Evaluation of Annual Bedding Plants for Resistance to White Mold

Michelle A. Grabowski¹ and Dean K. Malvick

Department of Plant Pathology, University of Minnesota, 1991 Upper Buford Circle, 495 Borlaug Hall, St. Paul, MN 55108

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Abstract. White mold, caused by the necrotrophic fungus *Sclerotinia sclerotiorum* (Lib.) de Bary, causes stem rot, crown rot, wilt, and death of many common herbaceous ornamental plants. Relatively little research has been done on management of white mold and resistance to this disease in ornamental crops. Plant varieties from four genera of widely available annual bedding plants with no reported history of white mold susceptibility were evaluated for potential resistance to *S. sclerotiorum*. All plants were inoculated with three isolates of *S. sclerotiorum* and evaluated for disease severity in a controlled environment and under field conditions. *Portulaca grandiflora* Hook. ‘Sundial Scarlet’, *P. grandiflora* ‘Sundial White’, *Pentas lanceolata* Forssk. ‘Graffiti Pink’, and *Scaevola aemula* R. Br. ‘Whirlwind White’ were highly susceptible to white mold in the controlled environment but had significantly lower disease incidence and severity than *Zinnia elegans* × *angustifolia* ‘Profusion White’, the susceptible control, under field conditions. *Impatiens hawkeri* W. Bull. ‘Sonic Red’, *I. hawkeri* ‘Sonic Amethyst’, *I. hawkeri* ‘Sonic White’, *I. walleriana* Hook. f. ‘Blitz 3000 Red’, *I. walleriana* ‘Blitz 3000 White’, and *I. walleriana* ‘Blitz 3000 Rose’ displayed abscission of diseased plant tissue as an unusual resistance response. Plants from all four genera that were evaluated became infected with *S. sclerotiorum* to a lesser extent than susceptible controls under field conditions and could be used as part of an integrated disease management program for white mold in ornamental plantings.

White mold, also called *Sclerotinia* stem rot, is caused by the fungus *Sclerotinia sclerotiorum*. This necrotrophic pathogen can infect over 400 species of plants from 75 families, including monocotyledons and dicotyledons and herbaceous and woody ornamentals (Boland and Hall, 1994). Gleason et al. (2009) listed over 30 genera of herbaceous perennials and Daughtrey et al. (1995) reported 10 genera of common flowering potted plants that are susceptible to *S. sclerotiorum*. Boland and Hall (1994) list over 50 susceptible genera in asteraceae alone. These lists are outdated because multiple new publications describe infection of new host plants by *S. sclerotiorum* (Chang et al., 1997; Garibaldi et al., 2001, 2008a, 2008b, 2008c; Gulya et al., 2006; Strauss and Dillard, 2009).

Annual bedding plants are widely used and highly valued in landscapes in private homes, public parks, and around commercial properties. The U.S. Department of Agriculture Agricultural Statistics Service reports that the value for wholesale annual bedding and garden plants totaled \$1.36 billion in 2012.

Many of the most popular annual bedding plants are susceptible to white mold, including petunia, zinnia, verbena, snapdragon, and salvia (Boland and Hall, 1994). Infection of annual bedding plants can cause significant plant death in ornamental beds in home and public landscapes. Although infection may start early in the season, plant death from white mold often occurs midsummer, when replacement plants are unavailable.

Infestation of an annual flower bed with *S. sclerotiorum* is particularly problematic because the fungus produces long-term resting structures called sclerotia (Bolton et al., 2006; Grau and Radke, 1984). Once introduced, the fungus can persist in soil and plant debris for decades with repeated infection each year. The close plant spacing and overhead irrigation used in most annual plantings create highly favorable environmental

conditions for sporulation and infection by *S. sclerotiorum* (Bolton et al., 2006; Grau and Radke, 1984).

Although white mold causes significant levels of stem rot, crown rot, wilt, and death of many common herbaceous ornamental plants (Bolton et al., 2006), relatively little research has been done on management of white mold in ornamental crops. Annual bedding plants are not commonly evaluated for resistance to white mold. There are several commonly used genera and species of annual bedding plants for which susceptibility or resistance to white mold has not been reported. Identification of genera or species with resistance to *S. sclerotiorum* would provide an effective means of managing white mold, requiring little extra labor or expenses. The objective of this study was to evaluate widely available annual bedding plant varieties with no reported susceptibility to white mold for potential resistance to this disease.

