Relationship between Silver Scurf Levels on Seed and Progeny Tubers from Successive Generations of Potato Seed

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ABSTRACT

The level of silver scurf on potato seed tubers on successive generations of potato seed tubers and their progeny tubers was investigated during 3 years in the field. The objective was to determine the importance of seed-borne inoculum on silver scurf development on the subsequent progeny tubers. Silver scurf incidence and severity increased with each generation. Coefficients of determination for disease levels among generations were significant and ranged from 0.89 to 0.97, indicating that seed tuber source accounted for a large proportion of silver scurf on progeny seed tubers. Incidence and severity of silver scurf also increased with decreasing time periods between potato crops in the field. In a field near Paterson, WA, where potatoes had not been previously grown, the severity of silver scurf increased on progeny tubers of cvs Russet Norkotah, Ranger Russet, and Shepody as disease severity increased on seed tubers of successive generations. Disease severity index significantly increased as disease incidence increased. The relationship between the two was best described using a curvilinear regression model.

RESUMEN

Durante tres años se investigó en el campo el nivel de costra plateada en tubérculos de semilla de papa, en generaciones sucesivas de tubérculos semilla y de su progenie. El objetivo fue determinar la importancia del inóculo trasmitido por la semilla en el desarrollo de costra plateada sobre los siguientes tubérculos progenie. La incidencia y severidad de la enfermedad aumentó en cada generación. Los coeficientes de determinación para los niveles de enfermedad entre generaciones fueron significativos y alcanzaron de 0.89 a 0.97, lo cual indica que la semilla fue la responsable de una gran proporción de costra plateada sobre la progenie de los tubérculos semilla. La incidencia y severidad de costra plateada también se incrementó con menores períodos de tiempo entre cultivos de papa en el campo. En un campo cerca de Paterson, WA, donde no se había sembrado papa anteriormente, la severidad de costra plateada se incrementó en los tubérculos progenie de Russet Norkotah, Ranger Russet y Shepody a medida que la severidad aumentó sobre los tubérculos semilla de generaciones sucesivas. El índice de severidad de la enfermedad se incrementó significativamente a medida que la incidencia aumentó. La relación entre los dos fue mejor descrita utilizando un modelo de regresión curvilínea.

INTRODUCTION

Silver scurf, caused by *Helminthosporium solani* Durieu & Montagne, is an increasingly important potato disease in many potato production areas because of the occurrence of fungicide-resistant strains, increased disease severity, and increased financial losses from blemished tubers (Bain et al. 1996; Hide and Hall 1993; Kawchuk et al. 1994; Merida and Loria 1994). Blemishes produced by *Helminthosporium solani* consist of buff or brown lesions that develop on tuber periderm and become silvery in appearance when wet. Severe infections obscure periderm pigmentation of red- or white-colored cultivars (Jellis and Taylor 1977). Increased water loss through infected areas can result in up to 13% less yield of marketable potatoes, which affect grower returns whether or

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not the tubers are sold to processors or fresh markets (Jellis and Taylor 1977; Lennard 1980; Read and Hide 1984). *Helminthosporium solani* lesions are particularly important on potato tubers sold on the fresh market because of low market tolerance for cosmetic blemishes (Goth and Webb 1983; Jellis and Taylor 1977; Merida and Loria 1994; Rodriguez et al. 1995).

Silver scurf is seed-tuber-borne, but the importance of this inoculum source in disease development in the field is not fully understood and is often overlooked. Evidence that seed tubers are an important source of inoculum comes from experiments showing the effectiveness of seed tuber fungicides in reducing silver scurf (Cayley et al. 1983; Denner et al. 1998; Frazier et al. 1998) and from the location of lesions on progeny tubers taken directly from the soil. Initial infections occur at the stem end of the progeny tuber, in the region around the stolon but not always adjacent to it (Jellis and Taylor 1977; Zimmerman-Gries and Blodgett 1974). Disease-free seed tubers typically produce disease-free progeny tubers (Zimmerman-Gries and Blodgett 1974).

