Effect of Seed-Tuber Generation, Soilborne Inoculum, and Azoxystrobin Application on Development of Potato Black Dot Caused by *Colletotrichum coccodes*

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ABSTRACT

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The effect of azoxystrobin on potato black dot and the role of seed- and soilborne inocula of *Colletotrichum coccodes* in the development of black dot were evaluated in the field using two potato seed generations (generation 1 and 3) of the susceptible cvs. Norkotah Russet and Russet Burbank over 3 years (2002 to 2004). Plants of Norkotah Russet and Russet Burbank treated with azoxystrobin had 13 and 23% higher yields, respectively, than nontreated plants in 2003. Disease severity on both cultivars was reduced 19 to 81%, and 22 to 81% on above- and belowground stem sections, respectively, when plants were treated with azoxystrobin. Plants of both cultivars that were treated with azoxystrobin had 9 to 26% less infected progeny tubers than the nontreated plants. These results indicated the efficacy of azoxystrobin to reduce black dot severity on both stems and progeny tubers. The roles of seed- and soilborne inocula in disease development were evaluated in 2003 and 2004 using generation 1 and 3 seed tubers. The incidence of *C. coccodes* in generation 1 mother tubers of Norkotah Russet and Russet Burbank were 2 and 16% in 2003, respectively, and 0 and 30% in 2004, respectively. The incidence of *C. coccodes* in generation 3 mother tubers of Norkotah Russet and Russet Burbank were 14 and 49% in 2003, respectively, and 12 and 38% in 2004, respectively. Generation 1 plants of Norkotah Russet had 36 and 13% greater yield than generation 3 plants in 2003 and 2004, respectively. In 2004, generation 1 plants of Norkotah Russet and Russet Burbank had 26 and 15% greater disease severity, respectively, on belowground stem than generation 3 plants. Generation 1 plants of Norkotah Russet had 7.5 and 11% more infected progeny tubers in 2003 and 2004, respectively, than generation 3. Significant differences for yield reduction and incidence of infected progeny tubers between the two seed generations were not recorded for Russet Burbank, suggesting that the effect of inoculum source of *C. coccodes* on black dot severity may be cultivar specific.

Additional keywords: seed-tuber generation, seedborne inoculum, soilborne inoculum

Potato black dot, caused by *Colletotrichum coccodes (*Wallr.) S. J. Hughes, is a concern in commercial potato production areas worldwide. The disease may cause up to 30% yield reduction on susceptible cultivars (24) and may damage tuber quality by causing skin blemishes, reducing tuber weight, and affecting tuber specific gravity (4).

Inoculum sources for infection of plants in the field by *C. coccodes* may be either one or a combination of soil-, seed-, and airborne inocula. Potato seed tubers that carry the pathogen internally in the vascular tissue, externally on the skin, or in a combination of both are one of the major inoculum sources. Seed tubers may spread the pathogen among and within potato production areas. Once introduced to the soil via infected seed tubers, the fungus may establish itself and become soilborne (13,14,16,19).

Potato seed tubers that are used commercially in Washington State are the progeny of plants produced in tissue culture from potato meristem cells. These pathogen-free plants are grown in greenhouses to produce pathogen-free progeny tubers that are referred to as "nuclear seed tubers". The latter are planted in the field and their progeny, which are the first potato tuber generation produced in the field, are referred to as generation 1 seed tubers. The progeny of generation 1 is referred to as generation 2, which will later produce the third generation of potato seed tubers in the field, known as generation 3. Generation 3 seed tubers are the most commonly used by commercial potato growers. In the field, each passing generation of progeny tubers accumulate seedborne pathogens; therefore, generation 3 seed tubers tend to have higher incidence of infection with *C. coccodes* than generations 1 and 2 seed tubers (14).

