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A major QTL for common bacterial blight resistance derives from the common bean great northern landrace cultivar Montana No. 5

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Abstract

Knowledge of the evolutionary origin and sources of pest resistance genes will facilitate gene deployment and development of crop cultivars with durable resistance. Our objective was to determine the source of common bacterial blight (CBB) resistance in the common bean Great Northern Nebraska #1 (GN#1) and GN#1 Selection 27 (GN#1 Sel 27). Several great northern cultivars including GN#1, GN#1 Sel 27, and Montana No. 5 (the female parent of the common x tepary bean interspecific population from which GN #1 and GN # 1 Sel 27 were derived) and known susceptible checks were evaluated for CBB reaction in field and greenhouse environments. These genotypes and CBB resistant and susceptible tepary bean including Tepary #4, the male parent and presumed contributor of CBB resistance to GN#1 and GN#1 Sel 27, were assayed for presence or absence of three SCAR markers tightly linked with independent QTLs conditioning CBB resistance. The parents and F_2 of Montana No. 5/GN #1 Sel 27 and Montana No. 5/Othello (CBB susceptible) were screened for CBB reaction and SCAR markers. CBB resistance in Montana No. 5 was comparable to that of GN#1 and GN#1 Sel 27. The SAP6 SCAR marker present in GN#1 and GN#1 Sel 27 was also present in Montana No. 5, and it co-segregated ($R^2 = 35\%$) with the CBB resistance in the Montana No. 5/Othello F_2 population. Although a few CBB resistant and susceptible transgressive segregants were found in the F_2 of Montana No. 5/GN #1 Sel 27 and later confirmed by F_3 progeny tests, SAP6 SCAR marker was present in all progenies. None of the tepary bean specific CBB resistance-linked SCAR markers were present in GN#1, GN#1 Sel 27, or Montana No. 5. A cluster analysis of 169 polymorphic PCR-based markers across three common bean and Tepary #4 indicated that GN#1, GN#1 Sel 27, and Montana No. 5 were closely related, and not related at all with Tepary #4. Thus, these results clearly indicate Montana No. 5, not Tepary #4, as the source of CBB resistance in GN#1 and GN#1 Sel 27.

Introduction

Common bacterial blight (CBB), caused by *Xanthomonas axonopodis* pv. *phaseoli* (Smith) Dye (*Xap*), is a major seed-borne disease of common bean (*Phaseolus vulgaris* L.) worldwide. Planting bacteria-free seed and growing resistant bean cultivars are the most effective means for controlling CBB. Developing cultivars with enhanced resistance to CBB is complicated, in part, by the paucity of available resistance sources (Beebe & Pastor-Corrales, 1991; Singh & Muñoz, 1999). The tepary bean (*P. acutifolius* A. Gray) possesses the highest level of resistance, whereas only low levels of resistance have been found in common and scarlet runner (*P. coccineus* L.) beans (Singh & Muñoz, 1999).

Honma (1956) is credited with the first interspecific cross between common and tepary bean. Embryo rescue was used to recover four self-fertile F_1 plants from a cross between great northern cultivar Montana

No. 5 (female) and Tepary #4 (male), which in turn produced a few F_2 seeds. Montana No. 5 was released as a cultivar in 1947 from a selection out of the common great northern landrace (Sutton & Coyne, 2002). The F_2 plants (Montana No. 5/Tepary #4) were selfed to produce a bulk F_3 population that was used to study segregation of CBB resistance, seed size, leaf length and width, and other plant traits. Segregation of plant traits was obviously skewed toward Montana No. 5, the *P. vulgaris* parent for data presented by Honma (1956) below.

Trait	Montana No. 5	F_3 mean (range)	Tepary #4
Seed size (g)	0.34	0.32 (0.20–0.47)	0.13
1^0 leaf length (mm)	72.6	75.7 (66–96)	46.3
1^0 leaf width (mm)	52.8	48.3 (41–63)	30.3

Disease score for reaction to CBB was normally distributed for 206 F_3 plants, but parental reactions were not reported. In a later study of 18 tepary bean accessions, the CBB reaction of Tepary #4 ranged from moderately resistant (12% = percentage infection of the inoculated leaf area) to susceptible (88% infection) with a mean of 41% against five *Xap* strains (Zaiter et al., 1989). Conversely, Zaiter et al. (1989) observed that six of the tepary bean accessions exhibited complete resistance (0% infection) to all five strains.