Relationships between silver scurf severity on the seed and disease severity on the progeny are not always consistent (Jellis and Taylor 1977; Kawchuk et al. 1994; Mooi 1959). Seed tubers of cvs King Edward and Ulster Sceptre that were severely, moderately, and slightly infected with silver scurf were tested to determine the resulting levels of infection of progeny tubers (Read and Hide 1984). Progeny tubers produced from severely infected seed tubers had less infection than tubers from moderately to slightly infected seed. Severely infected seed tubers had older lesions, which produced fewer spores than the younger lesions on the moderate or slightly infected seed (Jellis and Taylor 1977; Read and Hide 1984). Consequently, progeny tubers from moderately infected seed had the highest infection (Read and Hide 1984). Others have reported no significant differences in disease severity on progeny tubers from slightly, moderately, and severely infected seed tubers (Zimmerman-Gries and Blodgett 1974). Such conflicting results suggest that the relationship between the amounts of *H. solani* on seed- and progeny tubers is complex.

Soil-borne inoculum of *H. solani* was not considered a viable source of initial infection until recently (Bain et al. 1996; Firman and Allen 1995; Jellis and Taylor 1977), when Merida and Loria (1994) observed that *H. solani* survived saprophytically on many crops grown in rotation with potatoes. To avoid infection from seed tubers, previous studies have produced seed tubers from tissue culture plantlets or stem cuttings, thus eliminating the possibility of contamination from the seed tuber (Jellis and Taylor 1977; Merida and Loria 1994). These findings support and help to explain how fields where potatoes had never been grown, or were grown 1, 2, 3, or 4 years previously could produce potato crops with significant incidences of silver scurf even when the seed or stem cuttings used were free of disease (Bain et al. 1996).

Efforts to manage silver scurf are hampered because the method of spread of the pathogen and the relationship between silver scurf on seed and progeny tubers is unclear. Cultural practices, including planting pathogen-free seedpieces, may be important in minimizing disease severity, but these practices need to be commercially practical. The purposes of this study were to determine the relative importance of tuber-borne inoculum of *H. solani* on the development of silver scurf on progeny tubers and to quantify the relationship between disease incidence and disease severity indices for potato silver scurf.

MATERIALS AND METHODS

Tuber Assay for Silver Scurf

Seed tubers were evaluated for silver scurf prior to planting, as were the progeny tubers following harvest and after storage. Tubers were first washed with running water and soaked for 10 min in 0.05% sodium hypochlorite (NaOCl) at 70 C. Tubers were then assessed for the percentage of silver scurf and divided into one of six disease severity classes: 0 = tubers with no silver scurf symptoms; 1 = 1%-5%; 2 = 6%-25%; 3 = 26%-50%; 4 = 51%-75%; and 5 = 76%-100% surface area covered with visible lesions. These data were then used to calculate a disease severity index from the formula: DSI Σ (disease severity class $\times$ number of tubers in that class) $\times$ 100 / total number of tubers $\times$ 5 (Sherwood and Hagedorn 1958). DSI varies from 0 to 100, and a disease index of 0 means that all tubers were disease free, 100 means that all tubers were in the most severe class. Disease incidence was determined as the percentage of all tubers inspected that showed some incidence of silver scurf.

Levels of Silver Scurf in Successive Generations

Incidence and severity of silver scurf in successive generations (nuclear through generation 3) seed tubers were determined based on samples collected from four growers in Montana. All growers indicated that previous generations of

seed were used to grow the seed for the next generation, thus successive generations, and all seed tubers were grown without the use of seed treatments or at-plant fungicides. Three samples of successive seed tuber generations were collected in February of 1998 and four in 1999, and represented seed for those respective growing years. Four samples of seed tubers were also collected in October of 1999 and represented seed tubers that were planted in the spring of 2000. Growers who produced the seed tubers were identified by letters and labeled A-D.

Grower A provided successive generations of seed tubers for cv Russet Norkotah in 1998 and cvs Russet Norkotah and Shepody in 1999 and 2000. Grower B provided successive generations of Russet Burbank all 3 years. Grower C provided successive generations of Russet Burbank in 1998, but did not participate in the study in 1999 and 2000. Grower D did not participate in the study in 1998, but provided successive generations of seed tubers of Russet Burbank in 1999 and 2000. The following potato cultivars were studied each year: 1998—one successive generation of Russet Norkotah, and two of Russet Burbank; 1999—one successive generation of Russet Norkotah, one of Shepody, and two of Russet Burbank; 2000—one successive generation of Russet Norkotah, one of Shepody, and two of Russet Burbank. In 2000, grower B did not raise generation 3 Russet Burbank seed tubers because he sold all generation 2 seed to other growers. Table 2 identifies the growers, potato cultivar and generations that were used in each year of this study.

