

## QTL Analysis of ICA Bunsu-Derived Resistance to White Mold in a Pinto × Navy Bean Cross

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### ABSTRACT

**Breeding for genetic resistance to white mold [*Sclerotinia sclerotiorum* (Lib.) de Bary] in dry bean (*Phaseolus vulgaris* L.) is difficult because of low heritability. To facilitate breeding, researchers have sought to identify QTL underpinning genetic resistance to white mold. We identified two QTL conditioning ICA Bunsu-derived resistance to white mold in a pinto × navy bean (Aztec/ND88-106-04) recombinant inbred line (85 RILs) population. ND88-106-04 is a navy breeding line with resistance to white mold derived from ICA Bunsu navy. Aztec pinto is susceptible. The QTL were located to linkage groups B2 and B3 of the core map. The B2 QTL expressed in three of four field environments explaining 24.7, 9.0, and 8.7% of the phenotypic variation for disease severity score. The B3 QTL expressed in two of four environments, explaining 15.7 and 5.3% of the phenotypic variation. The B2 QTL was identified previously in ICA Bunsu × navy and ICA Bunsu × black bean RIL populations. The resistance conferred by the B2 QTL has a physiological basis due to association with stay green stem trait and lack of association with disease avoidance traits. The B3 QTL, undetected in previous studies, was associated with disease avoidance traits (canopy porosity, plant height), stay green stem trait, and maturity. The B2 QTL with stable expression in multiple environments and across genetic backgrounds will be most amenable to manipulation by breeders.**

**D**RY BEAN GROWN under irrigation and in temperate regions of the U.S. and elsewhere is extremely vulnerable to white mold disease. Pinto bean is the most important dry bean market class grown in the U.S., but is one of the most susceptible to white mold (Miklas, 2000). Bean growers surveyed in the Northern Great Plains and Michigan rated white mold as the most serious disease (Lamey et al., 2000; Webster and Kelly, 2000).

An integrated strategy encompassing crop rotation, minimum fertilizer rate, reduced irrigation, timely fungicide application, and less susceptible cultivar, is used to manage white mold disease in bean (Schwartz and Steadman, 1989). A few pinto cultivars, Chase (Coyné et al., 1994) and Maverick (Grafton et al., 1997), possess a low level of partial resistance to white mold. Pinto bean cultivars with moderate to high levels of partial resistance have not been developed yet because resistance is difficult to breed for, due in part to low heritability (Fuller et al., 1984; Lyons et al., 1987;

Kolkman and Kelly, 2002; Miklas and Grafton, 1992; Miklas et al., 2004; Park et al., 2001). The lack of adapted resistance sources also slows progress in breeding for improved resistance.

Moreover, breeding for genetic resistance is complex because it is conditioned by both avoidance and physiological mechanisms (Miklas et al., 2001). The use of avoidance mechanisms, including upright and open plant structure, less dense canopies and branching patterns, elevated pod set, and reduced lodging have been shown to reduce white mold damage (Schwartz et al., 1987; Kolkman and Kelly, 2002). These avoidance traits enhance penetration of the canopy by sun and aid air circulation, thereby creating a microclimate that is less conducive for infection and disease progression. Generally, most resistance detected by a laboratory or greenhouse screening method has a physiological basis. The stay green stem trait (Miklas et al., 2004), phytoalexin response (Sutton and Deverall, 1984; Miklas et al., 1993), and insensitivity to oxalic acid (Kolkman and Kelly, 2000; Chipps et al., 2005) represent physiological mechanisms implicated in genetic resistance to white mold disease.