The Great Northern Nebraska No.1 (GN#1) cultivar, selected for CBB resistance and good agronomic characteristics, derived from interspecific cross of Montana No. 5/Tepary #4 was released by Dr. O'Keefe in 1961 (Coyne, 1961). A later-maturing off-type plant with higher CBB-resistance found in GN#1 by Coyne et al. (1963) was subsequently named Great Northern Nebraska #1 Selection 27 (GN#1 Sel 27). The GN#1 Sel 27 cultivar is the source of CBB resistance in subsequent great northern cultivars developed at University of Nebraska including Jules (Coyne & Schuster, 1970), Harris (Coyne et al., 1980), Star (Coyne & Schuster, 1974a), and Starlight (Coyne et al., 1991), and in the pinto cultivar Chase (Coyne et al., 1994), among others (Sutton & Coyne, 2002). This source of CBB resistance has also been extensively used in the tropics and subtropics of Latin America and elsewhere (Singh & Muñoz, 1999).

The belief that the CBB resistance in GN#1 Sel 27 derived from tepary bean played a crucial role in the subsequent searches for higher levels of CBB resistance in tepary bean. Moreover, additional interspecific crosses between common and tepary bean were made (Haghighi & Ascher, 1988; Mejía-Jiménez et al., 1994; Thomas & Waines, 1984), and higher levels of CBB resistance were transferred into common bean (McElroy, 1985; Scott & Michaels, 1992). It is noteworthy that F_1 obtained from these and other (Pratt et al., 1985; Parker & Michaels, 1986; Scott & Michaels, 1990) subsequent interspecific hybridizations between common and tepary beans were always sterile, and required at least one or two additional backcrosses to *P. vulgaris* to obtain germplasm with partial to complete stable fertility. Even with one or two additional crosses using *P. vulgaris* as the recurrent parent, these backcross-derived interspecific populations never generated a commercial cultivar. Thus, many bean researchers have been skeptical that a commercial cultivar like GN#1 could derive from a *P. vulgaris* x *P. acutifolius* single cross, given the repeated difficulty for obtaining fertile progeny from other interspecific crosses (Hucl & Scoles, 1985).

Evidence that the primary source of CBB resistance in GN#1 Sel 27 might actually derive from Montana No. 5 came from two different sources simultaneously. A field trial conducted in Erie, North Dakota in 1999 revealed that Montana No. 5 exhibited a moderate level of CBB resistance comparable to other cultivars with GN#1 Sel 27 derived resistance (Miklas et al., 2002). In this trial, Montana No. 5 and Harris scored a 6, whereas the great northern cultivar Weihing and pinto cultivar Othello scored 8 and 9, respectively, using a rating scale from 1 to 9 where 1 = no visible symptoms and 9 = severely diseased (Singh & Muñoz, 1999). In the laboratory at Prosser, Washington, a SCAR marker ($SAP6_{820}$) tightly linked with a major QTL for CBB resistance derived from GN#1 Sel 27 (Miklas et al., 1996; 1999; 2000a), amplified in Montana No. 5 but not in any tepary bean accession. Conversely, other markers linked with CBB resistance (Bai et al., 1997; Jung et al., 1997; Pedraza et al., 1997; Yu et al., 2000a) have permitted researchers to trace back the tepary-derived genes conferring resistance to CBB to tepary bean as the donor parent (Miklas et al., 1999).

Our objective was to test the hypothesis that CBB resistance in GN#1 Sel 27 is derived from Montana No. 5, not tepary bean. A more complete understanding of the derivation of resistance in GN#1 Sel 27 will help breeders make more informed decisions about which parental sources of resistance to use to achieve higher levels of CBB resistance in future cultivars.

Materials and methods

To further examine the resistance response of Montana No. 5, it was screened for reaction to CBB infection in a field trial planted 5 June 2000 at the Western Regional Experiment Center in North Platte, Nebraska. The great northern cultivars GN# 1, GN#1 Sel 27, Harris, Weihing, UI 59, and Matterhorn (susceptible check) were included. UI 59, like Montana No. 5 is a selection out of the great northern landrace. The test consisted of single row plots, 1.8 m in length with rows 0.9 m apart. Experiment design was a randomized complete-block with four replications. Plots were irrigated as needed for normal plant growth. No herbicides were used and no fertilizer was added. The nursery was planted in a field that contained residue from common blight infected plants. Infected seed of the CBB-susceptible pinto UI 114 were planted around the test site. The trial was inoculated on 28 July with a bacterial suspension applied to the plants at 150 PSI until water soaking of the leaves was visible. The bacterial suspension was made using one three-day old petri plate (15 x 100 mm) of *Xap* strain EK-11 per 3.8 L of water. Disease reaction was rated 14 August as the visible percentage of leaf surface area infected with CBB. Analysis of variance was performed and mean separation was computed by an F-test protected-LSD at the 5% probability level using PROC GLM (SAS Institute, 1989).