Fifty tubers from each generation of each sample of seed tubers taken in February of 1998 and 1999, and 25 from each sample taken in October of 1999 were evaluated for *H. solani*. Levels of silver scurf incidence and severity among successive generations of seed tubers were analyzed for each grower's seed sample (Table 1). The field history for each seed sample was also obtained from the seed grower (Table 2). Each grower stored nuclear and generation 1 seed tubers together in the same storage facility with a circulated air system. Generations 2 and 3 were stored together in another facility with circulated air. Generations stored in the same facility were physically separated by wood panels. All storages maintained 90%-95% humidity and a temperature of 3-4 C.

In 1999, seed samples of generations 1, 2, and 3 seed tubers were planted in split-plot designs with four replications near Othello and Paterson, WA. The plots at both locations consisted of single rows, 9.2 m long. Seed tubers were spaced 25 cm within the row and 86 cm between the rows. Seedpieces were hand cut to 57 g.

Three of four seed samples were planted at Othello because of a shortage of seed tubers. Samples planted at Othello were cultivars Russet Burbank, Russet Norkotah and Shepody, all from three different growers. The trial was planted in Shano silt loam on 21 April, defoliated 9 September, and harvested 23 September. The field had not been planted to potatoes the previous 4 years. Time period from planting to harvest was 155 days. All tubers were stored in mesh bags in a refrigerated cooler with a circulating air system. Humidity was maintained between 85%-95% and the temperature was 7-8.8 C.

Four seed samples of generation 1 to 3 seed of Russet Burbank (two sources), Russet Norkotah (one source), and Shepody (one source) were planted near Paterson in Hermiston sand on 6 April, defoliated 17 September, and harvested 5 October. The field had never been in potatoes prior to this study. Time period from planting to harvest was 182 days. Plants at both locations were desiccated with 1.77 L/ha of Diquat.

TABLE 1—*Incidence and disease severity index of silver scurf on successive generations of potato seed tubers in 1998, 1999 and 2000 and coefficients of determination for the relationship of level of silver scurf among the seed generation.*[a]

	1998		1999		2000[c]	
Seed Generation	Inc (%)	DSI	Inc (%)	DSI	Inc (%)	DSI
Nuclear	0.0	0.0	1.5	0.3	0.0	0.0
G1[d]	17.4	4.4	9.0	2.8	5.0	1.0
G2	45.0	16.4	25.7	8.1	13.0	3.2
G3	53.9	21.0	29.0	9.2	13.0	3.2
R^{2bc}	0.97*	0.96*	0.94*	0.95*	0.90*	0.89

[a]One seed lot of Russet Norkotah and two of Russet Burbank were assayed in 1998, and one seed lot of Russet Norkotah, two of Russet Burbank, and one Shepody were assayed in 1999 and in the fall of 1999 for those seed tubers that would be planted in 2000. Fifty tubers were assayed from each sample in 1998 and 1999, 25 tubers were assayed from the 2000 seed tuber sample. In all years, the five seed lots came from four different commercial potato seed growers.

[b]Coefficients of determination for the independent variable seed generation and the dependent variables incidence and disease severity. Coefficients followed by (*) indicates significance at $P < 0.05$.

[c]Seed tubers for the year 2000 were assayed at harvest in the fall of 1999, seed tubers for 1998 and 1999 were assayed after 5 months in storage.

[d]Generations of seed are G1, G2, and G3 = generation 1-3, respectively.

TABLE 2—*Incidence and disease severity index of silver scurf on successive generations of Russet Norkotah, Russet Burbank and Shepody potato seed from three growers in 1998, 1999 and 2000.*

