Generation and Molecular Mapping of a Sequence Characterized Amplified Region Marker Linked with the *Bct* Gene for Resistance to *Beet curly top virus* in Common Bean

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ABSTRACT

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A random amplified polymorphic DNA (RAPD) marker directly linked (0.0 cM) with a resistance gene was identified in a snap bean recombinant inbred population (Moncayo $\times$ Primo) consisting of 94 $F_{5,7}$ recombinant inbred lines that had uniform segregation for disease reaction to *Beet curly top virus* (BCTV) across three field locations. Resistance was conditioned by a single dominant allele tentatively designated *Bct*. Seven hundred and fifty decamer primers were screened to obtain the linked

RAPD marker that was then converted to a sequence characterized amplified region (SCAR) marker SAS8.1550. The SCAR mapped within a cluster of resistance genes on linkage group B7 of the core map. A survey of 103 BCTV-resistant and -susceptible snap and dry bean genotypes was conducted using SAS8.1550. Results showed that the SCAR would be highly useful for marker-assisted selection of *Bct* in snap and dry bean originating from the Andean gene pool. Marker-assisted selection for *Bct* will expedite the development of BCTV-resistant cultivars and minimize the need for cumbersome pathogen tests.

Additional keywords: begomovirus, bulked-segregant analysis, polymerase chain reaction-based DNA marker.

Beet curly top virus (BCTV) is the type member of the family *Geminiviridae* in the genus *Curtovirus*, and is vectored in a persistent manner by the beet leafhopper *Circulifer tenellus* (Baker). The virus disease is endemic to the semiarid regions of the western United States and severely affects common bean (*Phaseolus vulgaris* L.) as well as over 300 other plant species including sugar beet (*Beta vulgaris* L.), tomato (*Lycopersicon esculentum* Mill.), pepper (*Capsicum frutescens* L.), squash (*Cucurbita* spp.), pumpkin (*Cucurbita pepo* L.), cantaloupe (*Cucumis melo* L.), and cucumber (*Cucumis sativus* L.) (2).

The occurrence of BCTV in bean was first described in California by Carsner in 1919 (3). He later determined that BCTV was the cause of a serious disease epidemic in 1924 in Twin Falls, ID (4). BCTV remains a persistent disease problem for bean production in the Pacific Northwest. Many susceptible cultivars grown in the Columbia Basin of Washington State in 2002 suffered yield losses of up to 100%; whereas, the disease was virtually nonexistent in the region the preceding year. Severity of disease epidemics of BCTV vary by year and location due to fluctuations in leafhopper populations and timing of migrations from winter weed hosts into new spring agricultural crops (2,5,6,9,21,34).

Symptoms in bean caused by BCTV include leaf malformation, curling, chlorosis, leatheriness, and severe plant stunting. Susceptible plants infected at the seedling stage typically wither and die. Symptoms may be attenuated in intermediate (partially) resistant cultivars or in susceptible plants infected at later growth stages, although yields may be significantly reduced.

Important original sources of resistance to BCTV in bean include California Pink (17) and Red Mexican landrace cultivars, and Burtner, a small white pea bean sometimes grown as a snap bean (7,8). The Red Mexican source of resistance was transferred to great northern, pinto bean, and improved Red Mexican cultivars in the 1930s and 1940s (13,22,32). Pioneer, released in 1943 as the first BCTV-resistant snap bean, derives resistance from Burtner (33). Many BCTV-resistant snap beans developed subsequently also derive resistance from Burtner (29).

Schultz and Dean (26) determined that resistance from Burtner and Red Mexican, when crossed with susceptible parents Dark Red Kidney or Bountiful snap bean, was dominantly expressed in the F_1 generation. They used a two-gene dominant and recessive epistasis model in which one gene was dominant and the other recessively inherited to explain segregation ratios observed for disease reactions among F_2 and F_3 progenies. Mackie and Esau (17) crossed Robust (a small white pea bean) with California Pink and reported that BCTV resistance could be readily introgressed in white beans, demonstrating that resistance was not completely linked with pink seed color as previously suggested. However, their findings of a larger proportion of pink versus white progeny with resistance to BCTV supports the potential existence of a loose linkage between seed coat color and resistance to curly top.

A few studies (17,27) report quantitative reactions of bean lines and cultivars to BCTV. For example, Mackie and Esau (17) graded reactions from 1 to 5, where 1 = no visible symptoms or apparent yield loss and 5 = plants killed or entirely unproductive. Silbernagel (27) reported three levels of reactions among 52 snap

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bean cultivars as very susceptible, moderately susceptible, and partially resistant. It was later shown under growth chamber conditions that tolerance in Goldcrop snap bean could be partially overcome in 4% of the plants tested at temperatures exceeding 31°C (29).