Materials and Methods

Isolate preparation

Three isolates of *S. sclerotiorum* from diverse hosts were arbitrarily chosen for this study. The isolates were ORN010, isolated from a petunia plant in Chaska, MN; FC002, isolated from a soybean plant in Renville County, MN; and VEG001, isolated from green bean in Spring Valley, WI. All isolates were grown on autoclaved carrots and stored as sclerotia. Sclerotia were later germinated on half-strength potato dextrose agar (PDA) (Difco, Sparks, MD) for use in inoculation trials. Isolates were never transferred on PDA more than three times between plant inoculations to maintain pathogenicity.

Studies of disease susceptibility in controlled environments

Plant species and varieties for these studies were chosen based on the known host list of *S. sclerotiorum* (Boland and Hall, 1994; Farr et al., 1989), the 2009 University of Minnesota annual flower trial list, and several commercial flower catalogs. Plant families were identified that had no known hosts to *S. sclerotiorum*, and genera of annual bedding plants within these families were chosen that are widely available to landscape managers (Table 1). The annual bedding plants were purchased in March as transplants from a local

Table 1. Plants species and varieties evaluated for susceptibility to white mold.

Scientific name	Variety	Family
<i>Impatiens hawkeri</i>	New Guinea Impatiens Sonic® Red	Balsaminaceae
<i>Impatiens hawkeri</i>	New Guinea Impatiens Sonic™ Amethyst '06	Balsaminaceae
<i>Impatiens hawkeri</i>	New Guinea Impatiens Sonic™ White	Balsaminaceae
<i>Impatiens walleriana</i>	Impatiens Blitz 3000™ Red	Balsaminaceae
<i>Impatiens walleriana</i>	Impatiens Blitz 3000™ Rose	Balsaminaceae
<i>Impatiens walleriana</i>	Impatiens Blitz 3000™ White	Balsaminaceae
<i>Pentas lanceolata</i>	Graffiti Bright Red®	Rubiaceae
<i>Pentas lanceolata</i>	Graffiti Pink®	Rubiaceae
<i>Portulaca grandiflora</i>	Moss Rose Sundial™ Scarlet	Portulacaceae
<i>Portulaca grandiflora</i>	Moss Rose Sundial™ White	Portulacaceae
<i>Scaevola aemula</i>	Whirlwind® White	Goodeniaceae
<i>Zinnia elegans</i> × <i>angustifolia</i>	Profusion White	Asteraceae

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¹To whom reprint requests should be addressed; e-mail magrabow@umn.edu.

garden center in 4-inch pots or nine packs. The plants in nine packs were transplanted into 4-inch pots and grown in a greenhouse with 12 h light at 25 °C and 12 h dark at 20 °C for 2 weeks before inoculation.

Studies in growth chambers were arranged in a completely random design with two factors, isolate and plant entry, replicated three times. One primary stem per plant was cut with a sterile razor blade just below the upper-most pair of fully expanded leaves. An 8-mm square of half-strength PDA containing the leading edge from a *S. sclerotiorum* culture was placed on the tip of the cut stem and covered by a sterile 0.6-mL microcentrifuge tube. One uninoculated control plant per plant entry was prepared for inoculation in a similar fashion but received only sterile half-strength PDA. *Zinnia elegans* × *angustifolia* ‘Profusion White’ was included as a known susceptible control.

All plants were incubated in a mist chamber with a 12-h photoperiod and misted 10 min every hour for 2 d after inoculation. After 2 d, plants were moved to a growth chamber (Environmental Growth Chambers, Chagrin Falls, OH) set to 18 °C for 14 h of light and 16 °C for 10 h of dark. Lesion length was measured at 4 and 14 d after inoculation (DAI) as a rating of disease severity. As a result of a unique and unexpected resistance response of abscission of infected stems, lesion length could not be accurately measured for impatiens and New Guinea impatiens. The following scale was used to rate all impatiens: 1 - stem abscission halted progression of disease; 0 - stem abscission did not halt progression of disease.

At 14 DAI, a 2.5-cm section of the inoculated stem centered on the leading edge of visible symptoms was excised, surface-sterilized, and plated on half-strength PDA to verify that *S. sclerotiorum* was the causal agent of the observed symptoms. The experiment was performed twice.

Field studies of disease susceptibility

All plant entries were transplanted in a randomized complete block design with six blocks at two locations in 2011 on the St. Paul campus of the University of Minnesota. Plants were purchased in 4-inch pots or nine packs from a local garden center and transplanted into one field plot on 8 June and the other field plot on 14 June. Each entry was transplanted in a 92 cm × 92-cm bed with 305 cm between plants. A single row of ‘Profusion Cherry’ zinnias was planted as a border around each block to help maintain even moisture within the beds and reduce edge effects. Plants were watered once a day for 20 min with an oscillating sprinkler. Osmocote 14-14-14 was applied 2 weeks after planting at a rate of 340 kg·ha⁻¹ to all plots.

