Breeding Common Bean for Resistance to White Mold: A Review

Howard F. Schwartz* and Shree P. Singh

ABSTRACT

Under favorable weather conditions white mold causes 100% loss of yield and quality of susceptible common bean (Phaseolus vulgaris L.) cultivars. The disease is endemic and widespread in North and South American countries including the United States, Canada, Argentina, and Brazil. Our objective was to review progress achieved in identifying sources of resistance in Phaseolus species, genetics, and breeding for resistance to white mold. We also describe an integrated genetic improvement strategy for resistance to the pathogen with germplasm enhancement and cultivar development using multiple-parent crosses and gamete selection methods of breeding. Substantial progress has been made in understanding pathogenic variation in the white mold fungus, developing screening methods, identifying sources of resistant germplasm, genetics of resistance, and introgressing resistance from the secondary gene pool, and breeding for resistance to white mold. Also, molecular marker-assisted selection for partial resistance is practiced. However, development of white mold resistant common bean cultivars in most market classes has been slow and localized. Breeding strategies for simultaneous and integrated genetic improvement of qualitatively and quantitatively inherited resistances to white mold and cultivar development are briefly described.

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Abbreviations: QTL, quantitative trait loci (or locus).

THE WHITE MOLD DISEASE

White mold, caused by Sclerotinia sclerotiorum (Lib.) de Bary, is among the most devastating and widely distributed fungal diseases of both green and dry common bean in North and South American countries including the United States, Canada, Argentina, and Brazil. White mold is also endemic in the cool humid highlands of Mexico, Guatemala, and similar production environments elsewhere. Crop losses due to white mold disease outbreaks in dry bean average 30% in the central high plains of the United States with individual field losses as high as 92% (Kerr et al., 1978; Schwartz et al., 1987). However, under favorable weather conditions 100% seed yield and quality losses occur on susceptible common bean cultivars (e.g., Argentina in 2011) (Singh and Schwartz, 2010). For every 1% increase in white mold incidence, seed yield was reduced by an average of 12 kg ha$^{-1}$ in pinto (medium-sized cream-spotted seeds, the largest market class of dry bean in North America) and by 23 kg ha$^{-1}$ in navy bean (small white seeds) in rain-fed production systems in North Dakota (del Río et al., 2004). A 3-yr survey in North Dakota reported that white mold was observed in 75% of the fields scouted in the largest dry bean production region of the United States (Hari-krishnan et al., 2006). White mold incidence is also widespread in the western United States, especially with the use of irrigation systems (e.g., furrow, drip, overhead sprinkler).

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White mold disease may occur on all aerial plant parts. Infected flowers may develop a white, cottony appearance as mycelium grows on the surface. Lesions on pods, leaves, branches, and stems are initially small, circular, dark green, and water soaked but rapidly increase in size, may become slimy, and may eventually encompass and kill the entire organ. Under moist conditions, these lesions may also develop a white, cottony growth of external mycelium. Affected tissues dry out and bleach to a pale brown or white coloration that contrasts with the normal light tan color of senescent tissue. The epidermis easily sloughs off when the stem or pod is rubbed. Colonies of white mycelium (immature sclerotia) develop into hard, black sclerotia in and on infected tissue. Entire branches or plants may be killed (Steadman and Boland, 2005).

The Fungus Sclerotinia sclerotiorum and Pathogenic Variability

Differences in aggressiveness of different pathogenic isolates from common bean from across the United States have been reported (Kull et al., 2004), but there were no significant crossover interactions. More recently, Otto-Hanson et al. (2011) reported large variation among S. sclerotiorum isolates within and between fields from multisite screening nurseries of common bean in the United States, using mycelial compatibility groupings and aggressiveness tests. Significant differences were found for pathogen isolate aggressiveness between, but not within, mycelial compatibility groups. Genetic polymorphisms for molecular markers were detected among populations of S. sclerotiorum in Brazil, but all isolates belonged to the same mycelial compatibility group (Meinhardt et al., 2002). Pascual et al. (2010) used four S. sclerotiorum isolates (two each aggressive and less aggressive) from northern Spain to screen 29 common bean germplasm lines for resistance. Six of the 29 lines had partial resistance to all four isolates. But there were no crossover interactions among the isolates and common bean genotypes. The lack of cultivars of different evolutionary origins with high levels of the monogenic white mold resistance has hampered the understanding of variation in virulence among pathogen isolates. Moreover, environmental effects and disease avoidance mechanisms (Ando et al., 2007; Coyne et al., 1974; Kolkman and Kelly 2002; Miklas et al., 2013; Saindon et al., 1995; Schwartz et al., 1987) often mask genetic differences for physiological resistance (Miklas and Grafton, 1992).

The genome of S. sclerotiorum has been sequenced (http://www.sclerotia.org/genome/, accessed 1 Jan. 2013). This resource now allows users to download the entire genome, protein set, or portions; perform nucleotide or protein Blast searches; interactively search for predicted genes based on name, location, homology information, protein domain, and multigene family; and graphically view the sequence annotated with genes, protein families, and regions of similarity to known sequences involved with pathogenic variation, fungal growth and survival, mycelial and apothecial development, response to disease management tactics including fungicides, and reaction to disease resistance and avoidance mechanisms.

The white mold fungus S. sclerotiorum infects over 400 plant species, mostly dicots (Boland and Hall, 1994; Purdy, 1979; Steadman and Boland, 2005). Mycelium is hyaline, septate, and branched, and additional morphological descriptors for specialized spores and reproductive structures have been well characterized (Pratt, 1992). Sclerotinia sclerotiorum is homothallic and exhibits clonality (Steadman and Boland, 2005). However, more recently Attanayake et al. (2012) reported considerable genetic and phenotypic variability among 40 isolates of S. sclerotiorum collected from a 1 m² area of the top 1.27 cm layer of soil in an alfalfa (Medicago sativa L.) field in southern Washington State and evidence of outcrossing (heterothallism) within interbreeding subpopulations of S. sclerotiorum.