The only effective control of BCTV in bean is genetic resistance. Numerous lines and cultivars with effective levels of resistance or tolerance to BCTV have been developed (32). However, breeding for resistance has been difficult because neither the occurrence nor the timing of epidemics in the field can be accurately predicted. Moreover, infection is usually sporadic and non-uniform across and within test plots even when disease pressure is severe, making it difficult for researchers to detect resistant lines and interpret studies on inheritance of resistance. Because the virus is not mechanically transmissible, greenhouse evaluations are difficult, requiring either viruliferous leafhoppers or *Agrobacterium tumefaciens*-mediated infectious clones to establish infection in test plants (10,31).

Given the difficulty of screening for resistance to BCTV in the field and greenhouse, marker-assisted selection (MAS) could provide an extremely useful tool for developing curly top disease-resistant bean cultivars in the absence of the pathogen. Our objectives were to study the inheritance of BCTV resistance segregating in a snap bean recombinant inbred line (RIL) population, identify tightly linked DNA markers with application for MAS of the resistance genes, and locate the genes on the molecular map.

MATERIALS AND METHODS

Population development. Ninety-four F_5 -derived F_7 ($F_{5,7}$) RILs were obtained from a cross between snap bean cvs. Primo and Moncayo. Primo, with large round pods, is susceptible to BCTV. Moncayo, with flat Romano-style pods, is highly resistant to BCTV. Moncayo derived curly top resistance from Burtner small white pea bean (7) via VR-Romano snap bean (28). The RILs were derived from individual F_2 plants by the single-seed descent breeding method (11).

Field experiments. Primo, Moncayo, 94 $F_{5,7}$ RILs, and Taylor Horticultural cranberry bean used as a susceptible check, were planted in replicated trials in the Columbia Basin region of Washington State in 2002 at 2-week intervals: 16 May at the Washington State University (WSU)-Roza Research Farm at Prosser (two replications), 31 May at the WSU Research Farm at Othello (three replications), and 13 June at the U.S. Department of Agriculture-Agriculture Research Service Research Site at Paterson (three replications). Experimental design was a randomized complete block. Each plot consisted of a single 3-m row length. Spacing between plots (rows) was 0.56 m. Seeding density was 234,000 seeds per hectare. The Othello and Paterson fields were fertilized, irrigated, and weeded to promote optimal plant growth. The Roza site was designed as a multiple stress nursery with compacted soil receiving no fertilizer, maintained at a 30% water deficit, and containing a high population of bean root rot pathogens.

Once occurrence of a severe BCTV epidemic was evident at Prosser, one F_1 seed from Primo $\times$ Moncayo and five residual F_1 seeds from the reciprocal cross Moncayo $\times$ Primo were germinated in individual pots in the greenhouse and subsequently transplanted to the field at the primary leaf stage. Simultaneously, four seeds of each of the parents, and VR-Romano and Tendercrop snap beans, which served as resistant and susceptible checks, respectively, were planted in the greenhouse and then transferred to the field. This test was conducted to determine whether resistance was dominant or recessively expressed in the F_1 generation.

To enable detection of quantitative resistance and associated quantitative trait loci (QTL), rating disease reaction on a scale from 1 to 9 was originally planned, where 1 = no symptoms and 0% of plants infected, 5 = moderate disease symptoms and up to

30% of plants infected, and 9 = severe disease symptoms with $\geq 99\%$ of the plants dead or dying. However, due to lack of intermediate reactions, a simple qualitative resistant (R) and susceptible (S) categorization of disease reaction was adapted. Although plots were assessed for curly top disease reaction throughout the growing season, only a final rating as either R or S was recorded at seed maturity, generally 100 days after planting. The presence or absence of BCTV in leaf tissue sampled from susceptible and resistant plants at the Prosser site was verified using the direct enzyme-linked immunosorbent assay (ELISA) system for geminiviruses (Agdia, Elkhart, IN) according to the manufacturer's instructions. Chi-square tests measured the fit for observed numbers of R and S RILs to expected ratios for a simply inherited trait.

