^b	;1CN	;N	2+3	;C	2+3
<i>Pc38</i>	CAV 2648-4/4*Pendek ^c	0;	;	;C	0;	;C
<i>Pc39</i>	CAV 5165/4*Pendek ^c	;	3	;	;	;C
<i>Pc40</i>	Pendek2*/CAV 4997 ^c	2+3	3	3	2+3	;C
<i>Pc45</i>	Pendek*4/CAV 5050 ^c	2+3	2+3	;	2+3	3
<i>Pc46</i>	Pendek*4/CAV 5115 ^c	2+3	2+3	2+3	2+3	2+3
<i>Pc48</i>	Pendek2*/CAV 5041 ^c	;C	;	;	2+3	;C
<i>Pc50</i>	CAV 2643/4*Pendek ^c	;	;	2+3	0;	0;
<i>Pc51</i>	Iowa Early isoline X-434 ^c	2+3	3	;	2+3	;
<i>Pc52</i>	Iowa Midseason isoline X-421 ^c	;C	;	0;	2+3	;
<i>Pc54</i>	Pendek*2/CAV 1832 ^c	2+3	2+3	;C	2+3	2+3
<i>Pc56</i>	CAV 1964/4*Pendek ^c	;1=C	;C	;C	;C	X(, 2)
<i>Pc58</i>	TAM-O-301 (P.I. 295919) ^c	;	;	X(, 2)	;C	X(, 2)
<i>Pc59</i>	TAM-O-312 (P.I. 296244) ^c	;	;	;	;	;
<i>Pc60</i>	Coker 227 ^d	2C	2C	X(, 2C)	;C	X(, 2)
<i>Pc61</i>	Coker 234 ^d	2C	2C	;	;	X(, 2)
<i>Pc62</i>	Fraser*2/CAV 4274 ^c	;1CN	;1	;C	;C	X(, 2)
<i>Pc63</i>	Fraser*2/CAV 4540 ^c	;	;	;	0	;C
<i>Pc64</i>	Makuru*2//CAV4248/2*Sun II ^c	;	;	;	;	3
<i>Pc68</i>	Makuru*2//CAV4904/2*Sun II ^c	0	0;	0;	0;	0;
<i>Pc94</i>	S42 ^f	;	0	0;	0	0
<i>Pc96</i>	RL 1730 ^g	0;	;	0;	0;	X

^a Roelfs (26).

^b Kiehn et al. (16).

^c Chong et al. (8). It should be noted that Kiehn et al. (16) listed the *Avena sterilis* donor lines of *Pc38*, *Pc40*, and *Pc50* as CAV 2468, CAV 4997, and CAV 2463, respectively.

^d Simons et al. (31).

^e Harder et al. (15).

^f Aung et al. (3); pedigree of S42 is SunII/RL1697.

^g Chong and Brown (6).

the agronomy department of the Small Grain Institute. These replicated trials consisted of commercial cultivars as well as the most promising breeding lines.

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 (32). 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 Cape and Western Cape regions, respectively, and 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 (SBLL) and four others (SGLL, PBBB+Pc35, SDQL, and JBBM+Pc35) detected at low frequencies only (Table 1).

In field tests, lines with *Pc40*, *45*, *46*, *51*, and *54* displayed severities of more than 80S, while lines with *Pc60* and *Pc61* had severities of 20MRMS and 15MRMS, respectively. All other *Pc* lines were completely resistant (data not shown).

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 KwaZulu-Natal, 13 from the Free State, and two from Lesotho. These collections resulted in the establishment and differentiation of 242 single-pustule isolates. From these, pathotypes SAPga 1 (*Pg3,9,10,13,16,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*) were identified (Table 2). The avirulence-virulence formulae of each pathotype are shown as proposed by Martens et al. (21). 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 3).

In the field, high (50S to 80MS S) stem rust severities on lines containing either *Pga*, *1*, *4*, *9*, *15*, *1+2*, *2+4*, or *4+5* were recorded. Although virulence occurred for *Pg2* in seedling tests, this line was not heavily rusted (30MS) under field conditions.