To date, no specific fungicide has been demonstrated to control potato black dot. Among the fungicides recently made available against black dot are the strobilurins. These are antifungal compounds, also known as Qo inhibitors (QoIs), which inhibit the respiratory cytochrome *bc1* complex in the mitochondria by blocking electron transport at the quinol-oxidizing (Qo) site (2,12,17,26,28). Fungicides of the strobilurin class are used commercially to control diseases caused by a wide range of fungal and fungal-like pathogens from the *divisions Oomycota, Ascomycota, and Basidiomycot*a (1,3,7,10,11,15,18,27). In the present study, we tested two hypotheses: (i) that azoxystrobin may reduce black dot under field conditions and (ii) that seed- and soilborne inocula of *C. coccodes* affect black dot development and severity differently. We hypothesized that exposing naturally infected certified potato seed tuber generations 1 and 3, which differ in incidence of initial inoculum, to soil naturally infested with *C. coccodes* would allow this evaluation.

MATERIALS AND METHODS

General. Trials were conducted in 2002, 2003, and 2004 at the Washington State University experimental station in Othello, WA. Trials were carried out on a Shano silt loam soil. Each year, trials were conducted in a different location of the station in fields where potatoes were grown previously and demonstrated high severities of black dot. All fields had 4 years of potato rotations (potato was grown every fourth year) with barley, mustard, wheat, or bean prior to planting. Experimental plots consisted of three rows, 4.5 m long and 86 cm wide, with seed pieces planted 25 cm apart within a row. Treatments were replicated four times in a randomized complete block design. The trial in 2002 was planted on 18 April, canopy was cut by mechanical flailing on 6 September, and trail was harvested on 16 September. The trial in 2003 was planted on 15 April, canopy was cut by mechanical flailing on 1 October, and trial was harvested on 3 October. The trial in 2004 was planted on 15 April, canopy was cut by mechanical flailing on 20 September, and trial was harvested on 22 September.

Plant material. Generation 1 and 3 seed tubers of the black dot-susceptible cvs. Norkotah Russet (short-season cultivar), and Russet Burbank (long-season cultivar)

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were used. Generation 1 seed tubers were grown in fields in which potato had not been grown previously, or had not been grown for at least 10 years and which, therefore were either disease free or carried low incidence of seedborne inoculum. Generation 3 seed tubers were grown in fields in which potato had not been grown for 4 to 5 years and carried a higher incidence of seedborne inoculum than generation 1 seed tubers. In 2002, generation 1 and 3 seed tubers of Norkotah Russet and Russet Burbank, respectively, were used. The Russet Burbank seed tubers in 2002 originated from two different seed sources (indicated as lot 1 and lot 2). In 2003 and 2004, seed-tuber generations 1 and 3 of both cultivars were used. Seed tubers were cut (60 g on average) and stored at 10ºC 3 days prior to planting.

Fungicide applications. Azoxystrobin (Quadris 2.08 SC; Syngenta) was applied with a CO2 backpack boom sprayer (TXVS-18 ConeJet nozzle, R&D Sprayers, Inc., Opelousas, LA) at a rate of 218.6 g/a.i./ha. Four applications were carried out: in furrow at planting time at a rate of 140 liters/ha, and 60, 83, and 93 days after planting to the foliage at a rate of 372 liters/ha.

Treatments. The trial in 2002 tested generation 1 seed tubers of Norkotah Russet with and without azoxystrobin application and generation 3 seed tubers of Russet Burbank (lots 1 and 2) with and without azoxystrobin application. The trial in 2003 tested generation 1 seed tubers of Norkotah Russet without azoxystrobin application and generation 3 of Norkotah Russet and generations 1 and 3 of Russet Burbank with and without azoxystrobin application. The trial in 2004 tested generations 1 and 3 of both Norkotah Russet and Russet Burbank with and without azoxystrobin application.

Evaluation of initial inoculum. Soil assays to evaluate initial soilborne inoculum were conducted in 2004. Three samples were taken randomly from each control plot. The presence of *C. coccodes* was evaluated by mixing 10 g of soil from each control plot (five subsamples) in 100 ml of autoclaved distilled water and shaken for 1 h. Soil suspension from each subsample (0.1 ml) was then spread onto 9-cm petri plats (five plats per subsample) with modified potato dextrose agar (PDA at 1 g/liter [Difco Laboratories, Detroit], Bacto agar at 15 g/liter [Difco], streptomycin sulfate at 0.1 g/liter [Sigma-Aldrich, St. Louis], and ethanol at 5 ml/liter] and incubated at 25ºC in the dark for 7 days.