A recent goal of many bean breeding programs has been to identify, tag, and map QTL for resistance to white mold to better understand the genetics underpinning partial resistance and to facilitate marker-assisted breeding for partial resistance obtained from different sources: navy 'ICA Bunsu', landrace G122 (PI 163120) from India, snap bean breeding line NY6020-4, and pompadour cultivar PC 50. Miklas et al. (2001) identified a QTL derived from G122 on linkage group B7 conditioning physiological resistance and on B1 contributing to disease avoidance. Similarly, two QTL from NY6020-4 were found on B6 associated with disease avoidance and on B8 conditioning physiological resistance (Miklas et al., 2003). Three major-effect QTL from PC-50 expressed in the greenhouse straw test and field were mapped to linkage groups B4, B7, and B8 (Park et al., 2001). Some of the resistance genes may be in common, as the B7 and B8 QTL from PC-50 map to the same general location as the B7 QTL from G122 and the B8 QTL from NY6020-4 (Miklas et al., 2006c). A preliminary report (Miklas, 2006) showed that marker-assisted backcrossing was successful in transferring white mold resistance conferred by B7 QTL from G122 and B8 QTL from NY6020-4 into susceptible pinto bean.

ICA Bunsu has been used extensively in breeding for white mold resistance in navy bean. The ICA Bunsu source of resistance segregating in crosses with suscep-

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**Abbreviations:** QTL, quantitative trait loci; RIL, recombinant inbred line; SRAP, sequence related amplified polymorphism; sCIM, simple composite interval mapping; MAS, marker-assisted selection.

tible navy 'Newport' was conditioned by QTL on linkage groups B2 and B7, and with susceptible black 'Raven' by QTL on B2, B5, B7 and B8 (Kolkman and Kelly, 2003; Ender and Kelly, 2005). The major-effect QTL residing on B2 and B7 were expressed in both populations. The QTL on B2 and B7 were confirmed by Kolkman and Kelly (2003) in a second navy bean population 'Huron'/Newport. The B2 QTL was associated with partial resistance expressed in the field; whereas, the B7 QTL was associated with resistance to oxalate in a greenhouse test (Kolkman and Kelly, 2000). The B7 QTL, independent of the QTL identified near the *Phs* locus by Miklas et al. (2001), was also significantly associated with agronomic traits including yield, seed size, lodging, and days to flower (Kolkman and Kelly, 2003).

To investigate the potential for ICA Bunsu as a source of white mold resistance in pinto bean improvement, Miklas et al. (2004) examined the inheritance of ICA Bunsu-derived resistance in a navy  $\times$  pinto bean recombinant inbred line population. They observed that partial field resistance had moderate heritability, was influenced by the environment, and was likely conditioned by genes with small effects. The stay-green trait where pods reach maturity but the plant remains green and physiologically active was associated with partial resistance in the population. A recombinant inbred line from the population, 'AN-37', which possessed pinto seed type and a high level of partial resistance to white mold was released as a germplasm line USPT-WM-1 (Miklas et al., 2006a). The objectives of this study were: i) to identify QTL conditioning partial resistance in the ICA-Bunsu derived navy  $\times$  pinto population developed by Miklas et al. (2004), and ii) to examine resistance-linked QTL for association with avoidance and agronomic traits.

## MATERIALS AND METHODS

A population of 85  $F_{5,8}$  recombinant inbred lines (RILs) generated by Miklas et al. (2004) from the cross Aztec/ND88-106-04 by single-seed descent method was used. Aztec is a semi-upright pinto bean (Kelly et al., 1992) susceptible to white mold. ND88-106-04 from the cross N85007/ICA Bunsu is an upright navy bean breeding line with partial resistance to white mold derived from ICA Bunsu (Tu and Beversdorf, 1982).

The 85  $F_{5,8}$  RILs were previously evaluated by Miklas et al. (2004) for reaction to white mold in four field environments: Hatton, ND, in 2001; Carrington, ND, in 2002; and in Paterson, WA, in 2001 and 2002. The planting dates, experimental design, plot size, planting density, weed control, fertilizer for optimum plant growth, and irrigation to maintain optimum moisture conditions for white mold infection, were as previously described.

