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A storage-protein marker associated with the suppressor of Pm8 for powdery mildew resistance in wheat

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Abstract A suppressor of resistance to powdery mildew conferred by *Pm8* showed complete association with the presence of a storage-protein marker resolved by electrophoresis on SDS-PAGE gels. This marker was identified as the product of the gliadin allele *Gli-A1a*. The mildew-response phenotypes of wheats possessing the 1BL.1RS translocation were completely predictable from electrophoretograms. The suppressor, designated *SuPm8*, was located on chromosome 1AS. It was specific in its suppression of *Pm8*, and did not affect the rye-derived resistance phenotypes of wheat lines with *Pm17*, also located in 1RS, or of lines with *Pm7*.

Key words *Triticum aestivum* · Rye · *Secale cereale* · 1BL.1RS translocation · 1AL.1RS translocation · Gliadin · Electrophoresis · *Erysiphe graminis*

Introduction

The 1BL.1RS wheat-rye translocation originally developed in Europe (Mettin et al. 1973; Zeller 1973) has been extensively studied (e.g. Bennett 1984; Heun and Fischbeck 1987; Singh et al. 1990; Gupta and Shepherd 1992; Hartmann et al. 1994) and widely utilised in wheat breeding programs throughout the world (Rajaram et al. 1983; Sreeramulu and Singh 1994). The frequent superior performance of wheat cultivars with this chromosome has been attributed to a number of factors, including disease resistance and wide adaptation (Rajaram et al. 1983; Villareal et al. 1991; McIntosh et al. 1993). However, the bread-making quality of wheats with the 1BL.1RS translocation has posed a potential problem since doughs produced from them have a tendency to become sticky especially with overmixing (Zeller et al. 1982; Martin and Stewart 1990; Singh et al. 1990).

Recent studies (Heun and Fischbeck 1987; Friebe et al. 1989; Heun and Friebe 1990; Lutz et al. 1992) drew attention to the fact that not all wheats with the 1BL.1RS translocation were resistant as seedlings to *Pm8*-avirulent isolates of *Erysiphe graminis* f. sp. *tritici*, the causal fungal pathogen of wheat powdery mildew. This led to suggestions that *Pm8* was not located on the 1RS chromosome segment, that the 1RS chromosome segment had been altered such that *Pm8* was deleted, or that there was genetic suppression of the resistance conferred by *Pm8*. Hanusova (1992) and Ren et al. (1997) showed that *Pm8* was present in the 1RS chromosome segment and that in many genotypes it was suppressed by a dominant gene located in the wheat genotype. In the present paper we describe studies by which the suppressor was associated with a storage-protein marker gene located on the short arm of chromosome 1A.

Materials and methods

Plant materials

F_7 bulks from F_6 single-seed-descent (SSD) lines, derived from individual F_2 plants of crosses Kohinoor 83/WW31 and Punjab 85/WW31, together with the respective parents were used to investigate the relationship between powdery mildew response and endosperm storage-proteins. These were previously scored for the presence or absence of the 1BL.1RS translocation based on rust responses and for powdery mildew response (Ren et al. 1997).

Studies were extended to backcross-derived lines based on a wide range of Australian wheat cultivars and advanced crossbreds as recurrent parents and four sources of the 1BL.1RS translocation, as well as cv Amigo as a source of the 1AL.1RS translocation possessing a gene for resistance to stem rust and *Pm17* for resistance to powdery mildew, and cv Transec as a source of *Lr25* for resistance to leaf rust and *Pm7* for resistance to powdery mildew (The and Bell

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1993). All backcross derivatives had been confirmed as possessing 1BL.1RS, 1AL.1RS or *Lr25,* based on rust tests.

Nine selections of Veery supplied by Dr. D. Mares, PBI Narrabri, but deriving from accessions held by the Australian Winter Cereals Collection, Tamworth, NSW, were also examined. A monosomic 1A stock of Glennson 81 (Veery #1) was kindly provided by Mr. A. J. Worland, Cereals Department, JI Centre, Norwich, UK.

Rust and powdery mildew tests

Seedling rust tests were performed by routine methods described in McIntosh et al. (1995).

