Usefulness of Gene *Pg10* as a Source of Stem Rust Resistance in Oat Breeding

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ABSTRACT

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Infection types produced by *Puccinia graminis* f. sp. *avenae* on plants of *Avena sativa* with the stem rust resistance gene *Pg10* are characterized by moderate-sized uredinia surrounded by an area of chlorosis and a larger variable zone of dark brown necrosis. This study was undertaken to assess the effectiveness of gene *Pg10* as a source of resistance to stem rust and to determine the interactions of this gene with other common *Pg* genes. A derived *Pg10* line was tested with 58 distinct pathotypes of *P. graminis* f. sp. *avenae* and was crossed to substituted single-gene lines carrying the resistance gene *Pg1*, *Pg2*, *Pg3*, *Pg4*, *Pg8*, *Pg9*, *Pg13*, *Pg15*,

Pg16, or *Pga*. The *Pg10* line showed moderate resistance to all 58 pathotypes, and there was no indication of specificity in virulence by any isolate. Gene *Pg10* was inherited independently of the other *Pg* genes and had a complementary effect on the expression of resistance by these genes. An effective level of resistance conferred by *Pg10* was demonstrated in a field nursery artificially inoculated with *P. graminis* f. sp. *avenae*. It was concluded that *Pg10* is a potentially useful source of stem rust resistance in oat breeding, with its main attributes being an apparent broad base of resistance, ease of combining with other *Pg* genes, and complementary effects on the expression of other *Pg* genes.

Additional keywords: gene interactions.

Gene *Pg10* was first identified (as gene G) in *Avena sativa* L. line CI 2824, a selection from 'Illinois' hulless (9). This gene was subsequently identified in the Wisconsin line X1588-2, which, in addition, was shown to carry genes *Pg3* and *Pg4* for stem rust resistance and *Pc95* for crown rust (*Puccinia coronata* Corda f. sp. *avenae* Eriks.) resistance (6). Line X1588-2 has been included as a supplemental line in the differential set used to identify survey isolates of *P. graminis* Pers.:Pers. f. sp. *avenae* Eriks. & E. Henn. at the Cereal Research Centre in Winnipeg for over 20 years. During this time, this line has not shown a fully susceptible reaction to any of the many thousands of field isolates inoculated to it and has performed well in nurseries in Wisconsin (6). When inoculated with pathotypes that are avirulent to either gene *Pg3* or *Pg4*, X1588-2 typically reacts with a highly resistant 1⁺ infection type (light chlorotic flecks with minute sporulation, according to the scale of Stakman et al. [11], with some necrosis). With pathotypes that are virulent to these genes, the infection type typically is a moderately resistant 1 to 3⁻ with a brown, somewhat watery-appearing ring of necrosis around the uredinia. Gene *Pg10* was subsequently isolated as a single gene in a 'Makuru' background (6). This study was undertaken to determine the potential effectiveness of gene *Pg10* against all isolates of *P. graminis* f. sp. *avenae* available at the Cereal Research Centre, the independence from and interactions with the *Pg* genes used as differentials in North America (8), and the performance of a *Pg10* line in an inoculated field nursery.

MATERIALS AND METHODS

Pathotypes of *P. graminis* f. sp. *avenae*. The pathotypes and their virulence formulas (effective/ineffective *Pg* genes) of the 58 iso-

lates of *P. graminis* f. sp. *avenae* used to determine the specificity of *Pg10* resistance are listed by Harder (5). This collection of pathotypes represents isolates collected since about 1952 from a wide geographic area in Canada and collectively covers virulence to most of the known sources of resistance (4,5). The pathotypes and their virulence formulas used to determine the genetic interactions of *Pg10* with other *Pg* genes were NA1, 1,2,3,4,8,9,13,16,a/15; NA26, 8,16,a/1,2,3,4,9,13,15; NA27, 9,13,15,16,a/1,2,3,4,8; NA28, 9,13,15,16/1,2,3,4,8,a; and NA55, 8,13,a/1,2,3,9,13,15,16.

