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Molecular markers linked to white rust resistance in mustard *Brassica juncea*

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Abstract White rust, caused by *Albugo candida* (Pers.) Kuntze, is an economically important disease of *Brassica juncea* (L.) Czern. and Coss mustard, particularly in India. The most efficient and cost-effective way of protecting mustard plants from white rust disease is through genetic resistance. The objective of this study was to identify RAPD markers for white rust resistance in an F_1-derived doubled-haploid (DH) population originating from a cross between white rust-susceptible and white rust-resistant breeding lines of *B. juncea* from the canola-quality *B. juncea* breeding project of the Agriculture and Agri-Food Canada-Saskatoon Research Centre. The DH population was used to screen for RAPD markers associated with white rust resistance/susceptibility using bulked segregant analysis. Two markers, WR2 and WR3, linked to white rust resistance, flanked the resistance locus *Ac2*$_1$ and were highly effective in identifying the presence or absence of the resistance gene in the DH population. These two markers were shown to be specific to the Russian source of white rust resistance utilized in this project. It is concluded that the availability of these RAPD markers will enhance the breeding for white rust resistance in *B. juncea*.

Key words *Brassica juncea* · RAPD markers · *Albugo candida* · White rust resistance

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Introduction

White rust, caused by *Albugo candida* (Pers.) Kuntze, is a widespread and destructive disease of cruciferous vegetables. It is also of economic importance in mustard (*Brassica juncea* L. Czern. and Coss.) and turnip rape (*B. rapa* L.) (Walker and Williams 1973; Saharan and Verma 1992; Buzza 1995; Kole et al. 1996). Yield losses of 30–60% have been reported in *B. rapa* due to white rust infection (Liu et al. 1996). The physiological specialization in *A. candida* is classified into ten races on the basis of their specificity to different plant genomes (Hill et al. 1988). However, host specificity in *A. candida* is not an absolute adaptation to a particular species, especially when the races are from hosts sharing a common genome (Liu et al. 1996). The predominant race of *A. candida* on *B. juncea* in western Canada is race 2 and the Canadian Oriental mustard cultivars Domo, Cutlass and AC Vulcan are resistant to this race whereas the landrace Commercial Brown is highly susceptible. A variant of race 2 (race 2V) was identified on the resistant cultivar Cutlass for which there is no resistance available in *B. juncea* (Rimmer and Buchwaldt 1995). Resistance to white rust in the *Brassica* species for which information is available is governed by simple Mendelian genes, including a single dominant gene in *B. juncea* against race 2 (Tiwari et al. 1988; Rimmer and Buchwaldt 1995), three dominant genes in *B. napus* against race 7 (Fan et al. 1983; Liu et al. 1996), and a single dominant gene in *B. rapa*, also against race 2 (Kole et al. 1996).

Molecular genetic maps generated from restriction fragment length polymorphisms (RFLP) are being developed in *B. juncea* (Upadhyay et al. 1996; Cheung et al. 1997). Knowledge about the location of the gene or genes for resistance to white rust on these maps is important for marker-assisted selection (MAS) for white rust-resistance, and will be valuable in identifying resistance genes from different germplasm sources.

Development of a MAS breeding strategy for white rust- resistant cultivars would be useful in canola-quality *B. juncea* breeding programs which are gaining strength in western Canada. Currently, one co-segregating RFLP marker for white rust resistance has been identified in *B. juncea* (W. Cheung, personal communication).

There is no published information on the commonly used technology of random amplification of polymorphic DNA (RAPD) markers for white rust resistance in *B. juncea*. The RAPD technique using the polymerase chain reaction (PCR) is relatively easy, inexpensive, fast, and is a technology that is commonly available in most molecular marker-assisted breeding laboratories. For these reasons this technique was used to identify RAPD markers for white rust resistance in a DH population of *B. juncea* that segregated for a white rust resistance gene. The utility of RAPD markers in identifying white rust resistance in different *B. juncea* germplasms was also tested, and the specificity of the identified markers for the Russian source of white rust resistance studied in the segregating population was examined.