The three isolates of *S. sclerotiorum* noted were grown on half-strength PDA. When cultures reached the edge of a 100-mm petri plate, the agar from three plates per isolate was cut into 10-mm squares using a sterile spatula and placed into a sterile 48 × 58-cm autoclavable bag (VRW Intl., Radnor, PA)

containing 2000 cc of sorghum seed. Before inoculation, sorghum seed was soaked in distilled water for 18 to 24 h, strained to remove excess water, autoclaved for 60 min, allowed to cool for 18 to 24 h, and autoclaved a second time for 60 min. The fungus was allowed to colonize the seed for 3 weeks at 23 °C and then the infested seed was dried at 35 °C for 48 h and ground to a course powder in a standard blender.

Because no affect resulting from isolate was identified in the controlled environment experiments, all three isolates were combined in field studies. Inoculum was created by mixing 1000 cc of ground seed from each of the three isolates. The inoculum mixture was then sprinkled evenly over the plant canopy by hand at a rate of 86.5 L·ha⁻¹ on 4 Aug. 2011 when the trial plants had grown together to achieve canopy closure and again 1 week later. Immediately after inoculation, plants were irrigated for 5 min. Irrigation was subsequently applied three times daily for 10 min for the duration of the experiment. After the inoculations, the daily maximum temperature ranged from 22.7 to 29 °C and the daily minimum temperature ranged from 15.5 to 23 °C.

Disease severity and incidence were recorded every 14 DAI until frost. Disease incidence was recorded as the number of plants per bed that were visibly infected with *S. sclerotiorum* based on signs and symptoms. Any plant with questionable symptoms was surface-sterilized and plated on half-strength PDA at the end of the experiment to determine if symptoms were caused by *S. sclerotiorum*. Disease severity was recorded as percent canopy missing as a result of death of plants within the nine plant beds. To compare disease progress over time, the summary variable area under disease progress curve was calculated for a 28-d period for Field 2 and a 42-d period for Field 1 (Madden et al., 2007). This difference in period of observation for the two fields was the result of delayed planting and an early frost in Field 2.

All varieties of *I. walleriana* were excluded from the final analysis of disease severity and incidence as a result of the confounding affects of severe infection by *Plasmopara obducens*, the causal organism of Impatiens Downy Mildew. *Pentas lanceolata* Graffiti Bright Red® was not included in the field trial as a result of a crop failure at the nursery.

Statistical analysis. Using Hartley’s Fmax test, it was determined that variances were homogeneous between both controlled environment experiments. As a result, data from

both experiments were combined and analyzed as a factorial design using JMP software (SAS Institute Inc., Cary, NC) to calculate an analysis of variance. Tukey’s honestly significant difference was used to separate means. Uninoculated controls were not included in statistical analysis but were maintained for comparison.

Data from field experiments were not normal and treatment variance was not equal. This could not be corrected with standard data transformations. As a result, non-parametric methods were used to analyze the data. Friedman’s test was used to calculate a χ^2 for the model and a *P* value for treatments. Treatments with a *P* value ≤ 0.01 were compared using Wilcoxon’s rank testing to determine differences between treatments.

Results

Studies in controlled environments. All inoculated plants became infected with *S. sclerotiorum*. The pathogen was successfully isolated from all symptomatic tissue. Disease severity varied among the plants tested (Table 2). *Scaevola aemula* ‘Whirlwind White’ had significantly greater disease severity than all other genera tested except the susceptible control ‘Profusion White’ zinnia. Disease severity of the *Portulaca grandiflora* and *Pentas lanceolata* varieties was significantly less than the susceptible control 4 DAI, but by Day 14, there was no significant difference between disease severity in these varieties and the susceptible control (Table 2). By Day 14 of the experiment, 100% of the susceptible control plants, ‘Profusion White’ zinnia, were infected and lesion length extended the entire length of the stem. *S. sclerotiorum* was recovered from the leading edge of all lesions tested. There was no significant difference in disease severity caused by the three isolates of *S. sclerotiorum*. In addition, there were no significant interactions between isolate and plant entry (data not shown).