A brief description of the phenotypic traits measured in these trials by Miklas et al. (2004) is given below. Disease reaction was scored from 1 to 9 based on combined incidence and severity of infection at physiological maturity (defined as 80% of the pods at harvest maturity), where 1 = no diseased plants and 9 = 80–100% diseased plants and/or 60–100% infected tissue (Miklas et al., 2001). Disease avoidance traits measured included: canopy porosity (Deshpande, 1992) at mid-pod fill and scored from 1 to 5, where 1 = a porous canopy with the soil surface highly visible, and 5 = dense canopy with

no soil visible; canopy height (cm) at mid-pod fill; and lodging scored at physiological maturity from 1 to 9; where 1 = no lodging and 9 = > 90% lodged. Lodging was not obtained for the WA 2002 trial. Maturity (d) was recorded as the number of days from planting to physiological maturity. Stay-green stem, only recorded for the ND trials, was scored from 1 to 5 at physiological maturity; where 1 = 0–20% green stem and 5 = 80–100% green stem. Plot yield ( $\text{kg ha}^{-1}$ ) and seed weight ( $\text{g } 100 \text{ seeds}^{-1}$ ) were measured.

A composite leaf tissue sample was collected from the first emerging trifoliolate leaves of four plants of each line. Genomic DNA was extracted using the FastDNA Kit (Bio 101, Vista, CA) according to the manufacturer's instructions. The purified DNA was adjusted to  $10 \text{ ng } \mu\text{L}^{-1}$  using a fluorometer before all PCR reactions. Equal amounts of DNA from the seven most resistant and seven most susceptible RILs as determined by average disease reaction across all four field tests were used to form resistant (R-bulk) and susceptible (S-bulk) DNA bulk samples, respectively. Similarly to the multi-trait bulking strategy described by Kolkman and Kelly (2003), other traits were considered in the formation of the DNA bulk samples, as above average yield and average maturity were sought for individual RILs comprising the resistant bulk. The multi-trait bulk segregant analysis was used to target identification of QTL conditioning resistance in an acceptable phenotype, lacking such undesirable traits as low yield and late maturity. Lines with inherently low yield will escape white mold disease primarily as a result of less biomass enabling air and light penetration of the canopy. Later maturity was significantly correlated with less disease in the Aztec/ND88-106-04 RIL population (Miklas et al., 2004).

The R- and S-bulks were screened against 650 decamer primers (Operon Technologies, Alameda, CA) for presence of random amplified polymorphic DNA (RAPD) markers. PCR consisted of 25  $\mu\text{L}$ -reactions containing 2 U Stoffel fragment DNA polymerase (Applied Biosystems, Foster City, CA), 1 X Stoffel buffer, 0.2  $\mu\text{M}$  primer, 5 mM  $\text{MgCl}_2$ , 200  $\mu\text{M}$  each dNTP, and 25 ng template DNA. Amplifications were performed on a Peltier Thermal Cycler PTC-200 (MJ Research Inc., Waltham, MA) programmed for 2 min at 94°C; 3 cycles of 1 min at 94°C, 1 min at 32°C, 1 min at 72°C; 30 cycles of 10 s at 94°C, 20 s at 37°C, 2 min at 72°C; 5 min at 72°C. RAPDs observed between the bulks were assayed across the individuals that comprised the bulks. RAPDs that cosegregated with disease reaction in at least 78% of the individuals comprising the DNA bulks were subsequently assayed across the entire population of 85 RILs.

Other phenotypic traits and markers that were mapped across the whole population included: the *I* gene for resistance to *Bean common mosaic virus* on linkage group B2 detected by segregation for top necrosis (ND88-106-04) vs. mosaic (Aztec) using strain NL-3 D and linked sequence characterized amplified region (SCAR) marker SW13.690; the *P* gene on B7 detected by segregation for white seed color (ND88-106-04) versus colored seed (Aztec); *Asp* gene on B7 conferring seed brilliance detected by shiny (ND88-106-04) vs. dull (Aztec) seed coat appearance; the SAP6.820 and BC409.1250 SCARs linked with a QTL for resistance to common bacterial blight [caused by *Xanthomonas axonopodis* pv. *phaseoli* (Smith) Vauterin et al.] on B10, although both parents lacked the resistance QTL the markers segregated anyway; and the *Ur-3* gene for resistance to rust caused by *Uromyces appendiculatus* (Pers.) Unger var. *appendiculatus* on B11 detected by hypersensitive fleck (ND88-106-04) vs. large pustule reaction (Aztec) to Race 53 and linked SCAR SK14.620. RAPDs identified between a different pair of R- and S-DNA bulks from the Aztec/

ND88-106-04 population designed for presence and absence of the *Ur-3* gene as part of another study were also mapped across the whole population and included in this study. The annealing temperatures and cycling profiles for the SCARs are listed online at <http://www.ars.usda.gov/SP2UserFiles/Place/53540000/miklas/SCARtable.pdf> (verified 23 Oct. 2006).