Interactions of *Pg10* with other *Pg* genes. The plants containing *Pg10* used for crossing in this study were F₅ plants derived from an X1588-2/Makuru F₂ population (6). Each generation was tested with pathotypes NA1 (avirulent to genes *Pg3* and *Pg4*) and NA27 (virulent to genes *Pg3* and *Pg4*), and plants showing only the typical intermediate infection type with necrosis characteristic of *Pg10* were selected to eliminate genes *Pg3* and *Pg4*. A single F₃ plant was chosen to produce a bulked F₄ generation. Seeds from the F₄ plants were planted, the plants again tested with pathotypes NA1 and NA27 to ascertain the presence of only *Pg10*, and two plants that had infection types characteristic of *Pg10* were selected as pollen parents for subsequent crosses. Crosses were then made to Rodney-0 (a line in which 0 denotes no stem rust resistance genes present) lines carrying the substituted single genes *Pg1*, *Pg2*, *Pg3*, *Pg4*, *Pg8*, *Pg9*, *Pg13*, *Pg15*, *Pg16*, and *Pga*. Of these genes, *Pg8*, *Pg9*, and *Pg13* are recessive (7), and the *Pga* resistance has been indicated as due to two complementary recessive genes (1). About 45 to 50 F₂ seeds from each cross were planted, and seedling plants were inoculated with a race avirulent to the gene in question (NA1 for *Pg1*, *Pg2*, *Pg3*, *Pg4*, and *Pg8*; and NA27 for *Pg9*, *Pg13*, *Pg15*, *Pg16*, and *Pga*), and a pathotype virulent to the genes (NA27 for *Pg1*, *Pg2*, *Pg3*, *Pg4*, and *Pg8*; NA26 for *Pg9*, *Pg13*, and *Pg15*; NA55 for *Pg16*; and NA28 for *Pga*) was inoculated to the same plants 1 week later. The plants were scored for infection types according to Stakman et al. (11) 14 days after each inoculation. Although the populations used were somewhat limited for formal inheritance studies, χ^2 tests were performed to indicate the independence of the other *Pg* genes from *Pg10* by estimating the fit of segregating plants to expected ratios. The

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inheritance of all of the *Pg* genes used in this study has previously been described (1,7).

Specificity of *Pg10* resistance. Eight to ten seeds of the bulked F_4 generation, along with X1588-2 and 'Makuru', were planted in each of 58 10-cm pots. Each pot of seedlings was inoculated individually 10 days after planting with each of 58 different pathotypes of *P. graminis* f. sp. *avenae*. The plants were grown in a greenhouse at about 20°C supplemented with high-pressure sodium lighting (approximately 300 $\mu\text{mol m}^{-2} \text{s}^{-1}$). Infection types were scored 14 days after inoculation.

RESULTS AND DISCUSSION

Expression of *Pg10* resistance. In greenhouse tests on inoculated leaves of plants with *Pg10* (backcrossed into a Rodney-0 background), the uredinium size typically corresponded to a 2 to 3 infection type, according to the scale of Stakman et al. (11). The uredinia were surrounded by a small area of chlorosis that, in turn, was surrounded variably by a zone of dark brown, somewhat-watery appearing necrosis (Fig. 1). The brown necrosis began to appear early in the infection process, before the uredinia erupted. The brown discoloration occurred to a variable extent around the uredinia and frequently appeared as a shadowy area in the leaf blade well away from the uredinia. The phenotypic expression of infection involving *Pg10* is not known for any other source of resistance to *P. graminis* f. sp. *avenae*. The expression was not affected by age of plants or host background. Plants with *Pg10* inoculated with *P. coronata* f. sp. *avenae* did not show the brown necrosis (D. E. Harder, unpublished data), thus, this reaction was specific to infection by *P. graminis* f. sp. *avenae* (Fig. 1).