Initial seedborne inoculum was tested in 2002, 2003, and 2004 by sampling 50 tubers from both cultivars. In the 2002 trial, the two Russet Burbank generation 3 seed lots (lots 1 and 2) were sampled jointly. Stolon ends of randomly selected seed tubers were cut and placed onto modified PDA and incubated at 25ºC in the dark. *C. coccodes* colonies were observed after 7 days of incubation. The number of infected stolon ends was counted and the incidence of infected seed tubers was calculated.

Evaluation of disease. Three parameters were used to evaluate disease, as follows. (i) Total yield weight of progeny tubers. (ii) Disease severity on stems. A week prior to harvest, 3 stems from each plot (12 stems per treatment) were selected randomly. Stems were washed with water to remove soil deposits and disinfected (1% NaClO for 10 min). Then, 5 cm of the aboveground and 5 cm of the belowground parts of the stems were each cut into six to eight (1-cm thick) segments. The segments were placed onto modified PDA and incubated at 25ºC in the dark for 14 days. Disease severity was evaluated separately for the aboveground and belowground part of stems and expressed as the percent infected segments. (iii) Disease incidence on progeny tubers. In all, 20 tubers from each plot were selected randomly at harvest (80 tubers per treatment). Tubers were washed with water to remove soil deposits and disinfected (1% NaClO for 10 min). Stolon ends were cut and placed onto modified PDA and incubated at 25ºC in the dark for 14 days. Incidence of infected progeny tubers was calculated.

Statistical analysis. Russet Burbank and Norkotah Russet were long- and short-season cultivars, respectively, and were exposed to the pathogen for different length of times during the growing seasons; therefore, each cultivar was analyzed separately. The main effects, azoxystrobin application and seed generation, were fixed. When analyzing the effect of azoxystrobin, plants from both seed generations treated with fungicide were contrasted collectively ($\alpha = 0.05$) to plants not treated with fungicide, except for Norkotah Russet in 2003, in which only generation 3 plants were contrasted due to the unbalanced design. The effect of seed generation was analyzed in 2003 and 2004 by contrasting only the plants not treated with azoxystrobin. The nontreated plants were used for this comparison due to the masking effect of azoxystrobin on disease development on treated plants. The general linear model procedure (Proc GLM) in SAS (SAS 9.1; SAS Institute, Cary, NC.) was used. Analysis of variance was followed by mean separation using Fisher's protected least significant difference ($\alpha = 0.05$). Comparisons of the incidence of infected mother tubers between generation 1 and 3 of Russet Burbank and Norkotah Russet was conducted using a statistical test for two binomial proportions ($\alpha = 0.05$).

RESULTS

Seed- and soilborne initial inoculum. Soil assays in 2004 did not detect *C. coccodes* in any of the sampled plots. Infection of mother tubers with *C. coccodes* of generation 1 Norkotah Russet and generation 3 Russet Burbank (lots 1 and 2) in 2002 was 0 and 41%, respectively. Generation 1 mother tubers of Russet Burbank were less ($P < 0.05$) infected than generation 3 in 2003 (16 and 49%, respectively), but not in 2004 (30 and 38%, respectively). Generation 1 mother tubers of Norkotah Russet were less ($P < 0.05$) infected than generation 3 in 2003 (2.3 and 14.3%, respectively) and in 2004 (0 and 12%, respectively).

Effect of azoxystrobin. Interaction between fungicide and seed generation was not detected ($P > 0.05$) for yield, disease severity, and infected progeny tubers. Plants of both Norkotah Russet and Russet Burbank treated with azoxystrobin had higher yields ($P < 0.05$) than nontreated plants only in 2003 (Table 1). Azoxystrobin-treated plants of Norkotah Russet and Russet Burbank yielded 23 and 13%, respectively, more than nontreated plants in that year (Table 1).