Cultivar reaction. Cultivars and advanced breeding lines were evaluated in the seedling stage for their reaction to crown rust pathotypes SBLL, SGLL, PBBB+Pc35, SDQL, and JBBM+Pc35 (Table 4) and stem rust pathotypes SAPga 1, SAPga 2, SAPga 3, and SAPga 4 (Table 5). The South African cultivars Drakensberg, Kompasberg, Sederberg, and Tafelberg were resistant to all crown rust pathotypes tested, and high levels of stem rust resistance were displayed by Drakensberg. Adult plant reactions of oat elite lines to natural infections of crown and stem rust during the 1999 season are given in Table 6. Similar to the seedling responses, Sederberg, Kompasberg, and Tafelberg were resistant to crown rust in the field. Line OX87:073-10-14 was the only entry not showing any stem rust symptoms. Because of the longer growth period and cold requirement of Drakensberg, this cultivar

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*

<i>Pg</i> gene	CI number/ line designation	<i>P. graminis</i> f. sp. <i>avenae</i> pathotypes			
		SAPga 1	SAPga 2	SAPga 3	SAPga 4
<i>Pga</i>	Rodney-0/ <i>Pga</i> ^b	;	;	;	3
<i>Pg1</i>	CI9318 ^c	2+3	2+3	2+3	3
<i>Pg2</i>	CI 9319 ^c	3	3	;	3
<i>Pg3</i>	CI 9320 ^c	X	2	X-	2
<i>Pg4</i>	CI 6661 ^c	3	3	0	3
<i>Pg8</i>	CI 9321 ^c	3	3	3	3
<i>Pg9</i>	CI 9322 ^c	;2	2+3	;1	3
<i>Pg10</i>	X1588-2/2*Makuru ^b	0	X-	0	;C
<i>Pg13</i>	CI 9212 ^c	;	;1	;C	;1C
<i>Pg15</i>	CI 9351 ^c	2+3	2+3	3	3
<i>Pg16</i>	CI 9352 ^c	;C	;	X-	;C
<i>Pg17</i>	IB3056/2*Makuru ^d	2+3	2+3	2+3	3
<i>Pg1+2</i>	Minn. Ag.331 ^c	3	4	;	3
<i>Pg2+4</i>	Garry ^c	3	4	;	3
<i>Pg4+8</i>	<i>Pg4+8</i> line ^c	2+3	2+3	;	3
<i>Pg2+4+9</i>	Can-Ot-184 ^c	X	2+3	;	3

^a Roelfs (26).

^b Harder (12); Martens et al. (21) listed *Avena sterilis* S-66AB667/Kyto as the original source of *Pga* resistance.

^c Martens et al. (20).

^d Harder et al. (14). *Pg17* confers adult plant resistance and was not used as a differentiating line.

^e Adhikari (1).

Table 3. Distribution of *Puccinia graminis* f. sp. *avenae* pathotypes in South Africa during 1997 and 1998

Region	1997				1998			
	SAPga 1	SAPga 2	SAPga 3	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 of isolates identified per region.

^b Total percentage of isolates identified in survey.

is not recommended for the winter rainfall area and was therefore not included in this trial.

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*, and 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 showed the presence of one dominant crown rust pathotype, SBLL, with virulence to the resistance genes *Pc40*, *Pc45*, *Pc46*, *Pc51*, and *Pc54*, on cultivated and wild oat in South Africa during 1997 and 1998. This confirms the preliminary data of Pretorius et al. (25), who described the common occurrence of the same pathotype. Domination by a single pathotype is unusual since *P. coronata* f. sp. *avenae* is characterized worldwide by high levels of virulence diversity (7,17,30). The origin of pathotypes SGLL, PBBB+*Pc35*, and SDQL is uncertain. With the exception of JBBM+*Pc35*, which was collected from the *Pc64* line included in a

disease nursery near Riviersonderend in the Western Cape in 1999, no indication of their existence was obtained from the rating of differential lines under conditions of natural infection in the field. All four were initially detected in the greenhouse as single, susceptible-type pustules on host lines completely resistant to pathotype SBLL. Furthermore, the fact that two of these pathotypes were avirulent to Overberg, the cultivar on which field collections were originally increased, suggests that they may have arisen through mutation. It is also possible, however, that contaminant spores may have inadvertently been collected either from resistant (;1) or susceptible (33+) reactions on Overberg. Based on these results, it is clear that Overberg is not an appropriate host for increasing field collections of *P. coronata* f. sp. *avenae*. The resistance of Overberg to pathotypes such as PBBB+*Pc35* and JBBM+*Pc35* will lead to biased results and not give an accurate description of pathogenic variability in South Africa.

Despite this, valuable information was obtained on which *Pc* genes are ineffective in South Africa. The pathotypes identified also made it possible to postulate or con-

firm the occurrence of *Pc39* in Fidler (7), *Pc40* in Wisconsin X1588-2, and *Pc51* in Euro, Maluti, Overberg, OX88I 075-106, Perdeberg, and Swartberg (Table 4). These postulations were based on the differential pathogenicity of tester isolates toward these genes.