The cosmopolitan necrotrophic fungus is endemic and seed transmitted (Tu, 1988) and produces sclerotia that survive in soil for five or more years (Steadman and Boland, 2005). It can be spread from field to field by internally infected seed, sclerotia mixed with seed, contaminated soil on farm equipment, and irrigation runoff water in addition to wind-blown ascospores (Steadman and Boland, 2005). Under suitable conditions of temperature, light, and moisture, sclerotia within 5 cm of the soil surface germinate to produce stipes and apothecia (Matheron and Porchas, 2005; Steadman and Boland, 2005). Ascospores are released from turgid asci, often simultaneously by “puffing.” Ascospores germinate and colonize flowers and other tissues that are senescing, and the mycelium from colonized tissues invades adjacent host organs. Senescing flowers and flower parts often fall onto pods, leaves, branches, and stems and provide nutrients required by the fungus to develop appressorium to penetrate these organs. Infected tissues are rapidly killed and become dry and bleached. Limited spread of the fungus from one plant to another may occur by mycelial growth between tissues in contact. Sclerotia form in or on infected tissues and may fall to the soil, remain in crop debris, or be removed with harvested seeds or pods. The fungus may continue to develop and cause disease in green beans in transit and in storage (nesting) (Schwartz and Steadman, 1989; Steadman and Boland, 2005).

Adequate control of white mold using fungicides, biopesticides (e.g., Coniothyrium minitans, Glomus intraradices, Sporidesmium sclerotivorum, Trichoderma viride), and other disease management strategies has been difficult (Agrios, 2005; del Río et al., 2004; Schwartz and Steadman, 1989; Steadman and Boland, 2005), especially on the susceptible indeterminate prostrate growth habit Type III (Singh, 1982b) common bean cultivars that have traditionally predominated in the western United States. However,
timing of fungicide applications between 50 and 100% bloom did significantly reduce white mold severity and yield loss for upright indeterminate navy and pinto bean grown under nonirrigated (del Río et al., 2004) and sprinkler irrigated (Schwartz et al., 1992, 1994) production systems. They also reported that net returns to producers were increased up to US$260 ha⁻¹ with low to moderate disease pressure and low market values for pinto bean.

Integrated management strategies (Forster et al., 2000; Schwartz and Pairs, 1999) rely on a combination of approaches including fungicides, deep plowing, and long-term crop rotations with nonhost crops such as corn (Zea mays L.), small grain cereals, and sorghum [Sorghum bicolor (L.) Moench]. White mold management practices such as use of wide-row spacing and low plant populations, reduced irrigation and fertilizer, and use of upright cultivars with open plant canopy may reduce white mold severity and incidence (Ando et al., 2007; Coyne et al., 1974; Kolkman and Kelly, 2002; Schwartz et al., 1987); however, they also reduce common bean yield and the economic return to producers (Park, 1993; Saindon et al., 1995). Nonetheless, Peachery et al. (2006) reported reduced white mold severity and increased pod yield in green bean due to increased-row spacing. Paula Junior et al. (2009) and Vieira et al. (2010) obtained similar results in dry bean from reduced within-row plant populations. Common bean breeding lines with moderate levels of white mold resistance provided adequate control and did not significantly respond to fungicidal applications (Miklas et al., 2013). Therefore, use of adequate levels of host-plant resistance is pivotal to integrated effective control of white mold disease.

GERMPLASM SCREENING METHODS

Often it is difficult to achieve high and uniform white mold pressure in the field year after year to permit adequate evaluation of plant germplasm for disease avoidance or physiological resistance. It may be difficult to discriminate between physiological resistance and plant architectural avoidance of white mold because both are confounded under field conditions (Miklas and Grafton, 1992). False positives for physiological resistance may result due to reduced disease severity in open plant canopies of upright determinate (Type I) and indeterminate (Type II) cultivars, especially when planted in wide-row and/or increased within-row plant spacing (Ando et al., 2007; Kolkman and Kelly, 2002; Miklas and Grafton, 1992).

Because only low levels or partial resistance to white mold exist in common bean, finding a reliable, cost-effective, and rapid method with high resolving power to detect physiological resistance has been a goal of several researchers (Cline and Jacobsen, 1983; Kim et al., 2000; Steadman et al., 2001; Terán and Singh, 2009b; Vuong et al., 2004). The germplasm screening methods for white mold in common bean can be classified into those using (i) direct intact or live plants, (ii) direct detached plant organs, and (iii) indirect approaches. For direct screening of live plants, researchers have used mycelial plug inoculation of cotyledons (Grau and Bissonnette, 1974; Kim et al., 2000; Kull et al., 2003), cut petiole (del Río et al., 2000), cut-stem or straw test (del Río et al., 2000; Petzoldt and Dickson, 1996; Terán et al., 2006; Vuong et al., 2004), mycelial-infected oat (Avena sativa L.) seed for stem inoculation (Adams et al., 1973; Terán and Singh, 2009b), mycelial-infected celery (Apium graveolens L.) for a 24- to 48-h limited-term stem inoculation (Hunter et al., 1981; Pennypacker and Hatley, 1995), mycelial-infected carrot (Daucus carota L.) for stem inoculation (Steadman et al., 2001), mycelial inoculation of foliage (Wegulo et al., 1998), mycelial-infected flower for stem inoculation (Schwartz et al., 1978; Terán and Singh, 2009b), and ascospore inoculation of flowering plants (Abawi et al., 1978; Cline and Jacobsen, 1983; Schwartz et al., 1978).

