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Quantitative trait loci associated with isolate specific and isolate nonspecific partial resistance to *Phoma macdonaldii* in sunflower

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Black stem, caused by *Phoma macdonaldii*, is one of the most important diseases of sunflower in the world. Quantitative trait loci (QTLs) implicated in partial resistance to two single pycnidiospore isolates of *P. macdonaldii* (MP8 and MP10) were investigated using 99 recombinant inbred lines (RILs) from the cross between sunflower parental lines PAC2 and RHA266. The experimental design was a randomized complete block with three replications. High genetic variability and transgressive segregation were observed among RILs for partial resistance to *P. macdonaldii* isolates. QTL-mapping was performed using a recently developed high-density SSR/AFLP sunflower linkage map. A total of 10 QTLs were detected for black stem resistance. The phenotypic variance explained by each QTL (R^2) was moderate, ranging from 6 to 20%. Four QTLs were common between two isolates on linkage group 5 and 15 whereas the others were specific for each isolate. Regarding isolate-specific and isolate-nonspecific QTLs detected for partial resistance, it is evident that both genetic effects control partial resistance to the disease isolates. This confirms the need to consider different isolates in the black stem resistance breeding programmes. The four SSR markers HA3700, SSU25, ORS1097 and ORS523_1 encompassing the QTLs for partial resistance to black stem isolates could be good candidates for marker assisted selection.

Keywords: *Helianthus annuus*, isolate specificity, partial resistance, QTL-mapping, sunflower black stem

Introduction

Phoma macdonaldii, the causal agent of black stem of sunflower is reported from different countries across the world. The disease has been spreading steadily since 1990 in France, and it is now the second most important sunflower disease after downy mildew (Alignan *et al.*, 2006). It causes premature ripening associated with yield losses of 10–30% (Penaud, 1996), and also reduction in oil content and thousand seed weight (Carson, 1991).

The fungus infects the plants by direct penetration or indirectly through wounds or plant structural openings such as lenticels and stomata (Roustae *et al.*, 2000a). To date, sunflower genotypes with different levels of resistance to this disease have been identified, but no fully resistant genotypes are available (Roustae *et al.*, 2000b).

Roustae *et al.* (2000b), using parental genotypes and their F_1 hybrids showed that the variation among genotypes studied was due to general combining ability and thus most of the variation was attributed to additive effects.

Recombinant inbred lines (RILs) were inoculated with an aggressive French isolate of *P. macdonaldii* and quantitative trait loci (QTLs) were identified (Rachid Al-Chaarani *et al.*, 2002). Bert *et al.* (2004), using F_2 - F_3 families, also detected QTLs controlling partial resistance to black stem. It has been recognized that significant differences in pathogenicity exist among different *P. macdonaldii* isolates on the same genetic material (Roustae *et al.*, 2000c). In the investigations cited above, the isolate specificity of partial resistance was not studied. Use of the QTL-approach to investigate isolate specificity of quantitative resistance has been reported in several research works (Leonards-Schippers *et al.*, 1994; Caranta *et al.*, 1997; Qi *et al.*, 1999; Arru *et al.*, 2003; Chen *et al.*, 2003; Zhu *et al.*, 2003; Calenge *et al.*, 2004; Cho *et al.*, 2004; Rocherieux *et al.*, 2004; Talukder *et al.*, 2004). Such an approach may throw light upon the existence of the minor-gene-for-minor-gene interaction for quantitative resistance postulated by Parlevliet & Zadoks (1977). The objective of the present study was to investigate isolate specificity and isolate nonspecificity of QTLs controlling partial resistance to two *P. macdonaldii* isolates using RILs coming from the cross between the sunflower parental lines, PAC2 and RHA266.

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Materials and methods

Sunflower genotypes and *P. macdonaldii* isolates

A set of 99 F₂ RILs derived from the cross between sunflower parental lines PAC2 and RHA266 was used in this study. The parental lines exhibit contrasting response in their partial resistance to two single pycnidiospore isolates of *P. macdonaldii* (MP8 and MP10) selected through purified isolates for this experiment. MP8 and MP10 isolates were derived from naturally infected plants in southwest and central regions of France in 1996 (Roustae *et al.*, 2000c). Conservation of isolates was achieved using the method described by Roustae *et al.* (2000c).

