A REVIEW OF THE EPIDEMIOLOGY OF SCLEROTINIA AND ATTEMPTS TO CONTROL IT IN POTATO [1/]

by
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SUMMARY

Results of our 3-year study indicate that Sclerotinia stem rot caused by Sclerotinia sclerotiorum is not an economically important potato disease and warrants little consideration for control. Wilt and death of plants in the field studied were caused mainly by Verticillium wilt. Very few plants and only a moderate amount of stems were killed by S. sclerotiorum. Foliage fungicides applied with a ground sprayer 8 times at 10-day intervals on non-fumigated plots or 6 times on plots fumigated for Verticillium wilt control failed to reduce Sclerotinia stem rot.

A review of literature indicates that chemical control of Sclerotinia diseases has never been very effective. Practices that either destroy the resting sclerotia or limit the environment favorable for infection would be more effective and less expensive. Sclerotia of S. sclerotiorum left on the soil surface with no tilling will be killed by wetting and drying and freezing over winter and inoculum levels will be effectively reduced the following year. Lengthening the period between irrigations and reducing irrigations as soon as Sclerotinia white mold or stem rot is observed will greatly reduce further infections. Infections and further damage will cease when the foliage remains dry 48 hours or longer. Crop rotation is of little value since sclerotia remain viable for at least 3 years and new sclerotia can form from old sclerotia in the soil. A foliar fungicide program is not recommended.

INTRODUCTION

Sclerotinia sclerotiorum attacks more than 360 species of plants in 64 families (45). It has been reported on potato in Florida (7, 16, 30), Idaho (J.R. Davis, personal communication), Maine (10), New York (35, 38), and Oregon (J. C. Zalenski, personal communication), in the U.S.A. and Britain (28), Canada (23) and Ireland (20, 28, 39). Potatoes rarely were affected by this pathogen until the 1950's (52). Sprinkler irrigation (42) intensifies the disease by providing the longer wet periods needed for infection. Yields and grades of potatoes were reduced by Sclerotinia stem rot in Florida (7) and New York (38) when early infections girdled and killed stems. Losses in western Ireland due to Sclerotinia stem rot were exceeded only by late blight (28).

We have observed a few plants infected with S. sclerotiorum in about every rill or sprinkler irrigated field examined in Washington since 1963. One field near Ruff, Wa. had considerable Sclerotinia in 1965. Since 1970 with increased irrigation by center pivot systems, increasing amounts of Sclerotinia have been reported by local growers throughout the Columbia Basin.

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S. sclerotiorum is a natural soil inhabitant in Washington and Sclerotinia stem rot develops even in the first cropping to potatoes in isolated areas. We observed Sclerotinia stem rot in June 8 miles from any other potato field on a Norgold Russet plant in a field being cropped to potatoes for the first time after 30 years of dryland wheat. About 30 native plants in desert areas and weeds associated with farm crops in the Columbia Basin are hosts of S. sclerotiorum (J. C. Zalenski, personal communication). These hosts apparently provide the initial inoculum for potato.

Whether Sclerotinia stem rot is of any economic importance has not been determined in Washington. Except for an occasional severe early June infection where many stems are girdled, usually only branches or at most a few main stems are killed by this organism. Wilting and early dying due to Verticillium wilt usually occurs at the same time Sclerotinia stem rot appears. Early dying is too often attributed to the Sclerotinia organism.

This paper will review the literature on the epidemiology of Sclerotinia stem rot and report our attempts at controlling this disease. Most work with S. sclerotiorum by other scientists has involved beans and lettuce so much of the reviews will relate to work with these crops.