Direct inoculation of excised common bean plant parts has been performed on detached leaves or flowers and excised stems using spore or mycelium suspensions (Chun et al., 1987; Leone and Tonneijck, 1990; Miklas et al., 1992a; Olivier et al., 2008; Steadman et al., 1997). Indirect methods include the use of pathogen filtrate to detect physiological resistance (Miklas et al., 1992b), oxalic acid (H₂C₂O₄) diffusion test (Tu, 1985), modified oxalate test (Kolkman and Kelly, 2000), and soluble stem pigment production in oxalic acid (Wegulo et al., 1998).

Many factors including germplasm screening methods may affect the outcome of breeding for white mold resistance in common bean. Hunter et al. (1981) compared three inoculation methods: spraying whole plants with ascospores, placing detached flowers infected with ascospores on the axes of leaves, and attaching celery pieces that are colonized with mycelia to stems. The ascospore method gave variable results and escapes occurred whereas the 24- to 48-h limited-term celery inoculation method detected partial resistance and was a more rapid and consistent test. Steadman et al. (2001) compared four greenhouse and/or laboratory screening methods with field evaluations at multiple locations. The screening methods were a detached leaf assay, straw test, oxalic acid, and modified limited term using infected pieces of carrot. Positive and significant correlations were observed between field evaluations and the straw test at two locations while oxalic acid and detached leaves were not correlated with other methods. Kull et al. (2003) using three cultivars of common bean and soybean [Glycine max (L.) Merr.] tested the efficacy of three screening methods to identify isolate aggressiveness and incidence of white mold. The screening methods used were (i) placing a mycelial plug on cotyledons, (ii) the cut-stem method, and (iii) inoculating detached leaves with a mycelial suspension. The cut-stem method was the most efficient
to identify susceptible and resistant white mold genotypes in dry bean and soybean. Carneiro et al. (2011) used the cut-stem method to evaluate 13 common bean cultivars against six isolates of *S. sclerotiorum* and to determine the minimum number of plants per plot required for the field test. They found no significant interaction between the pathogen isolates and cultivars, and eight plants per plot were adequate to assess the disease reaction. Because plant height, lodging, and days to flowering were positively correlated and seed weight and seed yield were negatively correlated with white mold disease severity in the field, it would be important to consider these factors and group common bean genotypes accordingly for field testing and seed yield measurements under white mold pressure to identify resistance (Ender and Kelly, 2005; Ender et al., 2008; Kerr et al., 1978; Miklas et al., 2013).

Terán and Singh (2009b) found significant differences among the modified cut-stem method, attaching an infected common bean flower to the stem, and attaching an infected oat seed to the stem. Significant interaction between year and screening method was detected. The cut-stem and infected common bean flower methods were the most consistent and highly correlated \((r > 0.70, P < 0.01)\). Furthermore, white mold scores increased with delayed postinoculation evaluations. In summary, there has been a gradual evolution in white mold screening methods, but the cut-stem or straw test using one or more mycelial plugs per inoculation and one or more inoculations per plant with the same or different pathogenic isolates is currently the most common and widely used method for detection of physiological resistance in the greenhouse in common bean worldwide (Singh and Terán, 2008).

Although ascospore infection in common bean principally occurs after flowering is initiated, mycelial infection in susceptible cultivars may occur anytime during the cropping season under favorable weather conditions and presence of the pathogen (Steadman and Boland, 2005). Therefore, screening for and identification of germplasm that condition resistance during the entire growing season is crucial for breeding and genetic studies. Moreover, given the fact that considerable pathogenic variability exists in *S. sclerotiorum* and common bean germplasm may respond differently to pathogenic isolates belonging to different mycelial compatibility groups, it is essential to screen germplasm against a broad spectrum of pathogen isolates found in the target production region. The aerial morphology of the common bean plant comprising the main stem and primary and secondary branches lends itself to multiple inoculations for a prolonged period covering the vegetative as well as reproductive periods. Therefore, multiple inoculations per plant using one or more mycelial plugs per inoculation with one or more isolates and periodic evaluations during the growing season may facilitate identification of highly resistant germplasm lines and cultivars.

**Sources of Resistance**

Plant architectural traits that impart a tall, upright growth habit and porous plant canopy help avoid white mold incidence and severity in disease-conducive environments in common bean (Ando et al., 2007; Coyne et al., 1974; Ender and Kelly, 2005; Fuller et al., 1984b, 1984c; Huang et al., 2003; Kolkman and Kelly, 2002; Miklas et al., 2013; Park, 1993; Saindon et al., 1995; Schwartz et al., 1987). The desirable upright indeterminate growth habit Type II and determinate growth habit Type I genotypes mostly occur in common bean germplasm from races Mesoamerica and Nueva Granada, respectively (Singh et al., 1991). However, not all genotypes with Type I or Type II growth habit avoid white mold disease equally and some are highly susceptible, for example, ‘Benton’ (Soule et al., 2011), ‘Newport’ (Kelly et al., 1995; Kolkman and Kelly, 2003), and ‘Raven’ (Ender and Kelly 2005; Ender et al., 2008; Kelly et al., 1994). A low level of white mold resistance is found in small-seeded Middle American common bean of the Mesoamerica race (e.g., AB 136, ‘ICA Bunsii’, which is synonymous with ‘Ex-Rico 23’, ‘ICA Pijao’) and wild bean (e.g., PI 318695) from Mexico (Ender and Kelly 2005; Graffon et al., 2002; Middleton et al., 1995; Miklas et al., 1992a, 1999; Mkwaia et al., 2011; Pascual et al., 2010; Singh et al., 2012; Steadman et al., 2001; Terpstra and Kelly, 2008; Tu and Beversdorf, 1982). Some large-seeded Andean common bean genotypes (e.g., A 195, CORN 501, CORN 601, CORN 606, Don Timoteo, G 122, Kaboo, L 192, MO 162, NY6020–4, PC 50, PI 313850, VA 19, and Xana) possess comparatively higher levels of white mold resistance (Griffiths et al., 2004; Griffiths et al., 2004, 2007; Maxwell et al., 2007; Miklas et al., 1999; Mkwaia et al., 2011; Pascual et al., 2010; Pérez-Vega et al., 2012; Singh et al., 2007a, 2012; Soule et al., 2011; Steadman et al., 2001, 2006). Dry bean breeding lines A 195 (Singh et al., 2007a) and VA 19 derive their white mold resistance from light red kidney ‘Red Kloud’ (Soule et al., 2011). Even higher levels of white mold resistance are found in *Phaseolus* species of the common bean secondary gene pool such as *Phaseolus cocineus* L. (e.g., G 35171, G 35172, PI 255956, PI 433246, and PI 439534), *Phaseolus dumosus* Mac-fad. (syn. *Phaseolus polyanthus* Gremm.) (e.g., G 35877), and *Phaseolus costaricensis* Freytag & Debouck (e.g., G 40604) (Adams et al., 1973; Gilmore et al., 2002; Hunter et al., 1982; Schwartz et al., 2006; Singh et al., 2009a, 2012).