Pathological tests and disease assessments

The responses of the RILs and parental lines to the two isolates were evaluated in two experiments. In each experiment the plant materials were inoculated with one of the selected isolates (MP8 and MP10). The experimental design was a randomized complete block with three replications. Each replication consisted of 10–12 seedlings. Pathological tests were performed using the method described by Darvishzadeh *et al.* (2007). Cotyledon petioles of seedlings were scored 7 days after inoculation according to the percentage of the petiole area exhibiting disease symptoms. A score of 1 (resistant) to 9 (susceptible) was given in relation to the proportion of petiole area showing necrosis as proposed by Roustae *et al.* (2000b), where: 1 = 0–5%, 2 = 6–10%, 3 = 11–20%, 4 = 21–30%, 5 = 31–40%, 6 = 41–60%, 7 = 61–80%; 8 = 81–99% and 9 = 100%, with necrosis spreading down the stem.

Data analysis and QTL mapping

Analysis of variance (ANOVA) of the disease data were performed using the general linear model (GLM) procedure in the SAS software (SAS Institute Inc.). The function 'FREQ' of SPSS software (SPSS/PC-10, SPSS Inc.) was used to analyse the frequency distribution of RILs and their parents for partial resistance to each *P. macdonaldii* isolate, scored 7 days after inoculation of petioles. The mean of RILs and that of the parents was compared. Genetic gain when the mean of the 10% most resistant RILs was compared with the mean of their parents was determined. Additive and environmental variances as well as narrow-sense heritability were calculated according to Kearsy & Pooni (1996) using least-square estimates of genetic parameters.

The linkage map used in this study is the improved map described recently by Poormohammad Kiani *et al.* (2007). Briefly, the improved map has incorporated 157 new microsatellite markers compared with its old version (Rachid Al-Chaarani *et al.*, 2004). Each linkage group was numbered according to the sunflower reference map (Tang *et al.*, 2002) and is presumed to correspond to one of the 17 chromosomes in the haploid sunflower genome

($x = 17$). The total map length is 1824.6 cM with mean density of 3.7 cM per locus.

QTLs were detected using the composite interval mapping (CIM) by QTL Cartographer, version 1.16 with model 6 of Zmapqtl (Basten *et al.*, 2002) and a LOD score threshold of 3.0. This model integrates two parameters for CIM: the number of markers which control the genetic background ($n_m = 15$) and a window size ($w = 15$) that will block out a region of the genome on either side of the markers flanking the test site. The inclusion of background markers makes the analysis more sensitive to the presence of a QTL in the target interval. QTLs identified for the two isolates were compared on the basis of overlapping support intervals; a decrease in the LOD score of 1.0 determined the end point of support interval for each QTL. Additive effects of the detected QTLs, the percentage of phenotypic variation explained by each one (R^2) as well as the total phenotypic variation explained in the model of composite interval mapping (TR^2) were estimated using the Zmapqtl programme of QTL Cartographer (Basten *et al.*, 2002).

Results

Disease observation

ANOVA showed highly significant effects for genotypes while no significant difference was observed among replications (Table 1). Parental lines showed a contrasting level of partial resistance to isolates (Table 2). PAC2 presented higher significant level of partial resistance to the MP8 isolate. By contrast, RHA266 showed a higher significant partial resistance to the MP10 isolate. Differences between the mean of RILs (\bar{X} RILs) and the mean of parents (\bar{X} P) were not significant for both isolates (Table 2). Significant differences were observed when the mean of parents (\bar{X} P) was compared with the mean of 10% selected most resistant RILs (10%SRILs) (Table 2). Narrow-sense heritability was 0.47 and 0.46 for MP8 and MP10, respectively. Frequency distribution of RILs and their parents for partial resistance to both isolates shows continuous patterns, suggesting that partial resistance is controlled by a polygenic system (Fig. 1). For isolate

Table 1 Mean squares for disease severity score in sunflower recombinant inbred lines (RILs) and their two parents infected by *Phoma macdonaldii* in controlled conditions

Source of variation	d.f. ^a	MP8	MP10
Genotype	100	11.13*	10.97*
Block	2	0.28 ^{ns}	0.75 ^{ns}
Residual	200	0.30	0.33
Total	302	3.89	3.85

Coefficients of variation (CV) are 10.45 and 10.09 percent for MP8 and MP10 isolate, respectively.

^ad.f. = degrees of freedom.

* = Significant at 0.001 probability level; ^{ns} = non significance.