EPIDEMIOLOGY

Source of Inoculum

Function and survival of sclerotia - S. sclerotiorum survives between crops as sclerotia. Sclerotia occur inside stems as hard flakes with a black outer rind, are easily seen, are variable in shape, and have dimensions up to .8 inch (45). Sclerotia form in dry, diseased potato stems and are scattered on the soil surface at harvest. Newly formed sclerotia must be preconditioned by high moisture and cool soil temperatures before they will germinate (6, 11). Continuous moisture for about 10 days and temperatures around 60°F are required for sclerotial germination. Sclerotia germinate and produce 1 or more slender, black thread-like stipes (stalks) which push through the soil surface and develop into beige, flat to slightly dished, thin, button-like sexual structures called apothecia (Fig. 1). Only sclerotia near the soil surface produce apothecia (4). Apothecia from sclerotia did not develop at a low 41°F or a high 86°F air temperature (1, 38). Even slight changes in moisture tension may prevent apothecial formation (17). Extreme drying (43) or freezing (38) of sclerotia will prevent germination. Therefore, many sclerotia on the surface of non-tilled soil surfaces will be killed by alternating drying, wetting and freezing. Alternate flooding and drying soils destroyed sclerotia in Florida (29). Dried sclerotia when rewetted leak nutrients and become parasitized by various microorganisms (27, 43, 47). Electron microphotographs of sclerotia buried deep in soil for long periods showed perforations in the rinds where microorganisms enter and multiply (27). The entire sclerotium must be destroyed, however, because any intact part can still germinate a stipe (21). Many weed hosts also may become infected and produce sclerotia (31). Sclerotia can be distributed by irrigation or run-off water (46, 48). Furthermore, sclerotia outside bean or potato fields produce apothecia and spores which can be spread by wind and water (46). Numerous sclerotia produce apothecia in irrigated winter wheat fields, especially if the field was previously cropped to potato. Even a 3-year cropping to crops other than bean did not reduce the sclerotial population in soil (47), possibly because new sclerotia can form from old sclerotia (5, 12).

Mycelium from sclerotia on the soil surface or aerial ascospores can infect dead bean blossoms (1, 11, 17, 40) and probably also old yellow and dying potato leaves in contact with sclerotia. Yellow and dying leaves occur on plants excessively shaded and overcrowded, or with depleted fertility but most frequently on plants affected by Verticillium wilt.

Production, dispersal and survival of the ascospore - Ascospores are sexual spores formed within apothecia above ground level. They require light for their development (26). Ascospores are forcibly discharged; shooting from .5 to 3 inches from apothecium when

subjected to slight moisture tensions (1, 2). The height of discharge enables ascospores to reach turbulent air and their dispersal may reach a few feet to several miles (9, 49, 50). A single apothecium may produce about 2.3 million ascospores (48). A mucilaginous material on the ascospores can cement them to host tissues encountered during flight (1).

Ascospores deposited on host tissues need not infect until wet conditions and a food source (dead leaves and flower parts) required for infection become available (1, 11, 17, 40). Under laboratory conditions, ascospores survived for 21 days at 7% relative humidity (RH) but less than 5 days at 100% RH (1, 38). Under field conditions, ascospores survived 12 days (17) and possibly for even 5 to 6 weeks (30).

The Infection Process and Disease Development

Mycelium arising from wind disseminated ascospores alone or growing on dead blossoms and leaves will not penetrate potato stem tissue unless certain conditions are met. Free moisture on leaves and stems is required for 48 to 72 hours before infection can occur (4). High humidity, even near 100% RH, is not sufficient for lesion initiation (4). Sclerotinia sclerotiorum white mold was more severe on varieties of bean with a dense canopy where free water due to rain or irrigation was retained longer than in varieties producing a less dense canopy (6, 13, 18, 34). Infections occur throughout the growing season on potato. Occasionally in June spores infect stem axils near the soil line and stems are girdled. Usually initial infections occur later on branches touching soil and only branches are pruned off the plant. Infections become more frequent in August as the density of the canopy increases and the period of wetness under the canopy becomes long enough for infection. Increased severity of the disease in August appears to be correlated with the rapid increase of secondary inoculum from infected lower, dying leaves which fall on and infect healthy green stem branches. Whether the initial infections of the lower, dying potato leaves are by ascospores, mycelium from secondary infections or mycelium directly from sclerotia on the soil surface has not been determined. However, on the basis of studies outside Washington ascospores are believed to be the principal inoculum (1, 2, 33, 34).

