Comparative QTL Map for White Mold Resistance in Common Bean, and Characterization of Partial Resistance in Dry Bean Lines VA19 and I9365-31

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ABSTRACT

White mold caused by the necrotrophic fungus *Sclerotinia sclerotiorum* (Lib.) de Bary limits common bean (*Phaseolus vulgaris* L.) production in temperate climates. Disease resistance has been identified, but breeding is hampered by a paucity of resistance sources and complex inheritance, as numerous quantitative trait loci (QTL) conditioning partial resistance have been found. Our objectives were to characterize the partial white mold resistance found in breeding lines I9365-31 and VA19 and to construct a comparative linkage map for all the white mold resistance QTL identified to date. Recombinant inbred line (RIL) populations 'Benton'/VA19 (BV) and 'Raven'/I9365-31 (R31) consisting of 79 F_6 and 105 F_5 RILs, respectively, were evaluated for white mold (WM) reaction in multiple greenhouse and field tests. Two QTL were found in BV, WM2.2 expressed in the greenhouse (R^2 = 33%) and the field (13%) and WM8.3 expressed in field (11%) only, and seven were found in R31 (WM2.2, WM4.2, WM5.3, WM5.4, WM6.1, WM7.3, WM8.4), three expressed in greenhouse tests and four in the field, ranging in phenotypic variance from 5 to 52%. These QTL were compared with 26 previously identified QTL, resulting in a comparative linkage map of 35 QTL, which coalesced into 21 distinct regions across nine linkage groups. Four QTL found in R31 were novel. Sequence characterized amplified region markers associated with WM2.2, WM8.3, and WM7.3 QTL were generated. The comparative linkage map provides a framework for integrating and interpreting future QTL studies concerning white mold resistance in common bean.

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Abbreviations: BJ, BAT93/Jalo EEP558 mapping population; BV, Benton/VA19 mapping population; CIM, composite interval mapping; DAP, days after planting; DG, DOR364/G19833 mapping population; EST, expressed sequence tag; GN, great northern; LG, linkage group; MAS, marker-assisted selection; PDA, potato dextrose agar; QTL, quantitative trait locus or loci; R31, Raven/I9365-31 mapping population; RAPD, random amplified polymorphic DNA; RIL, recombinant inbred line; RFLP, restriction fragment length polymorphism; SCAR, sequence characterized amplified region; SRAP, sequence related amplified polymorphism; SSR, simple sequence repeat (microsatellite); WM, white mold.

White mold disease caused by the necrotrophic fungus *Sclerotinia sclerotiorum* (Lib.) de Bary is a major constraint to common (dry edible and snap, garden, or green) bean (*Phaseolus vulgaris* L.) production worldwide. Currently, white mold is controlled with a combination of cultural practices and fungicide applications. Cultural control practices include increased plant and row spacing, orienting rows in the direction of prevailing winds, scheduling irrigations to allow plants to dry before nightfall, and avoiding excessive irrigation during blossom senescence. These cultural practices reduce disease severity but also contribute to lower yields in the absence of severe disease pressure.

Cultivars with upright architecture, reduced branching, reduced flowering, and resistance to lodging contribute to disease avoidance by creating a more open canopy that is less conducive to white mold development (Miklas et al., 2001; Schwartz et al.,

Published in Crop Sci. 51:123–139 (2011).
doi: 10.2135/cropsci2010.06.0356
Published online 22 Nov. 2010.

1987). Disease avoidance, however, can be overcome by moderate disease pressure and may also contribute to lower yields. Fungicides provide good disease control but are costly, and the timing and mode of application are critical for effective control (Miklas et al., 2004). For these reasons, the use of partially resistant cultivars in conjunction with cultural practices and fungicide applications has been recognized as the most effective approach to reducing the impact of white mold disease (Schwartz and Steadman, 1989; Steadman, 1979). This strategy has been difficult to implement owing to the lack of resistance sources and difficulties with introgressing resistance quantitative trait loci (QTL) into adapted cultivars (Miklas et al., 2006a; Miklas, 2007; Ender et al., 2008).

Since 2001 numerous QTL conditioning partial resistance to white mold have been identified and mapped in dry and snap bean (see reviews by Miklas et al., 2006b; Miklas and Singh, 2007). Sources for these QTL are 'ICA Bunsi' navy bean (Ender and Kelly, 2005; Kolkman and Kelly, 2003; Miklas et al., 2007), 'Huron' navy bean (Kolkman and Kelly, 2003), G122 (PI 163120) landrace from India (Maxwell et al., 2007; Miklas et al., 2001), 'PC-50' pompadour from Dominican Republic (Park et al., 2001), snap bean breeding line NY6020-4 (Miklas et al., 2003), and PI 318695 wild *P. vulgaris* from Mexico (Grafton et al., 2002; Terpstra and Kelly, 2008).

G122, PC-50, and NY6020-4 represent resistance sources from the Andean gene pool, and ICA Bunsi, Huron, and PI 318695 represent sources from the Middle American gene pool (see Singh et al. [1991] for an explanation of bean gene pools). Partial resistance is mediated by these collective QTL through two distinct mechanisms: avoidance, which inhibits establishment of the infection, and physiological resistance, which impedes spread of the pathogen through the plant.

The light red kidney bean breeding line VA19, purported by a private seed company to possess partial resistance, is better adapted (earlier maturity, uniform dry down, higher yield potential) in the United States than other Andean sources of resistance such as PC-50 and A 195 (Singh et al., 2007). Thus, VA19 may represent a more breeder-friendly source of partial resistance for introgression into 'Andean' market classes: kidney, cranberry, yellow, and some snap beans, for U.S. production.

Fewer resistance sources have been observed in the Middle American gene pool (Pascual et al., 2010). The small-seeded black bean germplasm I9365-31 was released by Miklas et al. (1998) as a potential Middle American source of partial resistance to white mold. I9365-31 derives from an interspecific population between common bean and scarlet runner bean (*Phaseolus coccineus* L.). Scarlet runner bean possesses high levels of resistance to white mold (Abawi et al., 1978; Myers et al., 2008; Schwartz et al., 2006) and could contribute to the partial resistance present in I9365-31.

In this study our objectives were to characterize the partial resistance to white mold in VA19 and I9365-31 using recombinant inbred line (RIL) populations screened in multiple greenhouse and field environments and populated with random amplified polymorphic DNA (RAPD), sequence related amplified polymorphism (SRAP), and simple sequence repeat (SSR) markers for QTL analysis. Another important objective was to develop a comprehensive genetic linkage map for all white mold resistance QTL to facilitate integration and comparison of QTL from this and future studies with previously reported QTL.

MATERIALS AND METHODS

Mapping Populations

A population of 79 F_6–derived RILs was generated by the single seed descent method from a cross between 'Benton' and VA19 (BV). Benton, which is highly susceptible to white mold, is a processing snap bean with determinate Type I (Singh, 1982) bush growth habit, heat tolerance, wide adaptation, mid-season maturity, and round pods with satisfactory length and color. VA19 developed by P. Ascher, University of Minnesota, possesses partial resistance to white mold and is a light-red kidney bean line derived from congruity backcrossing of *P. vulgaris* 'Red Cloud' to tepary bean (*P. acutifolius*) 'G40445' (PI 263590) (Anderson et al., 2002). The complete details concerning development of VA19 are unclear. The line does not exhibit any traits characteristic of tepary bean (i.e., narrow leaflets, small seed size, drought tolerance), and tepary bean as a species is very susceptible to white mold. Also, no cultivar named Red Cloud exists, but there is a 'Red Kloud' light-red kidney, developed at Cornell University in 1973, which has determinate Type I growth habit (Coyne, 1999) and is the likely parent of VA19.

A population of 105 F_5–derived RILs was similarly generated from a biparental cross between 'Raven'/I9365-31 (R31). Raven (Kelly et al., 1994) is a black bean cultivar with upright architecture and indeterminate short vine Type II growth habit. Raven is highly susceptible to *S. sclerotiorum* but is resistant to other diseases including *Bean common mosaic virus* (BCMV) and *Bean common mosaic necrosis virus* owing to the presence of the *I* and *bc-3* genes, anthracnose [*Colletrotrichum lindemuthianum* (Sacc. & Magnus)] from *Co-1* gene, and rust [*Uromyces appendiculatus* (Pers.) Unger var. *appendiculatus*] from *Ur-3* gene. Raven is representative of race Mesoamerica within the Middle American gene pool (Singh et al., 1991). I9365-31 is a black bean germplasm release from an interspecific cross between *P. vulgaris*★2/*P. coccineus* (Miklas et al., 1998). It was selected for high yield potential and partial resistance to white mold that could derive from the *P. coccineus* parent. I9365-31 possesses indeterminate Type III prostrate growth habit and *I* gene resistance to BCMV.

The microsatellite map for common bean developed by Blair et al. (2003) was used as the reference map for integrating QTL conferring partial resistance to white mold, identified in this study and in previous studies, to genomic regions within linkage groups. This microsatellite map links the DOR364/G19833 (DG) map from CIAT and BAT93/Jalo EEP558 (BJ) core map from Davis (CA) with the Florida restriction fragment length polymorphism (RFLP) map. The Florida portion of the microsatellite map was

not used for this study. Collectively, both BJ and DG are referred to as core maps throughout this study. The BJ linkage groups possessed 22 microsatellites as positioned by Yu et al. (2000) relative to 71 existing markers, and the DG linkage groups included 246 markers, of which 78 were microsatellites.

The QTL in BV and R31 were integrated to the reference map directly by assaying QTL-linked markers across 72 BJ RILs (courtesy of P. Gepts) or indirectly (comparatively mapped) via association of the QTL in BV and R31 with markers that were previously mapped in BJ or DG populations. The BJ marker dataset (http://www.plantsciences.ucdavis.edu/gepts/bjril7.htm; verified 12 Oct. 2010) for direct QTL integration consisted of 180 RFLP, RAPD, sequence characterized amplified region (SCAR), and gene-based markers and was similar to the marker dataset described in Freyre et al. (1998). Previous studies (Miklas et al., 2003; Kolkman and Kelly, 2003) similarly integrated QTL for partial resistance to white mold in BJ by assay of QTL-linked markers directly in BJ or by comparison with markers that were already mapped in BJ or DG. All QTL were located on the BJ core map portion of the microsatellite map from Blair et al. (2003) with the DG core map portion serving primarily as a bridge between white mold mapping populations and BJ.