Azoxystrobin application resulted in decreased disease severity on both aboveground and belowground stem sections all years (Table 1). In 2002, Norkotah Russet plants treated with azoxystrobin had 59% less disease severity ($P = 0.07$) on belowground stems than non-treated plants (Table 1). Russet Burbank plants treated with azoxystrobin in 2002 had 24% less disease severity ($P = 0.09$) on aboveground stem sections and 34% less disease severity ($P = 0.02$) on belowground stems than nontreated plants (Table 1). In 2003, Norkotah Russet and Russet Burbank plants treated with azoxystrobin had 19 and 25% less ($P < 0.05$) disease severity, respectively, on aboveground stem sections than nontreated control plants; and Russet Burbank plants treated with azoxystrobin had 29% less ($P < 0.05$) disease severity on belowground stem sections than nontreated control plants (Table 1). In 2004, Norkotah Russet and Russet Burbank plants treated with azoxystrobin had 49 and 81% less ($P < 0.05$) disease severity, respectively, on aboveground stem sections and 69 and 81% less ($P < 0.05$) disease severity on belowground stem section than nontreated control plants (Table 1).

Application of azoxystrobin generally decreased infection of progeny tubers of Russet Burbank and Norkotah Russet significantly ($P < 0.05$) (Table 1). In 2002, azoxystrobin nontreated plants of Norkotah Russet had 13.7% more ($P = 0.09$) infected progeny tubers than treated plants (Table 1). In 2003, Russet Burbank plants treated with azoxystrobin had 8.3% less infected progeny tubers than nontreated plants (Table 1). In 2004, azoxystrobin-treated plants of Norkotah Russet and Russet Burbank had 10.6 and 26% less ($P<0.05$) infected progeny tubers, respectively than nontreated plants.

Effect of seed generation. Yield of Norkotah Russet plants from generation 1

were 36 and 13% higher ($P < 0.05$) than generation 3 plants in 2003 and 2004, respectively (Table 2). Disease severity on aboveground stem sections did not differ significantly ($P > 0.05$) between the seed generations in either year for either cultivar (Table 2). However, significant differences for disease severity were recorded on belowground stems in 2004, with 25.6 and 15.2% more infection on generation 1 plants of Norkotah Russet and Russet Burbank, respectively, than on generation 3 (Table 2).

The incidence of infected progeny tubers differed significantly between seed generations in both 2003 and 2004 only for plants of Norkotah Russet (Table 2). Generation 1 plants had 7.5 and 11.2% more infected progeny tubers than generation 3 plants in 2003 and 2004, respectively (Table 2).

DISCUSSION

Application of azoxystrobin to potato plants of cvs. Russet Burbank and Norkotah Russet resulted in reduced black dot severity on stems and progeny tubers compared with nontreated control plants in all 3 years of the study. Plants treated with azoxystrobin had fewer infected progeny tubers and lower stem colonization than nontreated plants, demonstrating the ability of the fungicide to inhibit both stems and progeny tuber colonization.

Yield was increased with fungicide application on both cultivars in 2003, but not in 2002 and 2004. The lack of yield differences between azoxystrobin-treated and nontreated plants in 2002 and 2004 may have been due to the presence of *Verticillium dahliae* rather than azoxystrobin inability to control black dot. Furthermore, black dot effect on yield previously has been reported to be inconsistent and infrequent (21,23–25). Tsror et al. (24) indicated that, even in seasons in which high disease severity was recorded on dry stems at or just before harvest, yield reduction did not occur consistently. The effect of black dot on yield was reported to be associated mostly with abiotically stressed plants. In Israel, yield loss was recorded when plants infected with *C. coccodes* were exposed to high day temperatures and drought during the spring growing season (25) and in the autumn season when plants were exposed to a shorter day photoperiod (23,24). In the United States, Norkotah Russet and Russet Burbank plants grown in growth chambers in soil infested with *C. coccodes* at 23 and 28ºC had lower yield than plants grown at 18ºC (9). In the Columbia basin of Washington State, potato plants generally are grown from March to October, when day length is relatively long, until the end of the season, when disease symptoms and pathogen signs often appear. Potato plants in the Columbia basin of Washington State are kept relatively well hydrated and well fertilized. Therefore, under these growing conditions, plants are kept relatively stress free, which may explain the infrequent yield reduction in the Columbia basin and in our trials. However, because black dot is a monocyclic disease, building inoculum over seasons, a threshold of inoculum eventually may be reached, in which case consistent reduction in yield would become apparent. Therefore, we advise not dismissing the importance of black dot on the basis of infrequent yield reduction.