Materials and methods

Plant material

In 1991, a cross was made between the *B. juncea* breeding line J90-4317 (white rust susceptible) as female parent and line J90-2733 (white rust resistant) as male parent, with the objective of developing white rust-resistant, high oil content, canola-quality *B. juncea* (Rakow 1995). The white rust susceptible parent J90-4317 was derived from crosses involving the low-glucosinolate *B. juncea* line 1058 (Love et al. 1990), the Canadian Oriental mustard cultivar Cutlass, and two crosses with LDZ. LDZ is a BC_3 zero-erucic-acid line derived from the backcross [(Donskaja × ZEM1) × Donskaja*3] (Love et al. 1991; Rakow 1991) and is susceptible to white rust (Tiwari et al. 1988). Parental line J90-4317 was an F_3 derived, open pollinated F_4 line which was field tested at Saskatoon in 1990. The white rust-resistant parent J90-2733 was an F_3-derived, open-pollinated F_4 line derived from a cross between the two Russian Oriental mustard cultivars Donskaja and Jubilejnaja, made in 1986 with the objective of developing white rust-resistant, high oil content, parent material for the canola-quality *B. juncea* breeding program. The F_1 generation of the cross (J90-4317 × J90-2733) was used to produce a DH mapping population that segregated for white rust resistance utilizing microspore culture by [the Saskatchewan Wheat Pool (Saskatoon, SK).]

Reserve seed from the original 114 DH plants was used to score the white rust reaction of seedlings in each DH line. Reserve seed from 36 DH lines identified as resistant and 36 DH lines identified as susceptible was used for the molecular-marker analysis. An additional 42 white rust-susceptible DH lines from the above cross and several *B. juncea* cultivars were used for testing the utility in phenotypic classification and the specificity of the RAPD markers, respectively.

White rust evaluation

White rust disease screening was done in a growth chamber (18°C/15°C day/night temperature, 18 h photoperiod, 210 μE/m^2 per s light intensity). The inoculum was propagated in the growth chamber on the white rust-susceptible *B. juncea* cultivar Commercial Brown. Sporangia of *A. candida* were scraped from sporulating lesions of infected leaves, dried, and frozen at − 20°C. Three replicate pots of each parental and DH progeny lines with nine seedlings each were arranged in a randomised complete block design for inoculation with *A. candida* race 2. Twelve days after planting the test population, the stored sporangia of *A. candida* were germinated in sterile distilled water at 8°C for 4 h. The diluted suspension of zoospores (1.0×10^4/ml) was sprayed on the seedlings until run-off. The seedlings were misted (100% RH) in the dark for 24 h to promote plant infection. The pots were then returned to the above controlled conditions of the chamber and the seedlings were scored for the presence or absence of white rust pustules at 9 days after inoculation. Plants were classified as resistant only if they were completely devoid of pustules. The mean percent disease incidence was calculated for each of the parental and DH progeny lines.

DNA extraction and PCR

Leaves from six plants of each DH line and parental line were bulked, lyophilized, and ground to a powder in liquid nitrogen. DNA was extracted from 50 mg of dry tissue in a 1.5-ml microtube by adding 1 ml of 95°C extraction buffer [0.1 M Tris-HCl (pH 8.0), 10 mM EDTA, 1 M KCl] and incubating at 95°C for 10 min with occasional agitation. The homogenate was centrifuged to remove cell debris. The supernatant was treated with RNAse and DNA was precipitated with isopropanol. The DNA was washed with 70% ethanol, re-suspended in water, re-precipitated with ethanol, re-suspended in 75 μl of water then quantified by fluorimetry using Hoechst 33258 stain. Typical yields were 20–30 μg of DNA.