DNA extraction protocol. A composite leaf tissue sample (approximately 50 mg) was collected from the first emerging trifoliate leaves of four plants of each line evaluated. Genomic DNA was extracted with the FastDNA Kit (Bio 101, Vista, CA) according to the manufacturer's instructions. The procedure involved placing the leaf tissue in a microcentrifuge tube with DNA extraction buffer and homogenizing the tissue for two 30-s periods at an intensity setting of 5.0 in the FastPrep FP 120 homogenizer (Bio 101/Savant). The DNA was bound to a matrix using a binding buffer, washed, and then eluted with 100 μ l of sterile distilled water. The purified DNA was adjusted to 10 ng/ μ l with a fluorometer prior to all polymerase chain reaction (PCR) reactions.

Screening for random amplified polymorphic DNA markers, linkage analysis, and development of a sequence characterized amplified region marker. DNA bulked from eight curly top-resistant RILs (R-bulk) and eight curly top-susceptible RILs (S-bulk), respectively, were screened with 750 random decamer primers (Operon Technologies, Alameda, CA) for random amplified polymorphic DNA (RAPD) markers detected as amplified fragments present in one bulk but absent in the other. Only RAPD markers detected between the bulks that cosegregated ($>85\%$) with disease reaction across the RILs that comprised the bulk DNA were assayed across the entire population of 94 RILs. PCR consisted of 25- μ l reactions containing 15 mM Tris (pH 8.0); 3 mM $MgCl_2$; 200 μ M each of dATP, dCTP, dGTP, and dTTP; 500 μ M decamer primer; 50 ng of template DNA; and 2.5 units of AmpliTaq Gold DNA polymerase (Applied Biosystems, Foster City, CA). Thermocycling parameters were optimized and a final profile was employed that consisted of a single cycle of 5 min at 95°C; 40 cycles of 1 min at 94°C, 1 min at 37°C, and 1 min at 72°C; and a final extension for 7 min at 72°C. Reactions were resolved by agarose gel (1.4%, wt/vol) electrophoresis in TAE (40 mM Tris, pH 8.0, 20 mM sodium acetate, and 1 mM EDTA) buffer. Gels were stained with ethidium bromide, visualized under UV light, and recorded with an AlphaImager 2000 (Alpha Innotech, San Leandro, CA).

Linkage analysis of assayed RAPDs and disease reaction (R versus S) in the RIL population was performed by Mapmaker 3.0 (16) using Group and Order commands. Distances between linked loci (centimorgans [cM]) were based on recombination fractions using the Kosambi (15) mapping function. To integrate BCTV resistance segregating in the Primo $\times$ Moncayo $F_{5,7}$ mapping population with the *Phaseolus* core map (12), RAPDs linked with disease resistance were assayed across the BAT 93 $\times$ Jalo EEP 558 mapping population provided by P. Gepts (University of California, Davis).

Three RAPDs found tightly linked with curly top resistance were assayed across 71 snap and 32 dry bean breeding lines and cultivars with known susceptible, partially resistant, or resistant reactions to BCTV. Commercial snap bean genotypes were provided by H. Moran, Seminis, and Syngenta seed company breeding programs. Many resistance-linked markers in common bean can only be used for MAS in specific gene pools (14,19). In lieu of this potential for gene pool specificity of the resistance-linked

markers identified in this study, representative bean genotypes from both the Andean and Middle American gene pools (30) were included in the marker assays.

An RAPD marker tightly linked with resistance (0% recombination) was excised from an agarose gel. DNA was purified from the excised gel band using GeneLute Minus agarose spin columns (Sigma-Aldrich, St. Louis) and cloned into the vector pCR4-Topo using the Topo TA Cloning Kit (Invitrogen, Carlsbad, CA) according to the manufacturer's instructions. Plasmid DNA was purified from *Escherichia coli* using alkaline lysis (25) and clones containing the PCR product were identified by digestion with *EcoRI* and visualized by agarose gel electrophoresis. The DNA product was sequenced using the dideoxy-chain termination method and extended primers were subsequently designed based on the terminal sequences of the cloned marker for conversion to a sequence characterized amplified region (SCAR) primer pair. Thermocycling parameters were optimized and a final profile was employed that consisted of a single cycle of 5 min at 95°C; 30 cycles of 1 min at 94°C, 1 min at 68°C, and 1 min at 72°C; and a final extension for 7 min at 72°C.