A local isolate of *E. graminis* f. sp. *tritici* was used for powdery mildew tests. This isolate was avirulent on seedlings of reference stocks with *Pm8* (Ren et al. 1997). SSD lines, parents, and control lines were sown at two or three lines per 9-cm diameter pot and 8–10 seeds per line. Seedlings were inoculated either at the first leaf stage or at completion of rust tests 3–4 weeks after planting. Conidia of *E. graminis tritici* were applied by dusting or brushing from sporulating seedlings. Reactions were scored 10–14 days after infection either on a 0, 1 to 4 infection-type scale with 0 representing no visible symptoms for necrotic flecks, and 1 to 4 describing the sporulating areas, with 3 and 4 being interpreted as susceptible or alternatively, when controls were clearly distinguished, as resistant and susceptible. There was no evidence for the presence of any powdery mildew resistance gene other than *Pm8* in the materials examined.

Extraction of proteins

Unreduced total proteins were extracted from endosperm from the non-embryo halves of single kernels with 200 µl of extraction buffer [2.3% (w/v) SDS, 10% (v/v) glycerol, 10% sucrose, 0.05% bromophenol blue, 62.5 mM Tris-HCl, pH 6.8] in boiling water for two min. After centrifuging at 13 000 *g* for 5 min, 20 µl of supernatant was loaded into sample wells for electrophoresis. Tests were usually performed separately on four seeds per cultivar or line.

In an experiment involving the progenies of a Glennson 81 monosomic 1A stock, a similar electrophoretic procedure was performed on protein reduced by treatment with 2% mercaptoethanol from individual half-seeds of four putative nullisomics and a Glennson 81 control.

SDS-PAGE

Discontinuous one-dimensional SDS-PAGE was carried out using 10–17% acrylamide gels of 1 mm thickness. Gels contained 10–17% acrylamide, 1.34% bisacrylamide, 0.1%(w/v) SDS and 1 M Tris-HCl, pH 8.8. Electrophoresis was performed at a constant current of 7.5 mA/gel overnight. The gels were stained for about 1 h using Coomassie Brilliant Blue R-250, and then destained for about 30 min in de-staining solution (20% MeOH, 7% acetic acid, 3% glycerol). After de-staining, gels were immersed in water and the composition of endosperm proteins analysed.

Chromosome counts

Root-tips were removed from the germinating half-seeds of the progeny of a Glennson 81 monosomic 1A plant and pre-treated in ice water for 24 h prior to fixation (3 ethyl alcohol:1 acetic acid) for 24 h, hydrolysis in 1N HCl for 10 min and staining in leuco-basic fuchsin. Chromosome counts were made on squashes prepared from the stained tips. The seedlings were planted in pots and, at the 2-leaf stage, were inoculated with *E. graminis tritici* as above.

Results

Kohinoor 83/WW31 SSD lines

Thirty F_7 lines, previously shown to possess the 1RS-located genes *Lr26*, *Sr31* and *Yr9* on the basis of rust tests, were subjected to SDS-PAGE electrophoresis to confirm the presence of the rye gene *Sec-1* and the absence of the wheat gene *Gli-B1*. These genes, responsible for the production of low-molecular-weight glutenins, secalin and gliadin, respectively, are probably located at equivalent positions in the respective group-1 chromosomes of the two species. The same lines along with the parents were tested for response to powdery mildew (Table 1). Sixteen lines (group A) were homozygous-resistant producing symptoms typical of the Kohinoor 83 parent and of other lines possessing *Pm8* (Fig. 1) whereas 14 lines (group B) were homozygous-susceptible. The electrophoretograms for powdery mildew-resistant and susceptible lines differed in respect of the absence and presence, respectively, of a storage-protein band of approximately Mr=42.2 kDa temporarily designated as "suppressor band" (SB). This band was present in the WW31 parent.

To further examine this relationship between suppression of powdery mildew resistance and SB, one SSD line from Kohinoor 83/WW31 which was scored homozygous

Table 1 Relationship between the presence (+) or absence (–) of *Sec-1* and the suppressor band (SB), and disease response in SSD derivatives of Kohinoor 83/WW31

Item		No. lines	*Sec-1*[a]	SB	Reaction to	
					Leaf rust[a]	Powdery mildew
Parents						
Kohinoor 83		1	+	–	Resistant[b]	Resistant[c]
WW31		1	–	+	Susceptible	Susceptible
SSD lines						
Group	A	16	+	–	Resistant	Resistant
	B	14	+	+	Resistant	Susceptible
	C	30	–	–	Susceptible	Susceptible
	D	32	–	+	Susceptible	Susceptible