Following DNA sequencer procedures of Miklas et al. (2006b), 18 target region amplified polymorphism (TRAP) markers (Hu and Vick, 2003) were mapped across the entire population. The fixed primers for the TRAP protocol were based on published disease-related gene sequence from a *Helianthus* EST (LRR domain) 5'-GAAAGACGAAGGAA-CAGG-3', and a bean resistance gene analog RGA1 (NBS domain) 5'-CCTAAATGGGAGGAAGTG-3'. The same arbitrary primer 5'-GGAACCAAACACATGAAGA-3' from a sequence related amplified polymorphism (SRAP) primer list published by Li et al. (2003) was paired with each fixed primer.

Eleven AFLP markers that were linked with QTL conditioning resistance to white mold derived from ICA Bunsu in a cross with a susceptible black bean 'Raven' were assayed across the whole population of Aztec/ND88-106-04 RILs following the protocol of Ender and Kelly (2005). Only five of the 11 AFLPs from the Ender and Kelly (2005) study mapped in the Aztec/ND88-106-04 population, including EAGM-CAT.165 on linkage group B2 (named AFLP 1 in this study), EAGGMCAA.170 and EAGTMCAT.360 on B5, EACAMCCG.460 on B8, and EAGTMCTT.300 (named AFLP 10) on partial linkage group LG-5, were not integrated with the core map (Ender, 2003). AFLPs associated with the major QTL for resistance in the Bunsu/Raven population on B7 were monomorphic in the Aztec/ND88-106-04 population.

A partial linkage map of the mapped markers was constructed using Mapmaker 3.0 (Lander et al., 1987). A pairwise

linkage analysis of the marker data, imposing a minimum LOD score of 3.0 and maximum distance of 30 cM, was used to establish the linkage groups. Three-point and multi-point log-likelihood thresholds (LOD) of 2.5 and 2.0, respectively, were used to order the markers within linkage groups with the Order and Ripple commands. Centimorgan (cM) distances between linked loci were based on recombination fractions using the Kosambi (1944) mapping function. The mapping of QTL-linked RAPDs across the BAT 93/Jalo EEP 558 RIL population (BJ) was used to anchor partial linkage groups obtained in this study to the core *P. vulgaris* map (Freyre et al., 1998). Seventy-one of the RILs from the BJ population were available for this purpose.

Association of markers with disease score and other traits was determined by linear regression of the trait means on individual marker genotypes using PROC GLM (SAS Institute, 1987). An *F*-test significant at  $P \leq 0.01$  for a trait measured at a single location was used to indicate linkage between a marker and QTL. If significant for one location, a QTL was considered significant at  $P \leq 0.05$  level for the other locations.

For the partial linkage groups, simple composite interval mapping (sCIM) performed by MQTL (Tinker and Mather, 1995) across location means, was also used to detect QTL with main effects for partial resistance in the field as measured by disease score: for canopy porosity, canopy height, lodging associated with disease avoidance, for stay green stem, harvest maturity, yield and seed weight. A significant QTL was declared if the test statistic calculated by MQTL was greater than the significance threshold determined by permutation analyses (1000 permutations) of the data sets for each trait based on a 5% experiment-wise error rate (Doerge and Rebai, 1996). The test statistic calculated by MQTL was multiplied by 0.22 to equate to a LOD score.