Table 2 Genetic gain and heritability for partial resistance to *Phoma macdonaldii* isolates (MP8 and MP10) in sunflower recombinant inbred lines (RILs)

Items	Isolates		Items	Isolates	
	MP8	MP10		MP8	MP10
PAC2 (P1)	^a 2.54	6.40	$\bar{X}RILs^b - \bar{X}P^c$	0.86 ^{ns}	0.85 ^{ns}
RHA266 (P2)	6.20	3.30	10% SRILs ^d	1.88	1.96
P1-P2	-3.66*	3.10*	GG10% ^e = 10%SRIL - $\bar{X}P$	-2.46*	-2.89*
$\bar{X}P = (P1 + P2)/2$	4.37	4.85	LSD _{0.05} ^f	0.89	0.92
$\bar{X}RIL$	5.20	5.70	h^2g	0.47	0.46

^aAverage disease severity score of genotypes challenged by *P. macdonaldii* isolates MP8 and MP10, 7 days postinoculation, in three replications, each containing 24 cotyledon petioles.

^b $\bar{X}RILs$: mean of all RILs.

^c $\bar{X}P$: mean of parents.

^d10%SRILs: mean of the 10% selected most resistant RILs.

^eGG10%: genetic gain when the mean of 10% selected RILs is compared with the mean of parents.

^fLSD_{0.05}: least significant differences calculated using $t_{0.05}$ and error mean square of each experiment.

^g h^2 : narrow-sense heritability.

* and ns: significant at 0.05 level and non-significant.

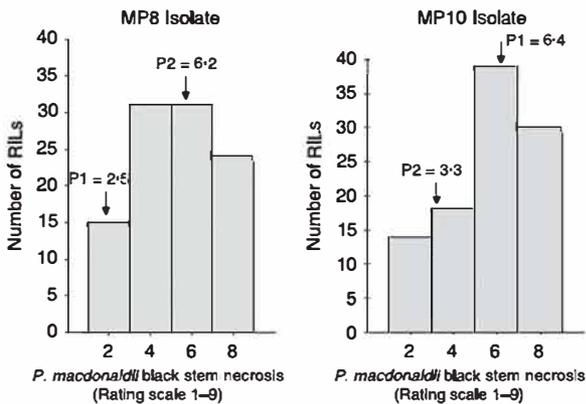


Figure 1 Frequency distribution of sunflower recombinant inbred lines (RILs) and their parents for partial resistance to *Phoma macdonaldii* isolates (MP8 and MP10), scored 7 days after petiole inoculation based on the percentage of the petiole area exhibiting necrosis symptoms.

Arrows show phenotypic values of parental lines (P1 = PAC2 and P2 = RHA266) to each *P. macdonaldii* isolate. Rating scale:

1 = resistant; 9 = susceptible.

MP10 the distribution of mean disease severity score was skewed toward susceptibility indicating that MP10 was more aggressive than MP8 on these genotypes (Fig. 1).

QTL analysis

Significant peak values of LOD scores, the position of their peaks, the percentage of phenotypic variance explained (R^2) and the estimate of QTL effects based on a composite interval mapping for QTLs associated with partial resistance to two *P. macdonaldii* isolates are summarized in Table 3. QTLs were designated as *bsr* corresponding to black stem resistance followed by the isolate name, linkage group and QTL number, respectively. These QTLs were mapped on the sunflower genome

(Fig. 2). A total of 10 QTLs were detected for partial resistance to two isolates. QTLs involved in partial resistance to the MP8 isolate are located on linkage groups 5, 9, 11, 15 and 17; and those for partial resistance to MP10 are located on linkage groups 1, 2, 5 and 15. Through the 10 detected QTLs, four QTLs locating two by two on linkage groups 5 and 15 provide partial resistance to both isolates tested (Fig. 2). The phenotypic variance explained by each QTL (R^2) is moderate, ranging from 6 to 20%, whereas a high percentage of total phenotypic variance ($TR^2 = 63-80\%$) is explained for partial resistance to two isolates when considering all the covariants in composite interval mapping. The sign of additive gene effects showed that favourable alleles for partial resistance come from both parents.