Interactions of *Pg10* with other *Pg* genes. The segregation of the F_2 progeny for resistance to *P. graminis* f. sp. *avenae* from crosses of the *Pg10* line with other *Pg* gene lines is shown in Table 1. In all crosses, there was segregation for resistant and susceptible plants. In the *Pg1/Pg10*, *Pg2/Pg10*, *Pg3/Pg10*, and *Pg4/Pg10* crosses inoculated with NA1, the segregation of plants fitted a 15:1 ratio (resistant/susceptible) expected for two dominant genes. Similarly, for the *Pg8/Pg10*, *Pg9/Pg10*, and *Pg13/Pg10* crosses inoculated with NA27, there was a marginally good fit to a 3:1 ratio (*Pg8/Pg10*) expected for a single gene, a 13:3 ratio (*Pg9/Pg10* and *Pg13/Pg10*) expected for a dominant and a recessive gene, and a 15:1 ratio expected for two dominant genes in the *Pg15/Pg10* cross. For the *Pg16/Pg10* cross inoculated with NA27, however, the fit to an expected 15:1 resistant/susceptible segregation ratio was poor ($P < 0.01$). Gene *Pg16* was originally isolated from the tetraploid *A. barbata* L. (2), and the *Pg16* parental line appeared to still carry a portion of the *A. barbata* chromosome. Brown et al. (2) indicated a reduced efficiency in the transmission of gametes involving *Pg16*, and this may, in part, have been responsible for the excess of susceptible plants in this cross. Also, Pavek and Myers (9) indicated partial dominance for *Pg10*, resulting in the misclassification of some heterozygous plants as susceptible. Poor transmission of *Pg16* and the partial dominance of *Pg10* in the current study may have resulted in the excess number of plants classified as susceptible, hence the poor fit to the expected segregation ratio. Generally, the fits to expected segregation ratios were marginal to poor, except for the crosses involving *Pg1* and *Pg4* inoculated with NA1 and for *Pg2* inoculated with NA27, in which the fits were good. The relatively small populations and the partial dominance of *Pg10* may have contributed to these results. The intent, however, was not to study inheritance, but to determine independence of *Pg10* from the other *Pg* genes. In all cases, there was segregation, and there was no evidence of linkage to any of the other *Pg* genes. It was concluded that gene *Pg10* is independent of *Pg1*, *Pg2*, *Pg3*, *Pg4*, *Pg8*, *Pg9*, *Pg13*, *Pg15*, and *Pg16*.

In the cross involving *Pga*, analysis of segregation was not attempted because of the apparent dependency of the expression of *Pga* resistance on the presence of two complementary recessive

genes (1). All of the F_2 plants were inoculated with pathotypes NA27 and NA28 (avirulent and virulent, respectively, to *Pga*) and subsequently grown to maturity. Of the 47 plants inoculated with NA28, 12 plants showed infection types characteristic of *Pg10*, and the remainder were uncertain as to their infection type or were susceptible. Of those inoculated with NA27, one plant showed the highly resistant 0; infection type characteristic of *Pga*. The latter plant and seven others that appeared to be homozygous for *Pg10*, based on their infection types when inoculated with NA28, were selected for progeny testing. Fifteen seedling progeny from each of these eight plants were inoculated with race NA27. The second leaves were inoculated 5 days later with race NA28. Any individual plant that showed a highly resistant 0; infection type to NA27 and the intermediate infection type with necrosis to NA28 should have *Pga* and *Pg10* combined. Of the eight plants progeny-tested, three segregated for the 0; and intermediate infection types, and one (the original plant showing the 0; infection type to NA27) was homozygous for the 0; infection type when inoculated with NA27. Among those plants with a 0; infection type, some plants showed

Fig. 1. Infection type produced by *Puccinia graminis* f. sp. *avenae* in the third leaves of a line of *Avena sativa* with resistance gene *Pg10* (backcrossed into the cv. Rodney), 14 days after inoculation. The uredinia typically are moderate in size (corresponding to a 2 to 3 infection type) and are surrounded by an area of chlorosis that, in turn, is surrounded by a zone of dark brown, watery-appearing necrosis that may extend to variable distances into the leaf tissue.

small, light brown, necrotic-appearing flecks, indicating the possible presence of *Pg10* (in plants with only *Pga*, any flecks that occurred as a result of infection were only faintly chlorotic, with no signs of necrosis). Subsequent tests on the second leaf with NA28 showed the intermediate infection type with necrosis in all plants that showed the 0; infection type to race NA27, indicating the presence of *Pg10*. All of the above plants that showed the 0; infection type to NA27 and the intermediate necrotic infection type to NA28 were grown to maturity. Progeny from these plants were again inoculated with NA27 on the seedling leaf and NA28 on the second leaf. All plants had a 0; infection type to NA27 and the intermediate necrotic infection type to NA28. It thus was evident that *Pga* and *Pg10* were combined in these plants, indicating that the *Pga* resistance and *Pg10* also are inherited independently.

The infection types characteristic for the *Pg* genes inoculated with avirulent pathotypes and the corresponding infection types for the same *Pg* genes in combination with *Pg10*, or when *Pg10* occurred alone, are shown in Table 2. Because the same plants were inoculated with pathotypes avirulent or virulent to each *Pg* gene other than *Pg10*, it was possible to determine which plants had the gene in question in combination with *Pg10*. When the *Pg* genes occurred in combination with *Pg10*, somewhat more highly resistant infection types were expressed than when the *Pg* gene occurred alone. The infections on plants with the combined genes were also accompanied by a variable expression of necrosis, indicating a further effect of *Pg10* on the expression of resistance. When the pathotype was virulent to the other *Pg* genes (NA27 to genes *Pg1* to *Pg8*; NA26 to genes *Pg9* to *Pg15*; and NA55 to gene *Pg16*), the expression of *Pg10* was similar to that in lines in which *Pg10* occurred alone. It thus appeared that *Pg10* enhanced the expression of the other *Pg* genes to those pathotypes that were used in the tests. The other *Pg* genes, however, appeared to have little effect on the expression of *Pg10*.

