Identification and Application of a Random Amplified Polymorphic DNA Marker for the *I* Gene (Potyvirus Resistance) in Common Bean

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ABSTRACT

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The dominant inhibitor *I* gene has recently become a liability for common bean (*Phaseolus vulgaris*) cultivars infected with temperature-insensitive, necrosis-inducing strains of bean common mosaic virus (BCMV). Although cultivars with the *I* gene can be protected from hypersensitive lethality by recessive resistance genes, the most broadly effective resistance gene combination (*I* and *bc-3* genes) is difficult to identify due to epistasis (dominance with epistasis of *bc-3*/*bc-3* over *I*/—; hypostasis). Our objectives were to identify a molecular marker linked to the *I* gene and to evaluate indirect selection with the marker to facilitate pyramiding of the *I* and *bc-3* resistance genes. Pairs of near-isogenic lines with and without the *I* gene were screened with random decamer primers in the polymerase chain reaction to identify linked random amplified polymorphic DNA (RAPD) markers. A single RAPD marker was identified ($OW13_{690}$, generated by a 5′-CACAGCGACA-3′ decamer) and found to be tightly linked in coupling with the *I* gene in five segregating populations (recombination from 1.3 ± 0.8 to 5.0 ± 2.2 centimorgans). Selections from complex backcross populations, made for the presence of *bc-3*/*bc-3* genotypes, were also assayed for $OW13_{690}$ to identify selections carrying the hypostatic *I* gene. Our results demonstrate the utility of RAPD markers when used as indirect selection criteria for pyramiding epistatic BCMV resistance genes.

Bean common mosaic virus (BCMV), a member of the potyvirus group (18), is the most widespread and economically important viral disease affecting the common bean (*Phaseolus vulgaris* L.). Seed transmission through susceptible bean plants is the most important factor responsible for the spread of the virus worldwide (5). Although chemical control of various aphid vectors may limit local spread of BCMV, and seed certification programs may reduce inoculum level, genetic resistance is the only economically viable method of control.

At least 10 different pathotypes of BCMV have been identified according to reactions on a series of differential host cultivars (2,3). This differential host series is grouped into two broad categories based on the presence or absence of the dominant inhibitor gene *I*, and then further classified according to the presence of strain-specific recessive resistance genes (*bc-1*, *bc-1*^{2}, *bc-2*, *bc-2*^{2}, or *bc-3*, together with strain-unspecific *bc-u*). The *I* gene, first described over four decades ago (1), has been deployed

widely in both snap and dry bean cultivars and provided effective resistance to BCMV for many years. Continued deployment of the *I* gene alone, however, has become a concern with the appearance of strains of BCMV that induce lethal systemic hypersensitive necrosis, either dependent (strains NL 2, NL 6) or independent (strains NL 3, NL 5, NL 8) of temperature (2). Temperature-insensitive, necrosis-inducing strains of BCMV, particularly NL 8 and to a lesser extent NL 3, have become a significant problem for the bean industry in the United States (4,9,13,20) and have necessitated a careful consideration of BCMV resistance gene deployment.

Our BCMV resistance breeding efforts have recently focused on two main objectives (11): immediate protection of *I* gene resistance in navy and black bean germ plasm and pyramiding of the *I* gene and recessive resistance genes, particularly *bc-2*^{2} and *bc-3*, in pinto and great northern genotypes adapted to Michigan. Greenhouse screening with the NL 3 strain of BCMV has greatly facilitated our efforts to protect the *I* gene with *bc-2*^{2} or *bc-3* resistance; breeding populations fixed for the *I* gene yet segregating for either *bc-2*^{2} or *bc-3* may be readily screened on the basis of the appearance of a "protected reaction" (*I*/− *bc-2*^{2}/*bc-2*^{2}, restricted necrotic local lesions; *I*/− *bc-3*/*bc-3*, no symptoms) versus local and systemic necrosis (*I*/− *Bc-2*^{2}/− or *I*/− *Bc-3*/−) following mechanical inoculation with NL 3. Epistatic interaction between the *I* gene and particularly *bc-3*, however, has presented a problem in our breeding efforts; *i*/*i* *bc-3*/*bc-3*, and *I*/− *bc-3*/*bc-3* genotypes may not be distinguished (without a test cross) in breeding populations that are segregating for both resistance genes (dominance with epistasis of *bc-3*/*bc-3* over *I*/−; hypostasis). Recent advances in DNA marker technology, particularly the random amplified polymorphic DNA (RAPD) assay (25,26), have made possible the elimination of laborious test crossing when pyramiding different resistance genes that exhibit epistasis. The objectives of our research were to 1) identify a RAPD marker tightly linked to the *I* gene, 2) determine linkage between the *I* gene and a linked RAPD marker in a variety of representative crosses in our breeding program, and 3) assess the usefulness of a linked RAPD marker in distinguishing *i*/*i* *bc-3*/*bc-3* and *I*/− *bc-3*/*bc-3* genotypes in breeding populations segregating for both the *I* and *bc-3* genes.