All varieties of *I. hawkeri* and *I. walleriana* developed a unique abscission resistance response to infection with *S. sclerotiorum*. Initially the inoculated stem developed soft rot at the point of inoculation, and occasionally blue-black steaks developed along the stem beyond the rotted area. This was most easily observed on varieties with green stems. At healthy nodes in advance of the infection, the plant created an abscission zone in the stem, and the stem above the abscission zone completely detached from the lower stem as shown in Figure 1. Nearly 100% of all three

Table 2. White mold lesion length ratings for annual bedding plants inoculated under controlled environment conditions.

Scientific name	Variety	Day 4 lesion length (cm)	Day 14 lesion length (cm)
<i>Scaevola aemula</i>	Whirlwind® White	7.45 a ^z	12.72 a
<i>Zinnia elegans</i> × <i>angustifolia</i>	Profusion White	5.99 b	9.68 ab
<i>Portulaca grandiflora</i>	Moss Rose Sundial™ White	3.74 c	6.71 bc
<i>Portulaca grandiflora</i>	Moss Rose Sundial™ Scarlet	3.54 c	7.87 bc
<i>Pentas lanceolata</i>	Graffiti Pink®	2.63 cd	6.53 c
<i>Pentas lanceolata</i>	Graffiti Bright Red®	2.22 d	7.86 bc

^zMeasurements with the same letter are not significantly different at $\alpha = 0.01$.

varieties of *I. hawkeri* completely recovered from infection through this resistance response (Table 3). All varieties of *I. walleriana* tested also developed an abscission layer, but *S. sclerotiorum* frequently infected the lower stem before complete abscission occurred and soft rot often progressed to the crown of the plant (Table 3). *S. sclerotiorum* was recovered from areas of the impatiens stem with clear soft rot symptoms both above and below the abscission layer. If the plant successfully abscised the stem section containing the infection, *S. sclerotiorum* was not recovered from the stem below the abscission layer.

Studies under field conditions. All plant entries tested under field conditions developed infection by *S. sclerotiorum*, but incidence and severity varied (Table 4). In contrast to observations in controlled environment conditions, all varieties tested had lower disease incidence and severity compared with

the susceptible control including the *Scaevola* hybrid. No consistent difference between plant entries was observed in disease incidence or severity. The abscission resistance response observed in *Impatiens* spp. under controlled environment conditions was also observed in both species in field conditions.

Discussion

This is a first report of susceptibility of *Impatiens hawkeri*, *Impatiens walleriana*, *Pentas lanceolata*, *Portulaca grandiflora*, and *Scaevola aemula* to white mold caused by *S. sclerotiorum*. This study shows that *Scaevola aemula*, *Portulaca grandiflora*, *Pentas lanceolata*, and *Impatiens* spp. are not immune to infection by *S. sclerotiorum* but have some resistance to infection under field conditions and suffer less canopy die-back than the susceptible control. As a result,

landscape managers could use these plants as part of an integrated disease management plan to reduce damage from white mold. In addition, these results indicate that trials conducted in controlled environmental conditions alone are not sufficient to evaluate plant species or varieties for susceptibility to white mold.

The stem abscission observed in *Impatiens* spp. in response to infection with *S. sclerotiorum* was unexpected, unique, and very effective in preventing disease development. Only one other report of abscission as a defense response to *S. sclerotiorum* was found. Sedun et al. (1989) reported that abscission of infected leaf petioles of *Brassica carinata* in response to infection by *S. sclerotiorum* prevented the infection from spreading to the main stem. This defense response was not observed in any other *Brassica* sp. tested. Ethylene and jasmonic acid are known signal molecules for disease defense and abscission in plants (Dostal et al., 1991; Estornell et al., 2013; González-Carranza et al., 1998; Sequeira, 1973; Sexton, 1976; Wilson et al., 1987), and it may be beneficial to further study the role that plant hormones may play in the resistance response between *Impatiens* spp. and *S. sclerotiorum*.

Varieties of *Scaevola aemula*, *Portulaca grandiflora*, and *Pentas lanceolata* demonstrated higher levels of resistance under field conditions than in a controlled environment growth chamber. In a similar study comparing greenhouse and field inoculations of soybean, sunflower, and dry bean, Vuong et al. (2004) demonstrated a correlation between disease severity in greenhouse and field evaluations of dry bean varieties but found no correlation in sunflower and varying results for soybean. In studies evaluating field and vegetable crops, genetic resistance is often attributed to varieties that have slower lesion length development (Blanchette and Auld, 1978; Sedun et al., 1989). Varieties of *Scaevola aemula*, *Portulaca grandiflora*, and *Pentas lanceolata* had lesion lengths equal to susceptible control plants 14 DAI, indicating a lack of genetic resistance to *S. sclerotiorum*. However, all of these plants had significantly lower disease incidence and severity than the susceptible control under field conditions throughout the growing season.