The PCR reactions included 10 ng of DNA, 1 U of *Taq* DNA polymerase (BRL, Mississauga, Canada), 50 mM KCl, 2.5 mM $MgCl_2$, 200 μM of each dNTP and 0.2 μM of primer (University of British Columbia, Vancouver, BC). The DNA amplification protocol was 95°C − 1:30 min (1 cycle); 95°C–20 s; 36°C–1 min; ramp 1°C/s to 72°C; 72°C–1 min (35 cycles); 72°C–7 min (1 cycle). All PCR products were separated in 2% (w/v) agarose gels in 1 × TAE by electrophoresis at 100 V for 3 h. Gels were stained in ethidium bromide and photographed on a digital gel-documentation system.

Bulked segregant analysis (BSA)

A total of 257 random, 10-base pair (bp) oligonucleotide primers were screened using the BSA strategy (Michelmore et al. 1991). The BSA included DNA of the parents J90-4317 (white rust-susceptible) and J90-2733 (white rust-resistant). The bulked segregants were prepared by combining equal amounts of DNA from each of nine random white rust-resistant and ten random white rust-susceptible DH lines.

Linkage analysis

The white rust resistance locus has been designated as $Ac2_1$ (Liu et al. 1996). Linkage between the $Ac2_1$ locus and the RAPD markers was done with Mapmaker/exp V3.0 software (Lander et al. 1987; Lincoln et al. 1992) by analysing marker segregation data in all 36 white rust-resistant and a random selection of 36 white rust-susceptible DH lines. The RAPD markers were grouped using a minimum LOD threshold of 3.0 and a maximum recombination fraction of 0.3 as linkage criteria.

Results

White rust reaction of DH lines

The white rust reaction was clear and uniform within the progeny population of the DH lines. Thirty one of the thirty six white rust-resistant DH lines showed an immune response on all seedlings. The remaining five lines showed a low degree of white rust incidence varying from 4 to 15% and were scored as white rust-resistant. There were 78 DH lines categorised as susceptible which showed a 100% incidence of white rust infection.

Marker and linkage analysis

Although the DH population did not segregate 1 : 1 for the disease reaction, there is strong evidence that the parent J90-2733 carries a single, dominant gene for white rust resistance. The accessions of *B. juncea* (Vniimk-405, Donskaja and Jubilejnaja) were all introduced into the Agriculture and Agri-Food Canada-Saskatoon Research Centre (AAFC-SRC) breeding program at the same time and from the same Russian breeding institute. Vniimk-405 was shown to carry a single, dominant gene for white rust resistance (Tiwari et al. 1988) and Jubilejnaja or Donskaja, the donor of white rust resistance to J90-2733, was presumed to carry the same white rust resistance gene.

Over 57% (147/257) of the primers amplified polymorphisms between the parents. There were 51/147 primers that amplified polymorphisms between the two bulks and the polymorphisms were considered as putative markers of the disease reaction. Fourteen primers amplified 15 putative marker fragments in the components of the bulks (a total of at least 18 DNA samples) with less than 25% recombinants. For example, primer WR2 amplified a 1540-bp fragment showing 2/18 recombinant DH lines, and primer WR3 amplified a 700-bp fragment showing 0/19 recombinant DH lines (Fig. 1A and B). The 15 RAPD marker fragments were selected for linkage analysis and five markers were found to be linked with the $Ac2_1$ locus (Fig. 2). The decamer primer sequences used to amplify these markers are listed in Table 1. Three of the RAPD markers, WR2, WR3 and WR4, identified the J90-2733 white rust resistance allele and two RAPD markers, WR1a and WR1b, amplified with the same primer, identified the J90-4317 white rust susceptibility allele.