MATERIALS AND METHODS

Genetic materials, BCMV evaluations. Two sets of near-isogenic lines (NILs), isolated from heterogeneous inbred populations (6), were used to screen for RAPD markers linked to the *I* gene. The NILs were developed from inbred pinto (F_8) and great northern (F_6) populations derived from crosses between breeding lines or cultivars resistant (*I*/*I*; local and systemic necrosis) and susceptible (*i*/*i*; mosaic) to the NL 3 strain of BCMV (isolate obtained from M. Silbernagel, USDA-ARS, Prosser, WA). The great northern population (coded 90T-39) was developed from a cross between resistant and susceptible breeding lines (X87210 [*I*/*I*] and P86270 [*i*/*i*]), and the pinto population (coded 90T-4011) was developed from a cross between a resistant breeding line and a susceptible cultivar (P86230 [*I*/*I*] and Sierra [*i*/*i*]). In both instances, the original source of the *I* gene was tropical black bean germ plasm as described previously (12). Homozygous single-plant selections subsequently were made from the pinto and great northern lines (providing F_9 and F_7 NILs, respectively) showing a heterogeneous resistance response (local and systemic necrosis versus mosaic) in greenhouse inoculations with the NL 3 strain of BCMV. RAPD-marker screening was conducted using a bulked-DNA sample from the NL 3-resistant and NL 3-susceptible pinto and great northern NILs.

Five segregating populations were developed to determine linkage between putatively linked RAPD markers and the *I* gene. The first of these populations, G91201 (*I*/*I*)/Alpine great northern (*i*/*i*), was developed from a cross made and advanced to the F_2 in the greenhouse. BCMV evaluations were conducted using 11 to 14 F_3 progenies derived from each of 105 greenhouse-grown F_2 plants. Tissue samples for DNA extraction were collected from individual F_2 plants. The other four segregating populations were developed from crosses made in the greenhouse and advanced to the F_2 in a common field environment (Saginaw, MI, summer 1991). The four segregating populations were developed from crosses between different navy bean representatives of race Mesoamerica (Seafarer [*I*/*I*]/Michelite [*i*/*i*], N84004 [*I*/*I*]/Michelite) and between representatives of race Mesoamerica and race Durango (Seafarer/UI-114 pinto [*i*/*i*], N84004/UI-114) (see reference 21 for discussion of races of common bean). F_2-generation plants (from 196 to 227 individuals, depending on seed quantity) from the four crosses were evaluated for BCMV in the greenhouse. Tissue samples for DNA extraction were collected from one primary leaf of each individual plant prior to inoculation of the other primary leaf with BCMV.

All BCMV evaluations were based on reaction to the NL 3 strain of BCMV (2). Inocula for mechanical transmission were prepared from infected foliar tissue homogenized with carborundum in a mortar and pestle in a 10 mM phosphate buffer (3 mM K_2HPO_4, 7 mM Na_2HPO_4; pH 7.2). Greenhouse-grown seedlings of all test materials were rub-inoculated with the viral homogenate at the primary leaf stage. Plants showing no veinal or interveinal necrosis 10 days after inoculation were reinoculated to eliminate potential escapes. After 21 days, the presence of replicating virus in surviving plants was confirmed by visual ratings of characteristic mosaic symptoms and by indirect enzyme-linked immunosorbent assay (ELISA) with a monoclonal antibody (I2) specific for serogroup A isolates of BCMV (17).