[a] The presence of *Sec-1* and *Lr26* indicates the presence of chromosome 1BL.1RS. Lines with *Lr26* also carried *Yr9* and *Sr31*
[b] Resistance attributed to *Lr26*
[c] Resistance attributed to *Pm8*

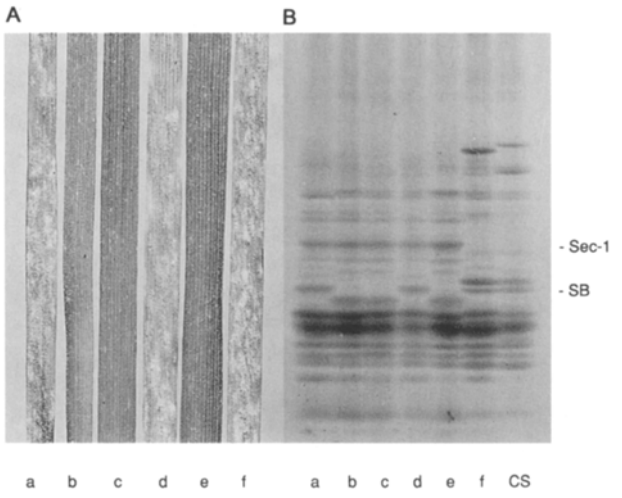

Fig. 1 **A** Powdery mildew responses of parents and SSD lines selected with 1BL.1RS from the cross Kohinoor 83/WW31. **B** SDS-PAGE electrophoretograms of unreduced proteins with Chinese Spring (CS) in the last lane. *Sec-1* indicates the presence of 1BL.1RS; SB is present in *CS*, *a*, *d and f*. For both **A** and **B** the genotype order is: *a to d* SSD lines K/WW#119, #114, #110, #107, *e* Kohinoor 83, *f* WW31

for 1BL.1RS, but segregating for mildew resistance in the previous study (Ren et al. 1996), was examined. The distal halves of 30 seeds were subjected to electrophoretic analysis whereas proximal halves containing the embryos were germinated and tested for powdery mildew response. Of the 27 seedlings that resulted, eight were resistant and lacked SB and 19 were susceptible and possessed SB. The same gels confirmed the presence of 1BL.1RS in all plants.

Sixty two SSD lines that lacked 1BL.1RS, and were susceptible to powdery mildew, were also subjected to electrophoresis; 32 lines (group D) possessed SB and 30 lines (group C) lacked it, demonstrating single-gene segregation within the wheat genome.

Two F_1 hybrids were generated and examined to verify the dominance of *Pm8* and of the suppressor. F_1 seedlings from K/WW#29 (1BL.1RS, SB–)×K/WW#16 (normal 1B, SB-) were resistant to powdery mildew, whereas those from K/WW#130 (normal 1B SB+)×K/WW#114 (1BL.1RS, SB–) were susceptible. These results were as predicted from the earlier studies.

Punjab 85/WW31 SSD lines

Sixteen lines with 1BL.1RS and nine lines lacking it were tested in the same way, as were lines from Kohinoor 83/WW31. Of the 1BL.1RS lines, eight carried SB and were susceptible to powdery mildew and eight which lacked SB were resistant. Among the nine lines identified as lacking 1BL.1RS, five possessed SB and four did not. It was of interest that one of this last group (*P/WW31* #10) not only lacked *Sec-1* but also lacked *Gli-B1*, while earlier results showed it to be lacking the rust-resistant genes. It possessed 42 chromosomes, but only two chromosomes showed satellites. The chromosomes of plants from this line were C-banded by Dr. A. Lukaszewski, University of California, Riverside, USA. The relevant chromosome carried a normal 1BL and a discernible 1RS segment extending at least to the characteristic band identifying the nucleolar organising region (NOR). Distal to the NOR there were no distinguishing bands, indicating the possibility of a terminal deletion or translocation to a wheat chromosome such as 1A with no distinctive C-bands in the terminal region. In any case, both *Sec-1* and the rust resistance genes had evidently been lost.