Infection hypha from germinating ascospores or mycelium on leaf surfaces form an appresorium (14, 40) or "infection cushion" (24). Direct penetration of host cuticle apparently occurs by mechanical pressure (3, 8) and by infection pegs from appresorium. Penetration requires about 24 hours (2). A vesicle is produced under the cuticle layer which then produces internal infection hyphae that spread into adjoining cells (2, 24). Host nutrition apparently plays a role in how rapidly they ramify within the host. Eventually hyphal tufts emerge from bean leaf stomata and form mycelial mats which develop into external sclerotia, whereas, in potato sclerotia are formed within infected stems.

In moist conditions such as under a dense plant canopy, initial infections produce water soaked lesions (4). These lesions enlarge within a few days and become covered with a dense cottony mycelial mat. Secondary infections may occur under these favorable conditions. Lesion expansion ceases and mycelial mats disappear if rains or irrigations discontinue, but growth will continue the next wet period. Infected potato stems and branches wilt rapidly when girdled by the fungus and soon die. Dry, dead stems have a white, papery look. They become hollow and rattle when shaken due to loose internal sclerotia. When walked upon, infected stems make a snapping sound as they are crushed. Sclerotia fall on the soil during harvest and provide for future propagation.

CONTROL

Cultural control - Attempts to control diseases caused by Sclerotinia sp. have not been very successful because of the aggressive pathogenicity of this fungus and the ability of its sclerotia to withstand adverse conditions (47).

Crop rotation has been advocated, however, a 3-year rotation to crops other than bean was not an effective control of white mold because sclerotia survived for 3 years or

more (12, 47). Leaving sclerotia on the soil surface without tillage or cover crop to dry (43) or freeze (38) may reduce yearly inoculum but soil erosion under these conditions could be a problem. Reducing numbers of irrigations, especially at the end of the growing season, reduced white mold on bean (47) and would reduce Sclerotinia stem rot of potato also but may reduce yield and grade. Efforts to reduce sclerotial inoculum within a field will be nullified if surrounding fields contain sclerotia or apothecia (9, 49, 50).

Chemical control - Foliar protectant sprays such as benomyl = Benlate® (methyl 1-(butylcarbamoyl) -2benzimi-dazolcarbamates), PCNB = Teraclor® (penta chloronitrobenzene), Botran® (2, 6-dichloro-4-nitroaniline) have been partially effective in controlling lettuce drop (25). Benomyl control of white mold of beans was successful by spraying blossoms (19). However if all above ground parts but the blossoms were sprayed with benomyl there was no control. In 1977 Botran was cleared for use by our State Department of Agriculture for control of Sclerotinia stem rot through sprinkler irrigation systems. Du-ter®, triphenylytin hydroxide, an early and late blight fungicide, has also been tried in the same way. Benomyl, although not registered on potatoes, has been considered for control. Rates of these three chemicals applied through a center pivot irrigation system would be diluted in 3, 000 to over 10, 000 gal of water per acre depending upon the water psi and speed of the center pivot. Unfortunately Easton (15) showed that these chemicals would not kill S. sclerotiorum mycelium or stop sclerotial formation if they were diluted in more than 500 to 1, 000 gal of water per acre.

Soil fumigation apparently inhibited sclerotial germination of S. sclerotiorum (22, 27, 38). Sub-lethal doses of soil fumigants are known to interfere with the defense mechanisms of resting structures of fungi (36). However, fumigation with a chloropicrin-methyl bromide mixture (66%:33% w/w) did not reduce viability of sclerotia in soil (27). Fumigation did increase the presence of Trichoderma spp. 2- to 5-fold because they have greater tolerance to soil fumigants than most other soil organisms (32). Trichoderma spp. are reported to colonize mycelium of S. schlerotiorum in vitro and degrade sclerotia; however, this degradation was very gradual and required more than 200 days (27). Lettuce drop caused by S sclerotiorum was actually increased in fumigated soils (37). In laboratory tests fumigation with DD® (1, 3-dichloro-propene; 3, 3-dichloropropene, 1, 2-dichloro-propane, 2, 3-dichloropropene and related C 3 hydrocarbons) increased stipe production from sclerotia in direct proportion to the rate of fumigant, which probably explains the increase in lettuce drop. A Vapam® (sodium-N-monomethyldithiocarbamatedihydrate) drench, 40 gal per 1/2 acre inch of water, killed sclerotia in the laboratory (38), but no studies have reported its control in the field.