RAPD procedures. The general procedures for DNA extraction and statistical (chi-square [χ^2] and linkage) analyses were as described previously (6,7,8,16) with minor modifications. The basic polymerase chain reaction (PCR) cycling profile that we have adopted consists of 3 cycles of 1 min/94 C, 1 min/35 C, 2 min/72 C; 31 cycles of 10 s/94 C, 20 s/40 C, 2 min/72 C; 1 cycle of 5 min/72 C; 1 s "auto-segment extension" (for extension phase of 31-cycle portion of PCR) utilizing a PE 480 DNA thermal cycler (Perkin Elmer Cetus, Norwalk, CT). Random decamer primers (mainly kits A through AE, Operon Technologies, Alameda, CA) were screened against the NILs to identify polymorphic DNA fragments. Prior to conducting linkage analyses (22), cosegregation of polymorphic DNA fragments and the *I* gene was confirmed by testing for the presence and absence of such DNA fragments in resistant and susceptible cultivars, respectively.

RAPD marker application. One of the objectives of our BCMV resistance breeding effort has been to pyramid the *I* and *bc-3* resistance genes into pinto and great northern genotypes adapted for production in the humid Great Lakes region of the American Midwest (12). In this program, 100 BC_3F_2 populations segregating for *bc-3* and the *I* gene were developed from a nonstructured backcrossing procedure where a diverse set of recurrent breeding lines (with and without the *I* gene) were used in backcrosses to BC_2F_1 plants (as opposed to homozygous *bc-3*/*bc-3* BC_2F_2 plants identified by BCMV screening following self-pollination). Greenhouse BCMV screening (with 10 to 20 BC_3F_2 plants) identified 55 populations (20 pinto and 35 great northern) that segregated for both *bc-3* and the *I* gene, 12 populations (9 pinto and 3 great northern) that segregated for *bc-3* alone, and 33 populations (18 pinto and 15 great northern) that segregated for the *I* gene alone and were therefore culled because surviving plants showed mosaic symptoms (*i*/*i*). Within each of the remaining 67 BC_3F_2 populations, one to four single-plant selections were made based on a symptomless response following inoculation with the NL 3 strain of BCMV. The selected lines (68 pinto and 96 great northern, 164 total) were advanced in the greenhouse where tissue samples for DNA extraction were collected as a bulk sample of three plants for each BC_3F_3 line. The presence and absence of the linked RAPD marker identified herein was determined for each of the 164 lines.

RESULTS

Identification and linkage of $OW13_{690}$. The screening of 460 random decamer primers against the susceptible and resistant

NILs identified one DNA fragment that cosegregated with the *I* gene (Fig. 1). This RAPD marker, designated $OW13_{690}$ (generated by a 5′-CACAGCGACA-3′ decamer), was found to be linked in coupling with the dominant *I* gene. Linkage analyses revealed a consistent and tight linkage between the *I* gene and $OW13_{690}$ in five different segregating populations (Table 1). The recombination values obtained varied from 1.3 ± 0.8 centimorgans (cM) for the Seafarer/UI-114 population to 5.0 ± 2.2 cM for the G91201/Alpine population. No significant differences in recombination values were observed among the four populations (Seafarer/UI-114, N84004/UI-114, Seafarer/Michelite, N84004/Michelite) that had been advanced to the F_2 generation by growing the F_1 in a common environment. In each population, segregation ratios consistent with completely dominant monogenic inheritance were observed for the $OW13_{690}$ RAPD marker and the *I* gene (Table 1).

We have previously reported on the differential utility of certain RAPD markers based on surveys for the presence or absence of a linked RAPD marker in representative genotypes of various market classes of common bean (8,16). In the present study (*data not shown*), we determined the presence and absence of $OW13_{690}$ in a collection of 40 common bean genotypes from seven different market classes (navy, black, pinto, great northern, pink, light red kidney, and dark red kidney). In each case, the presence or absence of $OW13_{690}$ was confirmed in genotypes that were known to possess or lack the *I* gene (Fig. 1).

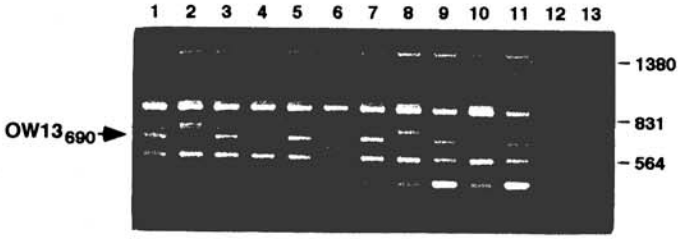

Fig. 1. Ethidium bromide-stained electrophoretic pattern of amplified DNA depicting $OW13_{690}$ RAPD marker. Key to individuals: 1) resistant (*I*/*I*) near-isogenic line (NIL), 2) susceptible (*i*/*i*) NIL, 3) G91201 great northern bean (*I*/*I*), 4) Alpine great northern bean (*i*/*i*), 5) Seafarer navy bean (*I*/*I*), 6) UI-114 pinto bean (*i*/*i*), 7) N84004 navy bean (*I*/*I*), 8) Michelite navy bean (*i*/*i*), 9) Montcalm dark red kidney bean (*I*/*I*), 10) Sierra pinto bean (*i*/*i*), 11) Redkloud light red kidney bean (*I*/*I*), 12) negative control (no template DNA added to polymerase chain reaction amplification), 13) molecular weight marker (λ *Hin*dIII/*Eco*RI; size of bands indicated in base pairs).

