New Tools for Managing Sclerotinia Stem Rot in Potato

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Sclerotinia stem rot, also called potato white mold, has historically been difficult to manage in potato fields in the Columbia Basin. The disease is favored by practices that promote lush, dense foliage; sprinkler irrigation; and long periods of high relative humidity that are often encountered in the potato canopy beginning at row closure. Satisfactory control of the disease has often not been achieved in the Columbia Basin even with two to three fungicide applications when applications were initiated at row closure.

Sclerotinia stem rot first appears as water-soaked spots usually at the point where stems attach to branches, on stems where blossoms have fallen, or on branches and stems in contact with the soil. A white cottony growth of fungus mycelium develops on the lesions, and the infected tissue becomes soft and watery. Lesions may then expand and girdle the stem, which causes the foliage to wilt. During dry conditions, lesions become dry and will turn beige, tan, or bleached white in color, and papery in appearance. Hard, irregularly shaped resting bodies of the fungus, called sclerotia, form in and on decaying plant tissues. Sclerotia are generally ¼ to ½ inch long, initially white to cream in color but becoming black with age, and are frequently found in hollowed-out centers of infected stems. Sclerotia will eventually fall to the ground and enable the fungus to survive until the next growing season.

Sclerotia are very durable and can survive in soil for at least 3 years. They require a conditioning period of cool temperatures before germination. During the growing season, sclerotia within 1 inch of the soil surface germinate when the canopy of the growing crop shades the ground and soil moisture remains high for several days. Sclerotia either germinate directly as mycelium, which may infect stems near the soil surface, or they produce fruiting bodies called apothecia (singular is apothecium). Apothecia are cup-shaped on their upper surface, about 0.5 inch in height, fleshy in texture, pale orange, pink or light tan in color. Millions of ascospores are formed in each apothecium and are ejected into the air. The ascospores are carried by air current up to several miles and colonize dead or dying plant tissue when moisture is present. Ascospores cannot infect green tissue directly, but need senescing or dead plant material as an energy base to produce mycelium and colonize green plant tissue. Fungus mycelium must be in contact with green plant tissue for infection to occur. Once infection has occurred, the disease develops most rapidly at moderate temperatures of 60 to77F.

Observations made in potato fields in the Columbia Basin in 2000 indicated that disease control could be improved if timing of initial fungicide application were based on the infection cycle of *Sclerotinia sclerotiorum*, the fungus that causes stem rot, as well as when the microclimate within the crop canopy favored disease

development. Consequently, sclerotinia stem rot was monitored throughout the growing seasons in six commercial fields in 2001 and four fields in 2002 to determine when infection occurs and to determine modes or methods of infection to improve timing of fungicide application. Disease progress in fields and lesion expansion on stems were quantified in each field; petri dishes with a semi-selective medium were exposed in fields four weeks after plant emergence to sample for air borne ascospores. In addition, fungicides were evaluated for efficacy in replicated plots in a commercial field of Ranger and on Russet Burbank in the greenhouse.

Ascospores of the fungus were collected on a semi-selective medium exposed to the air over potato canopies located throughout the Columbia Basin from mid-May, several weeks before row closure, through early July of 2001 and 2002 (Figures 1, 2 and 3). Stem infections did not occur until 10 to 14 days after row closure and initial blossom drop (Figures 4 and 5). *Sclerotinia* was frequently isolated from potato blossoms attached to plants in fields that did not show disease symptoms. Infection of stems occurred when contaminated blossoms fell on stems in the plant canopy, or when stems came in contact with colonized, fallen blossoms on the ground. Infections also came from direct mycelium growth from sclerotia. About 5% initial infections in one field in the north Basin came from sclerotia in the soil. In this field, lesions were first observed on the base of stems randomly scattered in the field with no evidence of colonized blossoms on the ground. Aerial infections from contaminated blossoms developed later in this field. Infections from sclerotia were less prevalent than from ascospores. However, either source of infection may cause numerous infections.