We began work in 1977 to determine if Sclerotinia stem rot of potato is an economically important disease of potato. Various experimental and registered foliage fungicides were evaluated for control of S. sclerotiorum. Some of them are registered and recommended for use but have not been properly evaluated in Washington. Soil fumigation for control of Verticillium wilt was included in our third year of study since it was present and appared to influence Sclerotinia stem rot.

MATERIALS AND METHODS

Experimental plots were located near Paterson, Wa. on four-125 acre fields where Sclerotinia stem rot has become an annual problem. The soil was a Hezel sand (41) which had raised two or more crops of potato under center pivot irrigation. These studies were conducted on the potato cultivar Russet Burbank.

In 1977 the fungicides Benlate, Benlate (slow release), Botran and Terraclor were sprayed on the soil surface and the lower one-third of the foliage with a PTO tractor sprayer with 24 ft boom. Polymer for the Benlate slow release chemical was furnished by G. Graham Allan, University of Washington. Plots, 30 ft by 30 ft in the experiment were sprayed on June 13 and June 23 prior to close of vines (Table 1).

In 1978 Du-ter, experimental fungicide DPX-4424, Benlate, Benlate (slow release) and Botran were sprayed on potato foliage twice and 8 times during the season by two men carrying a gasoline-engine-powered sprayer with a 9 ft boom. Plot size was 11.3 ft wide by 30 ft long (Table 2).

In 1979 Botran was applied 6 times by the same hand-carried sprayer but with a boom 24 ft long. Plot size was 40 ft wide by 30 ft long. Botran was applied on foliage either alone or in combination with soil fumigated with Telone C-17® (1,3-dichloropropene) or MC-33® (methylbromide plus chloropicrin) for control of Verticillium wilt on 2 farms (Table 3). The two fumigants were also applied without Botran. Telone C-17 was injected preplant by chisels spaced 9 inches apart and 9 inches in depth into soil previously chiseled once 12 inches in depth followed by rototilling 6 to 8 inches in depth. The soil was packed by rolling immediately following injection of fumigant. MC-33 was injected under black plastic tarps 17 ft^2 on March 14, 1979. Four tarps were used for each treatment site so they covered an area 34 ft^2. The tarps were buried in trenches 10 inches in depth to prevent escape of fumigant. The tarps were removed 6 days later and the field was not planted for about 2 weeks to allow escape of the toxic chemical.

A hygrothermograph (Cat. No. 5-594, Belfort Instrument Co., Baltimore, Md.) was located in a shelter under potato foliage to record continuous temperatures and relative humidities (RH). A catch bottle attached to a 2-3/4 inch diameter funnel was placed on a 4 ft stake near the hygrothermograph to collect irrigation water for calculating the amounts of water applied.

During all 3 years of experiments all spray and soil fumigation treatments were randomly located and replicated 6 times. The fields were planted in April. A 20 ft row was harvested from each treatment in September to determine yield and grade. Wilted or dead plants due to Verticillium wilt and stems with S. sclerotiorum were recorded in September.

RESULTS

Sclerotinia stem rot appears on a few plants the last of July or first of August. Incidence increased as lower leaves yellowed, died and fell on or near branches lying on the soil. Most plants by the last of August were severely wilted or dead and the plant canopy opened due to Verticillium wilt. Some Sclerotinia stem rot occurred during all 3 years, but only a few main stems were killed (Tables 1, 2, and 3). As the air temperature increased from mid-July to mid-August the hours over 90% RH decreased (Fig. 2, 1977; data for 1978 and 1979 not shown). The RH began to increase as temperature decreased after mid-August, but decreased again as plants wilted and died due to Verticillium wilt. Irrigation rates didn't appear excessive even during the period of stem infection by S. sclerotiorum (Fig. 2).