Specificity of *Pg10* resistance. Lines with *Pg10* reacted very uniformly to all 58 pathotypes that were inoculated, with infection types ranging from 1⁺ to 3⁻ (often on the same plant), and accompanied by the characteristic ring of dark brown necrosis. A common infection type was 2⁺-. The X1588-2 line reacted with a ;1- infection type to all pathotypes that were avirulent to either gene *Pg3* or *Pg4*, and with a 1⁺ to a 3⁻ infection type with necrosis to all other pathotypes. Rodney-0 was susceptible to all pathotypes. There was no indication in this test of pathogenic specialization involving *Pg10*. Also, Wisconsin 1588-2, which contains *Pg10* (6), has been included as a supplemental line in the differential set used to identify field survey isolates of *P. graminis* f. sp. *avenae* for over 20 years. During this time involving over 4,000 isolates of *P. graminis* f. sp. *avenae*, none were observed that were fully virulent to this line.

Performance in a field nursery. The *Pg10* line was included in a field nursery in 1997 that was inoculated with a mixture of six different pathotypes of *P. graminis* f. sp. *avenae*. The nursery contained a range of stem rust resistant and susceptible lines with which the *Pg10* line could be compared. The *Pg10* line was scored 30 MR to some MS-type infections (30 = percent infection, MR = moderately resistant, and MS = moderately susceptible). In comparison, susceptible lines were rated 80 S, and standard resistant oat cultivars such as 'Dumont', with genes *Pg2*, *Pg9*, and *Pg13*, were rated as 30 R to MR (D. E. Harder, unpublished data). These results indicate that *Pg10* as a sole resistance source provides relatively good protection against stem rust under artificially inoculated field conditions.

Conclusions and potential usefulness of *Pg10*. The *P. graminis* f. sp. *avenae* population in North America has been relatively stable for about the past 30 years, with few important changes in virulence (4). Many of the pathotypes with specific virulence that have occurred in the past can still be found on occasion in field

TABLE 1. Segregation of resistant and susceptible plants in F₂ progeny from crosses between a line of *Avena sativa* with *Pg10* and lines of *A. sativa* with other known *Pg* genes inoculated with pathotypes NA1, NA27, NA26, or NA55 of *Puccinia graminis* f. sp. *avenae*

Crosses involving <i>Pg</i> gene lines and <i>Pg10</i>	Pathotype ^a											
	NA1			NA27			NA26			NA55		
	R:S ^b	Ratio ^c	<i>P</i> ^d	R:S	Ratio	<i>P</i>	R:S	Ratio	<i>P</i>	R:S	Ratio	<i>P</i>
<i>Pg1</i>	43:4	15:1	0.7–0.5	28:19	3:1	0.02–0.01						
<i>Pg2</i>	38:5	15:1	0.2–0.1	31:12	3:1	0.7–0.5						
<i>Pg3</i>	43:1	15:1	0.3–0.2	25:19	3:1	<0.01						
<i>Pg4</i>	44:3	15:1	0.95–0.98	31:16	3:1	0.2–0.1						
<i>Pg8</i>				41:6	3:1	0.1–0.05	28:19	13:3	0.3–0.2			
<i>Pg9</i>				40:5	13:3	0.2–0.1	29:16	3:1	0.2–0.1			
<i>Pg13</i>				40:6	13:3	0.3–0.2	30:16	3:1	0.3–0.2			
<i>Pg15</i>				41:6	15:1	0.2–0.1	32:15	3:1	0.3–0.2			
<i>Pg16</i>				32:14	15:1	<0.01				29:17	3:1	0.1–0.05

^a All pathotypes are avirulent to *Pg10*, but vary in virulence to the other *Pg* genes as indicated by the expected segregation ratios.

^b Number of plants resistant/susceptible.

^c Expected segregation ratio.

^d P for χ^2 .