Comparison of South African pathotypes of *P. coronata* f. sp. *avenae* with those described from other parts of the world is difficult. The composition of differential sets as well as coding systems often differs among countries. According to the four-digit codes described by Chong et al. (8), none of the South African pathotypes resembled any of those from Mexico, the United States, or Canada. However, with regard to individual genes, virulence to *Pc39*, 40, 45, 46, 48, 51, 54, and 64 occurred in the latter countries as well as in South Africa. Although virulence to *Pc38*, 56, 58, 59, 62, and 68 has been reported elsewhere (8), these and other genes such as *Pc60*, 61, 63, 94, and 96 remain effective in South Africa. From Australian and New Zealand survey data, virulence to *Pc38*, 39, 58, 59, and 61 was common in 1998 and 1999, whereas virulence to *Pc68* was also detected in 1999 (23).

Table 4. Infection types^a produced on seedlings of South African cultivars as well as additional lines of *Avena sativa* by *Puccinia coronata* f. sp. *avenae* pathotypes SBLL, SGLL, PBBB+*Pc35*, SDQL, and JBBM+*Pc35*

Cultivars	<i>P. coronata</i> f. sp. <i>avenae</i> pathotypes				
	SBLL	SGLL	PBBB+ <i>Pc35</i>	SDQL	JBBM+ <i>Pc35</i>
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	;1	3	;1
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++
Wisconsin X1588-2	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 (26).

The lack of extensive pathogenic specialization in the local crown rust population may be due to the historic cultivation of highly susceptible cultivars and the susceptibility in wild oats, exerting little selection pressure for adaptation onto the oat crown rust population. 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 the use of Overberg excluded them from multiplication in the greenhouse.

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*, with only *Pg3*, *Pg10*, *Pg13*, and *Pg16* remaining effective (Table 2). *Pg12* was not included in the differential set but is represented in the cultivar Kyto (Table 5) (2). The effectiveness of *Pg17* to the South African rust population is not known. *Pg10* was previously identified as a useful source of resistance (12), whereas Šebesta (29) reported a lower frequency of virulence towards *Pg13*, *Pga*, and *Pg16* in Eurasia. On the other hand, none of the named *Pg* genes remains effective in Australia (23).

The 1997 survey was dominated by pathotypes SAPga 1 (43.1%) and SAPga 4 (30.4%), whereas pathotype SAPga 4 prevailed (67.7%) in 1998. The common isolation of pathotype SAPga 4 can be explained partly by the widespread cultivation of the cultivar Sederberg (approximately 80% of oat production) in the 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 since 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 predominantly northeasterly direction. Young oat plants in the summer rainfall area are subsequently infected, adding to the widespread distribution of SAPga 4.

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, OX881 075-106, Sederberg, and W94/4; *Pg2* and/or *Pg4* in Euro, Perdeberg, Potberg, and Swartberg; and *Pg9* in Pallinup and Victorian. According to Harder et al. (13) and Rothman (27), Wisconsin X1588-2 possesses resistance genes *Pg2*, *Pg4*, and *Pg10*. The data presented in this study show *Pg10* to be highly effective (Table 2), while Wisconsin X1588-2 is susceptible to pathotypes SAPga 2, SAPga 3, and SAPga 4 but resistant to SAPga 1 (Table 5). At present, this

discrepancy cannot be explained, and more tests with different seed stocks are necessary to interpret the *Pg10* and Wisconsin X1588-2 responses.

The information and oat rust pathotypes provided in this study should enable South African 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 cur-

Table 5. Infection types^a produced on seedlings of South African cultivars and lines of *Avena sativa* by *Puccinia graminis* f. sp. *avenae* pathotypes SAPga 1, SAPga 2, SAPga 3, and SAPga 4

Cultivar	<i>P. graminis</i> f. sp. <i>avenae</i> pathotypes			
	SAPga 1	SAPga 2	SAPga 3	SAPga 4
Alpha	;	;C	;C	2+3
Drakensberg	;	;C 1C	;	;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
OX881 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
Wisconsin X1588-2	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 (26).

Table 6. 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 and Piketberg (Western Cape) 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	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. [24]).

rently experienced will be minimized. It should be emphasized, however, that continual monitoring of these pathogens is necessary to provide meaningful contributions to oat rust control.

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