Over the past several years hundreds of germplasm accessions, cultivars, and breeding lines of common bean and *Phaseolus* species of the secondary gene pool have been screened for their reaction to white mold. All genotypes without exception were variable for white mold response irrespective of the aggressiveness of *S. sclerotiorum* pathogenic isolates (Schwartz et al., 2006; Singh et al., 2009a, 2012). Therefore, in addition to the range and mean white mold disease score it would be useful to consider the frequency of plants with resistant scores (i.e.,
leaves) (Terán et al., 2006) while selecting resistant genotypes. Furthermore, the response of white mold resistant genotypes has been observed to vary depending on the isolate used and evaluation environment (McCoy and Steadman, 2009; McCoy et al., 2012; Otto-Hanson et al., 2011; Steadman et al., 2006). These observations suggest that common bean researchers may want to carefully screen pure-line sources of parental germplasm with appropriate pathogen isolate or isolates across contrasting greenhouse and/or field environments before using them in pathogenetic diversity, genetic, and breeding studies. Some common bean genotypes useful for breeding for white mold resistance are listed in Table 1.

### GENETICS OF WHITE MOLD RESISTANCE

Genetic differences in physiological resistance to white mold in common bean (Kolkman and Kelly, 2000; Tu, 1985) and *P. coccineus* (Chippa et al., 2005) have been associated with sensitivity to oxalate. Abawi et al. (1978) and Schwartz et al. (2006) reported a single dominant gene controlling resistance to white mold in *P. vulgaris × P. coccineus* interspecific populations. In contrast, Myers and Stotz (2002) found the F1 between resistant (PI 255956) and susceptible (PI 153209) *P. coccineus* to be susceptible to white mold whereas the F1 between other susceptible and susceptible (PI 153209) accessions segregated in a 3:2 resistant:susceptible ratio (Myers and Stotz, 2002). The latter could be because of possible escapes in the F1 (as supported by the occurrence of only recessive white mold resistance segregation in the F2), or the resistant PI 255956 could have been variable for white mold response. Furthermore, two or more recessive white mold resistance genes segregated in the F2 of both populations thus suggest that white mold resistance in PI 255956 was controlled only by recessive genes. Molecular markers linked with these major white mold resistance genes in *P. coccineus* have not yet been identified and mapped.

Physiological white mold resistance and architectural avoidance in common bean are quantitatively inherited to moderate heritability (Fuller et al., 1984a; Miklas et al., 2004; Park et al., 2001). At least 35 quantitative trait loci (QTL) have been identified thus far in common bean that have been associated with resistance to white mold (Chung et al., 2008; Ender and Kelly, 2005; Kolkman and Kelly, 2003; Maxwell et al., 2007; Miklas et al., 2001, 2003, 2004, 2013; Mkwaia et al., 2011; Park et al., 2001; Pérez-Vega et al., 2012; Soule et al., 2011; Terpstra and Kelly, 2008). Chung et al. (2008), Mkwaia et al. (2011), and Soule et al. (2011) reported higher heritability for physiological resistance based on greenhouse tests than those based on field evaluations with low correlations between the two tests. Genchev and Kiryakov (2002), on the other hand, reported a single recessive allele controlled resistance to white mold in the greenhouse straw test, but a dominant allele controlled resistance in the field test of an A195 × ‘Lime Light’ inter–gene pool dry bean population.

Miklas et al. (2001) identified a major QTL near the *Pbs* locus on linkage group B7 that was responsible for 38% of the variance in resistance to white mold in the greenhouse straw test and 26% of the variance in the field test in the A55 (Middle American) × G122 (Andean) inter–gene pool dry bean population. An additional QTL responsible for canopy porosity (34% of variance) and disease avoidance (18% of variance) in the field also was mapped on linkage group B1 near the *fin* gene for determinate growth habit. Park et al. (2001) reported random amplified polymorphic DNA markers linked with as many as nine QTL responsible for white mold resistance in an Andean ‘PC50’ × XAN159 dry bean population of which three QTL mapped to linkage groups B4, B7, and B8, and each accounted for up to 12% of the variance in white mold resistance. Miklas et al. (2003) identified two QTL on linkage group B6 for white mold resistance in a Benton × NY 6020-4 green bean population. The QTL from NY 6020-4 was associated with increased internode length. Two white mold resistance QTL from Andean dry bean PC 50 mapped on linkage groups B7 and B8 near the QTL from the Andean dry bean landrace G 122