Greenhouse Screening

The BV and R31 RIL populations were evaluated for reaction to white mold in separate experiments. The 79 BV RILs and parents were screened for white mold reaction in three separate greenhouse environments at Prosser, WA, using the straw test described by Petzoldt and Dickson (1996). Straw Test 1 was conducted November 2004, Test 2 in April 2005, and Test 3 in June 2005. Three replicate plants of each RIL were included in each test, with the replications randomized in complete blocks. Greenhouses were maintained at 18°C at night and 25°C during the day with a 14-h photoperiod provided by sunlight and supplemental lighting. Plants were watered and fertilized for normal growth. Approximately 28 d after planting (DAP), the main stem was cut above or below the fifth node and immediately fitted with a plastic straw containing an agar plug of *S. sclerotiorum* mycelia.

Inoculum for the straw tests was prepared as follows: *S. sclerotiorum* culture T001.1 (collected from 'Newport' navy bean in Quincy, WA, in 1996) was germinated from a single sclerotium placed on a potato dextrose agar (PDA; Becton, Dickinson and Company, Franklin Lakes, NJ) plate, then subcultured on fresh PDA plates and grown until the mycelia reached the edge of the plate (approximately 3 d). Potato dextrose agar plates were poured using a Unispense Microprocessor Controlled Dispenser (Wheaton Science Products, Millville, NJ) to dispense 14 mL of agar into each dish to standardize the size of agar plugs used to inoculate the plants. The fungus was routinely incubated at 25°C in the dark for optimal growth. The entire plate of growing mycelia was used to prepare the agar plug inocula. Approximately 8 d after the plants were inoculated, their reaction to white mold was scored on a scale of 1 to 9, where 1 = no symptoms, 2 = invasion of the stem past the site of inoculation but not to the first node, 3 = invasion of the stem to the first node, 4 = invasion of the internode slightly past the first node, 5 = invasion to the middle of the internode, 6 = invasion to the second node, 7 = invasion slightly past the second node, 8 = invasion to the middle of the second internode and beyond, and 9 = total plant collapse (Petzoldt and Dickson, 1996).

In addition to the straw test, a novel, nonwounding greenhouse assay (Porter et al., 2009) was used to evaluate the 79 BV RILs and parents for white mold resistance, across four separate greenhouse runs (replicates), each containing two plants (subsamples) of each RIL. Replicates were planted 23 Feb., 2 Mar., 9 Mar., and 16 Mar. 2006. Thus, the four replicates were arranged in a randomized complete block design across space and time. Plants were maintained at 18°C at night and 25°C during the day, with a 14-h photoperiod provided by sunlight and supplemental lighting, and were watered and fertilized for normal growth. Approximately 28 DAP, a mini-agar plug (56.3 mm^3) colonized with *S. sclerotiorum* mycelia was wedged into the third node of each plant at the attachment point where the leaf branches from the main stem. For this test, the pathogen must penetrate natural structural defense barriers (cuticle and epidermal layers) before infecting stem tissues; therefore, additional mechanisms conferring partial resistance to white mold infection may be detected that are bypassed by the cut-stem straw test method.

The mini-agar plugs for the noninvasive greenhouse test were prepared as follows: Sclerotia from the same *S. sclerotiorum* isolate T001.1 used for the straw tests were transferred to PDA plates (poured as described above) and incubated in the dark at 25°C until they germinated, then subcultured onto fresh PDA plates. Mini-agar plugs were taken from the leading edge of the expanding colony using a dental amalgam carrier (AC1-JU Single End Jumbo 3-mm diameter amalgam carrier, Watertown, MA, Pulpdent.com) and used to inoculate the plants. Plants were then transferred to a humidity chamber covered with nylon mesh shade cloth that blocked 75% of the sunlight and maintained for 3 d at 100% relative humidity and 15°C (night) and 25°C (day) with a 14-h photoperiod. The low light intensity simulates cloud cover or dense canopy cover, which have been shown to favor white mold development in the field (Kraft and Pfleger, 2001). Plants were left undisturbed in the humidity chamber for 3 d, then returned to the greenhouse bench and rated for reaction to white mold 4 d later (7 d after inoculation) using a 1 to 9 scale similar to the straw test.

The 105 R31 RILs and parents were screened for white mold reaction in three separate straw tests using the same experimental design and procedures as described above, except that there were six replications per test. Straw Test 1 was conducted in April 2000, Test 2 in June 2000, and Test 3 in September 2000. Similarly, the nonwounding greenhouse assay was used to evaluate the 105 RILs and parents as described above, except that each replicate contained only one plant for each entry. Replicates were planted 8 May, 15 May, and 22 May 2008. Also, disease reaction was recorded 3 d after inoculation, in addition to the 7-d rating. The rating at 3 d was conducted to capture early differences in disease reaction, if any. A high correlation (0.78, $P < 0.001$) between 3- and 7-d ratings suggested the 3-d rating could be used to assess disease reaction in this test, but 7 d still allowed greater separation among lines (data not shown). The mean of the two ratings was used for subsequent analyses.

Field Screening

Reaction of 79 BV RILs, and the parents Benton and VA19, to *S. sclerotiorum* was assessed in the field during two growing seasons.

Both field tests were conducted at the USDA-ARS Cropping Systems Research Farm at Paterson, WA, in a field plot with a history for uniform *S. sclerotiorum* disease pressure and successful characterization of common bean lines for reaction to white mold (Miklas, 2007; Miklas et al., 2001, 2003, 2004). The soil is a Quincy sand (mixed, mesic Xeric Torripsamments). The field tests were planted 13 June 2003 and 16 June 2005. Both were treated identically, except there was more space available in 2003, allowing the susceptible pinto bean 'Burke' (Hang et al., 1998) to be planted every other row, while in 2005 Burke was planted every third row. For each field test, a randomized complete block design with three replications was used. A plot consisted of one row 3-m long. Plot and border rows were spaced 0.56-m apart. Planting density was 234,848 seeds ha^{-1}. To promote plant infection with *S. sclerotiorum*, approximately 6.3 mm of water was applied daily by overhead center-pivot irrigation from the onset of flowering to late pod fill. Nitrogen was foliar-applied weekly by chemigation at a rate of 22 kg ha^{-1} for 8 wk from the early seedling growth stage (about 18 DAP) to mid pod fill (about 74 DAP).

Reaction to white mold disease was measured at physiological maturity and was scored from 1 to 9 based on combined incidence and severity of infection, where 1 = no diseased plants and 9 = 80 to 100% diseased plants and/or 60 to 100% infected tissue (Miklas et al., 2001). Traits associated with disease avoidance—canopy height, lodging, and harvest maturity—were measured, although canopy height was only measured in 2003. Canopy height was measured in centimeters from the soil surface to the top of the canopy at mid pod fill, and lodging was measured on a scale of 1 to 9 (1 = no lodging and 9 = >90% lodged) at physiological maturity. Harvest maturity was recorded as DAP for this population as up to 19 d difference was observed in BV lines.

The 105 R31 RILs and parents Raven and I9365-31 were similarly assessed for white mold reaction in the field during two growing seasons. The field tests were planted 23 June 2000 and 15 June 2001. For each field test, a randomized complete block design with three replications was used. A plot consisted of four rows in 2000 and three rows in 2001. Row length, spacing, planting density, and management of the plots for R31 population were the same as described for the BV population except that no susceptible border rows were planted. For disease avoidance traits, canopy height and canopy porosity were measured. Canopy porosity (Deshpande, 1992) was recorded 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. Harvest maturity was not measured for this population because few differences in days to maturity were observed among R31 lines.

Statistical Analysis

Individual tests were analyzed by ANOVA (PROC GLM; SAS Institute, 2004) for randomized complete block designs. Error mean variances of disease reaction scores for the same tests conducted in separate environments were calculated using Bartlett's test (Armitage and Berry, 1994). To determine associations among phenotypic traits, simple correlation coefficients between means across environments were calculated using PROC CORR (SAS Institute, 2004). Narrow-sense heritability estimates were computed with variance components on an F_6 (BV)– or F_5 (R31)–derived line mean basis. Exact 90% confidence interval estimates were calculated following procedures of Knapp et al. (1985).

Marker Generation

The FastDNA Kit (MP Biomedicals, Solon, OH) was used to extract genomic DNA from a composite leaf tissue sample consisting of the first emerging trifoliate leaves of four plants from each RIL and the parents. The DNA was quantified using a spectrophotometer and adjusted to 10 ng μL^{-1} for use in SRAP, RAPD, and SSR polymerase chain reactions (PCR).

For both SRAP and RAPD markers, bulked-segregant analysis was used to select primer pairs to screen across the entire set of RILs (selective genotyping approach [Navabi et al., 2009]). Within each population equal amounts of DNA from the five most susceptible and five most resistant RILs (based on combined field and straw test reactions) were pooled to make resistant (R-bulk) and susceptible (S-bulk) DNA bulk samples.

The DNA bulks were used to screen ~500 decamer RAPD primers (R31 only) and 288 SRAP (BV and R31) primer pairs (representing all 18 forward × 16 reverse primer combinations from Li and Quiros [2001]) for polymorphic markers. Decamers and primer pairs that produced polymorphic RAPD and SRAP markers between the S- and R-bulks were then used to screen the individual RILs comprising the bulks. Random amplified polymorphism and SRAP markers that cosegregated with disease reaction in at least 70% of the individuals comprising the DNA bulks were subsequently assayed across the entire population of RILs (selective mapping approach [Miklas et al., 1996]).