This field resistance may be the result of plant architecture that creates an unfavorable environment for *S. sclerotiorum* in lieu of genetic defense mechanisms. Reduction of white mold disease severity resulting from plant architecture of bean has been observed in several studies (Blad et al., 1978; Park, 1993; Weiss et al., 1980). Blad et al. (1978) demonstrated that a dry bean variety with a “dense luxuriant canopy” had greater leaf wetness, cooler canopy temperature, and greater disease severity than a variety with an “open, upright growth form” with similar levels of genetic resistance. Park (1993) demonstrated that upright bean varieties had lower disease incidence than short bush types. An increase in leaf area and canopy cover of the soil was also associated with

Fig. 1. Abscission of an *Impatiens hawkeri* stem infected with *Sclerotinia sclerotiorum* prevented progression of the infection further down the stem.

Table 3. Percent of impatiens plants that recovered from infection by *S. sclerotiorum* as a result of abscission of infected stems and the average abscission rating² for each variety.

Scientific name	Variety	Percent plants recovered from infection	Abscission rating 14 DAI
<i>Impatiens hawkeri</i>	New Guinea Impatiens	100.0	1.00 a ²
	Sonic™ White		
<i>Impatiens hawkeri</i>	New Guinea Impatiens Sonic® Red	94.4	0.94 a
<i>Impatiens hawkeri</i>	New Guinea Impatiens Sonic™ Amethyst '06	94.4	0.94 a
<i>Impatiens walleriana</i>	Impatiens Blitz 3000™ Rose	72.2	0.72 ab
<i>Impatiens walleriana</i>	Impatiens Blitz 3000™ White	72.2	0.72 ab
<i>Impatiens walleriana</i>	Impatiens Blitz 3000™ Red	50.0	0.50 b

¹1 = stem abscission halted progression of disease; 0 = stem abscission did not halt progression of disease.

²Measurements with the same letter are not significantly different at $\alpha = 0.01$.

Table 4. Area under disease progress curve (AUDPC) for incidence and severity² of white mold in field-inoculated annual bedding plants.

Scientific name	Variety	Incidence AUDPC		Severity AUDPC	
		Field 1	Field 2	Field 1	Field 2
<i>Portulaca grandiflora</i>	Moss Rose Sundial™ Scarlet	13 c ³	0 b	1 d	0 b
<i>Portulaca grandiflora</i>	Moss Rose Sundial™ White	358 c	26 b	167 c	0 b
<i>Impatiens hawkeri</i>	New Guinea Impatiens Sonic™ Amethyst '06	13 c	0 b	0 d	0 b
<i>Impatiens hawkeri</i>	New Guinea Impatiens Sonic® Red	79 c	0 b	33 d	0 b
<i>Impatiens hawkeri</i>	New Guinea Impatiens Sonic™ White	52 c	0 b	0 d	0 b
<i>Pentas lanceolata</i>	Graffiti Pink®	700 b	93 b	109 c	19 b
<i>Scaevola hybrid</i>	Whirlwind® White	1184 b	39 b	657 b	6 b
<i>Zinnia elegans</i> × <i>angustifolia</i>	Profusion White	3163 a	2087 a	1268 a	1048 a

²As a result of delayed planting and early frost in Field 2, AUDPC was calculated over a 28-d period, whereas AUDPC for Field 1 was calculated over a 42-d period.

³Measurements with the same letter are not significantly different at $\alpha = 0.01$.

increased disease severity in bean crops (Blad et al., 1978; Weiss et al., 1980).

Scaevola aemula and *Portulaca grandiflora* both have narrow leaves that develop along recumbent stems. The overall plant canopy is open, and each individual stem receives sunlight and air movement. Soil was easily observed in beds below both plant entries in field plots indicating little shading of the soil surface. *Pentas lanceolata* has a tall, open upright plant form with narrow leaves and multiple branches arising from a primary stem. In contrast the susceptible control, 'Profusion white' zinnia has a short, dense, compact canopy with multiple stems grouped close together. The growth forms of *Scaevola aemula*, *Portulaca grandiflora*, and *Pentas lanceolata* could have accelerated the drying of stems, foliage, and soil after irrigation, rain, or dew, creating environmental conditions less favorable for infection by *S. sclerotiorum* and development of white mold.

This study reveals that various levels of resistance to white mold exist in readily available but understudied annual bedding plants. Resistance could come in the form of genetic resistance or as physical properties that make the plant a poor host. Evaluation of plants for resistance to white mold should therefore include studies under field conditions as well as in controlled environmental conditions.

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