TABLE 2. Infection types typically produced on lines of *Avena sativa* carrying different *Pg* genes or on plants from crosses involving *Pg10* with the other *Pg* genes inoculated with pathotypes NA1, NA27, NA26, or NA55 of *Puccinia graminis* f. sp. *avenae*

	Infection types on lines with single <i>Pg</i> genes				Infection types on lines with <i>Pg</i> genes combined with <i>Pg10</i>			
	NA1	NA27	NA26	NA55	NA1	NA27	NA26	NA55
<i>Pg1</i>	1	4	4	4	0;1 ^{Na}	2–3 ^{-N}		
<i>Pg2</i>	1	4	4	4	0;1 ^{-N}	2–3 ^{-N}		
<i>Pg3</i>	1	4	4	4	0;1 ^{-N}	2–3 ^{-N}		
<i>Pg4</i>	;1	4	4	4	0;1 ^{-N}	2–3 ^{-N}		
<i>Pg8</i>	1–1 ⁺	4	1–1 ⁺	1–1 ⁺		2–3 ^{-N}		;1 ^{-N}
<i>Pg9</i>	1–2 ⁺	1–2 ⁺	4	4		;1 ^{-N}		2–3 ^{-N}
<i>Pg13</i>	;1	1	4	1		;1 ^{-N}		2–3 ^{-N}
<i>Pg15</i>	4	1 ⁺ –2 ⁺	4	4		;1 ^{-N}		2–3 ^{-N}
<i>Pg16</i>	;1	1	1	4		;1 ^{-N}		2–3 ^{-N}

^a N = Brown necrosis accompanying the infection site.

survey collections (3), and virulences may be retained in the population for long periods (4). Gene *Pg13* currently provides the main effective resistance in oat cultivars in the rust area of the Canadian prairies. Genes *Pg9* and *Pg2* also occur in most of these cultivars. Historically, virulence to gene *Pg13* was quite high in the prairie region (4). The pathotypes that were virulent to *Pg13* (NA3 and NA7) were avirulent to *Pg2*. With the widespread use of the latter gene in oat breeding in the 1950s, and considering that the *P. graminis* f. sp. *avenae* population in the prairie region of North America is asexual with relatively stable virulence clusters (10), virulence to *Pg13* in this region virtually disappeared. This is a case in which an ineffective gene such as *Pg2* appears to have played an important role in extending the effectiveness of *Pg13* by preventing the reappearance of pathotypes in virulence clusters that are virulent to *Pg13*. In 1998, however, some stem rust was noted in fields in Manitoba on resistant cultivars such as 'AC Assiniboia'. Collections of *P. graminis* f. sp. *avenae* from 'AC Assiniboia' and some from wild oat elsewhere in Manitoba were identified as pathotype NA67, which has the avirulence/virulence formula *Pg16,a/1,2,3,4,8,9,13,15* (B. D. McCallum and D. E. Harder, unpublished data). A similar pathotype, NA75 (*Pg3,16,a/1,2,4,8,9,13,15*), was identified in the Great Plains region of the United States in 1996 (samples originally received from D. M. McVey, USDA Cereal Disease Laboratory, St. Paul, MN, and verified by D. E. Harder). These pathotypes could pose a threat to oat production in the plains region of North America, and it is urgent that additional resistance be incorporated into new oat cultivars. NA67 was included among the 58 pathotypes used to test the specificity of *Pg10*, and the reaction was moderate resistance with necrosis, similar to the reactions with all the other pathotypes. Except for *Pg16*, there are no other sources of resistance available in hexaploid oat that confer resistance to all pathotypes that have been found in the Great Plains region of North America. The usefulness of *Pg10* as a source of resistance in oat breeding may have been overlooked, possibly because of the intermediate infection types produced by lines with this gene. There was no virulence to *Pg10* identified among the 58 specific pathotypes tested, and there has been no evidence of specific virulence to X1588-2 (containing *Pg10*) among the field isolates of *P. graminis* f. sp. *avenae* collected and tested for over 20 years. In the test to determine the effectiveness of *Pg10* in the field, a useful level of resistance was demonstrated. It would not be advisable to use *Pg10* alone as a resistance source, but since it was shown to be independent of most other common *Pg* genes and, in

addition, appeared to complement their effectiveness, this gene would be a useful adjunct in combination with other *Pg* genes. Also, it would be relatively easy to test for the presence of *Pg10* in most combinations with other *Pg* genes in breeding populations because of the unique infection phenotype expressed by *Pg10*, and isolates of *P. graminis* f. sp. *avenae* are available at the Cereal Research Centre in Winnipeg that have specific virulence to all other *Pg* genes. With no specificity in virulence involving *Pg10* detected to date after extensive testing, it may offer a more durable resistance source and, as well, could contribute to longer term durability of other resistance genes.

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