The RAPD PCR (R31 only) consisted of 25 μL reactions containing 2 U Stoffel fragment DNA polymerase (Applied Biosystems, Foster City, CA), 1X Stoffel buffer, 0.2 μM primer (Operon Technologies, Inc., Alameda, CA), 5 mM $MgCl_2$, 200 μM each dNTP, and 25 ng template DNA. Amplifications were performed on a Peltier Thermal Cycler PTC-200 (MJ Research Inc., Waltham, MA) programmed for an initial cycle at 94°C for 2 min and then three cycles at 94°C for 1 min, 32°C for 1 min, 72°C for 2 min. This was followed by 30 cycles of 94°C for 10 s, 37°C for 20 s, and 72°C for 2 min, with a final 5 min extension at 72°C. All ramps to annealing and extension temperatures were set to 0.8°C sec^{-1}. Amplified products were separated on 1.4% agarose gels containing ethidium bromide (0.5 μg mL^{-1}) in 1X TBE buffer for 5 h at 3V cm^{-1} constant voltage. The SRAP PCR (BV and R31) was performed in a 12.5-μL reaction mix containing 1X AmpliTaq Stoffel buffer (Applied Biosystems, Foster City, CA), 5 mM $MgCl_2$, 0.8 mM dNTPs (Invitrogen, Carlsbad, CA), 1 U AmpliTaq Stoffel (Applied Biosystems), 37.5 ng of each primer (Li and Quiros, 2001), and 25 ng genomic DNA template. Thermalcycling was performed in an Applied Biosystems 9700 thermalcycler using calculated temperatures and consisted of 10 cycles of 94°C for 1 min, 35°C for 1 min, and 72°C for 1 min, followed by 35 cycles of 94°C for 1 min, 50°C for 1 min, and 72°C for 1 min, and ending with an additional extension step at 72°C for 7 min. Six microliters of each reaction were loaded on 10% acrylamide gels (BioRad Criterion gels; BioRad Laboratories, Hercules, CA) and electrophoresed in 1X TBE for 2.5 h at 100V.

Microsatellite PCR reactions (R31 only) were performed at the International Center for Tropical Agriculture (CIAT), using conditions described by Blair et al. (2003) and Grisi et al.

(2007). For SSRs, the parents were first screened for polymorphism, before the whole population of 105 RILs was assayed.

Genetic Linkage Maps and QTL Analysis

JoinMap4 software (Van Ooijen, 2006) was used to calculate separate genetic linkage for the BV and R31 RIL populations. Markers were assigned to a linkage group based on an independence logarithm of odds (LOD) score of 5.0 or higher. Linkage order was determined by the maximum likelihood mapping algorithm set on the default values, and linkage distances were calculated using the Kosambi mapping function. Markers linked to QTL (identified using WinQTLCart as described below) were mapped across the BJ population (described above) to anchor informative partial linkage groups obtained in this study to the *P. vulgaris* core map.

For each population, trait means across environments and partial linkage group markers and positions were entered in Windows QTL Cartographer 2.5 (Wang et al., 2007), and composite interval mapping (CIM) was used to identify the locations of QTL conditioning white mold resistance. The Standard Model (Model 6) was used with the following parameters: two (BV) or five (R31) control markers, 5-cM exclusion window size, 2-cM walk speed, and forward regression analysis. Permutation tests (1000) were used to estimate significant LOD thresholds for QTL detection (Churchill and Doerge, 1994). Linear regression (~single factor ANOVA) of individual markers and phenotypic means using PROC GLM (SAS Institute, 2004) was used to obtain R^2 values for describing the phenotypic variation explained by significant QTL-linked markers, calculated as (variance explained)/(total variance).

Sequence Characterized Amplified Regions

Selected markers underlying QTL were cloned and sequenced for development of SCAR markers. The RAPD and SRAP PCR were run as described above to amplify the marker of interest. The region of the agarose (RAPD) or polyacrylamide gel (SRAP) containing the polymorphic marker of interest was excised, put into a spin column from the FastDNA kit (MP Biomedicals), and frozen at −20°C. The gel slice was thawed while being centrifuged at maximum speed in a bench-top centrifuge, so that liquid containing the DNA collected in the catch tube, while the gel remained in the spin column. This DNA was used as template for a second PCR amplification (1 μL template in a 25-μL reaction) using the original marker primer(s). Standard PCR reagent mix and thermalcycling conditions were used, with an annealing temperature of 48°C. Polymerase chain reaction products from the second amplification were cloned using the TOPO-TA Cloning Kit (Invitrogen), as described by the manufacturer.

Plasmids were purified with the PureLink Quick Plasmid Miniprep Kit (Invitrogen) according to the manufacturer's instructions, except that plasmids were eluted from the spin columns with nanopure water. Plasmids were used as template for sequencing reactions using the BigDye Terminator v3.1 Cycle Sequencing Kits (Applied Biosystems) and universal primers, according to the manufacturer's instructions. DNA products in the sequencing reactions were ethanol-precipitated, rinsed once with 70% ethanol, then air-dried and sent to Washington State University's Center for Integrated Biotechnology to be analyzed on an ABI sequencer (Applied Biosystems). Sequences were used as queries for database searches. BLASTn (NCBI: http://www.ncbi.nlm.nih.gov/; verified 22 Oct. 2010) was used to search the "nucleotide collection (nr/nt)" and "non-human, non-mouse ESTs (est_others)" databases, and BLASTx (NCBI) was used to search the "non-redundant protein sequences (nr)" database.

The SCAR primers were designed from the sequences of selected markers, optimized to amplify a band only from the source parent, and screened against the respective BV or R31 RIL population. All SCAR PCR reactions were performed in a 12.5 μL reaction mix containing 1X AmpliTaq Stoffel buffer (Applied Biosystems), 0.2 mM dNTPs (Invitrogen), 0.5 uM each primer, 3.0 mM $MgCl_2$, and 1 U AmpliTaq Stoffel DNA polymerase (Applied Biosystems). Thermalcycling conditions consisted of an initial denaturing step at 94°C for 2 min, followed by 35 cycles of 94°C for 1 min, optimized annealing temperature (see results section) for 40 sec, and 72°C for 40 sec. A final extension step of 72°C for 2 min was performed before the reaction was cooled to 4°C. The amplified SCAR products were separated on 1.4% agarose gels as described above for the RAPD protocol.

RESULTS AND DISCUSSION

Heritability Estimates

The parents differed significantly for reaction to white mold, as expected, with the partially resistant parents VA19 and I9365-31 exhibiting lower disease severity scores and susceptible parents Benton and Raven higher disease scores for each test (Table 1). Means for the BV and R31 RIL populations were intermediate of the parental means. The frequency distributions for mean disease scores for the BV and R31 RILs were normal for all three tests (straw, nonwounding, and field) (data not shown).

Heritability estimates for disease score were consistent between straw and field tests for both populations, with estimates slightly higher under controlled conditions in the straw test. The heritability estimate for disease reaction in the nonwounding test was higher in the BV than the R31 population, perhaps because two plants were inoculated and rated per pot versus one plant per pot for R31 population. Heritability estimates for other traits measured were intermediate and consistent between populations for traits in common (i.e., canopy height). Together, these results support that partial resistance to white mold segregating in BV and R31 populations is quantitatively inherited. The intermediate to high heritability estimates obtained suggest that genes with major effects may contribute to the partial resistance observed in VA19 and I9365-31.

For both populations, disease reaction of the RILs measured in the greenhouse tests had low to moderate correlation with field reaction (Table 2). Low correlations between field and greenhouse tests occur, in part, because expression of physiological resistance in the field is confounded by disease pressure and avoidance traits, whereas greenhouse tests detect physiological resistance under uniform conditions. The low to moderate correlation between greenhouse and field reactions confirms the importance for using field trials to fully characterize

Table 1. Mean, range, and heritability estimates for avoidance traits (canopy height, lodging, and harvest maturity) and for reaction to white mold in the straw test, nonwounding greenhouse test, and field for two recombinant inbred populations BV (Benton/VA19, 79 $F_{6:8}$ recombinant inbred lines) and R31 (Raven/I9365-31, 105 $F_{5:7}$ recombinant inbred lines), and means for the parents, tested across multiple environments.

Trait (measurement)	Parent means†		Recombinant inbred population (BV)	
	Benton	VA19	Mean (range)	h^2 (90% CI‡)
Straw test (1–9§)	7.3 a	4.7 b	6.0 (2.3–9.2)	0.76 (0.67–0.79)
Nonwounding greenhouse test (1–9)	7.9 a	2.8 b	5.1 (2.6–8.1)	0.69 (0.59–0.74)
Field reaction (1–9)	6.1 a	2.8 b	4.3 (2.0–7.7)	0.71 (0.58–0.81)
Lodging (1–9)	6.8 a	4.5 b	4.9 (2.0–8.3)	0.64 (0.47–0.75)
Canopy height (cm)	49.0 a	74.0 b	59.4 (44.0– 70.0)	0.48 (0.17–0.55)
Harvest maturity (DAP¶)	99.3 a	99.7 a	98.5 (88.7–107.8)	0.89 (0.84–0.93)
	Raven	I9365-31	Recombinant inbred population (R31)	
Straw test (1–9)	7.0 a	3.9 b	5.8 (3.7–7.5)	0.78 (0.69–0.84)
Nonwounding greenhouse test (1–9)	7.5 a	3.5 b	4.8 (3.0–8.7)	0.37 (0.14–0.54)
Field reaction (1–9)	6.6 a	4.8 b	5.6 (3.8–7.5)	0.69 (0.42–0.78)
Canopy porosity (1–5#)	1.6 a	4.7 b	3.3 (1.7–4.6)	0.72 (0.61–0.79)
Canopy height (cm)	56.6 a	56.6 a	55.3 (46.8–62.4)	0.52 (0.34–0.64)

†Means within a row followed by different letters are significantly different at $P < 0.01$.

‡CI, confidence interval.

§Disease scores from 1 to 9 where 1 = no disease and 9 = completely diseased or dead plants.

¶DAP, days after planting.

#Rating from 1 to 5, where 1 = open canopy and 5 = completely closed canopy.

partial resistance segregating in genetic and breeding populations. Intermediate correlations between disease reactions in the straw and nonwounding tests indicate these greenhouse tests detect similar but also different mechanisms of partial resistance as well.

The association of late maturity with less disease in the BV population (Table 2) is consistent with previous studies (Kolkman and Kelly, 2003) and contributes to the difficulty of obtaining partially resistant cultivars with acceptable harvest maturity (Miklas et al., 2004). Although taller plants are a desirable trait, increased canopy height was slightly correlated with less disease in R31 ($P = 0.05$) but was not significant for BV. The negative correlation between reduced canopy porosity (high score) with decreased disease severity was unexpected because an open canopy (low score) is generally more capable of disease avoidance.