Australian backcross derivatives possessing 1BL.1RS

Because of the widespread interest and agronomic success of the 1BL.1RS chromosome in wheat production in many countries, this chromosome was introduced to a wide array of Australian cultivars and breeding lines over several years. Four cultivar donor sources were used. In many combinations only one cross or few backcrosses were undertaken prior to selfing and extraction of homozygous lines for distribution to wheat breeders. While the source and number of crosses to the final recurrent parents (cultivars and advanced crossbreds) were known, some of the intermediate steps in the crossing program were not recorded. However 81 lines that were homozygous for the 1BL.1RS chromosome were available for leaf rust and powdery mildew tests and for electrophoresis. The results are summarised in Table 2. Of 81 lines, 42 were resistant to powdery mildew and lacked SB, 29 were susceptible and carried SB, and ten lines segregated for reaction to powdery mildew and for SB.

Table 2 Donor sources of the 1BL.1RS chromosome, their SB status and response to powdery mildew (PM), and phenotypes for backcross derivatives

Source of 1BL.1RS	SB status	PM response	No. recurrent parents	Phenotypes of backcross derivatives[a]		
				SB–ve PM res	SB+ve PM sus	SB seg PM seg
Veery[b]	+	Sus	19	7	7	5
Skorospelka 35	–	Res	33	17	13	3
Aurora	–	Res	16	9	7	0
Disponent	–	Res	13	9	2	2
Totals			81	42	29	10

[a] –ve=SB absent, +ve=SB present, Res=resistant, Sus=susceptible, Seg=segregating
[b] Unknown source, but believed to be Veery#5

Table 3 Donors of 1BL.1RS to ten cultivars and advanced crossbreds, their SB phenotypes, and the SB phenotypes and powdery mildew responses of the backcross derivatives

Recurrent parent[a]		Donors[b]	Backcross derivatives	
Cultivar/line	SB		SB	PM
Condor	+/–	Au, Sk	+, +	S, S
Halberd	–	Au, Sk	–, –	R, R
Mokoan	–	Au, Sk	+, –	S, R
Oxley	–	Dis, Sk	–, –	R, R
Vulcan	–	Dis, V	–, –	R, R
CO1568	+	Au, Dis	+, +	S, S
CO1650	–	Au, Dis	–, –	R, R
RAC177	–	Au, Sk	–, –	R, R
TM56	+/–	Au, Sk	+, +	S, S
XL23	–	Dis, Sk	–, –	R, R

[a] Suppressor band + = present, – = absent
[b] Au = Aurora (SB–), Dis = Disponent (SB–), Sk = Skorospelka 35 (SB–), V = Veery (SB+)

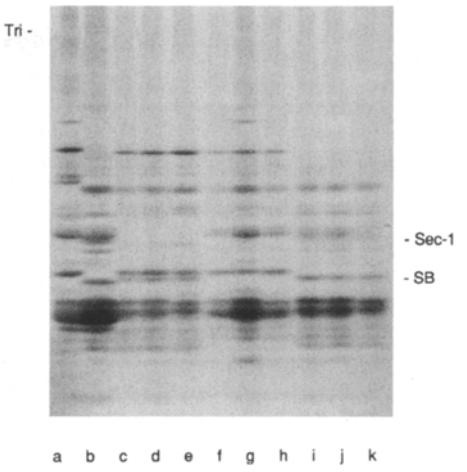

Fig. 2 SDS-PAGE electrophoretograms of unreduced proteins from backcross lines carrying 1BL.1RS or 1AL.1RS and relevant donor and recurrent parents. *Lane a* Amigo, SB–; *b* Skorospelka 35 (Sk), SB–; *c* Osprey, SB+; *d* Condor, SB+; *e* Bayonet, SB+; *f* Amigo/4*Osprey (accession 103058), SB–; *g* Amigo/6*Condor (146932) SB–; *h* Amigo/4/Bayonet (66996), SB–; *i* Sk/3*Osprey (35335) SB+; *j* Sk/6*Condor (50179), SB+; *k* Sk/4*Bayonet (22264), SB+

In Table 3 the SB identifications of the backcross derivatives, involving either named cultivars or experimental lines where two donor sources were used, and of the recurrent parents are compared. Except for Mokoan where the paired lines differed, the classifications of SB in the extracted lines and the recurrent parents were in agreement. In most of the other combinations they also agreed. Instances of non-agreement were not unexpected given that Veery possessed SB and the other three donors did not, as well as the unknown steps in the generation of some of the lines, the limited numbers of backcrosses from one to five (data not presented) and the heterogeneity for SB that occurred in a number of parents. However, there was perfect correlation between the presence of SB and suppression of powdery mildew resistance conferred by *Pm8*.