1998						1999						2000		
Grower	Cultivar	Seed Generation[a]	Inc (%)	DSI	Rotation[b]	Grower	Cultivar	Seed Generation	Inc (%)	DSI	Rotation	Inc (%)	DSI	Rotation
A[c]	Norkotah	Nuclear	0.0	0.0	grnhouse[g]	A	Norkotah	Nuclear	0.0	0.0	grnhouse	*	*	grnhouse
A	Norkotah	G1	18.0	4.4	8 yr	A	Norkotah	G1	16.0	4.8	10 yr	8.0	1.6	10 yr
A	Norkotah	G2	20.0	6.0	6 yr	A	Norkotah	G2	40.0	13.6	virgin	24.0	6.4	virgin
A	Norkotah	G3	62.0	23.2	4 yr	A	Norkotah	G3	36.0	11.2	4 yr	24.0	7.2	4 yr
B[d]	Burbank	Nuclear	0.0	0.0	grnhouse	A	Shepody	Nuclear	0.0	0.0	grnhouse	*	*	grnhouse
B	Burbank	G1	10.0	2.4	virgin[h]	A	Shepody	G1	0.0	0.0	10 yr	4.0	0.8	10 yr
B	Burbank	G2	14.0	4.0	10 yr	A	Shepody	G2	28.0	8.0	virgin	16.0	4.0	virgin
B	Burbank	G3	16.0	4.4	4 yr	A	Shepody	G3	48.0	17.6	3 yr	16.0	3.2	3 yr
C[e]	Burbank	Nuclear	0.0	0.0	grnhouse	B	Burbank	Nuclear	0.0	0.0	grnhouse	*	*	grnhouse
C	Burbank	G1	15.6	4.4	virgin	B	Burbank	G1	0.0	0.0	10+ yr	0.0	0.0	10+ yr
C	Burbank	G2	70.0	28.4	4 yr	B	Burbank	G2	8.0	1.6	10+ yr	0.0	0.0	10+ yr
C	Burbank	G3	80.0	37.6	4 yr	B	Burbank	G3	4.0	0.8	10+ yr	*	*	10+ yr
*	*	*	*	*	*	D[f]	Burbank	Nuclear	5.9	1.2	grnhouse	*	*	grnhouse
*	*	*	*	*	*	D	Burbank	G1	20.0	6.4	5 yr	8.0	1.6	5 yr
*	*	*	*	*	*	D	Burbank	G2	26.9	9.2	6 yr	12.0	2.4	6 yr
*	*	*	*	*	*	D	Burbank	G3	28.0	7.2	5 yr	12.0	2.4	5 yr

*No data obtained.
[a]Nuclear and generation 1 stored in a separate building from generations 2 and 3. Seed generations within a building were stored in separate bins.
[b]Rotation is the number of years since potatoes last grown in a field.
[c]Grower A crop rotation of barley, wheat and alfalfa.
[d]Grower B crop rotation of barley, wheat and canola.
[e]Grower C crop rotation of barley, wheat and mint.
[f]Grower D crop rotation of wheat, alfalfa and barley.
[g]Seed tubers grown in a greenhouse were planted from tissue cultured plants and grown in sterile soil.
[h]Virgin fields have never had potatoes grown in them.

Data for disease incidence and severity index for the successive seed generations were analyzed by linear regression (REG procedure of SAS [SAS Release 8.0, SAS Institute, Cary, NC]), with the amount of disease on the seed tuber as the independent variable and the amount of disease on the progeny tubers as the dependent variables. Data for disease severity index of progeny tubers from plots at Othello and Paterson were analyzed by ANOVA using the GLM procedure or by linear regression using the REG procedure of SAS. Treatment means for cultivar and generation were compared using protected Fisher's LSD with a critical value of $P \leq 0.05$. Othello data from the progeny tubers at harvest were transformed using log (DSI + 1) to achieve normal variances. Linear and curvilinear regressions were used to quantify the relationship of disease incidence and DSI using REG procedure of SAS. Disease incidence was used as the independent variable and DSI as the dependent variable.

RESULTS

Disease severity index and incidence of silver scurf from all growers and cultivars from combined data increased significantly ($P \leq 0.05$) on the successive generations of potato seed tubers tested in 1998 and 1999 (Table 1). In 2000, incidence of silver scurf increased significantly, but the DSI was not significant at the ($P \leq 0.05$). Disease pressure was less in 2000 and leveled off between generations 2 and 3 (Table 1). Coefficient of determination from regression analyses for the disease severity index of successive generations combined for each year were 0.96, 0.95, and 0.89 for 1998, 1999, and 2000, respectively. Silver scurf symptoms were not observed on nuclear seed in 1998 and 2000. Nuclear seed tubers in 1999 had a disease incidence of 1.5 and a disease severity index of 0.3 (Table 1). Disease incidence and severity indices for silver scurf increased with each successive generation in 21 out of 28, decreased in two and remained the same in six seed increases (Table 2).

