Timing Fungicide Applications for Managing Sclerotinia Stem Rot of Potato

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Sclerotinia sclerotiorum, the cause of Sclerotinia stem rot or white mold on potato infects more than 400 species in 75 plant families. In the Columbia Basin common cultivated crops susceptible to *S. sclerotiorum* include potato, bean, pea, carrot, cucurbits, and *Brassica* spp. . *Sclerotinia sclerotiorum* in the Columbia Basin shows a high level of genetic diversity, but isolates did not differ in sensitivity to registered fungicides (4). Increased disease incidences have been associated with cultivar architecture and morphology, high crop density, close row width, continuous plant surface wetness and excess nitrogen fertilization in potato and other crops (5, 6, 9, 10).

Prior to 2003, Sclerotinia stem rot was not satisfactorily managed in potatoes in the Columbia Basin of Washington and Oregon. Frequent sprinkler irrigation from center-pivot irrigation systems, high plant density, and dense crop canopies promoted prolonged plant surface wetness and high humidity and subsequent disease development. One to three fungicide applications were usually made in an attempt to control Sclerotinia stem rot on potato. The fungicides dichloran, iprodione, and quintozene (pentachloronitrobenzene) were registered and used. Initial application was made just prior to or at row closure according to labeled instructions and recommendations from Extension. Applications prior to row closure were made to achieve stem coverage within the potato canopy. Control was often inadequate.

Ascospores of *S. sclerotiorum* are incapable of direct infection of intact green leaves and stem tissues of potato and other crops, but they colonize flowers and other senescing tissues using them as energy sources to infect green tissues (2, 3). The importance of potato flower blossoms as a bridge for infection into potato stems was recently demonstrated (3). Airborne ascospores are deposited on open potato blossoms attached to the canopy. Infested flowers fall on stems and the ground, and fungal mycelia rapidly colonize the blossoms when humidity is high in the plant canopy. Stems or leaves contacting colonized blossoms then acquire the pathogen. Ascospores were found to cause the vast majority of observed lesions as opposed to infections near the soil line initiated by mycelium from soil-borne sclerotia. Flower removal before blossom drop or fungicide application at full or 100 % bloom of primary flower clusters drastically reduced disease incidence (3).

A potato shoot is composed of a main stem and branches. It can produce one or more inflorescences over time. The inflorescence is a cyme and also referred to as a cluster. The main stem of a potato shoot terminates in an inflorescence, which is called the primary cluster. Shoot growth continues by apical branching, and each branch terminates in a cluster of the corresponding order. Inflorescences or clusters on higher orders of branching are younger and flower later (13).

Fungicide application at full bloom of primary flower clusters has not been directly compared to application at row closure for control of Sclerotinia stem rot on potato. The purpose of this study was to evaluate the effectiveness of fungicide applications for control of Sclerotinia stem rot when fungicides were applied at row closure, following fungicide label recommendation, and at full bloom of primary flower clusters.

Materials and Methods

Trials were conducted at two commercial potato fields on a sandy-loam north of Pasco WA in 2003 and 2004. Fields were 125 acres in size and irrigated with center pivot systems. Certified potato seed was planted at 23–25 cm spacing within rows and at 86 cm spacing between-rows in mid-March of each year. Cultivars were Shepody in 2003 and Ranger Russet in 2004. Shepody is a determinate cultivar and Ranger Russet is an indeterminate cultivar. Plot size was three rows (2.6 m) wide and 9.1 m long. The grower irrigated, managed weeds and insects, and cultured the crop according to standard commercial practices. The grower did not apply fungicides for control of Sclerotinia stem rot to the section of the field containing the plots. Fungicides thiophanate-methyl (Topsin M 70 WP) at 1.05 lb ai/a, fluazinam (Omega 500F) at 0.25 lb ai/a, and boscalid (Endura) at 0.3 lb ai/a were used. In 2003, single applications of thiophanate-methyl and fluazinam were made at row closure on 30 May, and single applications of thiophanate-methyl, fluazinam and boscalid were made at 10% bloom of primary flower cluster on 2 June, at 100% bloom of primary flower clusters on 5 June, and at 10% drop of primary flower clusters on 9 June (Table 1). In 2004, single applications of thiophanate-methyl, fluazinam and boscalid were made at row closure on 2 June, at 100% bloom of primary flower clusters on 8 June, and at 20% drop of primary flower clusters on 15 June. The fungicides and fungicide application timings were arranged in a randomized complete block design with four replicates. Fungicides were applied using a CO_2 pressurized sprayer at a rate of 30gal water/a at 210 kPa, with a ConeJet hollow cone nozzle.

Disease incidence was assessed in the middle row of each plot by counting the number of stems with lesions 22-23 days after the last fungicide application. Percentage of control was calculated as 100 – ((mean number of stems with lesions from fungicide treatment/mean number of stems with lesions from non-treated control) x 100).