### Table 1. Some useful sources of partial resistance to white mold in common bean.

<table>
<thead>
<tr>
<th>Identification</th>
<th>Growth habit†</th>
<th>Seed type</th>
<th>Mean white mold score‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>Large-seeded Andean</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A 195</td>
<td>I</td>
<td>Beige</td>
<td>4.4</td>
</tr>
<tr>
<td>G 122</td>
<td>I</td>
<td>Cream mottled</td>
<td>5.3</td>
</tr>
<tr>
<td>NY6020-4</td>
<td>I</td>
<td>White</td>
<td>5.3</td>
</tr>
<tr>
<td>PC 50</td>
<td>I</td>
<td>Red mottled</td>
<td>6.0</td>
</tr>
<tr>
<td>VA 19</td>
<td>I</td>
<td>Light red kidney</td>
<td>4.5</td>
</tr>
<tr>
<td>Medium-seeded Middle American</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chase</td>
<td>III</td>
<td>Pinto</td>
<td>7.3</td>
</tr>
<tr>
<td>Eldorado</td>
<td>II</td>
<td>Pinto</td>
<td>–</td>
</tr>
<tr>
<td>USPT-WM-1</td>
<td>III</td>
<td>Pinto</td>
<td>7.0</td>
</tr>
<tr>
<td>USPT-WM-12</td>
<td>III</td>
<td>Pinto</td>
<td>–</td>
</tr>
<tr>
<td>Small-seeded Middle American</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ICA Bunsii</td>
<td>III</td>
<td>Small white</td>
<td>6.8</td>
</tr>
<tr>
<td>AB 136</td>
<td>IV</td>
<td>Small red</td>
<td>–</td>
</tr>
<tr>
<td>Interspecific breeding lines</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>92BG-7</td>
<td>III</td>
<td>Small black</td>
<td>6.3</td>
</tr>
<tr>
<td>I9365-31</td>
<td>III</td>
<td>Small black</td>
<td>5.3</td>
</tr>
<tr>
<td>VCW 54</td>
<td>II</td>
<td>Small purplish black</td>
<td>4.6</td>
</tr>
<tr>
<td>VRW 32</td>
<td>II</td>
<td>Small grayish brown</td>
<td>5.7</td>
</tr>
</tbody>
</table>

†Growth habit I represents determinate upright, II represents indeterminate upright, III represents indeterminate prostrate semiclimbing, and IV represents prostrate strong climbing.
‡Mean white mold disease score, in which 1 represents no infection, symptomless, or completely healthy, and 9 represents white mold passed the third node after inoculation or dead plant. These scores are based on inoculations with four isolates of *Sclerotinia sclerotiorum* and scored 35 d postinoculation in the greenhouse in Idaho in 2012 and scored 21 d postinoculation in Colorado in 2013 (courtesy of D. Viteri).
and green bean NY6020-4, respectively. Therefore, some of the same resistance QTL may be present in all three common bean genotypes (Kelly et al., 2003; Miklas and Singh, 2007; Miklas et al., 2006b, 2007, 2013; Soule et al., 2011). Maxwell et al. (2007) identified five QTL for greenhouse resistance on linkage groups B1, B2b, B8, and B9, which together accounted for 48% of phenotypic variance, and one QTL accounting for 12% of the variance for a field test in the G 122 × CO72548 Andean × Middle American inter–gene pool dry bean population.

Kolkman and Kelly (2003) reported white mold resistance QTL on linkage groups B2 and B7 in an ICA Bunsi × Newport Middle American dry bean population that were confirmed in another Middle American ‘Huron’ × Newport dry bean population. They also identified QTL that were associated with white mold avoidance traits, namely days to flowering, branching pattern, lodging, seed size, yield, and resistance to oxalate. Ender and Kelly (2005) detected QTL located on linkage groups B2, B5, B7, and B8 that explained 9.2 to 14.7% of the variance for white mold resistance in an ICA Bunsi × Raven Middle American dry bean population. Miklas et al. (2007) identified two QTL located on linkage groups B2 and B3 conditioning ICA Bunsi-derived resistance that individually accounted for 5.3 to 24.7% of phenotypic variance depending on the growing environment. The physiological resistance conditioned by the QTL on linkage group B2 was associated with the stay–green stem characteristic and the QTL on linkage group B3 was associated with disease avoidance traits including late maturity and the stay–green stem characteristic, which were introgressed from ICA Bunsi into pinto USPT-WM-1 (Miklas et al., 2006a). Of 10 QTL previously identified in independent dry bean populations, only three markers (QO09.950, Pls, and OC07.850) were associated with field and greenhouse straw tests in the G 122 × ‘Astral’ Andean population (Soule et al., 2011). In a more recent comparative QTL mapping study and characterization of partial white mold resistance in the dry bean breeding lines VA 19 and I9365–31, Soule et al. (2011) found two QTL in Benton × VA 19 and seven QTL in Raven × I9365–31 populations. Three significant QTL were detected from greenhouse tests and four QTL were detected based on the results of field tests in the Raven × I9365–31 population, which ranged in their contribution to phenotypic variance for resistance from 5 to 52%. In the Benton × VA 19 population, a single QTL (WM2.2) was expressed both in greenhouse (33%) and field (13%) tests and another QTL (WM8.3) was expressed only in the field (11%). These nine QTL and 26 previously reported QTL coalesced into 21 distinct regions across nine linkage groups in a comparative linkage mapping of which four QTL found in Raven × I9365–31 population were not previously reported (Soule et al., 2011). Furthermore, of 21 QTL six were detected only in the field test, seven only in the greenhouse straw test, and eight in both tests. The role and relative importance of white mold resistance QTL that are detected only in the greenhouse tests is not clearly understood at the moment in regards to their impact under field production systems. It may be that they condition physiological resistance to plants in the early vegetative growth stages when it is relatively difficult to measure the effects in the field. Nonetheless, both greenhouse and field tests under environments conducive to severe white mold disease would be required for breeding for high levels of white mold resistance that are highly effective during the entire growing season.