RESULTS AND DISCUSSION

A severe epidemic of BCTV in the Columbia Basin in 2002 enabled clear determination of disease reaction of bean genotypes. Primo, Taylor Horticulture, and 29 $F_{5,7}$ RILs from Primo $\times$ Moncayo exhibited severe curly top symptoms at the seedling stage resulting in plant death. In contrast, Moncayo and 65 $F_{5,7}$

RILs (Primo $\times$ Moncayo) exhibited no symptoms and were hence categorized as resistant. ELISA tests confirmed that the virus was absent in the resistant genotypes. Disease reactions of these genotypes were uniform across all replications and test sites. None of the six F_1 plants (Primo $\times$ Moncayo and reciprocal) exhibited disease symptoms, indicating that resistance was dominant. The transplanted parents and resistant and susceptible checks reacted to curly top as expected.

The observed ratio of 65 resistant and 29 susceptible RILs did not fit either 50% resistant to 50% susceptible (1:1) or 75% resistant to 25% susceptible (3:1) segregation ratios expected if one or two resistance genes, respectively, segregated in an RIL population. Because marker analysis identified only a single resistance gene (Fig. 1), skewed selection for RILs with BCTV resistance during development of the RIL population provides the only plausible explanation for the observed segregation ratio. During RIL development, two of the early generation increases took place under low curly top disease pressure in the field at Prosser, which would bias a false selection in favor of resistant RILs. Preferential transmission of gametes with the genomic region possessing the resistance allele during meiosis could also contribute to skewed segregation toward resistance.

Three dominant RAPD markers, AS8.1550 (Fig. 2), AH10.950, and I14.1700, detected between the R- and S-bulked DNAs had greater than 85% cosegregation with disease reaction among individual RILs comprising the bulks. Mapped across the entire population of 94 $F_{5,7}$ RILs, the three RAPDs were tightly linked in coupling (*cis*) with a single locus conditioning resistance to

Fig. 1. Partial linkage group of random amplified polymorphic DNA (RAPD) markers AH10.950 and I14.1700, sequence characterized amplified region (SCAR) SAS8.1550, and the dominant allele *Bct* conferring resistance to *Beet curly top virus* mapped in 94 $F_{5,7}$ recombinant inbred lines (RILs) Moncayo $\times$ Primo and position of I14.1700 RAPD and SAS8.1550 SCAR markers on linkage group B7 of the core map (80 RILs–BAT 93 $\times$ Jalo EEP 558). Markers with the prefix Bng (Florida) and D (Davis) are restriction fragment length polymorphism markers; *ChI* is the chalcone isomerase locus; and *Phs* is the phaseolin seed protein locus. Centimorgan (cM) distances are Kosambi. Quantitative trait loci (QTL) conditioning resistance to bacterial, fungal, and viral pathogens and a major allele for anthracnose resistance that map within the region are shown in the insert.

BCTV (Fig. 1). The tentative symbol *Bct* was assigned to this resistance allele given its dominant expression in the F₁ generation and ultimate derivation from Burtner. The designation of *Bct* is synonymous with the symbol *Ctv*, proposed by Provvidenti (24) for the dominant resistance gene in Burtner described by Schultz and Dean (26). The symbol *Bct* is preferable to *Ctv* because it better fits the example of gene symbols for virus resistance genes in the common bean gene listing (1).

The AS8.1550 RAPD marker (Fig. 1) was closest to *Bct* (0.0 cM) followed by I14.1700 (1.1 cM) and AH10.950 (5.0 cM). The less tightly linked RAPD markers AH10.950 and I14.1700 were present in most of the susceptible snap and dry bean lines and cultivars surveyed (data not shown) regardless of origin and therefore will have no direct application for MAS. Conversely, the AS8.1550 RAPD marker will be highly useful for MAS because it was present in all of the resistant and absent in most of the susceptible snap bean breeding lines and cultivars surveyed.

To facilitate detection of the AS8.1550 RAPD marker, it was converted to a SCAR marker designated SAS8.1550 (Fig. 2). The extended SCAR primer sequences are illustrated below and the underlined sequence represents the original AS8 decamer primer used in RAPD reactions: forward 5'-GGCTGCCAGTATCTTGTCTAACACC-3' and reverse 5'-GGCTGCCAGTGACGCAAT-TCTGCAG-3'.

The SCAR marker assay matched the known phenotype for 63 of the 71 snap beans surveyed (Table 1) and 15 of the 16 cranberry, kidney, and miscellaneous dry beans surveyed of Andean origin (Table 2). With regard to the eight snap bean genotypes that did not match phenotypes, the marker was present in susceptible genotypes in all cases. Although the eight susceptible snap bean lines were in advanced generations, it is possible that they continued to segregate for resistance such that a single resistant segregant with the marker may have been included in the four-plant bulked DNA sample assayed for each genotype. Alternatively, *Bct* may still be present in the susceptible genotypes but mutated to a nonfunctional status due to a potential instability of the gene. It is also possible that these eight susceptible snap beans possessed the resistance marker due to recombination events between the allele and marker during meiosis. However, if this were the case, more recombinants with BCTV resistance (*Bct*) and absent the marker should also have been observed. The landrace Jatu Rong (G122) from India was the only resistant Andean genotype (including snap bean) to lack the marker. An allelism test with *Bct* is warranted to determine whether G122 possesses novel resistance.