Level of silver scurf was associated with the time period between potato crops (Table 2). As time between potato crops in the rotation decreased among growers, silver scurf increased. In 1998, grower C had the shortest rotation and the highest silver scurf incidence and severity (80.0% and 37.6 DSI, respectively) in generation 3 seed. Grower A had the second shortest rotation and had less silver scurf (62.0% incidence and 23.2 DSI) than grower C, but more than grower B (16.0% incidence and 4.4 DSI) who had the longest rotation between potato crops. In 1999, the same pattern was observed in grow-

ers A and B, grower D went to a 5-year rotation at generation 1 and had an immediate increase in silver scurf that increased slightly in generations 2 and 3. Similar patterns of disease were observed in 2000; however, there was less disease than other years and disease incidence and severity leveled off between generations 2 and 3 (Table 2).

Progeny tubers from seed samples planted in Paterson had up to 41% incidence and 15.6 DSI for silver scurf. DSI on progeny tubers at harvest significantly increased as the DSI on seed tubers increased ($P = 0.03$). The coefficient of determination for silver scurf on seed tubers and silver scurf on progeny tubers at Paterson was 0.37 (Figure 1). At the Othello site in 1999, progeny tubers had less than 7% incidence and 1.4% DSI at harvest. Linear regressions for the relationship between level (severity or incidence) of silver scurf on the seed tubers relative to levels on the progeny tubers were not significant ($P > 0.05$) data not shown.

Disease severity index significantly increased as disease incidence increased for all data regarding seed to progeny tubers $P < 0.05$. The coefficient of determination for the relationship of DSI on disease incidence was 0.86 using linear regression and 0.93 for curvilinear regression. The regression equation for the curved line was $y = -2.34 + 3.64x - 0.036x^2$ (y variable DSI, and x variable disease incidence). Variation for DSI was greatest at high disease incidences (Figure 2).

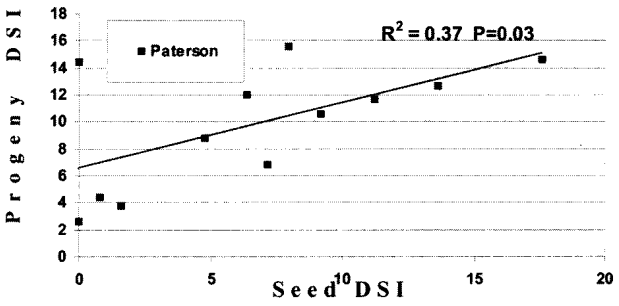

FIGURE 1.
Relationship of silver scurf severity on four successive generations of potato seed and their progeny tubers at Paterson, WA.

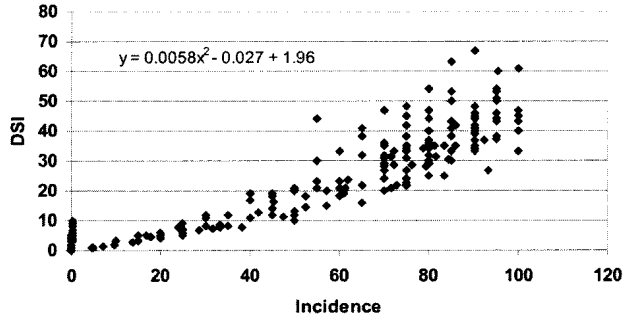

FIGURE 2.
Relationship between disease severity index and disease incidence.

DISCUSSION

Silver scurf severity on seed tubers had a major effect on the level of disease on subsequent progeny tubers in successive generations in this study. This confirms the importance of seed-borne inoculum. Successive generations had more disease than the previous generations in most seed tuber increases, as was also observed by research in England (Read and Hide 1984). If the disease on the seed tubers had no effect on level of disease of the progeny, then the amount of disease in successive generations would have remained constant given little or no effect from the soil. Data from the 2000 successive generations increased less than other years up to generation 2 when it leveled off. Edaphic factors likely had an influence on disease levels this year and may have reduced infections. However, the most likely reason for the decrease in silver scurf is the seed for 1998 and 1999 was evaluated after storage in February and the 2000 seed was evaluated in October following harvest but prior to long-term storage. Therefore, silver scurf did not have time to develop on the 2000 seed tubers. This may also account for the little to no increase in disease from generations 2 to 3 in 2000.