Australian backcross derivatives possessing 1AL.1RS and chromosome location of SB

An experiment was set up with the purpose of determining the effect of SB on a second rye-derived gene, *Pm17*, also located on chromosome 1RS. A number of Australian backcross derivatives with the 1AL.1RS chromosome derived from Amigo were available. All lines with the 1RS segment were resistant to powdery mildew irrespective of the status of SB established earlier for the recurrent parents. This suggested that *Pm17* was not suppressed. Further studies, however, established that SB could not be combined with *Pm17* indicating that SB and the gene responsible for suppression were located in chromosome 1AS. Some relevant results are given in Table 4 and illustrated in Fig. 2. Although neither Skorospelka 35 (1BL.1RS) nor Amigo (1AL.1RS) carry SB, it was present in the four recurrent parents to which both chromosomes were transferred. *Pm8* in the four backcross derivatives with 1BL.1RS was suppressed whereas *Pm17* in the 1AL.1RS lines was expressed. To further verify the distinctness of the backcross derivatives, the presence or absence of the storage-protein marker *Tri-A1* in chromosome 1AS was also recorded (Table 4).

Due to exclusion of SB from lines possessing 1AL.1RS the above tests were not a valid assessment of the effect of the suppressor on the expression of *Pm17*. F_1 hybrids involving 31 Australian cultivars and advanced crossbreds were available for assessment. The relationship between the presence or absence of SB and response to powdery mildew is shown in Table 5. It is clear that the presence of SB had no effect on the dominant resistance conferred by *Pm17*. The reason for the absence of SB in the Canna hybrid is not clear, but was presumably the result of undetected heterogeneity in the parent line.

Australian backcross derivatives with *Lr25* and *Pm7*

Pm7 (linked with *Lr25*) is located in a different rye-derived segment translocated to chromosome 4BS. Backcross derivatives of Condor, Teal and Oxley with *Pm7* were used to investigate the effect of SB on *Pm7*. Although Condor is heterogeneous for SB, four of its derivatives selected for *Lr25* were shown to carry SB. These four lines, along with the derivatives of Teal and Oxley which lack SB, were resistant to both diseases. It was apparent, therefore, that SB has no effect on the expression of resistance conferred by *Pm7*.

Formal location of SB and the suppressor of powdery mildew resistance in chromosome 1AS

At an advanced stage of the studies 23 half-seeds from a Glennson 81 (=Veery #1, SB+, powdery mildew suscepti-

Table 4 Chromosome status, protein phenotypes and response to powdery mildew (PM) of backcross derivatives of two donor lines, four recurrent parents and their backcross derivatives

Pedigree/cultivar	Line designation	Chromosome type	*Tri-A1*	*Sec-1*	SB	PM response
Skorospelka 35/						
4*Bayonet	22264	1BL.1RS	+[a]	+	+	S
6*Condor	50179	1BL.1RS	+	+	+	S
3*Osprey	35335	1BL.1RS	+	+	+	S
3*TM56	49053	1BL.1RS	+	+	+	S
Amigo/						
4*Bayonet	66996	1AL.1RS	–	+	–	R
6*Condor	146932	1AL.1RS	–	+	–	R
4*Osprey	103058	1AL.1RS	–	+	–	R
4*TM56	66762	1AL.1RS	–	+	–	R
Amigo		1AL.1TS	–	+	–	R
Skorospelka 35		1BL.1RS	+	+	–	R
Bayonet		Normal	+	–	+	S
Condor		Normal	+	–	+	S
Osprey		Normal	+	–	+	S
TM56		Normal	+	–	+	S

[a] + = present, – = absent, R = resistant, S = susceptible

Table 5 The relationship between the presence/absence (+/–) of suppressor (SB) band and seedling powdery mildew (PM) responses of F1 hybrids of Amigo and Australian wheat lines

Cultivar	Parent		F_1 Hybrid	
	SB	Response to PM	SB	Response to PM
Condor	–/+[a]	S	+	R
Bayonet	+	S	+	R
Spear	–/+[a]	S	+	R
Osprey	+	S	+	R
CSP44	+	S	+	R
Millewa	+	S	+	R
4 Others[b]	+	S	+	R
3 Others[b]	–/+[a]	S	+	R
Canna	+	S	–	R
Oxley	–	S	+	R
Cook	–	R[d]	+	R
15 Others[c]	–	S	+	R