The same cultivars, plus great northern UI 123 (also a selection from the great northern landrace), and the pinto Othello as an additional susceptible check, were examined for CBB reaction in the greenhouse at Fargo, ND in October 2000. The cultivars were planted in a randomized complete block design with three replications, with one plant per pot per replication. Inoculation of the middle leaflet of the third trifoliolate using a pair of tongs (with brads attached to the inside of one half and a sponge dipped in inoculum attached to the inside of the other half) was conducted 21 days after planting (DAP) using 10^7 cfu mL^{-1} of *Xap* strain ND1. A rating scale (Miklas et al., 1996) from 1 to 9, whereby 1 = no visible disease symptoms and 9 = > 90% necrosis or chlorosis of the inoculated area was used to score disease reaction 14 days after inoculation (DAI). Analysis of variance was performed and mean separation was computed by an F-test protected-LSD at the 5% probability level.

The same cultivars screened in the greenhouse, and a few resistant and susceptible tepary bean accessions including Tepary #4, were assayed for the SAP6 SCAR marker which is tightly linked with a QTL on linkage group B10 that conditions resistance to CBB that derives from GN#1 Sel 27 (Miklas et al., 2000a; 2000b). Protocols described by Miklas et al. (2000a) were followed for DNA extraction and PCR. The SAP6 SCAR is an 820 bp fragment amplified by the following primer pair: forward – GTCACGTCTCCT-TAATAGTA / reverse – GTCACGTCT CAATAG-GCAAA; at an annealing temperature of 55 °C. Note that this marker does not amplify well when template DNA concentration exceeds 25 ng per 25 ul PCR reaction volume. These materials were also assayed for presence of the SU91 (Pedraza et al., 1997) and BC420 (Yu et al., 2000a) SCAR markers (Miklas, 2003; Miklas et al., 2000b) which are tightly linked with independent major-effect QTL conditioning CBB resistance. Both of these QTL derive from tepary bean PI 319433 (G 40020 = CIAT designation) through the common bean XAN 159 which was developed from an interspecific population, *P. vulgaris*∗3/*P. acutifolius* (McElroy, 1985; Thomas & Waines, 1984). All three SCAR markers SAP6, SU91, and BC420 can be multiplexed in a single PCR with an annealing temperature of 58 °C (Miklas et al., 1999; 2000b).

Cluster analysis was performed to examine the amount of tepary bean germplasm that was introgressed into the common bean cultivars GN#1 and GN#1 Sel 27 believed to be derived from the original interspecific cross Montana No. 5/Tepary #4. A total of 169 PCR-based polymorphic DNA markers were scored as present (1) or absent (0) across the four genotypes: Tepary #4, Montana No. 5, GN#1, and GN#1 Sel 27. The scored markers consisted of 129 RAPDs (Miklas et al., 2000a) generated by 19 decamers (Operon Technologies, Alameda, CA); 36 polymorphic bands generated by 8 SSR primer pairs: PV-ag001, PV-gccacc001, PV-atgc001, PV-at003, PV-ctt001, PV-at007, PV-at004, and PV-at006 (Yu et al., 2000b); and 4 SCARs ($SU91_{700}$, $BC420_{900}$, $SAP6_{820}$, and $BC409_{1250}$) linked with QTL conditioning resistance to CBB (Miklas, 2003). The NTSYS-pc program version 2.02k (Rohlf, 1993) was used to set up a pairwise genetic distance matrix (ALL) for the 169 polymorphic PCR-based markers. The simple matching similarity correspondence coefficient was used. The ALL distance matrix was clustered based on the unweighted pair-group method with arithmetic averages (UPGMA).