The nuclear seed tubers of most samples in this study were disease free and those that were infected were most likely infected during storage. Rodriguez et al. (1996) similarly reported that disease-free tubers became infected after exposure in storages where the ventilation system was circulating air and moving conidia within the storage. Seed samples that were stored with older generations tended to have more disease than those that were stored separately (Table 2). Recommendations are to store seed generations in completely separate storage facilities, but this recommendation may be difficult to follow because of a limited number of storage units and the cost to build new ones. None of the growers sampled stored all generations in the same storage building. Nuclear and generation 1 seed tubers were stored in the same building using the same ventilation system but the generations were physically separated by wood panels. Generations 2 and 3 were stored in another building using the same ventilation sys-

tem, but the individual lots were again physically separated. All seed tubers were stored under similar conditions including humidity and temperature, which illustrates the importance of storing seed lots with little or no silver scurf separately from seed lots with higher levels of silver scurf.

Progeny tubers (generation 1) from nuclear seed tubers in 2000 in this study may have become infected in the soil during plant growth since silver scurf was observed on the G1 tubers at harvest. However, the nuclear tubers used as seed may have become contaminated with conidia of *H. solani* during storage or handling prior to planting. Progeny tubers from generations 1 through 3 that were harvested from fields where potatoes were grown in a long rotation (8-10 years) or had never been planted to potatoes tended to have less disease than those from fields in a shorter (4 years) rotation (Table 2). Bain et al. (1996) reported a similar reduction in severity of silver scurf as the length of time between potato crops increased. The soil could be an important inoculum source for early generation seed tubers if short rotations between potato crops are used, in that the seed lot could initially become infected. However, contamination during storage and handling pose a high risk for initial infection of seed lots (Frazier et al. 1998; Rodriguez et al. 1996).

The fields used to study silver scurf levels on successive generation of seed had been used to grow potatoes in the past. It is therefore, possible that soil-borne inoculum contributed to the amount of disease on progeny tubers and served as a means to initiate disease on clean seed. Kamara and Huguelet (1972) reported symptoms of silver scurf on progeny tubers that originated from disease-free seed tubers planted in infested soil. Soil-borne inoculum was not considered a source of infection until the mid-1990s. Merida and Loria (1994) reported that not only does *H. solani* survive in soil, but it can also colonize and sporulate on senescent tissue of non-host plants. Others have reported symptoms of silver scurf on tubers from fields where potatoes had not been grown for 3 years or had never been grown (Bain et al. 1996; Firman and Allen 1993). Evidence from this study indicates that infected seed tubers are the main source of infection of progeny tubers during the growing season. Soil did not have a major influence on overall disease levels.

Other cultural and/or management practices besides shorter rotation periods will also influence silver scurf levels on tubers. Use of fungicides would be very important as a disease-management tool to minimize initial inoculum; in this study none of the growers applied seed treatments or at-plant fungicides. Some crops used in rotation will be better alternative hosts than others (Merida and Loria 1994). All four growers included wheat and barley in their rotations, two also used alfalfa, one canola, and another mint. Sanitation, temperature and humidity management of storage facilities will also minimize silver scurf. Frazier et al. (1998) reported *H. solani* survival in soil and on some potato storage building materials up to 9 months, and others have reported decreased silver scurf levels with lower storage humidity and temperatures (Lennard 1980; Rodriguez et al. 1996).

There was a positive relationship between the amount of silver scurf on the seed tubers and the amount on the progeny tubers in the trial conducted near Paterson. Similar trends occurred at Othello in 1999, but were not significant at $P \leq 0.05$. Disease levels on progeny tubers were much lower at Othello than Paterson, and therefore any association between generation and disease level may have been missed. Edaphic variables such as soil type, moisture regime, and temperature as well as a relative abundance of organisms antagonistic to *H. solani* may explain the difference between the results obtained at the two test sites and may be the variables influencing disease levels within a location or between years. Fungicide seed treatments and at-plant applications were not made at Paterson or Othello.

Silver scurf incidence and disease severity index were closely related as expected. In situations where research resources are limited, only one of the two disease measures needs to be used. Disease incidence is the quickest and most straight forward measurement. More tubers could be evaluated for disease incidence than for disease severity index with the same amount of effort, thus increasing sample size. An advantage of disease severity index is the accounting for both disease severity and incidence in a single number expressed on a standardized scale.