Two of the fourteen partially resistant genotypes lacked the SCAR marker SAS8.1550, indicating that genes other than *Bct* may condition BCTV resistance in common bean. Variable expression of *Bct* as influenced by genetic background could explain presence of the SCAR in partially resistant genotypes.

All dry beans surveyed of Middle American origin possessed the marker regardless of disease reaction, but the number of susceptible genotypes surveyed was limited (Table 2). Susceptible genotypes of Middle American origin (navy, black, red, great northern, and pinto bean) have a relatively low incidence of infected plants that generally exhibit less severe symptoms and never result in complete crop loss. Conversely, snap bean, cranberry, kidney, and other large-seeded bean market classes originating from the Andean gene pool are the most susceptible of all beans to BCTV, exhibiting a high disease incidence and complete crop failure. The ubiquitous presence of SAS8.1550 in resistant Middle American germ plasm indicates that MAS of *Bct* using this SCAR marker will be limited to the Andean gene pool, fortunately, where it is most needed. Snap beans are primarily classified as Andean in origin, although many of them possess traits introgressed from the Middle American gene pool. Bush Blue Lake (BBL) and pole snap beans were categorized as Middle American in a genetic diversity study (23), which may explain

why the BCTV-susceptible BBL types such as BBL156, Benchmark, and SB4249 and pole bean SB4122 possess the SAS8.1550 marker (Table 1).

The RAPD marker I14.1700 and SCAR marker SAS8.1550 each mapped to the same region of the *Phaseolus* core map (BAT 93 × Jalo EEP 558) on linkage group B7 (Fig. 1) between the *Asp* locus for seed brilliance and *Phs*, the phaseolin seed protein locus (12,14). It is perhaps no coincidence that an RAPD marker of similar size (1,560 bp) and generated by the same AS8 decamer primer was mapped in the same general location on linkage group B7 in the Dorado × XAN 176 mapping population (20). The AS8.1560 marker was tightly linked with a QTL that explained 39% of the phenotypic variation for resistance (reduced mosaic symptoms) to the whitefly-transmitted begomovirus *Bean golden yellow mosaic virus* (BGYMV). The SAS8.1550 SCAR from Moncayo could not be mapped in the Dorado × XAN 176 population because it was monomorphic. The similar-sized SAS8.1550 SCAR and AS8.1560 RAPD markers, although mapped in the same region of B7, likely vary in sequence perhaps by only a few base pairs.

The proximity of the BGYMV QTL to *Bct* on the *Phaseolus* linkage map suggests that *Bct* may have a pleiotrophic effect by conditioning partial resistance to other geminiviruses (e.g., BGYMV). The proximity also suggests that separate genes with specialized resistance to different begomoviruses may have derived from an ancient resistance gene that had undergone duplication and divergence, which is the premise for resistance gene clusters (18). Experiments to determine *Bct* effects on BGYMV or other geminiviruses would be worthwhile.

Fig. 2. A, Agarose gel showing amplification products from polymerase chain reaction (PCR) of genomic DNA using the random amplified polymorphic DNA decamer primer AS8. Arrow indicates the 1,550-bp fragment linked with the *Bct* resistance allele, which was cloned and sequenced to design sequence characterized amplified region (SCAR) primer SAS8.1550. **B,** Amplification product from PCR of *Bct* genomic DNA for the SAS8.1550 SCAR primer pair. Lane M, 1-kb marker; lane 1, susceptible recombinant inbred line (RIL) parent Primo; lane 2, resistant RIL parent Moncayo; lane 3, susceptible RIL MP 5 from Moncayo × Primo population; and lane 4, resistant RIL MP 59 from Moncayo × Primo population.