Two crosses, Montana No. 5/Othello and Montana No. 5/GN#1 Sel 27, were conducted to examine the inheritance of CBB resistance in Montana No. 5. The re-

lationship (R^2) of the SAP6 SCAR marker in Montana No. 5 with CBB resistance was examined in the first cross but not the second because both parents Montana No. 5 and GN#1 Sel 27 possessed the marker. Fifty F_2 plants from Montana No. 5/Othello and 137 F_2 plants from Montana No. 5/GN#1 Sel 27, and the parents Montana No. 5, Othello, and GN#1 Sel 27, were inoculated with *Xap* DR7 strain (10^7 cfu ml^{-1}) using the multiple needle method (Andrus, 1948). Each population was screened in a separate greenhouse experiment in Lincoln, NE, in December 2000 and April 2001, respectively. Artificial lighting was used to supplement natural light to maintain a 14 h photoperiod. From 8 to 16 individual plants of the parents and check were tested for each experiment. The first fully expanded trifoliolate leaf was inoculated 21 DAP, and rated for percentage (0 to 100%) infection of the inoculated area (15 mm^2) 14 DAI. Percentage disease infection and 1 to 9 ratings of infection are similar. Regression (PROC REG) analysis (SAS Institute, 1989) measured the degree of association between SAP6 SCAR marker and CBB reaction in the Montana No. 5/Othello F_2 population, using disease reaction as the dependent variable and marker presence = 1 vs. absence = 0 as the independent variable. T-tests were used to determine significant differences ($P < 0.05$) among mean disease reaction (% infection) of the parental and check means. Frequency distributions of disease reactions in the F_2 populations were tested for normality using the Shapiro-Wilk test statistic (W) using PROC UNIVARIATE (SAS Institute, 1989). A probability of $P < 0.01$ was used to indicate lack of fit.

From the Montana No. 5/GN#1 Sel 27 cross, four F_3 families, two from transgressive resistant and two from transgressive susceptible F_2 plants, were further screened for CBB reaction in the greenhouse to verify F_2 phenotype. The two great northern parents and Othello as a susceptible check also were included in this test. The same screening method as described above was followed except separate leaflets from the first fully expanded trifoliolate leaf of each plant were inoculated with separate *Xap* strains, DR7 and SC4A. A randomized complete block design with three replicates of two pots per replicate and two plants per pot for a total of 12 inoculated plants per genotype was used. Analysis of variance was performed and mean separation was computed by an F-test protected-LSD at the 5% probability level.

Table 1. Mean CBB reaction of Montana No. 5 compared with two other great northern landrace cultivars, four great northern cultivars that putatively derive resistance from tepary bean via the interspecific cross Montana No. 5/Tepary #4 (Honma, 1956), and two susceptible checks

Genotype	Field N. Platte, NE (2000)	Greenhouse Fargo, ND (2000)
	(%)[a]	(1–9)
Great northern cultivar from landrace		
Montana No. 5	7	5.7
UI 59	29	3.7
UI 123	nt[b]	5.7
Great northern cultivar from cross		
Harris	14	3.0
Weihing	5	6.3
GN#1	10	5.0
GN#1 Sel 27	4	1.3
Susceptible check		
Matterhorn	75	8.0
Othello	nt	7.0
LSD (0.05)	23	3.1

[a] CBB reaction based on percentage infection of the inoculated area or based upon a 1 to 9 rating, where 1 = no visible symptoms and 9 = > 90% necrosis or chlorosis of the inoculated area.
[b] not tested.

Results

Montana No. 5 and all the great northern cultivars possessing resistance putatively derived from the interspecific cross, Montana No. 5/Tepary #4, expressed the same high level of resistance (29% infection or less) to CBB disease in the North Platte field trial (Table 1). Conversely, the susceptible check Matterhorn with no direct lineage to GN#1 Sel 27 had 75% infection, a highly susceptible disease reaction. Similar results were obtained in the greenhouse at Fargo except that GN#1 Sel 27 expressed a higher level of resistance than Montana No. 5. There was not enough viable seed of Tepary #4 for inclusion in these experiments; however, Tepary #4 would be considered moderately susceptible to CBB in comparison to tepary bean accessions with high levels of resistance (Zaiter et al., 1989).

The SAP6 marker amplified in all the great northern cultivars, even the susceptible check Matterhorn, but did not amplify in tepary bean (Table 2). Conversely, the SU91 and BC420 markers amplified in all the tepary accessions, but did not amplify in any of