Discussion

The RILs presented highly significant genetic variation for partial resistance to the two *P. macdonaldii* isolates used in this experiment (Table 1). Genetic variability for partial resistance to black stem has been previously reported in both field (Pérès *et al.*, 1994) and controlled conditions (Roustae *et al.*, 2000b; Rachid Al-Chaarani *et al.*, 2002; Bert *et al.*, 2004). The difference between the mean of RILs and the mean of parents was not significant indicating that RILs used in this study are representative of possible recombinations of the cross PAC2 × RHA266 (Table 2). The significant difference between the mean of 10% selected most resistant RILs and the mean of parents, considered as genetic gain, is evidence for transgressive segregation for partial resistance to *P. macdonaldii* isolates (Table 2, Fig. 1). The positive and negative signs of additive effect at the different loci (Table 3) indicate the contribution of both parental lines to partial resistance and confirm the transgressive segregation observed at the phenotypic level. Transgressive segregation has previously been reported by Rachid Al-Chaarani *et al.* (2002) and

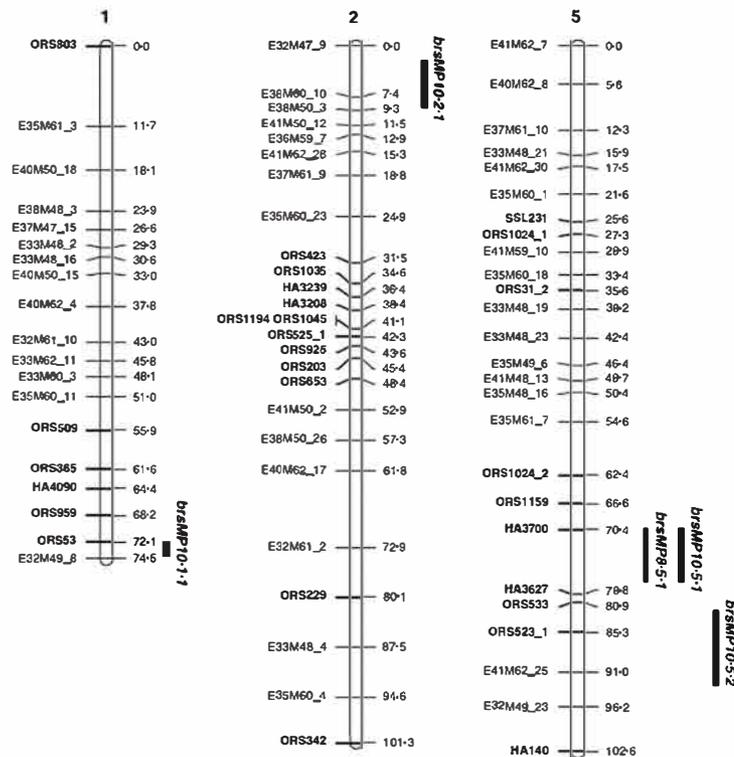


Figure 2 Linkage groups of the sunflower genome presenting QTLs for partial resistance to two isolates of *Phoma macdonaldii*. Vertical bars present the positions of QTLs. A decrease in the LOD score of 1.0, determined the end point of support interval for each QTL.

Bert *et al.* (2004) for partial resistance of sunflower to black stem with another isolate of *P. macdonaldii*. Narrow-sense heritability for partial resistance to MP8 and MP10 isolates was 0.47 and 0.46, respectively, which is similar to that presented by Rachid Al-Chaarani *et al.* (2002).

A total of 10 QTLs were detected for partial resistance to isolates MP8 and MP10 (Table 3). Among QTLs identified, three QTLs, *bsrMP8-9-1*, *bsrMP8-11-1* and *bsrMP8-17-1*, were only effective with isolate MP8 (isolate-specific) and three, *bsrMP10-1-1*, *bsrMP10-2-1* and *bsrMP10-5-2* only with isolate MP10 (isolate-specific). The other four QTLs were co-localizing (isolate-nonspecific). One of the QTLs detected for the MP8 isolate (*bsrMP8-5-1*) was co-localized with a QTL of the MP10 isolate (*bsrMP10-5-1*) on linkage group 5 (Fig. 2). These two co-localizing QTLs are important as they explain 16 and 13% of phenotypic variance for partial resistance to MP8 and MP10 isolates respectively (Table 3). Other co-localizing QTLs (*bsrMP8-15-1* and *bsrMP10-15-1*) located on linkage group 15, explain 8% of phenotypic variance of partial resistance to the two isolates (Table 3, Fig. 2). The identified QTLs in this experiment have been detected in favourable conditions for disease development (Roustae *et al.*, 2000c). In another programme at this laboratory, an experiment has been repeated with 12 genotypes challenged by seven isolates in the same favourable environmental conditions and the difference between the two tests was not significant (Darvishzadeh *et al.*, 2007). However, any changes in experimental conditions affecting disease development, may have an

effect on QTLs. Favourable alleles of QTLs for the two isolates tested come from both parental lines indicating that they are dispersed between the parents. Research in other host-pathogen systems also showed that susceptible parents could contribute to increasing resistance of progeny lines to diseases as a result of transgressive segregation (Cherif & Harrabi, 1993; Thomas *et al.*, 1995; Chen *et al.*, 2003; Chartrain *et al.*, 2004).