Contaminated blossoms falling from the top of the canopy were found to be the key inoculum bridge for infection. Foliar fungicides should be targeted at infection from ascospores via contaminated blossoms. Foliar fungicides with activity on stems include Omega, Endura, Rovral, and Botran. Current research suggests that such materials should be applied at initial full bloom. An additional application of a foliar fungicide could be made 7 days later, if needed. This is a narrow application window and applications made after blossom fall would be less effective. Sclerotia in soil can infect stems before blossom fall, but foliar fungicides are not likely to prevent these infections because the fungus (sclerotia and mycelium) is in and on soil.

In the past, fungicide applications at row closure have not been completely effective because blossom contamination was not prevented. Lesions develop on stems about 3 days after inoculation. Application at row closure is about 10 to 14 days before lesions are visible on stems and 7 to 11 days before inoculation. Fungicides applied at row closure are more likely to be washed from stems and broken down by environmental factors than when applied at or just before initial full bloom.

Chemical Control of Sclerotinia Stem Rot

Several fungicides, fungicide mixtures and blossom removal before blossom fall were evaluated for control of Sclerotinia stem rot in replicated plots in a commercial potato field of Ranger Russet located north of Pasco in 2002. Materials were applied

using a CO_2 backpack sprayer at initial full bloom on 11 June 2002 (30 GPA @ 30 psi). A second application of some fungicides was made 7 days later on 18 June (Table 1). Plots were 3 rows by 30 feet. Treatments were replicated four times in a randomized complete block design.

Incidence of stem rot was significantly reduced with one application of Bass 510 (Endura), Omega, Rovral, Topsin M, and by removing blossoms before blossom fall (Table 1). The effectiveness of removing blossoms supports the hypothesis that contaminated blossoms play a major role in stem infections. Endura and Omega are new fungicides with good efficacy on stem rot and timing was effective at initial full bloom (Table 1). Chemigation of Rovral, Botran, Quadris and mixture of these materials with Blocker was ineffective in reducing stem rot when applied at row closure, which was 10 days before initial full bloom, in a test in a commercial field in 2001. Initial full bloom appears to be a better application timing than row closure; however, applications must occur before more blossoms fall.

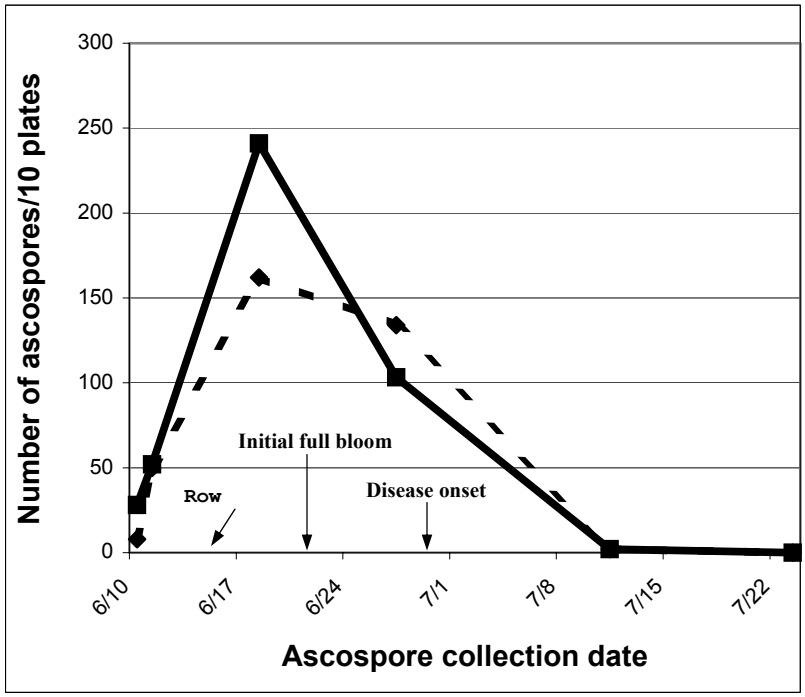

Figure 1. Number of ascospores captured in two fields of Ranger north of Pasco 2002.