No double crossover products were observed between the two white rust resistance markers (WR2 and WR3) that included the $Ac2_1$ locus. This would have resulted in the occurrence of white rust-susceptible DH lines with both markers or white rust-resistant DH lines without both markers. Similarly, no double crossovers were detected between markers WR2 and WR1a which flank $Ac2_1$. However, when the markers were considered individually, some DH lines showed recombination between the markers and the $Ac2_1$ locus, resulting in the white rust-susceptible genotypes having

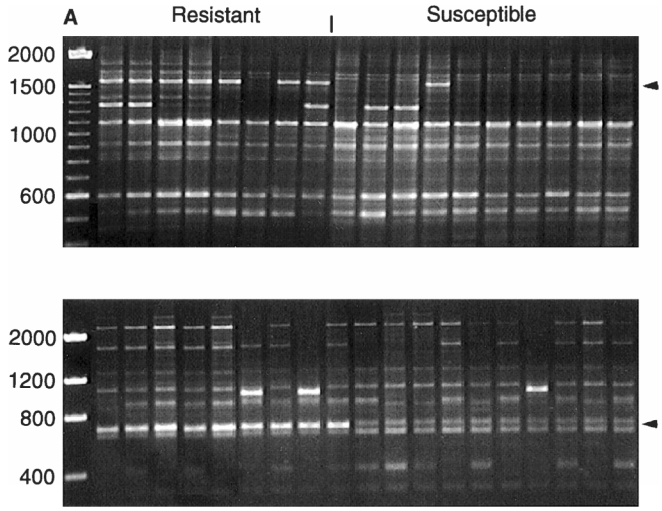

Fig. 1 RAPD profiles of white rust-resistant and susceptible *B. juncea* doubled-haploid lines from the cross J90-4317 × J90-2733. PCR primers amplified fragments of DNA (indicated with *arrowheads*) that were linked with the white rust resistance locus $Ac2_1$ and these two markers, WR2 (A) and WR3 (B), flank the $Ac2_1$ locus. Molecular-weight markers (bp) are shown on the left.

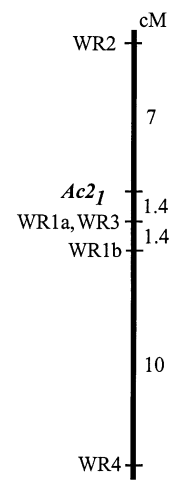

Fig. 2 A genetic map of the RAPD markers from *B. juncea* that surround the white rust resistance locus $Ac2_1$. Markers WR1a and WR1b are linked to the white rust-susceptible allele from J90-4317, and markers WR2, WR3 and WR4 are linked to the white rust resistance allele from J90-2733. Map distances are converted to Kosambi cM distances

Table 1 Summary of molecular markers for white rust resistance/susceptibility in *B. juncea*

Primer sequence (5′ → 3′)	Parental allele	Map locus
GCGTGACCCG	J90-4317	WR1a
GCGTGACCCG	J90-4317	WR1b
AGGCAGACCT	J90-2733	WR2
AATAACCGCC	J90-2733	WR3
TTGCTGGGCG	J90-2733	WR4

a marker for resistance (Table 2) or resistant genotypes having a marker for susceptibility.

Utility and source specificity of RAPD markers

Forty two additional DH lines susceptible to race 2 of *A. candida* produced from the same cross were screened for the presence of the five markers. All 42 DH lines showed the presence of the closest susceptible allele marker WR1a and the absence of the closest resistance allele marker WR3 (Table 2). Due to the small sample size, no recombinant class was observed for the above markers. A varying degree of misclassification of the disease reaction phenotype was observed with the other RAPD markers which was directly related to their distance from the $Ac2_1$ locus. However, when the flanking markers WR2 and WR1a, or markers WR2 and WR3, were considered together, 37 out of 42 DH lines were correctly identified as white rust-susceptible and no lines were misclassified (Table 2).