So far, two studies have been undertaken for mapping QTLs controlling partial resistance to black stem disease in sunflower (Rachid Al-Chaarani *et al.* 2002; Bert *et al.* 2004). In both studies the French MP6 isolate was used. In the first study, using an AFLP map, Rachid Al-Chaarani *et al.* (2002) detected seven QTLs for partial resistance to black stem with moderate phenotypic variance (R^2) ranging from 6 to 17%, on linkage groups 3, 4, 8, 9, 11, 15 and 17, which correspond to linkage groups 1, 7, 16, 10, 14, 17 and 8, respectively, in the new improved map. One of the detected QTLs for partial resistance to the MP8 isolate in the study reported here (*bsrMP8-17-1*) was co-localized with a QTL for the MP6 isolate (*bsr.15-1*) (Rachid Al-Chaarani *et al.*, 2002). In another study, using an RFLP/AFLP map of F_3 families, Bert *et al.* (2004) reported four QTLs for partial resistance of sunflower to black stem. However, the lack of SSR markers and common linkage group nomenclature in their map makes it difficult to compare the location of QTL detected in this study and those detected by Bert *et al.* (2004).

Other research on the host-pathogen interaction systems also indicated that some QTLs for quantitative

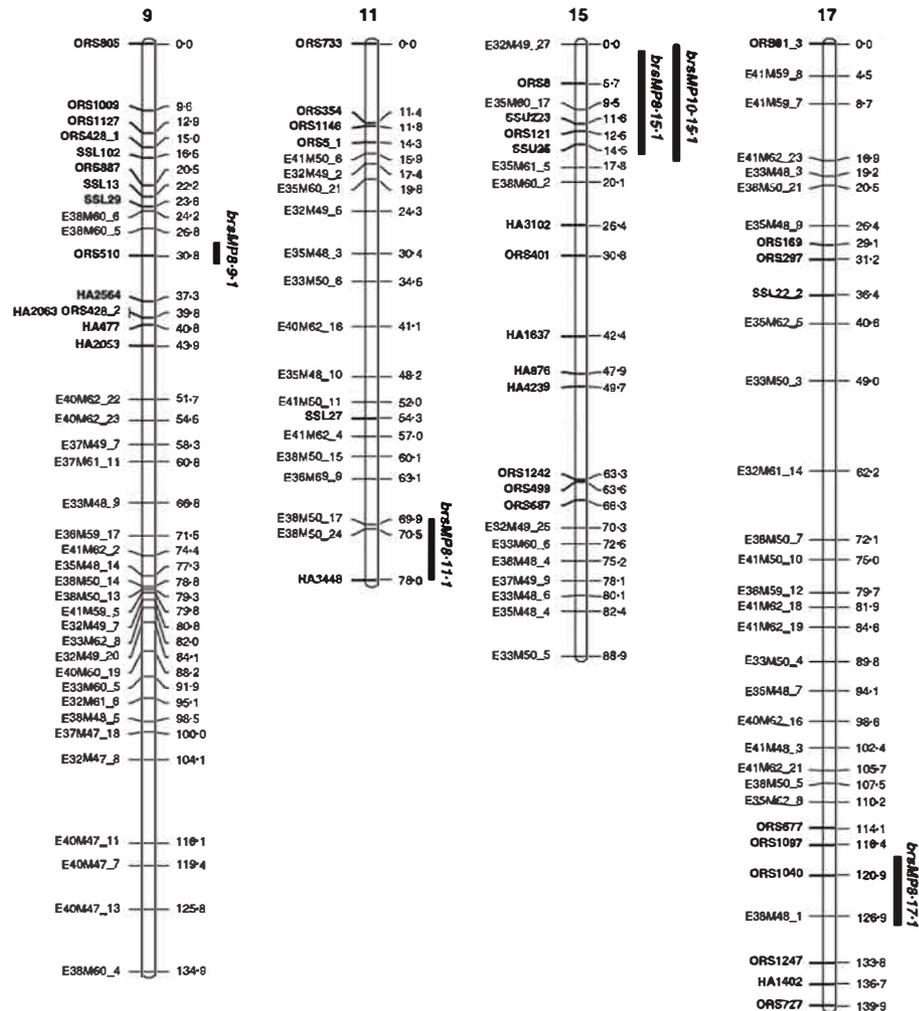


Figure 2 Continued

Table 3 QTLs detected for partial resistant to *Phoma macdonaldii* isolates in sunflower recombinant inbred lines (RILs) using composite interval mapping (CIM). Markers linked to QTLs common for two isolates are presented in bold

Isolate	QTL	Linkage group	Marker	^a Position	LOD	Additive effect	^b R ²	^c TR ²
MP8	<i>bsrMP8-5-1</i>	LG5	HA3700	74.30	4.2	0.91	0.16	0.72
	<i>bsrMP8-9-1</i>	LG9	ORS510	30.80	3.4	0.69	0.08	0.63
	<i>bsrMP8-11-1</i>	LG11	E38M50_24	76.50	4.8	0.81	0.14	0.69
	<i>bsrMP8-15-1</i>	LG15	SSU25	14.46	4.0	-0.63	0.08	0.65
	<i>bsrMP8-17-1</i>	LG17	ORS1097	120.40	5.5	-0.87	0.15	0.68
MP10	<i>bsrMP10-1-1</i>	LG1	ORS53	74.00	4.8	-0.64	0.06	0.72
	<i>bsrMP10-2-1</i>	LG2	E38M60_10	7.40	8.0	0.87	0.15	0.72
	<i>bsrMP10-5-1</i>	LG5	HA3700	76.40	4.8	0.75	0.13	0.73
	<i>bsrMP10-5-2</i>	LG5	ORS523_1	89.20	5.6	0.95	0.20	0.80
	<i>bsrMP10-15-1</i>	LG15	SSU25	14.46	4.9	-0.60	0.08	0.72

^aExpressed in Kosambi CM, from the north of linkage group (LG).

^bPercentage of individual phenotypic variance explained, value determined by QTL Cartographer, version 1.16 (Basten *et al.* 2002).