Mkwaila et al. (2011) could not detect QTL for field resistance but identified two QTL each in ‘Tacana’ × PI 318695 (on linkage groups B4 and B7) and Tacana × PI 313850 (on linkage groups B2 and B9) inbred backcross lines using the greenhouse straw test. They also validated two previously mapped QTL (on linkage groups B2 and B4) and identified a QTL on linkage group B2 in the Tacana × PI 318695 population that accounted for 19 to 37% variation for seed yield under white mold pressure over 3 yr. Tacana is a small–seeded black bean and PI 318695 a wild bean, both from Mexico, and PI 313850 is a large–seeded Andean landrace, and all three have partial resistance to white mold (Ender and Kelly, 2005; Grafton et al., 2002; Miklas et al., 1999). Pérez-Vega et al. (2012) identified four major regions on linkage groups 1, 6, and 7 that were associated with partial resistance to white mold in a Xana × Cornell 49–24–2 recombinant inbred line population. The large–seeded Xana exhibited similar levels of resistance as PC 50, A 195, and G 122 to five different isolates of *S. sclerotiorum*. Furthermore, QTL involved in the control of morphological traits, namely plant height, first internode length, and first internode width, were co–located at the same relative position on linkage groups 1, 6, and 7, respectively. In a more recent characterization of white mold avoidance, Miklas et al. (2013) integrated 27 QTL for partial white mold resistance (Table 2), 36 for disease avoidance traits, and 16 for root traits, using comparative mapping. Furthermore, 36 disease avoidance QTL coalesced into 18 genomic regions influencing plant architectural traits such as canopy height, internode length, canopy porosity, and lodging, and 13 of these regions co–located with 13 of partial white mold resistance QTL. Therefore, readers interested in more complete information on white mold avoidance and resistance QTL and their map positions should refer to Miklas et al. (2013) and Soule et al. (2011).

**GERMLASM ENHANCEMENT**

Partial physiological white mold resistance has been reported in small–seeded Middle American and large–seeded Andean common bean, wild bean, and in the *Phaseolus* species of its secondary gene pool, namely *P. coccineus*, *P. dumosus*, and *P. costaricensis* (Adams et al., 1973; Gilmore et al., 2002; Grafton et al., 2002; Hunter et al., 1982; Maxwell et al., 2007; Middleton et al., 1995; Miklas
et al., 1992a, 1999; Mkwaila et al., 2011; Pascual et al., 2010; Schwartz et al., 2006; Singh et al., 2009a, 2009b, 2012; Steadman et al., 2001; Terpstra and Kelly, 2008; Tu and Beversdorf, 1982). Attempts have been made to use these individual and combined resistant germplasm as described below.

**Introggression of Resistance from Phaseolus coccineus and Phaseolus costaricensis**

Hunter et al. (1982) reported a group of F$_5$ breeding lines and Miklas et al. (1998) reported four breeding lines, namely I9365-3, I9365-5, I9365-31, and 92BG-7, derived from *P. vulgaris × P. coccineus* interspecific populations that possessed moderate to high levels of white mold resistance. Singh et al. (2009a, 2009b) developed white mold resistant interspecific breeding lines VCW 54 and VCW 55 by congeneric backcrossing between a tropical small-seeded black common bean cultivar ICA Pijao and *P. coccineus* accession G 35172. VCW 54 has a high level of resistance and VCW 55 has an intermediate level of resistance. However, introgressing white mold resistance from *P. coccineus* accessions G 35171 and PI 433246 and *P. dumosus* accession G 35877 was not successful, possibly because of elimination of whole chromosomes and/or segments carrying the white mold resistance genes and/or QTL and related incompatibility problems common in interspecies crosses (Manshardt and Bassett, 1984; Singh et al., 2009a). White mold resistance derived from *P. vulgaris × P. coccineus* populations appeared to be higher and more stable across multiploecy greenhouse and field environments (Steadman et al., 2001) than that available in pinto ‘Chase’ (Coyne et al., 1994) and breeding line USPT-WM-1 (Miklas et al., 2006a). Singh et al. (2009a, 2012) developed white mold resistant interspecific breeding line VRW 32 by recurrent backcrossing of ICA Pijao with *P. costaricensis* accession G 40604.

**Transferring Middle American White Mold Resistance to Other Market Classes of Common Bean within the Primary Gene Pool**

Miklas et al. (2006a) used marker-assisted selection and a combination of bulk and single-seed-descent breeding methods to develop pinto USPT-WM-1, which has a low level of physiological resistance to white mold derived from navy bean cultivar ICA Buni (Table 1) (Terán and Singh, 2009a, 2010a, 2010b). Similarly, Ender et al. (2008) used marker-assisted selection for white mold resistance QTL located on linkage groups B2 and B7 in two common bean populations involving ICA Buni. Furthermore, Kolkman and Kelly (2003) and Mkwaila et al. (2011) were able to enhance white mold resistance by including seed yield and plant architectural traits measured under white mold pressure in the selection process. More recently, Kelly et al. (2012) developed pinto bean cultivar Eldorado and Miklas et al. (2012) developed germplasm line USPT-WM-12 solely based on field tests under white mold pressure that exhibited higher levels of white mold resistance in the greenhouse straw test than USPT-WM-1 (McCoy et al., 2012).