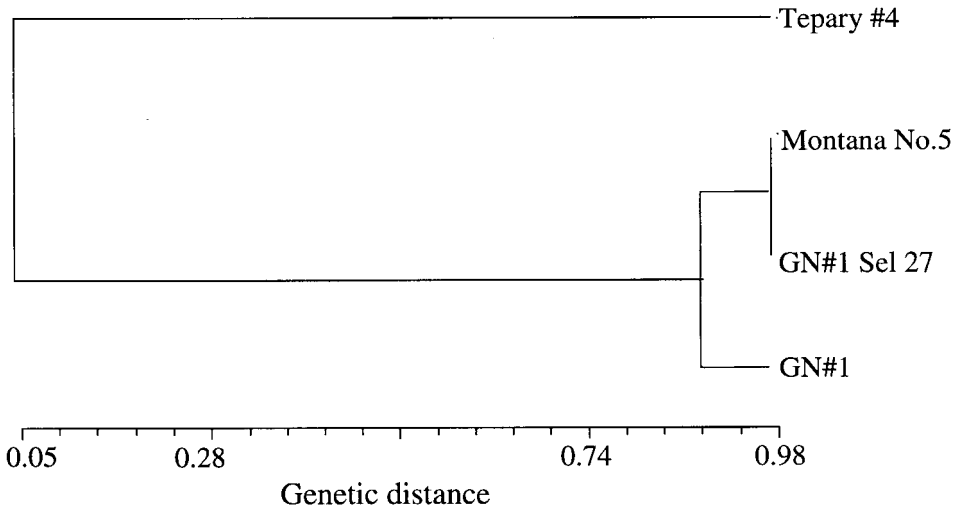

Figure 1. Cluster diagram based on genetic distance calculated from presence (1) or absence (0) of 169 polymorphic PCR-based markers across three common bean cultivars and one tepary bean genotype using the UPGMA method.

the common bean cultivars. Cluster analysis based on genetic distance, revealed a lack of presence of Tepary #4 genome in GN#1or GN#1 Sel 27 (Figure 1). Interestingly, GN#1 Sel 27 was more related to Montana No. 5, than to GN#1 from which it was derived.

Disease reaction (percentage of inoculated leaf area showing CBB symptoms) was normally distributed among the 50 F_2 plants from the cross Montana No. 5/Othello (Figure 2). The resistant disease reactions of Montana No. 5 (9% infection) and GN#1 Sel 27 (4% infection) were similar and significantly different from the susceptible parent Othello (92% infection). Regression analysis revealed that the SAP6 marker present in Montana No. 5 co-segregated with disease resistance in this F_2 population, as it explained $R^2 = 35\%$ ($P < 0.0001$) of the phenotypic variation for disease reaction. Mean disease reaction of the F_2 plants possessing the marker was 24% infection and those without SAP6 was 63% infection.

Disease reaction was also normally distributed among the 137 F_2 plants from the cross Montana No. 5/GN#1 Sel 27 (Figure 3). Disease resistance of GN#1 Sel 27 (20% infection) was slightly greater than Montana No. 5 (43% infection), with both parents expressing significantly better resistance than the susceptible Othello (100% infection). Warmer conditions and perhaps greater light intensity (Beebe, 1989; Arnaud-Santana et al., 1993) in the spring greenhouse contributed to more severe infection in this test. Disease reaction of the F_3 progeny from transgressive F_2 plants supported the occurrence of transgressive segregation for increased partial resistance in F_3 progeny line #9 and increased susceptibility in F_3 #14 to the DR7 strain (Table 3). Less separation of disease reaction occurred among F_3 lines and parents inoculated with *Xap* SC4A strain, because this strain is less virulent than DR7.

Discussion

The moderate resistance expressed by Montana No. 5 (Tables 1 and 3; Figures 2 and 3), combined with moderately susceptible reactions for Tepary #4 in the literature (Zaiter et al., 1989), suggests Montana No. 5 is the primary source of CBB resistance in progeny (GN#1, GN#1, Sel 27, Jules, Harris, etc.) derived from the interspecific cross Montana No. 5/Tepary #4 (Honma, 1956), not tepary bean as previously thought. In fact, the resistance expressed by Montana No. 5 in most experiments was comparable to the level of resistance expressed by GN#1 Sel 27.

Montana No. 5 and many of the other resistant cultivars possessed the SAP6 marker (Table 2). For Weihing (Coyne et al., 2000), which possesses less CBB resistance than Harris, the major-effect QTL on B10 is likely absent because the linked SAP6 marker is absent. The SAP6 marker is found in susceptible dry bean, primarily of Mesoamerican origin (unpublished