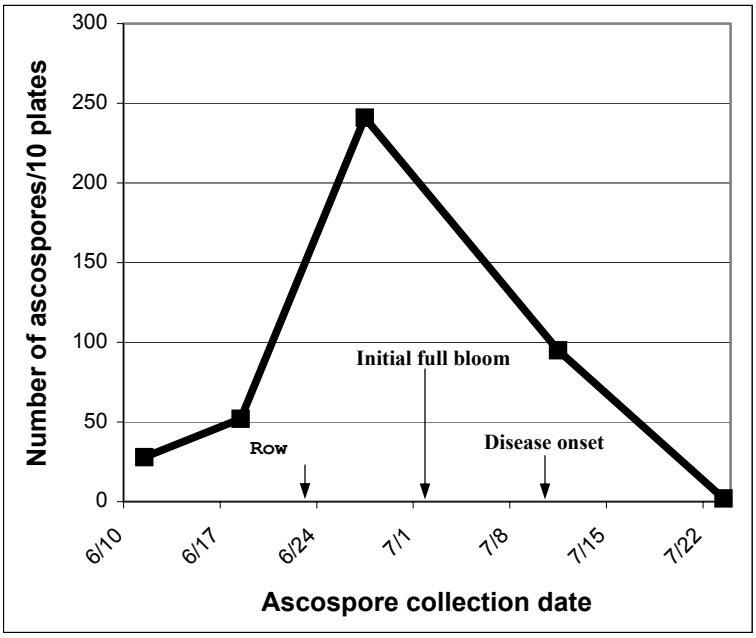

Figure 2. Number of ascospores captured in field of Shepody north of Pasco in 2001.

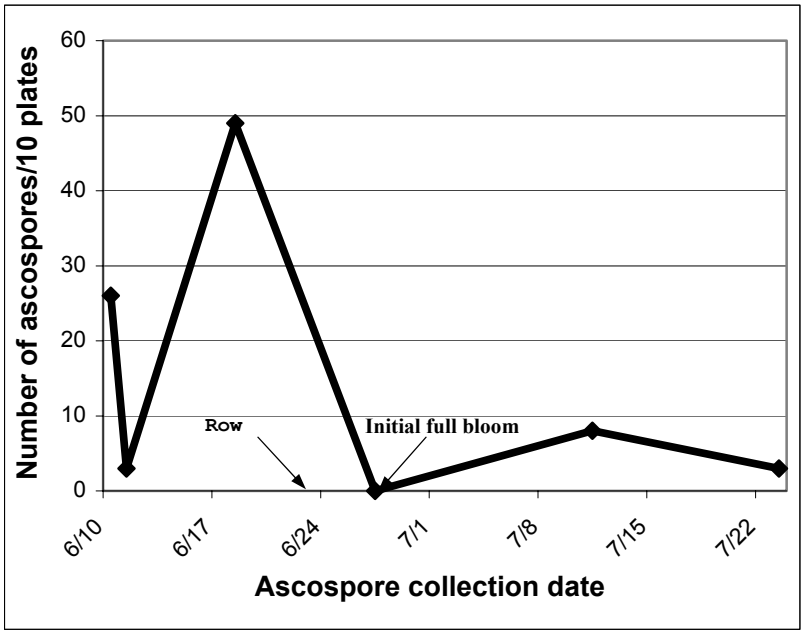

Figure 3. Number of ascospores captured in field of Umatilla near Warden in 2002.