In addition to *Bct* and BGYMV resistances, a major gene for resistance to anthracnose (*Colletotrichum lindemuthianum*), and QTL for resistance to common bacterial blight (*Xanthomonas axonopodis phaseoli*), white mold (*Sclerotinia sclerotiorum*), and ashy stem blight (*Macrophomina phaseolina*) map in the same region of B7 (14), which further supports the presence of a resistance gene cluster in this region of B7 (Fig. 1). *Bct* is also located ap-

proximately 25 cM from the *P* locus that conditions seed coat color (recessive *p* gives white seed color). This linkage between *Bct* and *P* in coupling may explain the skewed segregation for more pink-seeded than white-seeded recombinants with curly top resistance in the Robust (white-seeded) × California Pink population (17).

The *Bct* resistance allele derives from Burtner small white bean and was first described by Schultz and Dean (26). This gene has

TABLE 1. Resistant (R), susceptible (S), and partially resistant (PR) commercial snap bean breeding lines and cultivars assayed for the sequence characterized amplified region (SCAR) marker SAS8.₁₅₅₀ linked in coupling with the putative *Bct* gene for resistance to *Beet curly top virus*

Genotype	Disease reaction	SCAR ^a	Genotype	Disease reaction	SCAR ^a
8100508	R	+	Hystyle	R	+
8100518	PR	+	Leon	R	+
8104379	R	+	Malibu	R	+
8104389	R	+	Masai	R	+
Alberta	R	+	Medinah	R	+
Amboto	R	+	Minuette	S	—
Astrel	R	+	Mirada	PR	+
Astun	PR	+	Moncayo	R	+
Banjo	R	+	Normandie	S	+
BBL156	S	+	NY6020-4	PR	+
Benchmark	S	+	Oregon 91G	PR	+
Benton	S	—	Palati	R	+
Bogota	S	—	Palati (sister)	PR	+
Brio	R	+	Paulista	R	+
Castano	R	+	Pix	PR	+
Castel	S	+	Primo	S	—
Celtic	R	+	Prosperity	S	—
Charon	R	+	Rapids	S	+
Concesa	R	+	Rhapsody	R	+
Corella	R	+	Rocdor	S	—
Dandy	S	—	Savannah	R	+
Derby	R	+	SB4122	S	+
Doral	R	+	SB4230	R	+
Dusky	PR	+	SB4240	R	+
Eagle	S	—	SB4249	S	+
Ebro	S	—	SB4253	R	+
Envy	S	—	SB4254	R	+
Espada	S	—	Shade	R	+
EZ-Pick	S	—	Summit	S	—
Festina	R	+	Sunburst	S	—
Flo	R	+	Tendercrop	S	—
Fury	R	+	Totem	S	—
Goldcrop	R	+	True Blue	S	+
Grenoble	R	+	VR-Romano	R	+
Hercules	S	—	Warrior	PR	—
Hialeah	S	—			

^a Marker present = +; marker absent = —.

TABLE 2. Resistant (R), susceptible (S), and partially resistant (PR) dry edible bean breeding lines and cultivars assayed for the sequence characterized amplified region (SCAR) marker SAS8.₁₅₅₀ linked in coupling with the putative *Bct* gene for resistance to *Beet curly top virus*

Andean				Middle American			
Type	Genotype	Disease reaction	SCAR ^a	Type	Genotype	Disease reaction	SCAR ^a
Cranberry	95-8186-C	R	+	Navy	Sanilac	S	+
	Cardinal	R	+		ND88-106-04	PR	+
	Etna	S	—		Norstar	PR	+
	Hooter	S	—	Black	Raven	R	+
	Taylor Horticulture	S	—		UI 911	PR	+
	USCR-7	S	—	Red	Rufus	R	+
	USCR-9	S	—				
	UI 686	PR	—	Great northern	UI 16	R	+
	Kardinal	R	+		UI 31	R	+
	USWA-33	R	+		UI 61	R	+
Kidney	USWA-39	R	+		Matterhorn	S	+
	CELRK	S	—		UI 59	S	+
	Foxfire	S	—		USWA-12	S	+
	Red Kloud	S	—	Pinto	Othello	R	+
	Montcalm	PR	+		UI 114	R	+
Miscellaneous	G122	R	—		Aztec	S	+
	Jalo EEP 558	S	—				

^a Marker present = +; marker absent = —.

been widely used during the past half-century to combat BCTV in common bean. The SCAR SAS8.1550 assay is sensitive and specific for the *Bct* resistance gene in snap and dry bean originating from the Andean gene pool. The SCAR will be applicable for high throughput marker detection systems. Therefore, MAS for *Bct* resistance to BCTV using the SAS8.1550 marker should considerably reduce the time currently required for screening germ plasm using field trials or *Agrobacterium*-mediated inoculation and expedite development of resistant cultivars.

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