Note that based on homogeneity of variance (Bartlett's test) among repetitions (straw test, field) and replications (nonwounding test), data above were combined across the different experimental environments for all phenotypic tests, with one exception. Straw Test 1 data for the BV population was discarded altogether because of a significantly different error variance.

Table 2. Pearson correlation coefficients between white mold disease tests and between disease avoidance traits and disease severity in the field for Benton/VA19 (BV) and Raven/I9365-31 (R31) recombinant inbred lines populations, respectively, using means combined across environments.

	Field test		Straw test	
	BV	R31	BV	R31
Nonwounding test (1–9†)	0.21	0.23*	0.56***	0.48***
Field test (1–9)			0.25*	0.44**
Lodging (1–9)	0.14			
Canopy height (cm)	–0.18	–0.21*		
Canopy porosity (1–5)		–0.26**		
Harvest maturity (DAP‡)	–0.67***			

*Significant at $P < 0.05$.

**Significant at $P < 0.01$.

***Significant at $P < 0.005$.

†Disease scores from 1 to 9, where 1 = no disease and 9 = completely diseased or dead plants.

‡DAP, days after planting.

Selective Mapping

Forty-eight SRAP primer pairs, selected using bulked-segregant analysis, were assayed against the entire BV RIL population, resulting in 69 discernible polymorphic markers. Thirteen markers segregated identically with other markers and therefore were excluded from linkage group analysis. The remaining 56 markers mapped to four partial linkage groups. Two of the four BV linkage groups (LG), which integrated with LG 2 and LG 8 of the core map, contained QTL conferring partial resistance to white mold (Fig. 1).

There were 126 markers assayed across the 105 R31 RILs. The markers consisted of 51 RAPDs and 31 SRAPs identified by bulked-segregant analysis, 40 SSRs identified by polymorphism between the parents, 2 SCARs from the BV population, and 2 traits: resistance to *Clover yellow vein virus* (ClYVV), which was linked with *bc-3* gene (Larsen et al., 2008), and resistance to Race 1 of *Pseudomonas syringae* pv. *phaseolicola* causal agent of halo bacterial blight (unpublished data). Of the 126 markers, 110 were mapped across 10 linkage groups. Six linkage groups (2, 4, 5, 6, 7, and 8) contained seven QTL contributing to partial white mold

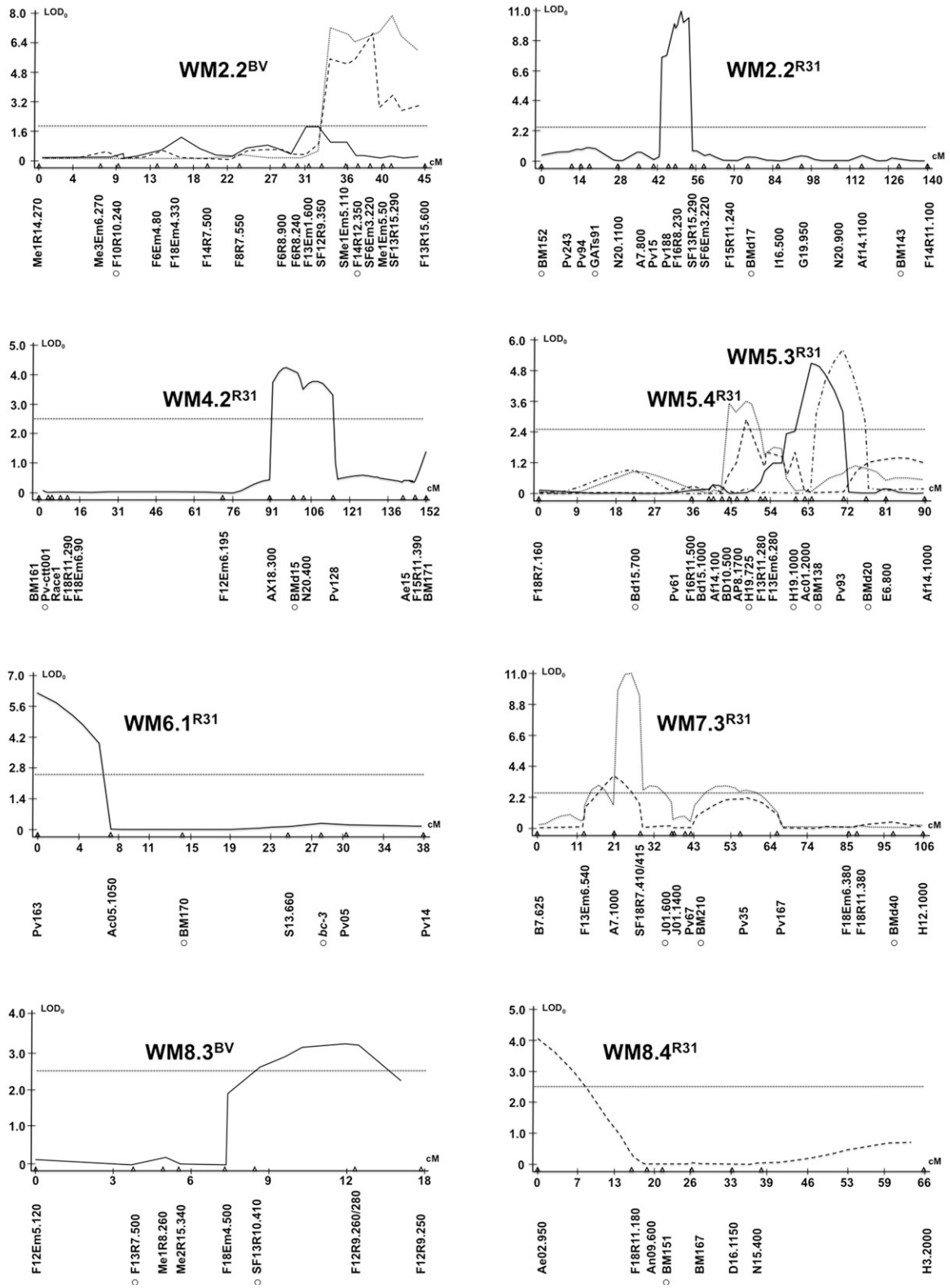

Figure 1. Composite interval mapping logarithm of the odds (LOD_0) graphs depicting quantitative trait loci (QTL) conferring white mold resistance identified in Benton/VA19 (BV) and Raven/I9365-31 (R31) recombinant inbred line populations. Traits shown are field test (solid line), straw test (dotted line), nonwounding test (dashed line), and plant height (dot-dash line). A significance threshold LOD_0 score determined by permutation tests is depicted on each graph as a dashed horizontal line. The significance threshold of higher value is presented for those graphs depicting more than one trait. Positions of markers are indicated with triangles, and markers used to anchor the partial linkage groups to the core maps are tagged with small circles. The QTL are named according to procedures in Miklas and Porch (2010; Fig. 2). Markers with a Pv or BM prefix are simple sequence repeat markers, F or M prefixes represent sequence related amplified polymorphism markers and are preceded by an S if converted to a sequence characterized amplified marker, and one- or two-letter prefixes are random amplified polymorphic DNA markers. Base pair size of markers is indicated after the period.

Table 3. List of sequence characterized amplified region (SCAR) markers generated from sequence related amplified polymorphism (SRAP) markers associated with quantitative trait loci (QTL) conferring white mold resistance in the Benton/VA19 (BV) and Raven/I9365-31 (R31) recombinant inbred line populations.

SCAR (original SRAP)	Primer sequence 5′-3′	QTL	Trait R^2	Annealing temp	Product size	Parental allele
			%	°C	bp	
		BV population				
SMe1Em5.110 (Me1Em5.120)	F-CCAAACCGGATAGTCTAAAC R-GTACGAATTAACTGACTATG	WM2.2	ST† 29 NWT 25 Field 12	55	110	VA19
SF12R9.350 (F12R9.370)	F-ATCTTAGCCGGAGCTGAGAC R-ACGAATTTGAGATGGTTTAC	WM2.2	ST 33 NWT 27 Field 11	65	350	Benton
SF6Em3.220 (F6Em3.250)	F-GCGTACGAATTGACATACACC R-CACAAGCCGGATATATCTTATC	WM2.2	ST 30 NWT 26 Field 9	60	220	Benton
SF13R15.290 (F13R15.330)	F-AGCCGGCACTGATAAAATTTG R-TCCGTAATTCCACATTCTCC	WM2.2	ST 30 NWT 27 Field 8	67	290	Benton
SF13R10.410 (F13R10.420)	F-GACACCGTACGAATTAACTCATTTTG R-CGAATCTTAGCCGGCACCGAAATGG	WM8.3	Field 11	60	410	VA19
		R31 population				
SF18R7.410/415 (F18R7.440)	F-ACCGTACGAATTTGCTTAAGTG R-GATCCAGTTACCGGAAT	WM7.3	ST 51 NWT 22	45	410/415	Codominant

†ST, straw test; NWT, nonwounding test.

resistance in the R31 population (Fig. 1). There were more linkage groups generated in the R31 than the BV population, in part because the 40 SSR markers assayed in R31 were selected to cover the complete genome as far as was possible given low polymorphism levels.

Sequence Related Amplified Polymorphism Cloning and Sequence Characterized Amplified Region Markers

Because SRAPs are designed to amplify open reading frames, they provide an opportunity for identification of candidate genes underlying QTL. Twelve markers in the BV population near QTL positions were cloned and sequenced: 10 for the LG 2 QTL and 2 for the LG 8 QTL. Sequences were deposited in GenBank (accessions FJ597978–FJ597982 and FJ748892–FJ748898) and were used as queries to search the online nucleotide, expressed sequence tag (EST), and protein databases. Seven (accessions FJ748892, FJ748894, FJ748896, FJ748898, FJ597978, FJ597980, and FJ597981) of the 12 sequences did not contain any regions of similarity to known sequences in the databases, while 4 (accessions FJ748893, FJ748895, FJ597979, and FJ597982) had similarity to transposable elements and 1 sequence (accession FJ748897) was similar to uncharacterized ESTs from *P. vulgaris* and *P. coccineus* libraries. Although the SRAP nucleotide sequences did not reveal candidate resistance genes they were useful for developing five SCAR markers with potential use for marker-assisted breeding, four linked to the LG 2 QTL and one to the LG 8 QTL (Table 3). Sequences of the five SRAPs used to develop SCAR markers correspond to GenBank accession numbers FJ597978–FJ597982.