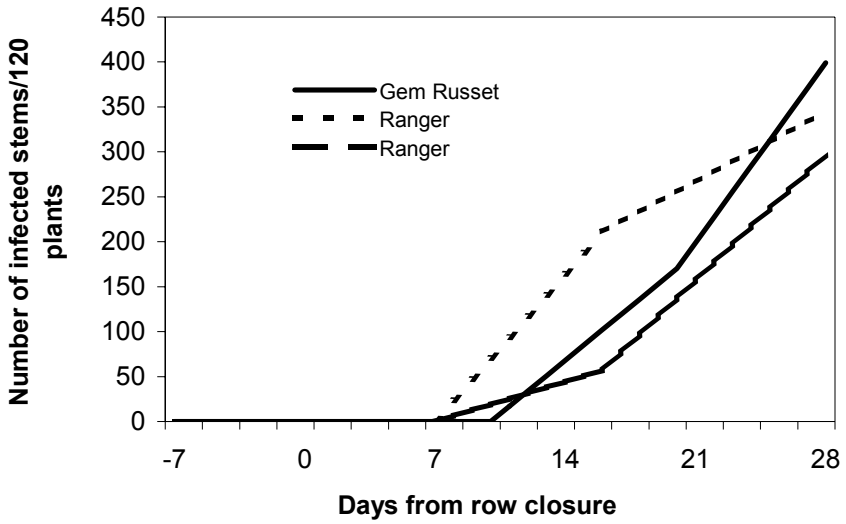

Figure 4. Disease progress curves of Sclerotinia stem rot in commercial fields of cvs. Gem Russet southwest of Pasco, and Ranger Russet north of Pasco. Initial full bloom occurred 10-14 days after row closure.

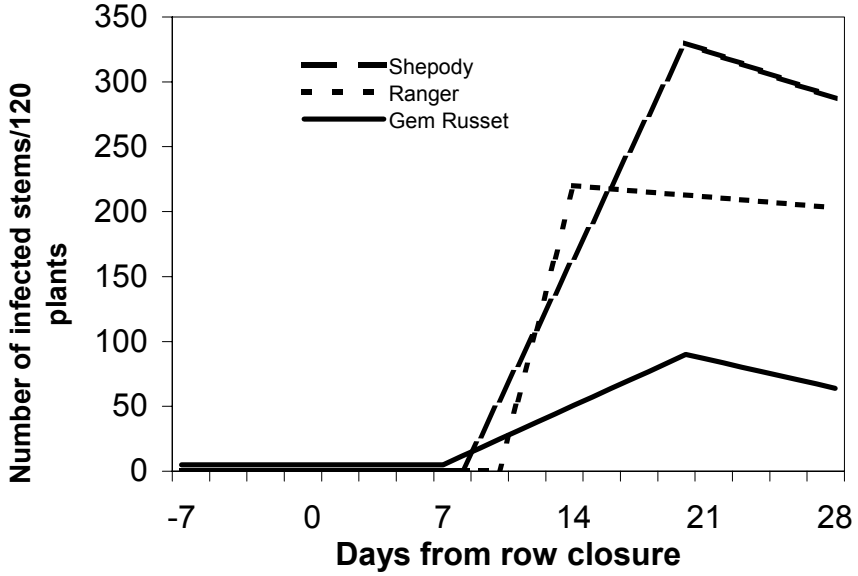

Figure 5. Disease progress curves of Sclerotinia stem rot in commercial fields north of Pasco planted with cvs. Shepody, Ranger Russet and Gem Russet. Initial full bloom occurred 10-14 days after row closure.

Table 1. Incidence of sclerotinia stem rot when Ranger Russet potatoes were treated with fungicides at initial full bloom in replicated plots in a commercial field by Pasco, Washington.

Treatment	Rate	Applications	Infected stems/30 ft.
Control			87 a
Rovral + oil	16 oz	1	33 b
Roval – high	32 oz	1	27 bc
Bas 510 low	0.286 lb	1	25 bc
Bas 510 med	0.357 lb	1	25 bc
Blossom Removal		3	25 bc
Topsin M	1.5 lb.	2	25 bc
Rovral + oil	16 oz.	2	23 bc
Omega	0.25 lb*	1	16 cd
Bas 510 (Endura)	0.429 lb	1	12 d

*ai/acre. Others are product/acre.