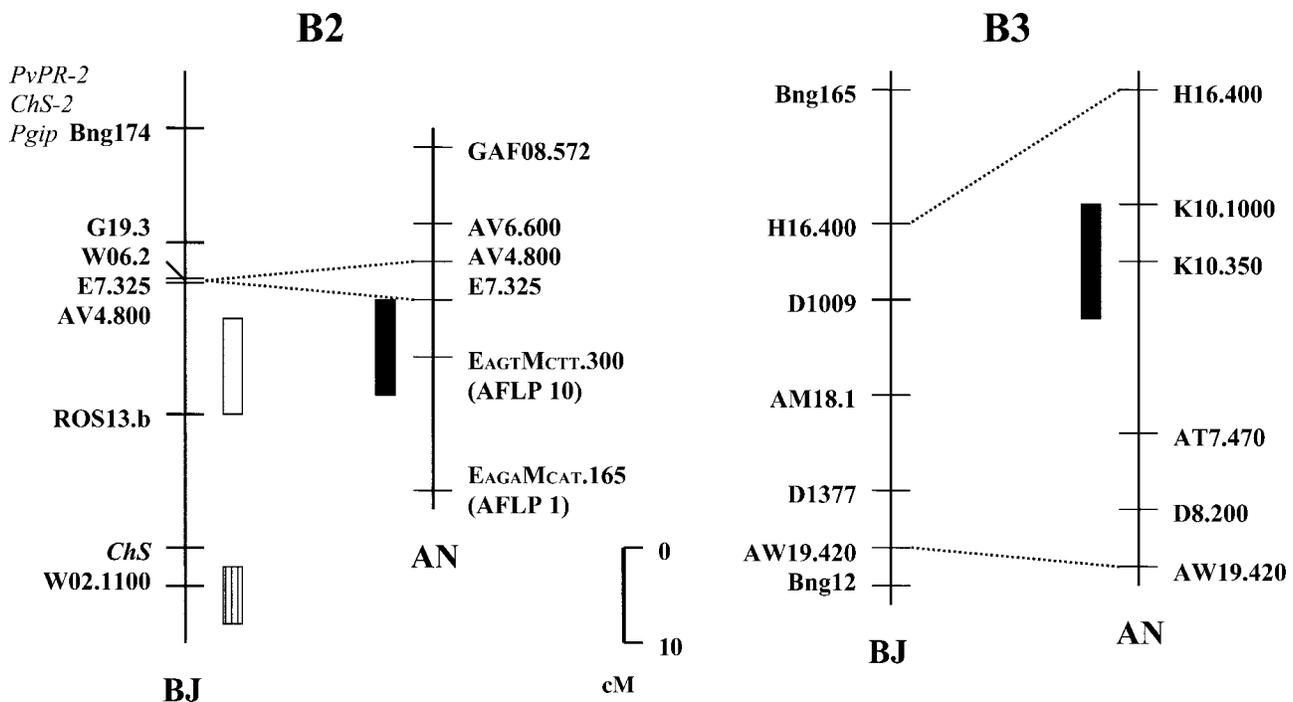
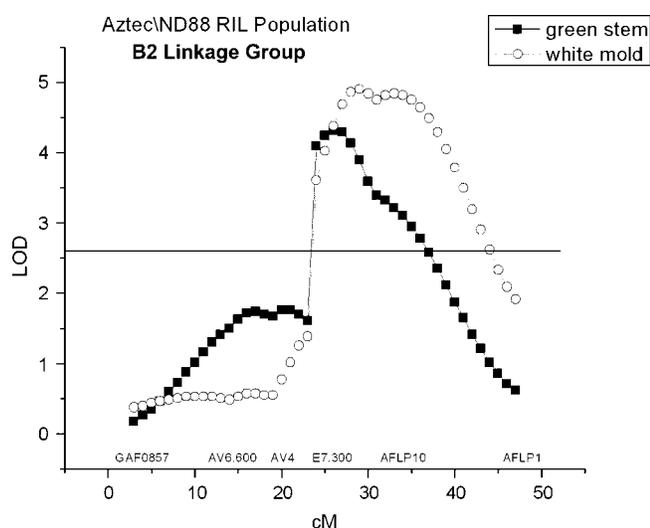


Fig. 1. Integration of partial linkage groups in Aztec/ND88-104-04 (AN) RIL population with the BAT93/Jalo EEP558 (BJ) core map. QTL locations conferring resistance to white mold in AN population depicted by solid bar, in BJ map extrapolated from QTL in Bunsu/Newport (Kolkman and Kelly, 2003) and Bunsu/Raven (Ender and Kelly, 2005) populations by open bar, and in BJ map extrapolated from QTL in PC50/XAN-159 population (Park et al., 2001) by cross-hatch bar. Framework RFLP markers for the core map have D and Bng prefixes (Freyre et al., 1998), defense related genes are italicized, and all other markers are RAPDs. For the AN linkage groups, GAF indicates a TRAP marker (Miklas et al., 2006b), E...M... markers are AFLPs with size of the amplified fragment indicated after the period, and all other markers are RAPDs with sizes similarly indicated.