**Use of Andean Sources of White Mold Resistance**

Griffiths (2009) and Griffiths et al. (2004) developed a series of dry and green bean breeding lines with partial white mold resistance by crossing within the Andean gene pool and using conventional breeding methods. Singh et al. (2007a) developed A195 with a high level of white mold resistance from an Andean intra–gene pool and using conventional breeding methods. Miklas (2007) used molecular marker-assisted selection to introgress white mold resistant QTL located on linkage group B7 in G 122 and linkage group B8 in NY6020-4.
Inter–Gene Pool Transfer and Pyramiding of White Mold Resistance within Common Bean

While genetic studies have been conducted for some sources of white mold resistance found in the Andean and Middle American gene pools of common bean and wild bean, resistance from one gene pool has neither been introgressed into cultivars of the other gene pool nor pyramided together to develop breeding lines with higher levels of resistance. From screening done thus far it appears that the Andean genotypes (e.g., A 195, G 122, PC 50, VA 19) possess comparatively higher levels of resistance than the Middle American sources of resistance (e.g., ICA Buni, ICA Pijao, AB 136), probably because some white mold resistance QTL are located in the same linkage group as is the Phs gene for determinate growth habit or the Psh gene for phaseolin seed protein (Miklas et al., 2013; Soule et al., 2011), which may directly or indirectly help avoid the disease in some Andean germplasm. Furthermore, inter–gene pool and interracial incompatibility may occur in some crosses (Singh and Gutiérrez, 1984; Singh and Molina, 1996) and often it is difficult to recover desirable seed and plant characteristics and high pod or seed yield potential from biparental (and other) crosses between the two gene pools (Kornegay et al., 1992; Miklas et al., 2013; Welsh et al., 1995). Therefore, for inter–gene pool transfer of white mold resistance, it is recommended that >50% of the genetic contribution of the germplasm should be that of the specific gene pool being improved (Singh, 2001). Similarly, for pyramiding high levels of white mold resistance it is advisable to carry out bidirectional selection (i.e., independent selection for both the Andean and Middle American genotypes) from the early generations in each genetically broad–based interracial and inter–gene pool population (Singh, 2001).

Pyramiding White Mold Resistance from Across Phaseolus Species

Lyons et al. (1987) obtained a 31% gain in white mold resistance through recurrent selection in sequential multiple–parent crosses. Terán and Singh (2009a) realized an average of 20.5% gain in two inter–gene pool double-cross populations using gamete selection. However, in the same two populations there was only 10% gain in white mold resistance in one population and 5% in another using recurrent selection (Terán and Singh, 2010a, 2010b).

Combining white mold resistance available in pinto cultivars Chase (Coyne et al., 1994) and Eldorado (Kelly et al., 2012) and breeding lines USPT-WM-1 (Miklas et al., 2006a) and USPT-WM-12 (Miklas et al., 2012) with that introgressed from P. coccineus (e.g., I9365–31, VCW 54), P. costaricensis (e.g., VRW 32), large-seeded Andean (e.g., A 195, G 122, PI 313850), small-seeded Middle American (AB136, PI 318695), and green bean (NY6020–4) may result in breeding lines with improved levels of white mold resistance. Accordingly, Terán and Singh (2009a, 2010a, 2010b) pyramided high levels of white mold resistance into pinto bean breeding lines using double–inter–gene pool populations and gamete and recurrent selection methods.

Some white mold resistances of Andean (e.g., Don Timoteo, Kaboon, PC 50, PI 313850, Xana) and Middle American (e.g., AB 136, ICA Pijao) common bean (Mkwaila et al., 2011; Park et al., 2001; Pascual et al., 2010; Singh et al., 2012; Soule et al., 2011) and wild bean (e.g., PI 318695) (Mkwaila et al., 2011; Terpstra and Kelly, 2008) and those introgressed from the Phaseolus species of the secondary gene pool (e.g., I9365–31, VCW 54, VRW 32) (Miklas et al., 1998; Singh et al., 2009a, 2009b, 2012) have not yet been systematically pyramided into common bean genotypes, and their usefulness has not been tested against different pathogenic isolates of S. sclerotiorum across production environments. Therefore, concerted efforts must be made to systematically pyramid white mold resistance and avoidance traits from Phaseolus species and introgress the highest levels of stable resistance into popular cultivars. This may require sustained collaboration among researchers in sharing germplasm and conducting genetic studies and breeding efforts.

CULTIVAR DEVELOPMENT

For successful common bean cultivar development, high levels of white mold resistance must be combined with high seed (dry bean) or pod (green bean) yield and quality with broad adaptation to production environments, desired plant type, harvest maturity, and resistance to other biotic and abiotic stresses. Therefore, selection for total plant performance and simultaneous improvement of multiple agromoctrnic traits become essential in a cultivar development program, which sets it apart from germplasm enhancement or trait introgression programs discussed above, where genetic improvement for a single trait (e.g., white mold resistance) at a time is emphasized. Consequently, it is a challenge to develop successful common bean cultivars.

Integrated genetic improvement (Singh, 1999, 2001) involving multiple–parent (i.e., three or more parents) crosses and gamete selection (Singh, 1994) that fully uses laboratory, greenhouse, and field facilities across contrasting environments should expedite cultivar development. Also, the task of cultivar development becomes comparatively easier if all parents to be used in multiple–parent crosses are well adapted in the production region or regions and have similar growth habit, harvest maturity, and seed or pod characteristics as popular cultivars. For multiple–parent crosses, enhanced germplasm lines for specific traits, elite breeding lines combining multiple desirable traits, and popular cultivars to be improved should be carefully selected after a thorough evaluation of a range of production and quality traits. Similarly, the genetic contribution of any parent in a cross, especially those contributing a quantitatively inherited trait such as white mold and/or common bacterial blight.
resistance, must not be less than 10% (Singh, 2001). Therefore, three-way and modified double crosses (i.e., a cross between an elite genotype and $F_1$ of a three-way cross) (Singh, 1982a) would be preferable over double crosses. Furthermore, the three-way $F_1$ could be screened for desirable dominant and codominant traits and/or molecular markers and only selected plants with desired genes and/or QTL crossed further to make modified–double–cross families (i.e., gamete selection) (Singh, 1994). Similarly, gamete selection could be practiced with other types of multiple–parent crosses. Gamete selection should increase the frequency of desirable recombinants in subsequent generations and help reduce population size. The amount of $F_1$ seed produced for each multiple–parent cross varies depending on the number of parents used in each cross, the genetic distance among parents, and inheritance and number of traits to be improved (Singh, 1992, 2001). Researchers should keep in mind that an average of 50 breeding lines of each market class with resistance to white mold and other abiotic and biotic stresses need to be tested for seed yield, harvest maturity, plant type, canning and cooking quality, and other traits across production region or regions to identify a superior cultivar (Singh and Schwartz, 2010).