Benlate, Benlate (slow release), Botran, DPX-4424, Du-ter and Terraclor applied twice and 8 times at 10 to 14 day intervals did not lessen the amount of Sclerotinia stem rot nor increase yield and grade (Tables 1, 2, and 3). Botran sprayed on foliage of plots fumigated to reduce Verticillium wilt did not less Sclerotinia stem rot (Table 3). The combination treatment of Botran foliage sprays and tarped fumigation with MC-33 increased yield on Farm #1 but not on Farm #2. Fumigation with MC-33 but not Telone C-17, delayed Verticillium wilt on both farms. The combination treatment of Botran and MC-33 fumigation reduced U.S. No. 1 tubers on Farm #2, probably due to reduced stands caused by toxicity of MC-33 remaining in soil at planting.

Discussion on Control

We found a number of branches but only a few main stems girdled by Sclerotinia stem rot (Tables 1, 2, and 3). Fallen, dead leaves were often colonized with S. sclerotiorum and even though they serve as a food base for the infection process they do not cause any real loss. Simulated hail studies have shown that Russet Burbank can lose 25% of its foliage, especially

after blooming, with little or no reduction in yield (44, 51). Therefore the branches killed by Sclerotinia stem rot in our study probably would not be expected to result in reductions in yield or grade.

Verticillium wilt caused by Verticillium albo-atrum, microsclerotial type, (G. D. Easton, unpublished data) was the primary causal factor for the wilted and dead plants in these fields. Verticillium wilt killed plants, removed the plant canopy and lessened the time that the free water was present which is required for infections by S. sclerotiorum.

Even 8 ground sprayings of fungicides on foliage at 10-day intervals did not reduce the the amount of Sclerotinia stem rot (Table 2). It should be noted, however, that the lack of any effect by fungicide sprays might be due to extenuating circumstances. Sprinkler irrigation might have washed the fungicides off the foliage so they became ineffective. Perhaps so little injury was caused by Sclerotinia stem rot that the effects of controlling the disease were minimal. Also, the inoculum (wind blown ascospores) from outside the treated plots could have rapidly reexposed the plots. Outside inoculum has been considered a factor affecting chemical control of even large fields (1). However, if inoculum from adjoining lands were a factor in our studies, then more disease should have occurred in our untreated control plots.

Suggestions for Reducing Sclerotinia Stem Rot

1. Allow sclerotia from an infected crop to remain on soil surface so they can be killed by drying (43) and wetting and freezing before tilling of soil (38). This practice is probably not practical in light textured soils due to wind and water erosion where cover crops are not present.

2. Watch field closely in June and if early infections occur, reduce irrigation to allow drying under the canopy.

3. After the canopy covers the ground apply larger quantities of water but less often to reduce 48 hour periods of free water needed for infection to occur (4, 11).

4. Reduce irrigation rates at first indication of a white mold on fallen, dead leaves.

5. Application of fungicides to foliage is not recommended.

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Table 1. Effect of fungicides on Sclerotinia control, plant death and production of Russet Burbank potato near Paterson, Wa. in 1977.

Fungicide[1/]	Rate/a	Number Plants With Sclerotinia[2,3/] (Sept. 7)	Cwt/a[3/]	Percent U.S. No. 1 Tubers[3/]
Benlate 50 WP[4/]	1 lb a.i.	13	359	50
Benlate 50 WP[4/] (slow release)	1 lb a.i.	11	327	48
Botran 75W[4/]	6 lb a.i.	15	335	65
Terraclor 2EC	6 lb a.i.	14	302	48
Not treated	—	14	339	50

[1/] Sprayed on lower 1/3 of plant and soil prior to vine close on June 13 and at cultivation lay-by on June 30 at 20 psi in 30 gal fungicide-water solution per acre.

[2/] Number of dead plants per 20 ft of row with paperous white stems and sclerotia of Sclerotinia sclerotiorum. Death presumably due to Verticillium wilt.

[3/] Data not significant at P = .05 according to F test.

[4/] Spreader activator (X-77) at 5 ml/gal was added.

Figure 1. Life Cycle of Sclerotinia sclerotiorum on potato.