**Fig. 2.** Simple composite interval mapping (sCIM) by MQTL (Tinker and Mather, 1995) for detection of QTL on linkage group B2 conditioning resistance to white mold disease severity in the Aztec/ND88-106-04 RIL population across four environments and stay green stem trait across two environments. The horizontal line represents the average of the 5% significance level for each trait based on 1000 permutations.

## RESULTS AND DISCUSSION

A total of 125 loci were mapped across the Aztec/ND88-106-04 RIL population. There were 4 genes (*I*, *P*, *Asp*, and *Ur-3*), 5 AFLP, 4 SCAR, 18 TRAP, and 94 RAPD markers generated by 51 decamer primers. Thirty-four decamer primers detected a RAPD between R- and S-bulks for white mold disease score and met the criteria for mapping across the whole population. Seven partial linkage groups were constructed from 70 of the loci, but only the two partial linkage groups with QTL conferring partial resistance to white mold are shown (Fig. 1). The partial linkage groups that possessed QTL integrated with linkage groups B2 and B3 of the core map (Freyre et al., 1998). The RAPD markers AV4.800 and E7.325 (B2) and H16.400 and AW19.420 (B3), mapped in both Aztec/ND88-106-04 and BAT93/Jalo EEP558 populations, enabling integration of the two partial linkage groups. Integration of the QTL on B2 is supported

further by the location of EAGM<sub>CAT</sub>.165 AFLP marker on linkage group B2 near RAPD marker OP15.1800 by Ender and Kelly (2005).

### QTL on Linkage Group B2

A QTL for partial resistance to white mold found on linkage group B2 (defined by AFLP marker EAGT<sub>MCTT</sub>.300; Fig. 1 and 2) was expressed in three of the four test environments (Table 1), accounting for 24.7% of the phenotypic variation for disease score for 2001 and 9.0% for 2002 in Paterson, and 8.7% for Carrington. AFLP markers EAGT<sub>MCTT</sub>.300 and EAGM<sub>CAT</sub>.165 were linked (15.8 cM) in this study. For the ICA Bunsu/Raven population, both EAGT<sub>MCTT</sub>.300 and EAGM<sub>CAT</sub>.165 markers were associated with QTL for resistance, but were not linked (Ender, 2003). The EAGM<sub>CAT</sub>.165 marker, positioned on linkage group B2 near RAPD marker OP15.1800, explained 8.7% of the phenotypic variation across two environments (Ender and Kelly, 2005). The same QTL, observed by Kolkman and Kelly (2003) near RAPD marker BC20.1800 in repulsion phase linkage, explained 11.7% of the variation in ICA Bunsu/Newport population tested across three environments and 40% in Huron/Newport population (consisting of 28 RILs) tested across four environments. Our data combined with previous results validate the existence of a major QTL conferring partial physiological resistance to white mold, between framework loci *ChS* and *Bng174* of the core map (Freyre et al., 1998; Miklas et al., 2006c).

Huron navy bean (Kelly et al., 1994), which lacks ICA Bunsu in its pedigree, possesses a major QTL in the same genomic region of linkage group B2, suggesting that multiple sources of the QTL may exist. A minor-effect QTL from the Andean source, PC-50, detected in only a single environment, maps in the same vicinity of the B2 linkage group as the ICA Bunsu- and Huron-derived QTL (Park et al., 2001). The QTL detected in the PC-50/XAN159 population is located near RAPD W02.1100, which is about 10 cM from where the ICA Bunsu-derived QTL is located (Fig. 1). As recognized by Kolkman and Kelly (2003) and Ender and Kelly (2005), proximity of these QTL on B2 with *Chalcone synthase*, patho-

**Table 1.** Examination of the relationship of QTL conferring white mold resistance on linkage groups B2 and B3 with disease avoidance and agronomic traits across four environments.