For the R31 population, given the proximity of SSR markers to QTL on most linkage groups (Table 4, Fig. 2), a SCAR was only developed from a SRAP marker associated with a QTL on LG 7 (Table 3). The sequence for this marker was deposited in GenBank (accession HM358996), and it did not align with any stored sequences in the databases.

Comparative Mapping QTL for White Mold Resistance

A comprehensive linkage map of QTL conferring resistance to white mold identified in previous studies (Miklas et al., 2001, 2003, 2007; Park et al., 2001; Kolkman and Kelly, 2003; Ender and Kelly, 2005; Maxwell et al., 2007) was developed to ascertain novelty of the QTL identified in the BV and R31 populations (Fig. 2, Table 4). This map provides a historical perspective and baseline for naming all the QTL identified previously, herein, and henceforth. In fact, this map provided impetus for the recent establishment of comparative map QTL nomenclature guidelines for common bean (Miklas and Porch, 2010).

Park et al. (2001) observed QTL for partial resistance to white mold on LG 10 and LG 11, but owing to minor effect and lack of repetition these QTL were omitted from the comprehensive map developed herein. Our map consists of 9 linkage groups (1 to 9) that possess QTL for white mold resistance out of a possible 11 for the species ($2n$ = 22, for *P. vulgaris*). The nine QTL identified in BV and R31 integrated to six of the nine linkage groups. Five of the QTL were associated with previously identified QTL and four were novel. Although linkage groups 1, 3, and 9 did not possess QTL from BV or R31, they were included in Fig. 2 and the discussions, below, albeit in less detail, to support the comparative map as a more comprehensive reference map. The map reflects reorientation of certain linkage groups 1, 2, 3, 4, 6, and 9 on the basis of chromosome arm length (short arm on top), in adoption of the new nomenclature guidelines for common bean chromosomes and linkage groups (Pedrosa-Harand et al., 2008).

Table 4. Comprehensive list of quantitative trait loci (QTL) conditioning partial resistance to white mold in common bean (*Phaseolus vulgaris* L.) from previous studies and identified in Benton/VA19 (BV) and Raven/I9365-31 (R31) recombinant inbred line populations (italic type). BJ, BAT 93/Jalo EEP558; DG, DOR364/G19833.

QTL	Pop†	Traits‡	R^2§	Nearest marker	
				Source population	Core map BJ or DG
			%		
WM1.1¶	AG	Field (CP, avoidance)	18 (34)	*fin*	*fin*
WM1.2	GC	ST	20	PatMaca300	V20.3 or ROU15
WM2.1	PX	ST	7	W02.1100	BMd18
WM2.2	BN	Field	12	BC20.1800	ROS13b
	HN	Field	40	BC20.1800	ROS13b
	BR	Field	9	O15.1800	*Chs*
	AN	Field	25	AFLP10	ROS13b
	BV	*Field, ST, NWT*	*13, 35, 36*	*Me1Em5.50*	*ROG19b*
	R31	*Field*	*32*	*F16R8.230*	*ROS3c*
WM2.3	BR	Field	10	O12.1600	O12.1
	GC	ST	15	EacaMaat220	U12.2
WM3.1	AN	Field (CP, avoidance)	16 (36)	K10.350	AM18.1
WM4.1	PX	ST, Field	5, 5	U10.900	*Rbcs*
WM4.2	*R31*	*Field*	*14*	*BMd15*	*BMd15*
WM5.1	PX	ST	11	D05.1100	D1157
WM5.2	BR	Field	11	EactMcat85	unknown
WM5.3	*R31*	*Field, (PH, avoidance)*	*21 (14)*	*BM138*	*BM138*
WM5.4	*R31*	*ST, NWT*	*8, 5*	*H19.725*	*W16.3*
WM6.1	B60	ST, Field	12, 10	SAU5.1350	PO1.3
	R31	*Field*	*12*	*Pv163*	*PO1.3*
WM7.1	AG	ST, Field	38, 26	*Phs*	*Phs*
	PX	ST, Field	9, 16	J09.950	*Phs*
WM7.2	BN	Field	17	EaggMctt85	Bng047
	BR	Field	15	EaacMctt223	Bng047
WM7.3	*R31*	*ST, NWT*	*51, 22*	*SF18R7.410/415*	*Bng204*
WM8.1	PX	Field (PH, avoidance), ST	9 (15), 24	A017.1050	ROS3a
	CG	Field	12	PagMact254	BMd25
WM8.2	PX	ST	12	H19.1250	Bng73-AH05.1
WM8.3	B60	Field, ST	26, 38	B10.1100	D15.1
	GC	ST	7	EacaMaga228	BMd25-F082G
	BV	*Field*	*11*	*F12R9.260/280*	*D15.1*
WM8.4	BR	Field	9	EagaMctg190	L04.1
	GC	ST	11	PatMaac500	D11.2
	R31	*NWT*	*8*	*Ae02.950*	*BM151*
WM9.1	GC	ST	13	BM154	BM184

†Pop, population. The populations in which the QTL have been identified are abbreviated AG = A55/G122 (Miklas et al., 2001), PX = PC-50/XAN-159 (Park et al., 2001), BN = Bunsi/Newport and HN = Huron/Newport (Kolkman and Kelly, 2003), BR = Bunsi/Raven (Ender and Kelly, 2005), B60 = Bunsi/NY6020-4 (Miklas et al., 2003), AN = Aztec/ND88-106-04 (Miklas et al., 2007), GC = G122/CO72548 (Maxwell et al., 2007), and BV = Benton/VA19 and R31 = Raven/I9365-31 (current study).

‡NWT = nonwounding test; ST = straw test; CP = canopy porosity; PH = canopy height.

§The R^2 values were rounded to the nearest whole number, and most represent values obtained by regression analysis (single-factor ANOVA) with significance levels ranging from $P < 0.05$ to $P < 0.001$. For studies that reported R^2 values for individual environments, the environment with the highest value is listed. For NWT, ST, and Field, values represent amount of phenotypic variation explained for disease score. Values within parentheses represent avoidance traits that co-located with field resistance.

¶The QTL were named based on recent QTL nomenclature guidelines (Miklas and Porch, 2010). For example, WM2.3 represents the third QTL for white mold resistance identified on linkage group 2. It was originally identified in the BR mapping population, and the same QTL as determined by comparative mapping was subsequently observed in GC population.

Linkage Group 1

Two QTL were previously identified on LG 1. The first QTL identified on this linkage group, WM1.1^{AG}, conditioned field resistance in the AG (A55/G122) RIL mapping population (Miklas et al., 2001). Field resistance for WM1.1 was associated with canopy porosity, a disease avoidance trait, which was influenced by the *fin* gene for determinate (bush) growth habit. The AG RILs with bush growth habits tended toward porous canopies conducive to disease avoidance.

The second QTL on LG 1, WM1.2^{GC}, was discovered in the GC (G122/CO72548) RIL mapping population (Maxwell et al., 2007). This QTL conditioned partial resistance in the greenhouse straw test. The resistance allele was derived from G122, a well-known source for partial resistance. WM1.2 was located 20 cM from the *fin* locus in the GC population; however, orientation of WM1.2 with *fin* in BJ or DG core maps is unknown because markers in common between GC and the core maps were lacking for this region.