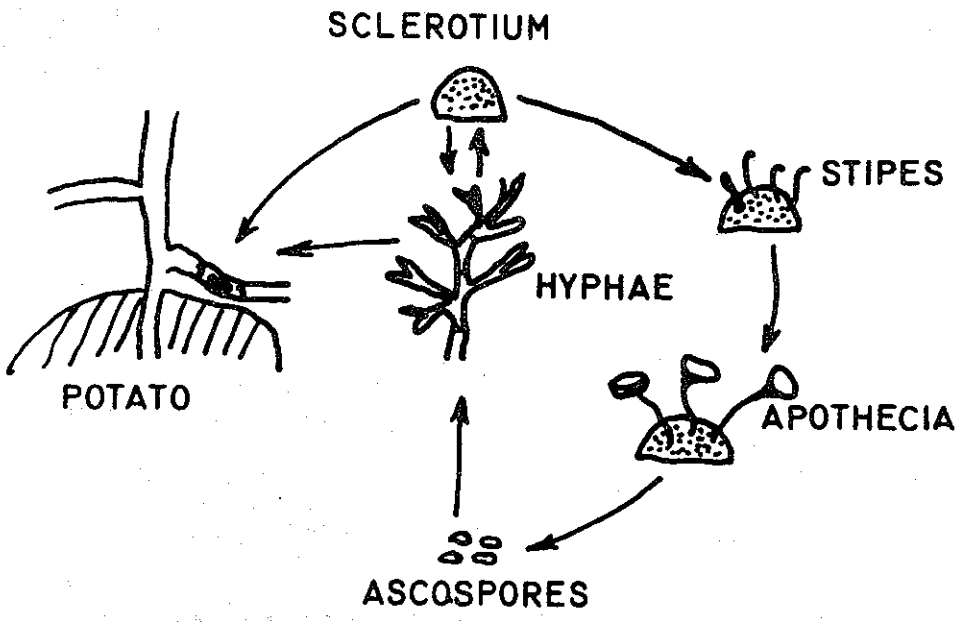

Table 2. Effect of foliage applications of chemicals on Sclerotinia control, Verticillium wilt, and production of Russet Burbank potato near Paterson, Wa. in 1978.

Chemicals	Rate/a	Number of Sprayings	Wilted and Dead Plants (Aug 21)	Dead Plants (Sept. 6) Without Sclerotinia sclerotia[1]	Dead Plants (Sept. 6) Stems With Sclerotinia sclerotia[1]	Yield Cwt/a[2]	Percent U.S. No. 1 Tubers[2]
Benlate 50 WP	1.5 lb	2[3]	14 a[5]	15 a	0	448	51
Benlate 50 WP	1.5 lb	8[4]	12 a	14 a	0	411	52
Benlate 50 WP (slow release)	1.5 lb	2	14 a	16 a	0	481	50
" "	1.5 lb	8	14 a	14 a	0	476	50
Botran 75W	2.0 lb	2	11 ab	15 a	0	413	43
Botran 75W	2.0 lb	8	17 a	15 a	.1	433	43
DPX-4424 50 WP	1.5 lb	2	7 c	11 bc	0	417	58
DPX-4424 50 WP	1.5 lb	8	16 a	17 a	0	401	49
DPX-4424 50 WP	3.0 lb	2	12 a	15 a	0	381	58
Du-ter 47.5 WP	10 oz	8	10 b	8 c	3	433	55
Not treated	-	-	15 a	16 a	0	440	52

[1]Number from approximately 24 plants per plot.

[2]Data not significant at P = .05 according to F test.

[3]Sprayed foliage on June 12 near vine close and on June 22 at 20 psi in 50 gal of fungicide-water solution/a.

[4]Sprayed on foliage on June 12, 22, July 5, 13 and 24, August 2, 14 and 24.

[5]Vertical means followed by the same letter of the alphabet are not significantly different according to Duncan's Multiple Range Test at P = .05.

Figure 2. Mean inches of irrigation applied weekly and mean temperature and hours over 90% relative humidity (RH) underneath potato vines in center pivot irrigated field near Paterson, Wa.in 1977. Sclerotinia stem rot appeared the first week of August.