Application of $OW13_{690}$. Analysis of the BC_3F_3 lines showed a close agreement between the appearance of unprotected *I* gene reaction (systemic necrosis with NL 3 strain of BCMV) in a BC_3F_2 population and the presence of the $OW13_{690}$ RAPD marker in a family of single-plant selections made from that population on the basis of a symptomless response following inoculation with the NL 3 strain of BCMV (data summarized in Table 2). Among 55 BC_3F_3 families selected from populations segregating for the *I* gene, 50 families (91%) had at least one selection that showed the $OW13_{690}$ RAPD marker. Conversely, among 12 BC_3F_3 families selected from populations that showed no unprotected *I* gene reaction, no families were found that contained selections showing $OW13_{690}$. The five families that lacked $OW13_{690}$ (yet the *I* gene was confirmed as segregating alone in BC_3F_2) were each derived from populations where the *I* gene was segregating at a very low frequency because of susceptibility (*i*/*i*) of the final parent used in the backcross.

DISCUSSION

The *I* gene has long provided effective resistance to BCMV in several major market classes of common bean and, despite recent concerns arising from the appearance of temperature-insensitive necrosis-inducing strains of BCMV, efficiently serves to eliminate infected seed as the primary source of inoculum. Breeding for BCMV resistance has traditionally proceeded in a stepwise fashion with incorporation of the *I* gene as the first line of defense. Breeders that utilize temperature-insensitive, necrosis-inducing strains of BCMV (e.g., NL 3) are forced to delay screening to the F_3 generation because of hypersensitive lethality of *I*/− genotypes in the F_2 generation. In market classes where the *I* gene has been extensively incorporated, breeding strategies for BCMV resistance have largely involved protecting the *I* gene with one or more strain-specific recessive resistance genes. In market classes where the *I* gene is less common, however, bean breeders have had to choose whether or not to incorporate the *I* gene as a component of their BCMV resistance breeding strategy. A major objective of our breeding strategy has involved pyramiding of the *bc-3* gene and the hypostatic *I* gene to provide effective and durable resistance in market classes where neither gene is commonly found. Since the *bc-3* gene alone conditions resistance to all BCMV strains, it is not possible to detect accompanying resistance sources without a test cross. It would be im-

TABLE 1. Chi-square (χ^2) and linkage analyses of five common bean F_2 populations segregating for the $OW13_{690}$ RAPD marker and bean common mosaic virus (BCMV) resistance conferred by the dominant allele at the *I* locus

Population	Expected ratio[a]	Observed frequency	*I* Locus		$OW13_{690}$		cM ($r \pm SE$)[b]
			χ^2	*P*	χ^2	*P*	
Seafarer/UI-114	9:3:3:1	163:2:1:57	0.07	0.79	0.18	0.67	1.3 ± 0.8
N84004/UI-114	9:3:3:1	172:3:3:49	0.42	0.51	0.42	0.51	2.8 ± 1.1
Seafarer/Michelite	9:3:3:1	175:4:1:42	3.46	0.06	1.95	0.16	2.5 ± 1.1
N84004/Michelite	9:3:3:1	140:5:0:51	0.06	0.80	1.15	0.28	2.5 ± 1.1
G91201/Alpine[c]	3:6:3:1:2:1	32:43:3:0:2:25	2.45	0.29	0.00	1.00	5.0 ± 2.2

[a]Expected ratios: 9:3:3:1, 9 *I*/− +$OW13_{690}$: 3 *I*/− −$OW13_{690}$: 3 *i*/*i* +$OW13_{690}$: 1 *i*/*i* −$OW13_{690}$;
3:6:3:1:2:1, 3 *I*/*I* +$OW13_{690}$: 6 *I*/*i* +$OW13_{690}$: 3 *i*/*i* +$OW13_{690}$: 1 *I*/*I* −$OW13_{690}$: 2 *I*/− −$OW13_{690}$: 1 *i*/*i* −$OW13_{690}$.