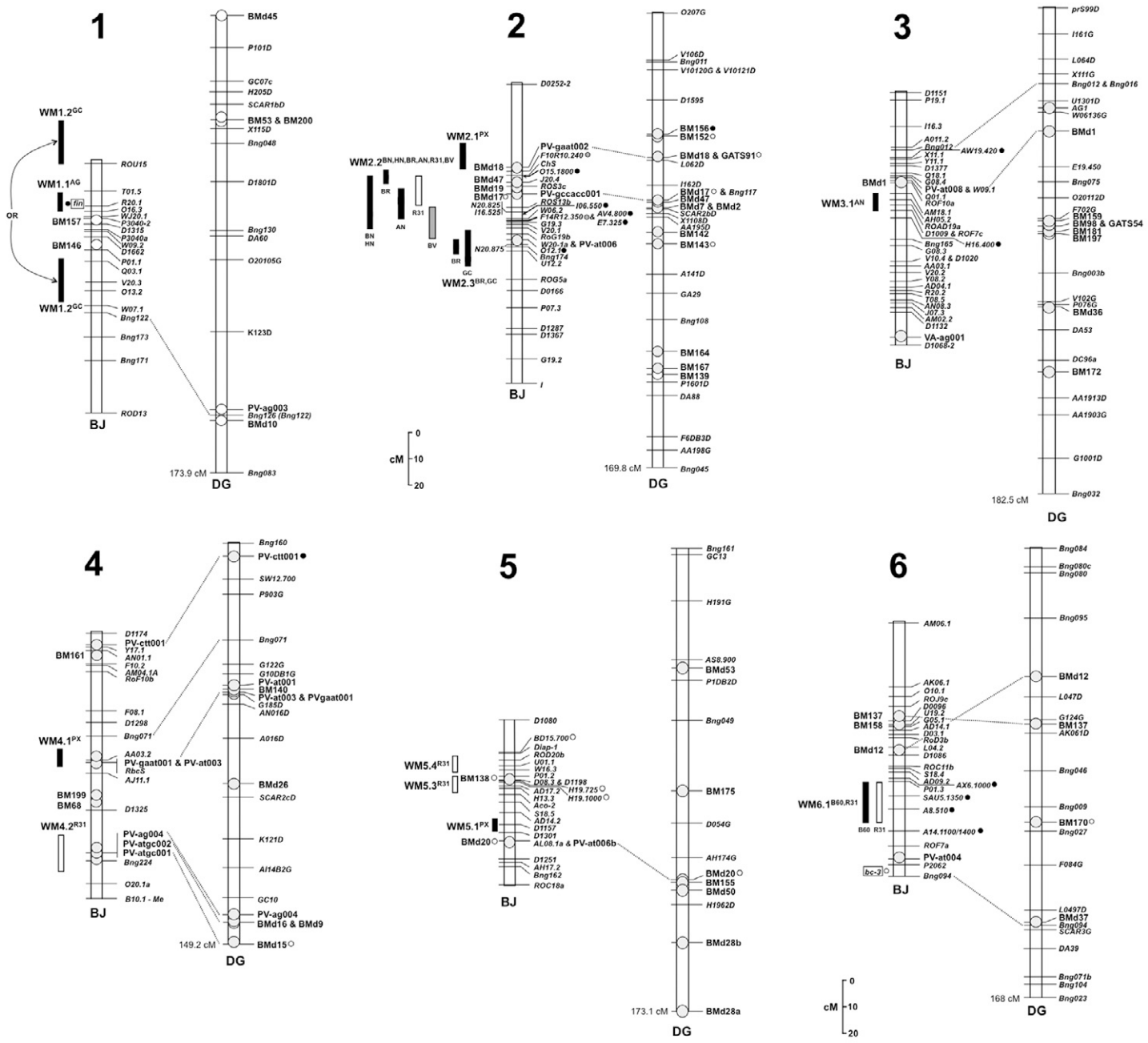

Figure 2. (continued on next page) Linkage groups 1 through 9 of common bean (*Phaseolus vulgaris* L.), showing multiple quantitative trait loci (QTL) conferring white mold resistance identified in dry bean germplasm lines VA19 (Benton/VA19 [BV] population) light red kidney and I9365-31 (Raven/I9365-31 [R31]population) small black, in comparison with QTL identified in previous studies. The BV QTL positions are represented by gray bars, R31 QTL by open bars, and QTL from previous studies by black bars. The QTL nomenclature used follows recent guidelines for comparative QTL mapping (Miklas and Porch, 2010). The previously identified QTL shown were mapped in populations Bunsi/Raven (BR; Ender and Kelly, 2005), Bunsi/Newport and Huron/Newport (BN and HN; Kolkman and Kelly, 2003), G122/CO72548 (GC; Maxwell et al., 2007), A55/G122 (AG; Miklas et al., 2001), Bunsi/NY6020-4 (B60; Miklas et al., 2003), Aztec/ND88-106-04 (AN; Miklas et al., 2007), and PC-50/XAN-159 (PX; Park et al., 2001). Markers tagged with a small circle were used to anchor partial linkage groups to the core maps: open circles anchor R31, gray circles anchor BV, and black circles anchor partial linkage groups from previous studies. The BAT 93/Jalo EEP558 (BJ) and DOR364/G19833 (DG) core maps from Blair et al. (2003) were used. Additional markers have been inserted at estimated positions based on the BJ map from Freyre et al. (1998), and markers mapped in BJ during the current study are also included. Large open circles within the core maps indicate simple sequence repeat (SSR) marker positions. Black dotted lines indicate connections between core maps. All linkage groups have been oriented and named according to Pedrosa-Harand et al., 2008. Note that on linkage group 1, WM1.2^{GC} is shown with two possible positions because complete orientation of the GC partial linkage group could not be determined. See Fig. 1, Blair et al. (2003), and Freyre et al. (1998) for description of the markers.

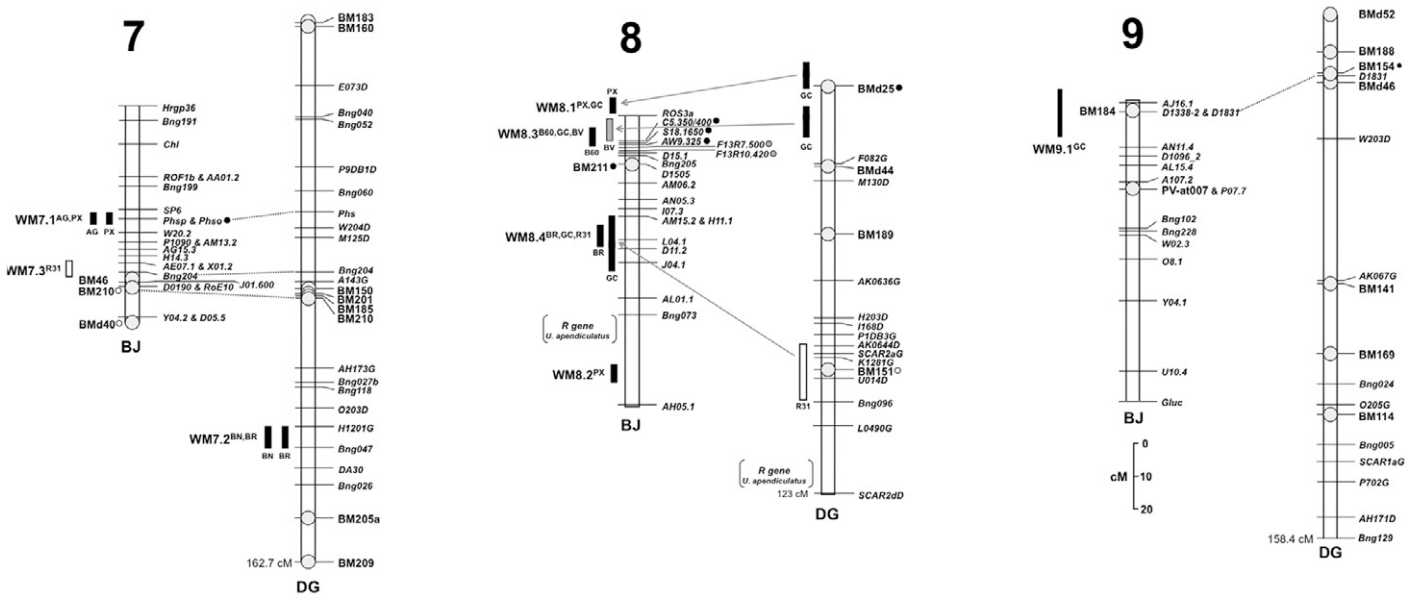

Figure 2. Continued.

Linkage Group 2

Nine different mapping populations, including BV and R31 (current study) detected QTL conferring partial resistance to white mold in the same general vicinity of LG 2 that spans about 40 cM in the BJ and DG core maps. Comparative mapping, origin of the resistance alleles, and function of the alleles, considered simultaneously, suggest presence of three independent QTL within this region.

The first QTL for LG 2, WM2.1^{PX}, was discovered in the PX (PC-50/XAN-159) RIL mapping population by Park et al. (2001). This QTL is distinct because (i) it represents a resistance allele from the Andean gene pool (PC-50 parent), (ii) was detected in the straw test but not the field, and (iii) the location for this QTL is different from those subsequently mapped to LG 2.

The second QTL, WM2.2^{BN,HN,BR,AN,BV,R31}, was detected first in the BN (Bunsi/Newport) and HN (Huron/Newport) mapping populations (Kolkman and Kelly, 2003). The QTL conditions partial field resistance and in BN derives from Bunsi navy bean and in HN from C-20 navy bean, both of which belong to the Middle American gene pool. Bunsi, a well-known resistance source, exhibits susceptibility in the straw test, so it was no surprise that WM2.2 only conditioned field resistance. Field resistance for WM2.2 was validated in the BR (Bunsi/Raven; Ender and Kelly, 2005) and AN (Aztec/ND88-106-04; Miklas et al., 2007) RIL mapping populations, which segregate for the same Bunsi source of resistance.

Interestingly, QTL discovered in BV and R31 populations (Fig. 1) map to the same region as WM2.2. The resistance allele from I9365-31 was expressed solely in the field, which fits the profile for WM2.2. Conversely, the VA19 resistance allele is of Andean origin and was expressed in both the greenhouse and the field, with greater expression in the former. WM2.2 in BV had the same level of expression in the straw (35%) and non-wounding (36%) greenhouse tests suggesting that both tests equally detect the gene conferring partial resistance at this locus. The resistance allele from VA19, of Andean origin, is likely distinct from the resistance alleles from the Middle American sources (Bunsi, I9365-31), which map to the same WM2.2 QTL region.

The SF13R15.290, SF6Em3.220, and SMe1Em5.110 SCARs linked with WM2.2 in the BV population (Table 3) were assayed in the R31 and AN (Miklas et al., 2007) populations, respectively, where they located within the same WM2.2 QTL region. Of these BV SCAR markers, SF13R15.290 was most closely associated with WM2.2 in the R31 and AN populations. For R31, SF13R15.290 and SF6Em3.220 explained 28% ($P < 0.0001$) and 24% ($P < 0.0001$), respectively, of the phenotypic variation in field disease reaction. Using the field data for the AN population from Miklas et al. (2007), SF13R15.290 and SMe1Em5.110 explained 17% ($P < 0.0001$) and 11% ($P = 0.002$), respectively, of the variation in disease reaction for the 2001 trial conducted in Washington. Ender et al. (2008) used the RAPD marker BC20.1800 for marker-assisted selection (MAS) of WM2.2 allele derived from Bunsi. The four WM2.2-linked SCAR markers generated in BV should expand capacity for MAS of resistance alleles for this prominent WM2.2 QTL.

The third QTL, WM2.3^{BR,GC} on LG 2, is represented by QTL identified in BR (Ender and Kelly, 2005) and GC (Maxwell et al., 2007) populations, which locate on the outer edge of the WM2.2 region in the vicinity of SSR marker BM143. Both WM 2.2 and WM2.3 QTL were observed in the BR population to be distinct and separate QTL. The Bunsi and G122 alleles for WM2.3 conditioned field and straw test reactions, respectively.

Plant architectural traits conferring resistance through avoidance do not map near any of the LG 2 QTL, indicating that resistance mediated by these QTL is physiological. The stay-green stem trait, associated with the Bunsi type of field resistance to white mold, mapped to WM2.2 in the AN population. Plants with stay-green stem trait, whereby plant tissues (stems primarily but leaves too) remain green even though pods have reached harvest maturity, likely remain physiologically active and more able to combat white mold infection, compared to susceptible plants with tissues that become desiccated as plants mature.

As recognized in previous studies (Miklas et al., 2007; Kolkman and Kelly, 2003; Ender and Kelly, 2005), the QTL region on LG 2 is in close proximity to chalcone synthase *ChS*, pathogenesis-related protein *PvPR-2* (Walter et al., 1990), and polygalacturonase-inhibiting protein *Pgip* (Toubert et al., 1992). These three genes are involved in plant defense against fungal pathogens, thus may be involved in host defense against *S. sclerotiorum*. In addition, QTL for resistance to web blight [caused by *Thanatephorus cucumeris* (Frank) Donk] and common bacterial blight map in the same region (Miklas et al., 2006b). Fine mapping in this genomic region is warranted given the numerous QTL from diverse sources that map within the region.