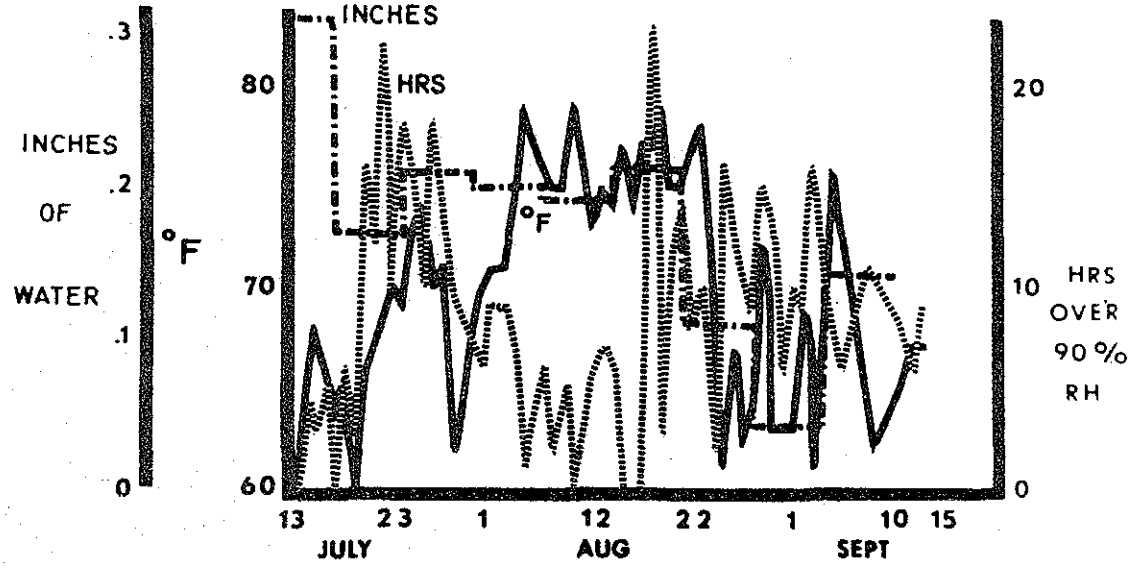

Table 3. Effect of foliage sprays of Botran alone and with soil fumigation on Sclerotinia control, Verticillium wilt control and production of Russet Burbank potato on farms near Paterson, Wa. in 1979.

Treatment	Rate/a	Number Wilted Plants (Sept. 5)[1]		Stems W/ Sclerotinia sclerotia (Sept. 17)[1]		Percent U.S. No. 1 Tubers		Yield Cwt/a	
		#1[2]	#2[2]	#1	#2	#1	#2	#1	#2
Botran	14 lb[3]	24 a[6]	22 a[6]	0 b[6]	1[7]	49[7]	44 c[6]	392 b[6]	484[7]
MC-33	960 lb[4]	19 b	11 b	3 a	3	41	48 ab	446 b	561
Telone C-17	25 gal[5]	22 a	21 a	2 ab	4	40	51 ab	408 b	538
Botran + MC-33	2 lb + 960 lb	16 c	12 b	3 a	.5	40	39 d	516 a	561
Botran + Telone C-17	2 lb + 25 gal	23 a	21 a	2 ab	2	45	35 e	431 b	554
Not Treated	-	23 a	22 a	1 b	2	45	47 bc	431 b	492

[1]Number per 20 ft row per plot.

[2]Farm near Paterson, WA.

[3]Sprayed foliage with 4 lb actual/a on July 2 and with 2 lb actual/a on July 19 and 30, August 9, 20, and 27 at 20 psi in 50 gal of fungicide-water solution/a.

[4]Injected under 4 tarps 17 ft^2 on March 14.

[5]Injected into soil through shanks 9 inches in depth and 9 inches apart on March 12 followed by immediate packing.

[6]Vertical means followed by the same letter of the alphabet are not significantly different according to Duncan's Multiple Range Test at P = .05.

[7]Vertical means not significant at P = .05 according to F test.