Table 2 Phenotypic misclassification of white rust susceptibility with RAPD markers in a set of 42 white rust-susceptible *B. juncea* doubled-haploid lines from the cross J90-4317 (susceptible) × J90-2733 (resistant)

Marker	Number of DH lines		Misclassification (%)
	Marker present	Marker absent	
WR1a	42	0	0.0
WR1b	41	1	2.0
WR2	5	37	11.0
WR4	6	36	13.9
WR3	0	42	0.0
WR3 + WR2	0	37	0.0

The specificity of the RAPD markers for the Russian source of white rust resistance to race 2 was tested on eight *B. juncea* cultivars released in western Canada and a germplasm line from Russia (Table 3). The flanking markers WR2 and WR3 correctly identified the cultivar Scimitar as the only line in the collection with a known Russian source of white rust resistance to race 2 (Woods and Petrie 1989). The RAPD marker (WR3) tightly linked to the white rust resistance allele at $Ac2_1$ was absent in the other three white rust-resistant cultivars Domo, Cutlass and AC Vulcan, all of which were developed from a common base population of Oriental mustard. Likewise, the markers WR1a and WR1b linked to the white rust-susceptible allele were observed only in the white rust-susceptible Russian germplasm line Stalingradska and in the susceptible parent of the DH population J90-4317. The white rust-susceptibility markers were absent in all four white rust-susceptible Canadian cultivars, none of which had any Russian parentage (Table 3).

Discussion

Utility of RAPD markers for MAS

Two white rust resistance and two susceptibility markers were mapped within an interval of 10 cM that included the $Ac2_1$ locus (Fig. 2). Use of these markers together in a breeding program will make selection for the white rust resistance gene very accurate, unlike using the single markers that show recombination with the gene. The possibility of a white rust-susceptible allele being wrongly selected by the flanking white rust resistance markers was practically nill because of the extremely low frequency of double crossing over

Table 3 Pedigrees of *B. juncea* cultivars, their reaction to infection with *A. candida* race 2, and RAPD marker genotypes

Cultivar	Pedigree (released)[a]	Reaction[b]	RAPD marker				
			WR1a	WR1b	WR2	WR3	WR4
Commercial Brown	Introduction	S	–	–	+	–	–
Lethbridge 22A	Selection from mustard landrace (1974)	S	–	–	+	–	–
Blaze	Selection from Commercial Brown (1976)	S	–	–	+	–	–
Forge	Montana Blonde × Chinese line (1989)	S	–	–	–	–	+
Stalingradska	Russian germplasm	S	+	+	+	–	–
J90-4317[c]	[Cutlass × (1058 × LDZ) × LDZ[d]]	S	+	+	–	–	–
J90-2733[c]	Donskaja × Jubilejnaja[e]	R	–	–	+	+	+
Scimitar	Blaze × Vniimk-405[f] (1987)	R	–	–	+	+	–
Domo	Selection from a broad based *B. juncea* population (1977)	R	–	–	+	–	+
Cutlass	Selection from Domo (1985)	R	–	–	–	–	+
AC Vulcan	Selection from Cutlass (1993)	R	–	–	+	–	+

[a] Source: Production and marketing branch, Plant Products Division, Ottawa, Canada
[b] S = susceptible, R = resistant to *A. candida* race 2
[c] Parents of DH population in this study
[d] LDZ = [(Donskaja × ZEM1) × Donskaja*3]
[e] Putative Russian source of white rust resistance
[f] Russian source of white rust resistance

between them. The frequency of occurrence of double crossing over involving the *Ac2*$_1$ gene within an interval of 8.4 cM between markers WR2 and WR3 in this population is 9.0×10^{-4}, assuming no interference, but the frequency may actually be even lower because of natural interference. The utility of white rust resistance markers WR2 and WR3 flanking the *Ac2*$_1$ locus was confirmed when 37 out of 42 white rust-susceptible DH lines were correctly identified as susceptible by the absence of both white rust resistance allele markers; the remaining five lines were not misclassified as resistant since these lines showed the absence of at least one of the white rust resistance allele markers (Table 2).