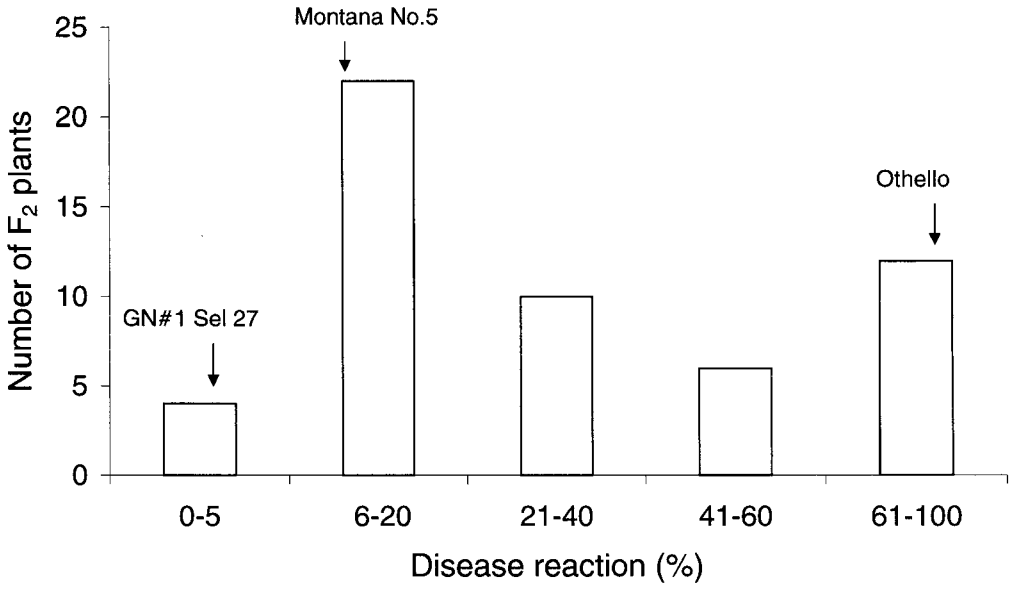

Figure 2. Histogram depicting distribution of CBB reaction (0 to 100% of the inoculated leaf area infected) frequency in F_2 population of Montana No. 5/Othello (W-test for normality = 0.85, $P > 0.0001$).

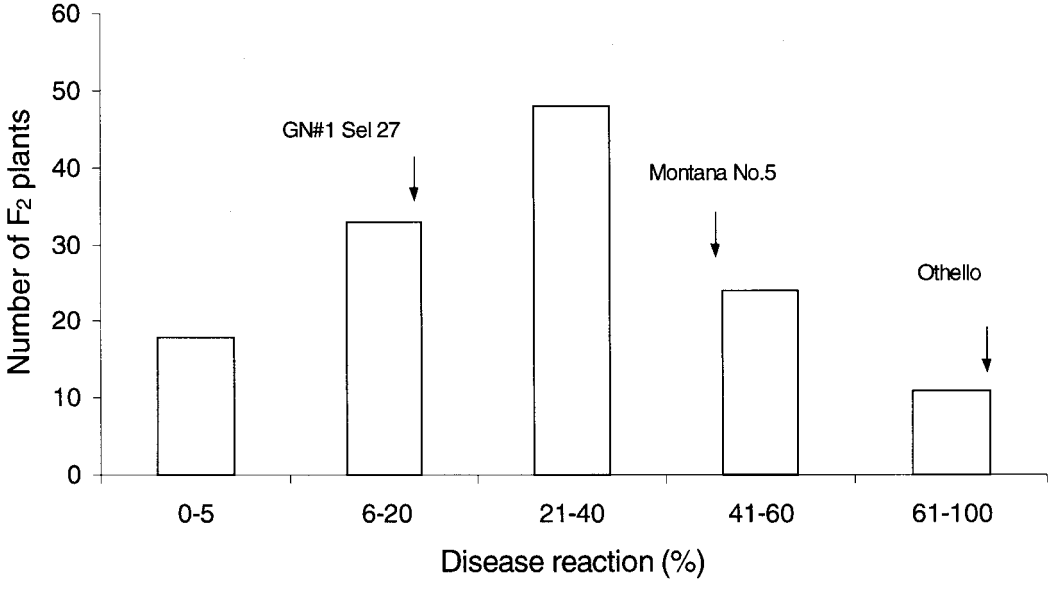

Figure 3. Histogram depicting distribution of CBB reaction (0 to 100% of the inoculated leaf area infected) frequency in F_2 population of Montana No. 5/GN#1 Sel 27 (W-test for normality = 0.95, $P > 0.0001$).