Transfer of silver scurf from seed to progeny tubers was evident by the increase of disease in successive generations of seed tubers and from the increase in silver scurf on progeny tubers relative to silver scurf levels on the seed in trials conducted in commercial fields. This study provides further evidence that seed-borne inoculum is the main source of silver scurf infection on progeny tubers. Because silver scurf is mainly seed-tuber-borne, fungicide seed tuber treatments or in-furrow applications of efficacious fungicides over the seed-pieces should provide a degree of protection against disease on the progeny tubers. Management of silver scurf requires an

integrated approach in the field and storage, through each generation of seed tuber production, to the production and storage of ware and processing potatoes.

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LITERATURE CITED

Bain PS, VS Bisht and DA Benard. 1996. Soil survival and thiabendazole sensitivity of *Helminthosporium solani* isolates from Alberta, Canada. Potato Res 39:23-30.

Cayley GR, GA Hide, PJ Read and Y Dunne. 1983. Treatment of potato seed and ware tubers with imazalil and thiabendazole for control of silver scurf and other storage diseases. Potato Res 26:163-173.

Denner FDN, CP Millard and FC Wehner. 1998. The effect of seed- and soilborne inoculum of *Colletotrichum coccodes* on the incidence of black dot on potatoes. Potato Res 41:51-56.

Firman DM and EJ Allen. 1993. Effects of windrowing, irrigation and defoliation of potatoes on silver scurf (*Helminthosporium solani*) disease. J Agric Sci 121:47-53.

Firman DM and EJ Allen. 1995. Transmission of *Helminthosporium solani* from potato seed tubers and effects of soil conditions, seed inoculum and seed physiology on silver scurf disease. J Agric Sci 124:219:234.

Frazier MJ, KK Shetty, GE Kleinkopf and P Nolte. 1998. Management of silver scurf (*Helminthosporium solani*) with fungicide seed treatments and storage practices. Amer J Potato Res 75:129-135.

Goth RW and RE Webb. 1983. Maintenance and growth of *Helminthosporium solani*. Am Potato J 60:281-287.

Hide GA and SM Hall. 1993. Development of resistance to thiabendazole in *Helminthosporium solani* (silver scurf) as a result of potato seed tuber treatment. Plant Path 42:707-714.

Jellis GJ and GS Taylor. 1977. The development of silver scurf (*Helminthosporium solani*) disease of potato. Ann Appl Biol 86:19-28.

Kamara AM and LE Huguelet. 1972. Host range and overwintering of *Helminthosporium solani*. Am Potato J 49:365. (abstr)

Kawchuk LM, JD Holley, DR Lynch and RM Clear. 1994. Resistance to thiabendazole and thiophanate-methyl in Canadian isolates of *Fusarium sambucinum* and *Helminthosporium solani*. Am Potato J 71:185-192.

Lennard JH. 1980. Factors affecting the development of silver scurf (*Helminthosporium solani*) on potato tubers. Plant Path 29:87-92.

Merida CL and R Loria. 1994. Survival of *Helminthosporium solani* in soil and *in vitro* colonization of senescent plant tissue. Am Potato J 71:591-598.

Mooi JC. 1959. A skin necrosis occurring on potato tubers affected by black dot (*Colletotrichum atramentarium*) after exposure to low temperatures. Eur Potato J 2:58-68.

Read PJ and GA Hide. 1984. Effects of Silver Scurf (*Helminthosporium solani*) on seed potatoes. Potato Res 27:145-154.

Rodriguez DA, GA Secor, NC Gudmestad and K Grafton. 1995. Screening tuber-bearing solanum species for resistance to *Helminthosporium solani*. Am Potato J 72:669-679.

Rodriguez DA, GA Secor, NC Gudmestad and LJ Franci. 1996. Sporulation of *Helminthosporium solani* and infection of potato tubers in seed and commercial storages. Plant Dis 80:1063-1070.

Sherwood RT and DJ Hagedorn. 1958. Determining common root rot potential of pea fields. Univ of Wisconsin Agric Exp Stn Bull 531.

Zimmerman-Gries S and EC Blodgett. 1974. Incidence and tuber transmission of silver-scurf on potatoes in Israel. Potato Res 17:97-112.