Trait†	Marker	Linkage group	Paterson WA, 2001	Paterson WA, 2002	Hatton ND, 2001	Carrington ND, 2002	Additive effect‡
<i>R</i> <sup>2</sup> (F-test <i>P</i> value)							
Disease score (1–9)	AFLP 10	B2	24.7 (< 0.0001)	9.0 (0.009)	ns§	8.7 (0.01)	–0.68
	K10.350	B3	5.3 (0.041)	ns	15.7 (0.0003)	ns	–0.48
Porosity (1–5)	K10.350	B3	21.2 (< 0.0001)	35.7 (< 0.0001)	17.0 (0.0002)	7.1 (0.018)	0.78
Lodging (1–9)	AFLP 10	B2	ns	NM¶	ns	9.9 (0.006)	–0.30
	K10.350	B3	8.3 (0.01)	NM	ns	ns	0.07
Plant height (cm)	K10.350	B3	ns	11.1 (0.003)	13.4 (0.009)	ns	2.84
Harvest maturity (d)	K10.350	B3	17.3 (0.0001)	33.4 (< 0.0001)	32.1 (< 0.0001)	15.4 (0.0003)	3.60
Stay green (1–5)	K10.350	B3	NM	NM	8.8 (0.008)	12.4 (0.0014)	0.52
Yield (kg ha <sup>–1</sup> )	K10.350	B3	ns	13.8 (0.0007)	6.3 (0.025)	ns	–114

† Disease score where 1 = none and 9 = most severe; canopy porosity where 1 = open and 5 = closed; lodging where 1 = none and 9 = completely lodged; stay green stem trait where 1 = most dry and 5 = most green.

‡ Additive effect from allelic substitution of ND88-106-04 allele for Aztec allele.

§ ns = nonsignificant.

¶ NM = trait not measured.

genesis related protein *PvPr-2* (Walter et al., 1990) and polygalacturonase-inhibiting protein *Pgip* (Toubert et al., 1992), suggests that one or more of these pathogen defense-related genes may be involved in host defense response to *S. sclerotiorum*. The same genes may combat other pathogens, as QTL for resistance to web blight [caused by *Thanatephorus cucumeris* (Frank) Donk] and common bacterial blight map in the same region (Miklas et al., 2006c). The involvement of general pathogen defense-related genes in the partial resistance response is supported by preliminary transcript expression profiling of EST microarrays which have shown such genes to be up-regulated in response to *S. sclerotiorum* infection in canola (*Brassica napus* L.) (Hegedus et al., 2005).

In addition to proximity to fungal defense-related genes, partial resistance conferred by the B2 QTL likely has a physiological basis because it is not influenced by disease avoidance traits (open canopy, plant height). The association of this QTL with reduced lodging at Carrington was not consistent across locations (Table 1), and was not detected by composite interval mapping (Fig. 2). The ND88–106–04 allele contributed to both reduced disease severity and reduced lodging. This B2 QTL was also associated with lodging in other populations (Kolkman and Kelly, 2003; Ender and Kelly, 2005). In those populations reduced lodging was contributed by the white mold susceptible parents (Newport and Raven) with upright architecture; whereas, reduced disease severity was contributed by the white mold resistant parent ICA Bunsu with a more prostrate indeterminate plant growth habit. Furthermore, interval mapping in those populations depicted a distance of about 10 to 12 cM between the LOD peaks for lodging and disease severity traits; thus, as suggested by Kolkman and Kelly (2003), separate mechanisms contributing to less disease from each parent were located in the same general region of the linkage group.

Composite interval mapping showed a strong association between the B2 QTL and expression of the stay green stem trait, which further supports a physiological basis for the partial resistance conferred by this QTL. Plants with stay green trait, described as pods reaching harvest maturity while the branches and stems remain green, are still likely to be physiologically active and engaged in plant defense response (Miklas et al., 2004).