Table 2. Survey of SCAR markers[a] SAP6, SU91, and BC420 across Montana No. 5, UI 59, UI 123, XAN 159, great northern cultivars with CBB resistance putatively derived from the interspecific cross Montana No. 5/Tepary #4, susceptible dry bean checks, and resistant and susceptible tepary beans

Genotype	SCAR marker[b]		
	SAP6	SU91	BC420
Montana No. 5	+[b]	–	–
UI 59	+	–	–
UI 123	+	–	–
XAN 159	–	+	+
Great northern			
GN#1	+	–	–
GN#1 Sel 27	+	–	–
Harris	+	–	–
Weihing	–	–	–
Susceptible common bean			
Othello	–	–	–
Matterhorn	+	–	–
Resistant tepary bean			
Tepary #4	–	+	+
G40001 (PI 196932)	–	+	+
G40020 (PI 319433)	–	+	+
PI 440795	–	+	+
Susceptible tepary bean			
G40110	–	+	+
Mex-114	–	+	+

[a] SAP6 is linked with CBB resistance derived from GN#1 Sel 27, and SU91 and BC420 are linked with CBB resistance derived from PI 319433 tepary bean via the common bean XAN 159 from an interspecific cross.
[b] + and – represent presence and absence of the marker, respectively.

Table 3. Mean CBB reaction (% infection of the inoculated area) to *Xap* isolates DR7 and SC4A in a greenhouse test of the parents, a susceptible check, and four F_3 families derived from transgressive resistant (#7 and #9) and susceptible (#10 and #14) F_2 plants of Montana No. 5/GN#1 Sel 27

Genotype	DR7	SC4A
	%	%
Parents		
GN Montana #5	56	34
GN#1 Sel 27	57	25
F_3 lines		
#7	45	18
#9	29	9
#10	81	59
#14	84	46
Susceptible check		
Othello	100	87
LSD 0.05	24	27

data), and in Matterhorn (Kelly et al., 1999) which is partially derived from Race Mesoamerica germplasm. Thus, careful interpretation of presence/absence and use of SAP6 as a selectable marker is warranted. The SU91 and BC420 markers linked to independent QTL for CBB resistance derived from tepary bean PI 319433 via XAN 159 common bean were observed in the tepary bean accessions assayed. Because these markers were present in both resistant and susceptible tepary beans, SU91 and BC420 are not necessarily diagnostic for CBB resistance within *P. acutifolius*; however, they are diagnostic for CBB resistance in common bean.

The co-segregation ($R^2 = 35\%$) of SAP6 with CBB resistance in the Montana No. 5/Othello cross, provides clear evidence that Montana No. 5 possesses the same major-effect QTL (linked with SAP6 marker) as GN#1 and GN#1 Sel 27 and most other CBB resistant lines subsequently derived from them. Tighter co-segregation between SAP6 and CBB resistance was observed in the RIL mapping populations, Dorado/XAN 176 ($R^2 = 65\%$, Miklas et al., 1996) from which SAP6 was developed, and G122/Montcalm ($R^2 = 65\%$, unpublished results). Montcalm, developed from the GN#1/Red Kidney cross (Adams & Saettler, 1974), derives SAP6 marker and partial resistance to CBB from GN#1. Given the quantitative nature and strong effect of environment on expression of CBB resistance, the tighter co-segregation observed between SAP6 and CBB resistance for the inbred (RIL) populations probably results from more accurate assessment of phenotype due to replicated testing, as compared to non-replicated observations for F_2 populations.

A difference in degree of co-segregation would also be expected to occur between RIL and F_2 populations if the QTL allele linked with SAP6 had a dosage effect whereby two doses of the resistance allele ex-

pressed greater resistance than one dose. Additive effects from the combination of independent resistance sources have been observed (Beebe, 1989; Beebe & Pastor-Corrales, 1991; Silva et al., 1989). If such a dosage effect existed, then weaker co-segregation between SAP6 and CBB resistance would be expected for the F_2 population, because both homozygous and heterozygous individuals for the QTL would possess the dominant SAP6 marker.

Only one DNA fragment of similar size was amplified between Tepary #4 and GN#1, which indicates that genomic regions derived from tepary bean were generally lacking in GN#1 or GN#1 Sel 27 (Figure 1). We did not determine if the same-sized fragment from Tepary #4 and GN#1 had the same sequence. Bands of similar size but with diverse sequences are often reported as a limitation for using RAPDs in genetic distance studies because relatedness between any two individuals could be overestimated (Thorman & Osborn, 1992).