Results

Initial disease symptoms on stems presumably from airborne inoculum became evident on 17 June 2003 and on 22 June in 2004. This was 17 days after row closure and 12 days after full bloom of primary flower clusters in 2003, and 20 days after closure and 17 days after full bloom of primary flower clusters in 2004.

Incidence of Sclerotinia stem rot was less ($P < 0.05$) when plots were treated at row closure with fluazinam than with thiophanate-methyl, and at 10% bloom of primary flower clusters with boscalid than with thiophanate-methyl in 2003 (Table 1). Disease incidence did not vary ($P > 0.05$) among the three fungicides when applied at 100% bloom of primary flower clusters in 2003 (Table 1) or when applied at row closure, 100% bloom of primary flower cluster, and 20% drop of primary flower clusters in 2004 (Table 2).

Incidence of Sclerotinia stem rot was significantly less both years when thiophanate-methyl, fluazinam, or boscalid were applied to potato foliage at full bloom of primary flower clusters than at row closure or not applied (control) (Tables 1 & 2). Mean percentage of control for the fungicides combined, relative to the non-treated control, was 43 and 48% in 2003 and 2004, respectively, when application was made at row closure; whereas, it was 77 and 83%, in 2003 and 2004, respectively, when application was at full bloom of primary flower clusters.
Mean disease incidence for the fungicides combined did not differ ($P > 0.05$) when applications were at 10% bloom, 100% bloom, and 10% drop of primary flower clusters in 2003 (Table 1), but disease incidence was higher ($P < 0.05$) when application was made at 20% drop than at 100% bloom of primary flower clusters in 2004 (Table 2)

Discussion

Control of Sclerotinia stem rot was significantly improved when fungicides were applied at full bloom of primary flower clusters compared to applications at row closure. Infested flower blossoms likely served as a bridge for infection and fungicides applied when flowers were exposed to air borne ascospores subsequently reduced infections on stems. Similar observations were made when bean flowers were protected with benomyl at full bloom, which prevented white mold development even under optimal environmental conditions (2, 12). Applications of benomyl made after full bloom on beans failed to provide effective stem rot control because flowers had already acquired the inoculum (8, 9)

A difference in application timing of a few days made a difference in degree of control. Fungicides applied at row closure did not cover the flower blossoms because blossoms were absent at that time, and fungicides applied at 20% blossom drop were too late to effectively prevent stem infections from infested blossoms. Data in 2003 indicated that the window for effective applications was somewhat wider than just at 100% bloom, but data in 2004 demonstrated that the window width truncated before 20% drop of primary flower clusters.

Fungicides applied at row closure are likely washed from stems and partially degraded before effective inoculum on colonized blossoms is present; whereas, applications at full bloom of primary flower clusters are made just before infection would otherwise occur from dropping contaminated blossoms. We have observed colonization periods (time from inoculation with colonized blossoms to initial stem lesions on potato stems) of three days in greenhouse experiments. This is consistent with the rapid development of lesions that is often noted soon after contaminated blossoms drop in a humid potato canopy. In light of the current findings, we recommend amending fungicide labels and disease management recommendations for control of Sclerotinia stem rot of potato to take into consideration the data presented in this study.

Development of initial lesions of *S. sclerotiorum* on potato stems relative to row closure and full bloom of primary flower clusters was the same as in a previous study (3). In both studies, stem rot initially appeared on potato stems 14 to 20 days after row closure and 5 to 7 days following blossom fall.

The fungicides used in this study, when applied at full bloom of primary flower clusters, did not vary in efficacy and all three effectively reduced Sclerotinia stem rot; whereas fluazinam was more effective than thiophanate-methyl in reducing number of stems with lesions when applied at row closure in 2003, and boscalid was more effective than thiophanate-methyl when applied at 10% bloom of primary flower clusters in 2003.

Sclerotia of *S. sclerotiorum* in soil can infect nearby stems by the soil surface before blossom fall, and foliar fungicides tested in this study would not be expected to prevent these infections. This is because the inoculum exists in or on the soil, and soil around the lower stems prevents contact of the fungicide with the stem.

Improvements in management of Sclerotinia stem rot occurred in commercial potato fields in the Columbia Basin during the 2003 and 2004 growing seasons when growers, based on our previous work (3), applied a fungicide at full bloom of primary flower clusters. These observations are in reference to seasons before 2003 when initial application was made at row closure.

One fungicide application was sufficient for satisfactory control when application was at full blossom of primary flower clusters in the 2003 and 2004 seasons; whereas, two and three applications often did not give satisfactory control when applications were initiated at row closure before 2003. The fungicide fluazinam was registered for the 2003 season and boscalid was registered for 2004 in the Columbia Basin and these products have aided in effectiveness of control. However, timing of fungicide application is particularly crucial for effective control.

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