Molecular markers for white rust resistance have been identified recently in *B. rapa* and *B. napus*. The RFLP markers flanking the white rust resistance locus in *B. rapa* are expected to be useful for an efficient introgression of white rust resistance (Kole et al. 1996). In contrast, the RFLP markers linked to white rust resistance in *B. napus* do not flank the gene for white rust resistance, which is the terminal locus mapped on the linkage group (Ferreira et al. 1995). A single marker may be used for the selection of resistant types but the accuracy will be dependant on linkage with the resistance gene. Similarly, the lack of flanking markers for a blackleg resistance gene in *B. napus* among 11 RAPD and two RFLP markers was identified as the main reason for their reduced utility in MAS for blackleg resistance (Mayerhofer et al. 1997).

Source specificity of RAPD markers

The source of the white rust resistance gene *Ac2*$_1$ employed here is of Russian origin. One of the Russian progenitors (Jubilejnaja or Donskaja) of line J90-2733 used in the present study most likely carried a white rust resistance allele similar to that in the Russian white rust-resistant cultivar Vniimk-405, since the three Russian cultivars were developed at the same breeding institute. The source of the white rust resistance in J90-2733 is probably Jubilejnaja since the zero-erucic-acid BC_3 Donskaja line, LDZ, in the pedigree of J90-4317 was susceptible to white rust (Rakow 1991). An analysis of pedigrees confirmed that the Canadian cultivar Scimitar was the only one that used a Russian source (Vniimk-405) for white rust resistance (Woods and Petrie 1989) (Table 3).

The molecular and pedigree data indicated that markers WR3, WR1a and WR1b specifically identified the genomic region that included the Russian white rust resistance allele from Jubilejnaja and the susceptible allele from LDZ, the zero-erucic-acid Donskaja, for the following reasons: (1) only Scimitar possesses a related Russian line in its pedigree and resembled the genotype of the white rust-resistant parent J90-2733 for markers WR3, WR1a and WR1b; (2) the white rust-susceptible lines Stalingradska and J90-4317 both have Russian progenitors and amplified the white rust susceptibility markers WR1a and WR1b, and (3) none of the white rust-susceptible Canadian cultivars amplified the susceptible allele markers WR1a and WR1b (Table 3). The resistance to white rust in Domo, Cutlass and AC Vulcan suggested that another source of white rust resistance was present in these *B. juncea* cultivars.

Since J90-2733 and Scimitar (derived from crosses with Vniimk-405) show common RAPD markers surrounding the *Ac2*$_1$ locus, these data strongly support the presumption that J90-2733 carries a single dominant gene for white rust resistance, similar to Vniimk-405, and thus the strategy adopted for molecular mapping with this DH population was valid and accurate.

The implementation of these markers was considered with regard to making selections in an F_2 population for true breeding, white rust-resistant, plants. The recombination frequencies of markers WR2 and WR3 with the *Ac2*$_1$ locus were 0.069 and 0.014 respectively. There would be an equal frequency of gametes (0.458) with the flanking markers WR2 and WR3 carrying the white rust resistance allele and those lacking these markers carrying the white rust susceptibility allele in the F_1 plants of a cross segregating for the Russian source of white rust resistance. This would result in 71% of the F_2 population having the dominant white rust resistance allele that is identified by the presence of the flanking markers WR2 and WR3, because of the dominant nature of RAPD markers. Within these F_2 plants having both the WR2 and WR3 markers, 35% would be resistant and homozygous dominant for the *Ac2*$_1$ locus and the remainder would be heterozygous resistant. Plant breeders interested in selecting true-breeding, white rust-resistant F_2 plants from a cross between susceptible and resistant plants need to consider the above calculations in order to decide the appropriate size of an F_2 population to use for MAS of this trait.

Work is underway to develop the markers as amplicon-specific diagnostic tests through sequence analysis of the amplified fragments. It would also be important to study if these markers identified white rust resistance in other *Brassica* species which could be the donors of *Ac2*$_1$ in the Russian source of resistance.

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