### QTL on Linkage Group B3

A QTL for partial resistance to white mold identified on linkage group B3, nearest RAPD marker K10.350, was expressed in two environments explaining 5.3% of the variation for disease score for Paterson in 2001 and 15.7% for Hatton (Table 1). Disease avoidance and physiological mechanisms likely underlie the partial resistance expressed by this QTL, because the same genomic region was associated with canopy porosity across all environments, canopy height in two environments, stay green stem trait in two environments, and lodging in one environment. In addition, the same genomic region was associated with harvest maturity in all environments and yield in two environments. The genomic region for this QTL is more than 20 cM from the minor-effect QTL for

resistance to white mold located on B3 near RAPD marker G03.1150 (Park et al., 2001).

The ND88–106–04 allele for the B3 QTL, as defined by K10.350 RAPD marker, contributed favorably to reduced disease severity, increased plant height, and increased green stem trait. Conversely, the Aztec allele contributed favorably to increased yield, earlier maturity, and a more open canopy. Composite interval mapping depicts a 19 cM span of the genome which influences disease avoidance (open canopy) from the Aztec parent near K10.350 marker and physiological resistance (stay green stem) from the ND88–106–04 parent near AT7.470 marker (Fig. 3). For this genomic region, the challenge for breeders will be to select progeny which recombine favorable traits contributed by each parent in coupling-phase linkage.

### CONCLUSIONS

In summary, the QTL for partial resistance derived from ICA-Bunsu navy bean located on linkage group B2 is stably expressed across multiple environments and in different genetic backgrounds including pinto, navy (Kolkman and Kelly, 2003) and black bean (Ender and Kelly, 2005). The effect of the B2 QTL has a physiological basis due to association with stay green stem trait, and lack of association with disease avoidance traits in this and previous studies.

The RAPD markers, 015.1800 and BC20.1800, identified previously (Kolkman and Kelly, 2003; Ender and Kelly, 2005), and the AFLP 10 (EAGT/MCT.300) identified herein, provide initial markers for testing the effectiveness of marker-assisted breeding for the B2 QTL to improve white mold resistance. It may be worthwhile to backcross the B2 QTL into a susceptible background using marker-assisted selection (MAS) to validate the effect of the QTL in absence of other re-

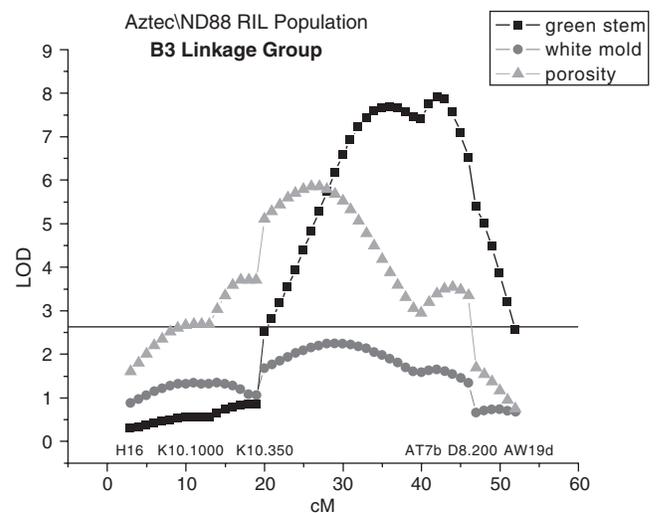


Fig. 3. Simple composite interval mapping (sCIM) by MQTL (Tinker and Mather, 1995) for detection of QTL on linkage group B3 conditioning resistance to white mold disease severity in the Aztec/ND88–106–04 RIL population across four environments, canopy porosity across four environments, and stay green stem trait across two environments. The horizontal line represents the average of the 5% significance level for each trait based on 1000 permutations.

sistance factors, validate the effectiveness of MAS for the QTL itself, and to generate near-isogenic inbred lines for eventual analysis of candidate genes underlying the QTL. Eventually, those markers found to be tightly linked with the B2 QTL should be converted to SCAR markers to facilitate use by different programs.

The B3 QTL for partial resistance, as determined by regression analysis, had a minor effect in two environments. Composite interval mapping determined the QTL did not have a main effect; and, to date, this same B3 QTL has not been detected in any other populations. These results for B3 QTL suggest further research would be required to validate the importance of this genomic region in breeding for resistance to white mold disease in common bean.

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