Strategies and selection methods used for managing segregating populations and families for the development of breeding lines combining white mold resistance and other desired traits for cultivar development may vary depending on the overall objectives of the program and available resources. Miklas et al. (2013) discouraged genotypic selection solely based on white mold disease avoidance QTL until their effects can be validated in commercially acceptable high yielding genetic backgrounds. Furthermore, it is challenging to note that partial physiological white mold resistance is detected in greenhouse tests, plant architectural avoidance traits are fully expressed only in the field, all QTL controlling each trait may not be expressed in both environments, and both traits must be combined together for effective management of white mold disease. Therefore, seed yield measured from replicated trials in environments free from all stresses including white mold and under white mold disease pressure should be an integral and reliable measure of the response of common bean genotypes to the disease (Ender et al., 2008; Kolkman and Kelly, 2003; Miklas et al., 2013; Mkwaila et al., 2011). Also, this should allow identification of broadly adapted high yielding genotypes across environments with or without white mold pressure. Therefore, use of both greenhouse and field testing from the early generations may be worthy of consideration. Sequential inoculation of the same plant in the greenhouse with multiple pathogens (Terán et al., 2013) may facilitate the selection process and reduce costs. For example, inoculation of one primary leaf with *Beauveria bassiana* (an aphid–vectored potyvirus) and the other with the rust pathogen (*Uromyces appendiculatus* (Pers.;Pers.) Unger) followed by inoculation of the first trifoliolate leaf with the common bacterial blight pathogen (*Xanthomonas axonopodis pv. phaseoli* (Smith) Vauterin et al.) and the fourth or fifth internode with *Sclerotinia sclerotiorum* may allow for simultaneous selection for resistance to multiple diseases. Progenies of selected plants could then be tested in the field under severe white mold pressure for combined effects of physiological resistance and plant architectural avoidance traits, harvest maturity, seed yield, seed quality, and other traits in subsequent generations. The selection process could be repeated as necessary for the development of uniform and stable breeding lines for overall plant performance. Often a three-stage program for testing improved breeding lines is used to identify new cultivars. The three stages typically consist of (i) nonreplicated adaptation nursery at contrasting sites covering the production region or regions, (ii) multilocation replicated yield trials, and (iii) multilocation strip– or semicommercial field plots (Singh, 1992). Each of these stages or their modifications may take 1 to 3 yr.

## CONCLUSIONS AND FUTURE PROSPECTS

White mold is one of the most devastating diseases of common bean in many production environments of North and South America and worldwide. There is considerable information available on the pathogen, *S. sclerotiorum*, its genetic and phenotypic variability among pathogenic isolates, and the sequence of its genome. Disease management through the use of chemicals and other management practices often has been difficult and costly. White mold resistant cultivars are desirable for more effective and integrated management of the disease and to reduce production costs. Germplasm screening methods have evolved over time, and currently the cut–stem or straw test is most widely used for the greenhouse system to detect physiological resistance. Partial physiological white mold resistance is found in small-seeded Middle American (e.g., ICA Buni, AB 136, PI 313850) and large-seeded Andean (e.g., A 195, G 122, PC 50, PI 318695, VA 19, Xana) common bean, wild bean (e.g., PI 318695), and *Phaseolus* species of the secondary gene pool such as *P. cocineus* (e.g., G 35172, PI 255956, PI 439534) and *P. costaricensis* (G 40604). Also, plant architectural traits such as upright determinate growth habit Type I and upright indeterminate Type II with open porous canopy, stay–green stem characteristics, lodging resistance, and late maturity help avoid or minimize white mold incidence and severity. Partial physiological resistance to white mold is controlled quantitatively and by major dominant and recessive genes. Twenty-seven QTL for partial physiological white mold resistance and 36 QTL that coalesced into 18 genomic regions for white mold avoidance traits have been reported. Of the latter, 13 co-located with 13 QTL for physiological resistance. Genomic selection solely for white mold avoidance traits is discouraged until their effects are validated in high yielding cultivars.

White mold resistance from small-seeded white bean (ICA Buni) has been successfully transferred into pinto bean...
(e.g., USPT-WM-1, USPT-WM-12, Eldorado). Similarly, white mold resistance from *P. coccineus* (e.g., 92BG-7, I9365-31, VCW 54) and *P. costaricensis* (e.g., VRW 32) has been introgressed into common bean. Furthermore, high levels of physiological white mold resistance from within the primary gene pool and from across *Phaseolus* species has been pyramided in pinto and other types of common bean. Stability, effectiveness, and usefulness of the recently introgressed and pyramided resistances remain to be determined across greenhouse and field environments against pathogen isolates of varying aggressiveness and their deployment in cultivar development. Efforts, therefore, need to continue to combine high levels of white mold resistance with other desirable traits to incorporate into future releases as promising cultivars of different market classes of common bean. Integrated genetic improvement using multiple-parent crosses, gamete selection, and combined use of laboratory (marker-assisted selection), greenhouse (direct disease screening), and field environments for testing for seed or pod yield, maturity, plant type, and other agronomic traits with and without white mold and other stresses is suggested for development of high yielding broadly adapted cultivars.

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