[a] Heterogeneous. In all cases the actual subline used as parent was classified as +

[b] Unnamed advanced crossbreds

[c] Includes named cultivars (Hartog, Banks, Egret, Matong, Mokoan, Dagger, Bindawarra, Jacup, Miling, Torres, Flinders and Tatiara) and three unnamed advanced crossbreds

[d] Resistance due to *Pm6*

ble) monosomic 1A stock were subjected to protein analysis while, independently, the germinated seedlings were subjected to chromosome counts and a test for powdery mildew response. The chromosomal status was also predicted from the intensities of the relevant bands on the electrophoretograms. Six plants were predicted to be disomic, 13 monosomic and four nullisomic for 1A based on the intensities and absence of SB. Reduced proteins from the four putative nullisomics and a Glennson control enabled the classification of the *Glu-A1* locus on chromosome 1AL. Three plants lacked the *Glu-A1* band and were presumed to be nullisomic 1A, whereas one, like Glennson 81, possessed *Glu-A1a* indicating the presence of the long arm of chromosome 1A.

The above chromosome-number predictions were confirmed by cytology except that two of the plants predicted to be disomic were scored as 2n=43. The plant lacking SB and having *Glu-A1a* possessed 40 normal chromosomes and a telocentric. The disomic and monosomic seedlings were susceptible to powdery mildew, whereas the three nullisomics and the monotelocentric were resistant. The combined results of protein analysis and chromosome counts showed that the monosomy involved chromosome 1A and that the 40-chromosome powdery mildew-resistant plants were nullisomic for this chromosome. Moreover, the fact that these plants and the powdery mildew-resistant plant with 40 + t chromosomes lacked SB, showed that SB must be located in chromosome 1AS.

Association of SB with the Gli-A1 locus

Having established the location of SB in chromosome 1AS it was possible to relate the present results with published lists of alleles at the *Gli-A1* locus in Australian wheats (Metakovsky et al. 1990; Gupta et al. 1993; Gupta et al. 1994). The identifications for SB in named cultivars and selected lines from the current study are compared in Table 6 with allelic identifications for the *Gli-A1* and *Glu-A3* loci available from other sources. The data indicate that powdery mildew suppression is likely to be associated with the allele *Gli-A1a*.

The SDS-PAGE method of protein separation used in this study did not identify variation at the *Glu-A3* locus which is closely linked with *Gli-A1*.

Table 6 Relationship between the suppressor band (SB) and alleles identified by other workers for the *Gli-A1* and *Glu-A3* loci

Cultivar/line	SB	*Gli-A1* allele[a]	*Glu-A3* allele[b]
Aroona	+	.[c]	c
Bayonet	+	a	c/e
Canna	+	.	c
CSP 44	+	a	c
Kelalac	+	.	c
Lilimur	+	.	c
Millewa	+	a	c
Osprey	+	a	c
Wyuna	+	.	c
Condor	+/–	a/g	b/c
Egret	+/–	a/l	c/e
Banks	–	g	b
Bindawaara	–	.	b
Bodallin	–	.	e
Cook	–	g	b
Dagger	–	b	c
Gutha	–	.	b
Halberd	–	m	e
Hartog	–	g	b
Jacup	–	.	b
Lance	–	b	c
Lark	–	.	b
Lowan	–	.	d
Miling	–	.	b
Mokoan	–	f	e
Oxley	–	g	b
Spear	–	m	e
Tatiara	–	b	c
Teal	–	f	e
Vulcan	–	.	b

[a] Gupta et al. (1993) and Metakovsky et al. (1990)
[b] Most data same as [a], some cited from the 'Genejar' database (Cornish G.B., personal communication)
[c] not recorded

Ren et al. (1997) reported that only #6 of nine selections of Veery was resistant to powdery mildew. When subjected to electrophoresis this line was the only selection which did not carry SB.