^cPercentage of phenotypic variance explained by the QTLs given all the covariants, determined by QTL Cartographer, version 1.16 (Basten *et al.*, 2002).

resistance were isolate specific. In the *Solanum tuberosum* – *Phytophthora infestans* system, six out of the 11 detected QTLs showed specificity to two *P. infestans* races (Leonards-Schippers *et al.*, 1994). The *Capsicum annuum*–Potyvirus host–pathogen system also showed isolate-specific effects for QTLs detected (Caranta *et al.*, 1997). Qi *et al.* (1999) found a similar situation for partial resistance to leaf rust in barley. They found three QTLs in common for effective resistance to two isolates at the adult stage and five remaining QTLs were isolate specific. In mapping QTLs for partial resistance to rice blast (*Magnaporthe grisea*) most of identified QTLs were isolate-specific (Talukder *et al.*, 2004). Parlevliet (1976) reported small but significant genotype–isolate interactions in partially resistant barley lines to *Puccinia hordei*. This led him to propose the ‘minor-gene-for-minor-gene’ hypothesis to explain quantitative resistance. Indeed, the examples mentioned above together with the present data support this hypothesis. Several hypotheses have been addressed for the specificity effect of QTLs detected with different isolates (Rocherieux *et al.*, 2004). The resistance response implicates a great number of genes that could be interconnected and be required in different signalling pathways (Feys & Parker, 2000).

In conclusion, regarding isolate-nonspecific and isolate-specific QTLs detected for partial resistance to two *P. macdonaldii* isolates, it is suggested that both specific and nonspecific genes or genomic regions control partial resistance to *P. macdonaldii* isolates. The results of this study confirm the need to consider different isolates in the black stem resistance breeding programmes. Pyramiding isolate-nonspecific together with isolate-specific QTLs could increase the level of resistance to a wide range of isolates. SSR markers such as HA3700, SSU25, ORS1097 and ORS523_1, linked to QTLs for partial resistance to black stem isolates could be good candidates for marker-assisted selection.

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