Linkage Group 3

To date, only one QTL has been mapped on LG 3. The WM3.1^{AN} QTL identified in the AN population (Miklas et al., 2007) conditioned field resistance and was likely influenced by disease avoidance traits (canopy porosity and height) and late maturity, which mapped to the same region. The reviews by Kelly et al. (2003) and Miklas et al. (2006b), which show a QTL for white mold resistance from the BN population on linkage group 3, is an error.

Linkage Group 4

Two QTL have been positioned on LG 4, WM4.1^{PX}, identified previously in PX population by Park et al. (2001), and WM4.2^{R31}, identified in the R31 population (this study). The WM4.1 QTL, located near the *RbcS* gene in BJ, was detected with minor effect in both the field and straw tests. The resistance allele in PX at this locus derives from the Andean partial resistance source PC-50.

The second QTL on LG 4, WM4.2^{R31}, is located near SSR marker BMd15 in the R31 population. The BMd15 marker is positioned at the distal end of LG 4 in the DG population and located by RFLP marker Bng224 in BJ. The WM4.2 allele from I9365-31 conditioned partial field resistance (14%, $P < 0.0001$) and did not co-locate with QTL for disease avoidance traits (Fig. 1).

Linkage Group 5

Four QTL have been placed on LG 5, two previously and two from this study. The WM5.1^{PX} QTL, identified in PX (Park et al., 2001) and detected solely in the straw test, was comparatively mapped near RFLP marker D1157 in BJ. The WM5.2^{BR} QTL conferred partial resistance in the field and was associated with a yield QTL in the BR population (Ender and Kelly, 2005). Although the authors place WM5.2 QTL on LG 5, we were unable to comparatively map this QTL in either the BJ or the DG core map, so the genomic relationship or lack thereof between WM5.2 and WM5.1 could not be determined. The WM5.2 QTL was named anyway to acknowledge the chronological order of discovery for QTL on this linkage group.

Two QTL identified in the R31 population locate in a separate region from WM5.1 (Fig. 2). WM5.3^{R31} QTL, nearest SSR marker BM138, was expressed in the field (21%, $P < 0.0001$) and co-located with the disease avoidance trait canopy height (14%, $P < 0.0001$). WM5.4^{R31} QTL, nearest RAPD marker H19.725, was detected in the straw (8%, $P < 0.007$) and nonwounding (5%, $P < 0.03$) tests, suggesting that both tests measure the same resistance allele, at least for this locus. The WM5.3 and WM5.4 QTL were positioned about 20-cM apart in R31 as determined by CIM. The alleles conferring resistance for both QTL derive from the resistance source I9365-31.

Linkage Group 6

A single QTL has been identified on LG 6. The WM6.1$^{B60, R31}$ QTL was first identified in the B60 (Benton/NY6020-4) RIL mapping population (Miklas et al., 2003) and then subsequently in the R31 population (this study). WM6.1 QTL is located near SCAR marker SAU5.1350 in the B60 population, which was subsequently mapped between RFLP markers Bng094 and D1086 in the BJ population. The resistance allele for this QTL derived from NY6020-4 and conditioned partial resistance in both the straw test (12%) and field (10%) but was associated with QTL for disease avoidance traits lodging (15%) and canopy height (20%).

WM6.1 QTL in R31 (12%, $P < 0.0005$) was detected solely in the field and was not associated with disease avoidance traits measured in the R31 population. The resistance allele at this locus was derived from I9365-31. The WM6.1 QTL in R31 was comparatively mapped in BJ on the basis of orientation with SSR marker BM170 and *bc-3* gene relative to the orientation of BM170 and RFLP marker Bng094 in the DG population. Bng094 is linked with *bc-3* (Johnson et al., 1997). To attempt more definitive mapping, the SCAR SAU5.1350 and other RAPD markers linked with WM6.1 in B60 (Miklas et al., 2003) were assayed in the R31 population, but none were polymorphic.

Linkage Group 7

Three QTL have been located on LG 7, two previously identified and one discovered in R31 population. The WM7.1^{AG,PX} QTL, discovered first in the AG population (Miklas et al., 2001) and then subsequently in the

PX population (Park et al., 2001), is located near the *Phs* seed protein locus. The resistance allele for WM7.1 QTL is derived from the Andean parents G122 and PC-50 in the respective AG and PX populations. Interestingly, the WM7.1 QTL was not detected in the GC population, which segregates for the same G122 source of partial resistance (Maxwell et al., 2007). WM7.1 in AG conditioned partial resistance in the straw test (38%) and the field (26%). WM7.1 in PX likewise conditioned partial resistance in both the straw test (9%) and the field (16%). Marker-assisted backcrossing of the resistance allele from G122 for WM7.1 into susceptible pinto bean, using *Phs* as the selectable marker, was successful in transferring partial resistance but not recovery of pinto beans with acceptable agronomic performance (Miklas, 2007). The WM7.1 QTL from G122, as detected by the *Phs* marker, was also expressed in a susceptible snap bean background, as observed in the G122/'Astrel' RIL population (Chung et al., 2008).

The WM7.2^{BN,BR} QTL on LG 7 was discovered first in the BN (Kolkman and Kelly, 2003) and subsequently in the BR population (Ender and Kelly, 2005), which segregates for the same Bunsi-derived partial resistance to white mold. This QTL in BN was associated with field resistance, resistance to oxalate in a greenhouse test, and other agronomic traits including yield, seed size, days to flowering and lodging. The oxalate test can be used to screen for partial resistance to white mold (Kolkman and Kelly, 2000) but is not widely used, in part, because the test is cumbersome. WM7.2 in BR was similarly associated with field resistance, seed size, days to flowering, and lodging but also harvest maturity. WM7.2 maps toward the distal end of LG 7 near RFLP marker Bng047 in DG, and RFLP marker D1107 in BJ (Freyre et al., 1998). The QTL does not map to BJ in the integrated core map from Blair et al. (2003) used for this study (Fig. 2) because the long arm of LG 7 for BJ is incomplete. The AFLP marker EaacMctt130 in the BN population was used for MAS of WM7.2 by Ender et al. (2008). They showed that MAS for WM7.2 and WM2.2 alleles from Bunsi source, simultaneously, was effective in selecting progeny lines from the Bunsi/'Midland' population with partial resistance to white mold.

The WM7.3^{R31} QTL conferred partial resistance in both straw (51%, $P < 0.0001$) and nonwounding (22%, $P < 0.0001$) greenhouse tests (Fig. 1). Given the major effect for this QTL, a SCAR marker SF18R7.410/415 (Table 3) was developed from a SRAP marker nearest the peak response for WM7.3 in the R31 population. The SCAR will facilitate validation of WM7.3 QTL in other populations and could be useful for marker-assisted selection.

Linkage Group 8

Four QTL were established on LG 8 in previous studies. The WM8.1^{PX,GC} and WM8.2^{PX} QTL were discovered in the PX population (Park et al., 2001), and WM8.1 was subsequently observed in the GC population (Maxwell et al., 2007). WM8.1 in PX, linked with the *C* locus, was associated with field (9%) and straw test (24%) reactions. WM8.1 in PX was associated with avoidance traits, indicating presence of a closely linked but distinct avoidance QTL, or a relationship between physiological and avoidance resistance traits. WM8.1 in GC was associated with field reaction only (12%). This QTL is located at the proximal end of LG 8 in the BJ and DG populations.

The WM8.2 QTL in PX was linked with the H19.1250 RAPD marker and explained 12% of the variation in the straw test. WM8.2 has not been observed in any other QTL mapping populations.

The WM8.3^{B60,GC,BV} QTL was first observed in the B60 mapping population (Miklas et al., 2003), then subsequently in GC (Maxwell et al., 2007), and then in the BV population (Fig. 1). The resistance allele derived from NY6020-4 in the B60 population was expressed in both the field (26%) and straw tests (38%). The QTL in GC explained 7% of the variation in straw test reaction, and the resistance allele was from the susceptible parent CO72548. WM8.3 in BV was only expressed in the field, explaining 11% ($P < 0.003$) of the variation in disease reaction. The resistance allele was derived from the resistant parent VA19. WM8.3 did not co-locate with QTL for disease avoidance traits measured in the PX, GC, or BV populations, suggesting resistance conferred by this QTL has a physiological basis.

The SS18.1650 SCAR and AW9.1200 RAPD markers linked with WM8.3 in the B60 population were useful for marker-assisted backcrossing the resistance allele from NY6020-4 into the susceptible great northern dry bean market class, where it conditioned a 17% reduction in disease severity averaged across straw and field tests and recipient populations (Miklas, 2007). A SCAR marker SF13R10.410 tightly linked with WM8.3 QTL in the BV population, and developed in the current study (Table 3), was assayed in the B60 population, where it mapped to the exact same location as AW9.1200 RAPD. The SCAR SF13R10.410 provides breeders with an additional marker with potential for marker-assisted selection of favorable alleles for the WM8.3 QTL.

The WM8.4^{BR,GC,R31} QTL was observed previously in the BR and GC populations and in the R31 population in this study (Fig. 1). WM8.4 in BR explained 9% of the variation in disease severity for one of two field trials and was most closely linked to marker EAGAMCTG190 (Ender and Kelly, 2005). This QTL was associated with lodging in the BR population. WM8.4 has not been detected in other populations segregating for the Bunsi-derived white mold resistance (Kolkman and Kelly, 2003; Miklas et al., 2007). WM8.4 in GC was linked with the PATMAAC500 AFLP marker and contributed to partial resistance in the straw test (11%), which derived from the susceptible parent CO72548. WM8.4 in R31 was detected

Table 5. A survey of five quantitative trait loci (QTL)–linked sequence characterized amplified region (SCAR) markers from BV (Benton/VA19) recombinant inbred line population across a selection of lines, cultivars, and *Phaseolus* species with partial resistance (PR) or susceptibility (S) to white mold.