[b]Linkage distance between $OW13_{690}$ and the *I* gene calculated using Linkage-1 (22).

[c]BCMV evaluation of G91201/Alpine population done with F_2-derived F_3 progenies, allowing characterization of 1:2:1 genotypic segregation ratio for *I* locus and linkage analysis using 1:2:1 and 3:1 ($OW13_{690}$) ratios.

TABLE 2. Presence (+$OW13_{690}$) and absence (−$OW13_{690}$) of RAPD marker linked to the *I* gene in pinto and great northern BC_3F_3 families derived from 67 BC_3F_2 populations identified as segregating for the *bc-3* gene and either segregating or not segregating for the dominant *I* gene[a]

Type	*I* Gene segregating in BC_3F_2			*I* Gene not segregating in BC_3F_2		
	Total	+$OW13_{690}$	−$OW13_{690}$	Total	+$OW13_{690}$	−$OW13_{690}$
Pinto	20	17	3	9	0	9
Great northern	35	33	2	3	0	3
Total	55	50	5	12	0	12

[a]Evaluations based on inoculation of 10 to 20 BC_3F_2 individuals with NL 3 strain of bean common mosaic virus (BCMV).

practical to detect desirable *I*/− *bc-3*/*bc-3* genotypes in early generation lines when hundreds of individuals would require test crossing. To this end, we have identified a RAPD marker linked to the *I* gene that will enable marker-assisted selection for the hypostatic *I* gene in selections previously identified as resistant (homozygous *bc-3*/*bc-3*) based on inoculations with the NL 3 strain of BCMV. The value of the $OW13_{690}$ RAPD marker has already been demonstrated in our program, where the presence of $OW13_{690}$ was confirmed in over 90% of the BC_3F_3 families previously selected for resistance (*bc-3*/*bc-3*) from BC_3F_2 populations segregating for both the *I* and *bc-3* resistance genes.

In previous studies, we identified RAPD markers for resistance genes that showed tight linkage in experimental mapping populations yet little association with susceptibility and resistance in cultivars of the gene pool from where the resistance gene was initially identified (8,16). Although the underlying mechanisms affecting recombination are complex (24), we postulated that those observations resulted from linkage disequilibrium between chromosomal regions derived from divergent genetic backgrounds (8). With respect to the *I* gene, its utilization as a source of BCMV resistance in the United States has followed two different paths from distinctly different sources: from the garden bean cultivar Corbett Refugee (1) and from landrace tropical black bean germ plasm (10). In our present studies, we had hypothesized that recombination frequencies in our mapping populations would vary depending on the origin of the *I* gene (Seafarer, from Corbett Refugee; N84004, from tropical black beans) and the origin of the susceptible parent (Michelite, race Mesoamerica; UI-114, race Durango). Our observation of statistically similar recombination values among the four segregating populations advanced to the F_2 in the same environment (Seafarer/UI-114, N84004/UI-114, Seafarer/Michelite, N84004/Michelite) does not disprove the hypothesis of recombination suppression between more genetically divergent backgrounds of common bean. If linkage disequilibrium was responsible for our previous observations, its apparent absence in the present study suggests that disequilibrium is less with intra-gene pool crosses of common bean. It is also possible that the chromosomal position of the *I* and $OW13_{690}$ loci may be responsible for the similarity of recombination values obtained in our study. A recently published genetic linkage map based on restriction fragment length polymorphism (19) assigned the *I* gene to the end of the second largest linkage group of the map (D2). Recombination or chiasma frequencies are known to be greater in more telomeric chromosomal positions (24), and the telomeric position of the *I* and $OW13_{690}$ loci may have affected recombination in addition to possible disequilibrium between chromosomal regions of diverse origin.

Three forms of evidence support the use of $OW13_{690}$ as a reliable marker for breeding applications: the tightness and consistency of linkage with the *I* gene, its association with resistance regardless of *I* gene source or bean market class (Fig. 1), and its absence and presence in selections derived from backcross populations segregating for one (*bc-3*) or both (*I* and *bc-3*) BCMV resistance genes. The use of this marker may also help to elucidate more basic properties of the *I* gene with regard to possible allelism with other types of potyvirus resistance in common bean (14, 5) and its association with color-intensifying effects (B locus) that are undesirable in certain seed types (23).

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