Discussion

Friebe et al. (1989) reported seedling susceptibility to powdery mildew in European wheat cultivars Olymp, Florida and Heinrich, which possess the 1BL.1RS translocation, when tested with pathogen cultivars avirulent on other lines with *Pm8*. Although they suggested reasons to account for the incomplete association of powdery mildew resistance and the presence of 1BL.1RS they also noted that these lines possessed only one major 1RS-controlled protein band when extracts were electrophoresed on PAGE gels. However, cv Disponent which expressed resistance to powdery mildew gave a similar result. In this and a previous study (Ren et al. 1997) we provide evidence that all wheats with the 1BL.1RS translocation derived from early work in Germany (Kattermann 1937, cited in Zeller 1973) actually possess *Pm8* in the 1RS segment, but that in many wheats this resistance is suppressed by a gene in wheat chromosome 1AS. The suppressing allele showed complete association with a specific storage-protein detected on SDS-PAGE gels and was identified as *Gli-A1a*. Each gliadin locus in wheat is apparently responsible for a number of bands separated in SDS-PAGE gels. In the present study we focussed only on a single band which occurs at approximately Mr 42.2 kDa and which is not indicated on any of the photographs published by Singh and Shepherd (1988), Gupta and Shepherd (1992) and The et al. (1992).

Ren et al. (1997) noted a high frequency of powdery mildew susceptibility (*Pm8* suppression) in CIMMYT-distributed germplasm with 1BL.1RS compared with European wheat cultivars possessing the same translocation chromosome. Although Payne et al. (1987) attempted to implicate a role for particular gliadin fractions in aspects of flour and dough quality, there is no evidence to support the selection of *Gli-A1a* in favour of alternative alleles. The *Gli-A1* locus is closely linked with the *Glu-A3* locus which, according to Gupta and Shepherd (1988), may have a more significant influence on industrial quality. The wheats identified here as carrying SB and *Gli-A1a* (Table 6) carry mainly *Glu-A3c*. While Bayonet appears to be homogeneous for SB and *Gli-A1a*, it is heterogeneous for alleles *c* and *e* at *Glu-A3*. Cultivars Condor and Egret are heterogeneous for all three loci and, although not established in this study, the *Gli-A1a* and *Glu-A3c* are likely to be in coupling phase in both stocks. However, *Glu-A3c* occurred in three South Australian wheats (Dagger, Lance and Tatiara) which were identified as having *Gli-A1b* (Gupta et al. 1993) but lacking SB (our results). It appears, therefore, that the high frequency of suppression did not result from selection for some aspect of quality.

In wheat genetic nomenclature for genes determining protein traits, the allele of the widely accepted reference line Chinese Spring is usually designated "*a*". Chinese Spring carries the SB band and preliminary results indicated that *Pm8* is ineffective in that background. In our study the SB (*Gli-A1a*) band in Chinese Spring and the gliadin (*Gli-A1c*) linked to *Glu-A1c* in other wheats (Gupta et al. 1994; Gupta, personal communication) could not be distinguished. Using an ACID-PAGE system Gupta et al. (1993) earlier designated the gliadin allele in these wheats as *Gli-A1a*. Clearly the alleles are difficult to distinguish and Metakovsky (1991) actually considered them to be closely related and probably differing by a mutation affecting mobility.

Our observation of the chromosome location of the suppressor of *Pm8* was a serendipitous outcome of an attempt to determine if the effect of that gene was specific to *Pm8* or whether it had a suppressive effect on the expression of other resistance genes derived from rye. Because *Pm17* and the suppressor were mutually exclusive in homozygous stocks, the test had to be performed on heterozygotes where there was no evidence for suppression (although we have not formally excluded the possibility that it acts as a reces-

sive suppressor of *Pm17*). Furthermore, there was no suppression of the resistance conferred by *Pm7*.

It is of interest that the suppressor (designated *SuPm8*, a wheat gene) of *Pm8* (derived from rye) was located to a homoeologous chromosome, possibly in the vicinity of the polyallelic *Pm3* locus. Although suppression of resistance may be more common in interspecific crosses and polyploids, relative to diploid species, there are few indications that such genes are more likely to involve related chromosomes. R.P. Singh (personal communication) recently mapped a suppressor of *Lr23* (a *T. turgidum*-derived resistance, McIntosh et al. 1995), located in chromosome 2B, to a likely homoeologous region of chromosome 2D.

The genetic linkage between *SuPm8* and *Gli-A1a* must be extremely close. Because the suppressor is dominant we predict that mutagenic treatment of a suppressed genotype possessing 1BL.1RS will yield segregates that are mildew-resistant with the specificity of *Pm8*. This experiment is already under way using a powdery mildew susceptible accession of Veery as the source genotype. Resistant mutants will be examined for the status of the *Gli-A1* locus in consideration of the unlikely possibility that *SuPm8* and *Gli-A1a* may be the same gene.

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