Line	White mold reaction	WM2.2 QTL				WM8.3 QTL
		SMe1Em5.110 (coupling)†	SF12R9.350 (repulsion)	SF13R15.290 (repulsion)	SF6Em3.220 (repulsion)	SF13R10.410 (coupling)
Parents						
VA19 (kidney)	PR	+‡				+
Benton (snap)	S		+	+	+	
Andean gene pool						
Red Kloud (kidney)	PR	+				+
Red Hawk (kidney)	S	+				+
Montcalm (kidney)	S	+				+
Cardinal (cranberry)	S	+				
G122 (red mottled)	PR	+				+
Snap beans (mixed gene pools)						
NY6020-4	PR				+	+
Medinah	S		+	+	+	
Top Crop	S		+		+	
Hystyle	S		+	+	+	+
OSU 5640	S		+	+	+	
Middle American gene pool (race Durango)						
Othello (pinto)	S		+	+	+	
Winchester (pinto)	S			+	+	
Maverick (pinto)	S			+	+	+
Aztec (pinto)	S	+		+		
PS02-011A-39 (pinto)	PR			+	+	
USPT-WM-1 (pinto)	PR		+			
Matterhorn (GN§)	S	+		+		
PS02-029C-26 (GN)	PR	+		+		+
PS02-029C-40 (GN)	PR	+		+		+
Middle American gene pool (race Mesoamerican)						
ND88-106-04 (navy)	PR		+			
Bunsi (navy)	PR		+			
Raven (black)	S		+	+	+	
I9365-31 (black)	PR					
A 55 (black)	S		+			
Phaseolus coccineus (scarlet runner bean)						
Wolven Pole	S					
PI 255956	PR					
Phaseolus acutifolius (tepary bean)						
PI 319443 (Serowi)	S					
PI 263590 (G 40045)	S					
G 40199	S					

†Coupling and repulsion represent linkage orientation of the SCAR to the resistance allele from VA19.

‡A plus sign (+) indicates presence of the marker and minus sign (–) indicates absence.

§GN, great northern.

solely by the nonwounding test, explaining 8% ($P < 0.005$) of the variation in disease reaction. Given relatively minor and inconsistent expression of WM8.4 QTL across the different populations and screening tests, further investigation regarding the effect of this QTL on partial white mold resistance is warranted.

Linkage Group 9

A single QTL WM9.1^{GC} has been found on LG 9. WM9.1, identified in the GC population, confers partial resistance in the straw test (13%). This QTL is located near SSR marker BM184 and RFLP marker D1831 in the BJ and DG populations.

Germplasm Survey for Sequence Characterized Amplified Region Markers

To ascertain potential application for marker-assisted breeding, the SCARs linked with WM2.2 and WM8.3 QTL from BV were assayed across a wide array of germplasm representing different origins and different reactions

to white mold disease (Table 5). Overall, the SCARs appear less useful for marker-assisted breeding in the Andean gene pool because most lines surveyed possessed the coupling and lacked the repulsion markers regardless of disease reaction.

Both QTL will be selectable in susceptible snap beans surveyed because they lacked the SCAR maker in coupling and possessed two to three of the SCARs in repulsion linkage. Similarly, both QTL will be selectable in the Middle American beans surveyed, with few exceptions. For example, only marker-assisted selection for absence of repulsion marker SF13R15.290 may be suitable for transferring the WM2.2 allele from VA19 into the great northern (GN) market class. The PS02-029C GN lines possess partial resistance obtained by marker-assisted backcrossing the WM8.3 allele from NY6020-4 to the recurrent parent Matterhorn (Miklas, 2007). Thus, these lines should and do exhibit presence of SCAR SF13R10.410 from VA19 because the marker is also linked with the NY6020-4 resistance allele in the B60 population. Selection against SF13R15.290 and SF6Em3.220 SCARS may be useful for transferring the WM2.2 resistance allele from I9365-31 into snap and Middle American beans.

None of the SCARs were detected in *P. coccineus* or *P. acutifolius* accessions tested, which reflects likely *P. vulgaris* origin of the partial resistance governed by the WM2.2 and WM8.3 QTL in VA19 kidney bean. The novel QTL identified in the R31 population are being examined for marker-assisted breeding potential in a separate study.

SUMMARY

QTL from BV and R31 Populations

Partial white mold resistance in VA19 light red kidney and in I9365-31 black bean were characterized and shown to be quantitatively inherited as supported by QTL analyses. For BV population, selective mapping detected the previously identified QTL WM2.2 on LG 2 and WM8.3 on LG 8. WM2.2 was expressed in both greenhouse tests and the field, which differs from previous resistance alleles characterized for this locus that were expressed solely in the field. Perhaps the Andean origin for VA19 versus Middle American origin of the previously described alleles contributes to the different effects. The same WM2.2 QTL was detected in the R31 population but only in the field, which fits the pattern of expression for the Middle American allelic sources. Four SCAR markers linked with WM2.2 in BV were generated, one in coupling, three in repulsion, and primarily assorted with gene-pool status except in some snap beans, and dry beans from race Durango.

The WM8.3 QTL was expressed only in the field for the BV population, compared with expression in both field and greenhouse or just greenhouse in other populations from previous studies. A SCAR marker linked in coupling with WM8.3 in BV was generated, so now there are two SCARs with potential utility for marker-assisted selection for this QTL, SF13R10.410 from this study and SS18.1650 from a previous study (Miklas et al., 2003).

Seven QTL were selectively mapped in the R31 population. Four QTL were expressed solely in the field, WM2.2 mentioned above and WM4.2, WM5.3, and WM6.1. The WM5.3 QTL likely confers disease avoidance because of its co-location with a QTL for canopy height detected in the same R31 population. The field resistance conferred by the other three QTL could be physiological in nature even though they were not detected by the greenhouse straw or nonwounding tests. Examples of field resistance QTL with a physiological basis include the Bunsi-derived WM2.2 and WM7.2 QTL that were associated with green stem trait (Miklas et al., 2007) and resistance to oxalate (Kolkman and Kelly, 2003), respectively. Expression of a QTL solely in the field could also result from association with avoidance traits that were not measured in the R31 population such as lodging or branching density.

The WM5.4, WM7.3, and WM8.4 QTL in R31 were expressed in the greenhouse assays but not in the field. Disease avoidance traits can confound expression of physiological resistance mechanisms in the field. It is surprising that the WM7.3 QTL with large effect in the greenhouse tests was undetected in the field. Other QTL, WM2.2 in BV, WM7.1 in AG, and WM8.3 in the B60 population, with large effect in the straw test were also detected in the field. Marker-assisted backcrossing the WM7.3 resistance allele from I9365-31 into a susceptible near-isogenic background to ascertain breeding value is warranted given the large and consistent effect for this QTL in the original R31 mapping population.

The WM4.2, WM5.3, WM5.4, and WM7.3 QTL from R31 are novel to this study. Whether these QTL represent introgressed genes from *P. coccineus* is unknown. Major effect for the WM7.3 QTL suggests that this resistance could derive from *P. coccineus* because major gene resistance appears to be the precedent for this species. Abawi et al. (1978) reported single dominant gene inheritance for white mold resistance for *P. coccineus* in interspecific crosses with *P. vulgaris*. Likewise, Schwartz et al. (2006) observed single dominant gene inheritance for partial resistance to white mold in the greenhouse straw test for two interspecific F_2 populations obtained from hybridizations between susceptible pinto bean cultivars × resistant *P. coccineus* accessions. In a preliminary study, two major QTL explaining 86% of the phenotypic variation for disease reaction in the straw test were identified in an F_2 mapping population derived between a susceptible *P. coccineus* cultivar Woven Pole and resistant *P. coccineus* accession PI 255956 (Myers et al., 2008).

Nonwounding Greenhouse Screening Method

The usefulness of the nonwounding assay to detect novel QTL conditioning partial resistance related to initial

infection, perhaps at the level of the cuticle or epidermis, was not adequately tested in this study. The phenotypic pools of BV and R31 RILs used for bulked-segregant analysis were based on disease response only in the field and straw tests. Whole linkage maps for the BV and R31 populations, once constructed, may enable further detection of QTL specific to the nonwounding tests. Conversely, the AG population from Miklas et al. (2001) consisted of a whole linkage map, but QTL specific to the nonwounding test (recently conducted but unpublished) were not detected. Results from this study clearly indicate, however, that the nonwounding and straw tests detect many of the same QTL. All three QTL detected by the straw test, WM2.2, WM5.4, and WM7.3, were also detected by the nonwounding assay. There was one QTL, WM8.4 in R31, detected solely by the nonwounding assay. Further research is needed to determine whether WM8.4 QTL is related to initial infection mechanisms that are circumvented by inoculation of the cut stem in the straw test.

Comparative QTL Map

Thirty-five QTL conditioning partial resistance to white mold from nine common bean RIL populations, 26 previously reported and 9 from this study, were comparatively mapped to the BJ and DG core maps as integrated by Blair et al. (2003). Precise interval mapping of these QTL in relation to each other is generally lacking because the QTL identified in the original populations were not linked to a universal set of evenly spaced markers. Instead, relative positions of the QTL to two or more of the same framework markers on the BJ or DG core maps were used to group the QTL. The integration of SCARs and other QTL-linked markers from the original mapping populations in the core maps helped to group QTL, as did assaying QTL-linked markers across other white mold mapping populations as described above. Using these types of information, the 35 QTL were coalesced into 21 independent QTL across nine chromosomes. With more precise mapping, it is expected that subsequent studies will group or split apart some of these QTL assignments. But for now, names were assigned to the 21 QTL using the recent QTL nomenclature guidelines established by the Bean Genetics Committee (Miklas and Porch, 2010).

Six of the comparatively mapped QTL were detected solely in the field, seven only in the straw test, and eight in both field and greenhouse tests. Three QTL, WM1.1, WM3.1, and WM5.3, expressed only in the field were associated with disease avoidance traits. Eight QTL, WM2.2, WM2.3, WM6.1, WM7.1, WM7.2, WM8.1, WM8.3, and WM8.4, validated in multiple populations, represent excellent candidates for marker-assisted breeding. In fact, four of them, WM2.2, WM7.1, WM7.2 and WM8.3, have already shown potential utility for marker-assisted breeding (Miklas 2007; Ender et al., 2008). Overall, the comparative linkage map represents a useful resource for interpreting past, present, and future QTL and fine-mapping studies concerning partial resistance to white mold in common bean.

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