Preferential transmission of *P. vulgaris* alleles due to gametic selection (Guo et al., 1991) could explain why tepary genome was not readily detected in GN#1 or GN#1 Sel 27, using 169 PCR-based markers. For a more compatible *P. vulgaris* x *P. coccineus* interspecific F_2 population (Guo et al., 1991), only 60% of the RFLP probes tested showed preferential transmission, so complete reversion to *P. vulgaris* alleles in interspecific populations may eventually occur if selection for traits from the donor *Phaseolus* species is not practiced each generation. The lack of fertile F_1 and cultivars from subsequent *P. vulgaris*/*P. acutifolius* hybridizations, suggests Honma (1956) was fortunate to obtain fertile F_1's and a commercial cultivar (GN#1) so easily from the Montana No. 5/Tepary #4 hybridization.

The inheritance of CBB resistance of Montana No. 5 (Figure 2) appears to be quantitative but with at least one major-gene effect, because although the distribution for disease reaction among F_2 individuals was continuous in the Montana/Othello population, a bimodal shape in favor of resistance was visible. If resistance (0 to 60% infection) and susceptibility (61 to 100% infection) were separated accordingly, then a Mendelian 3 to 1 segregation ratio in favor of resistance would have been observed. Inheritance of CBB resistance in Montana No. 5 generally fits the 'quantitative inheritance but with some major-gene effect' previously reported for GN#1 Sel 27 (Coyne & Schuster, 1974b) and derived lines BAT 93 (Nodari et al., 1993), XAN 176 (Miklas et al., 1996; 2000a), and BAC 6 (Jung et al., 1996).

Although the 'major-effect' SAP6-linked QTL for CBB resistance is common to both Montana No. 5 and GN#1 Sel 27, a completely normal distribution for disease reaction in the Montana No. 5/GN#1 Sel 27 population indicates that differences due to minor resistance genes exist between the two genotypes (Figure 3). Differences exist despite the nearly-complete genetic relatedness observed between them (Figure 2). GN#1 Sel 27, with its slightly higher level of resistance, is a likely source of additional minor gene(s) for CBB resistance. The observance in F_2 and confirmation in F_3 progenies of transgressive segregation for resistance and susceptibility in this cross provides additional support for the presence of minor gene differences between Montana No. 5 and GN#1 Sel 27 (Table 3). The F_3 line #9 with transgressive segregation for a higher level of resistance may be useful in breeding for enhanced CBB resistance.

Until the late 1930's and early 1940's Idaho, Montana, Nebraska, and Wyoming were the principal great northern producing states in the USA (Mimms & Zaumeyer, 1947). Moreover, popular cultivars were either the landraces or selections from the landraces. The early great northern landrace cultivars grown in these western states were obtained directly from the Mandan native American tribe of North Dakota (Dean, 2000). The University of Idaho (UI) *Bean common mosaic virus* resistant cultivar Nos. 1, 56, 59, 60, 73, 77, 81, 95, and 123 were selections made in the great northern landraces (Pierce, 1934). Of these the most popular and long lived UI 59 and UI 123 were also tested for field and greenhouse reactions to CBB (Table 1) and for the presence or absence of SAP6 SCAR marker (Table 2). Both cultivars had the marker and similar CBB resistance as GN#1, GN#1 Sel 27, and Montana No. 5. The hot and humid environment prevalent in most growing seasons in North Dakota is highly conducive for CBB disease development, and extremely high levels of CBB infection occur in susceptible cultivars. Thus, CBB resistant great northern landraces either introduced from Middle America or similar mutants arising from a susceptible landrace cultivar would have had a selective advantage and become predominant in the region.

This rather indirect evidence and the results of our study clearly suggest that Montana No. 5 was the primary source of CBB resistance in the Montana No. 5/Tepary #4 interspecific cross made by Honma (1956), not the Tepary #4 parent as previously thought.

Thus, the major effect QTL for CBB resistance linked with SAP6 marker and derived from Montana No. 5 is present in GN#1, GN#1 Sel 27, and derived lines Montcalm, Jules, Harris, BAC 6 and XAN 176, among others.

Common bacterial blight resistance in Montana No. 5 is different from that of PI 207262, a small-seeded common bean landrace from Mexico (Coyne & Schuster, 1974b). The combination of Montana No. 5 and PI 207262 CBB resistances, as obtained in the tropical black bean XAN 112 (Beebe, 1989; Beebe & Pastor-Corrales, 1991) and a few other tropical lines, should be introduced into pintos, great northerns, kidneys and other medium and large seeded market types. The systematic combination of Montana No. 5 resistance with other *Phaseolus* species resistance should continue to produce cultivars with superior levels of CBB resistance.

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