Table 1. Main effect contrasts between azoxystrobin-treated and nontreated plants of Norkotah Russet and Russet Burbank potato in 2002, 2003, and 2004 field trials conducted at the Washington State University station, Othello

				Disease severity (stem colonization) (%)[a]								
	Yield (t/ha)[b]			Aboveground			Belowground			Infected progeny tubers (%)[c]		
Year, cultivar[d]	Non	Treated	*P*	Non	Treated	*P*	Non	Treated	*P*	Non	Treated	*P*
2002												
NK	70.2	69.4	0.21	43.0	15.8	0.13	77.5	18.3	0.07	13.7	0.0	0.09
RB (lots 1 & 2)	80.3	87.3	0.12	27.0	2.6	0.09	42.0	7.6	0.02	3.1	1.3	0.29
2003												
NK-G3	42.0	55.0	0.04	19.8	1.0	0.01	35.5	14	0.3	1.3	1.3	1.0
RB	84.2	96.5	0.02	33.0	7.8	0.01	36.8	7.9	0.01	8.8	0.5	0.01
2004												
NK	88.7	93.1	0.37	49.0	0.0	<0.0001	69.2	0.63	<0.0001	11.9	1.3	0.003
RB	122.5	129.7	0.14	87.1	6.3	<0.0001	84.6	3.8	<0.0001	26.0	0.0	0.001

[a] Aboveground and belowground parts of the stem (5 cm each, 12 stems per treatment) were cut into six to eight segments and placed onto potato dextrose agar (PDA; 1 g/liter). The incidence of infected segments was evaluated. Non = nontreated.
[b] Total yield of progeny tubers in metric tons per hectare.
[c] The stolon end of the tuber (80 tubers per treatment) was cut, and placed onto PDA (1 g/liter). The incidence of infected tubers was evaluated.
[d] Azoxystrobin comparison: single degree of freedom comparisons ($\alpha = 0.05$) for main effect (azoxystrobin) were conducted separately for each cultivar. The general linear model procedure (Proc GLM) in SAS (SAS 9.1; SAS Institute, Inc., Cary, NC) was used. NK = Norkotah Russet, RB = Russet Burbank, G1 = generation 1, and G3 = generation 3.

Table 2. Main effect contrasts between azoxystrobin-nontreated plants of generation 1 and generation 3 Norkotah Russet and Russet Burbank potato in 2003 and 2004 field trials conducted at the Washington State University station, Othello

				Disease severity (stem colonization) (%)[a]								
	Yield (t/ha)[b]			Aboveground			Belowground			Infected progeny tubers (%)[c]		
Year, cultivar[d]	G1	G3	*P*	G1	G3	*P*	G1	G3	*P*	G1	G3	*P*
2003												
NK	65.9	42.0	0.0078	10.3	19.8	0.1	36.8	35.5	0.95	8.8	1.3	0.03
RB	82.0	86.4	0.5	28.0	38.3	0.4	36.0	37.5	0.9	7.5	10.0	0.5
2004												
NK	95.0	82.5	0.02	48.0	50.0	0.95	82.0	56.4	0.01	17.5	6.3	0.009
RB	122.6	122.5	0.8	91.0	83.2	0.3	92.2	77.0	0.03	28.8	23.3	0.7

[a] Aboveground and belowground parts of the stem (5 cm each, 12 stems per treatment) were cut into six to eight segments and were placed onto potato dextrose agar (PDA, 1 g/liter). The incidence of infected segments was evaluated. NK = Norkotah Russet, RB = Russet Burbank, G1 = generation 1, and G3 = generation 3.
[b] Total yield of progeny tubers in metric ton/hectare.
[c] The stolon end of the tuber (80 tubers per treatment) was cut and placed onto PDA (1 g/liter). The incidence of infected tubers was evaluated.
[d] Generation comparison: single degree of freedom comparisons ($\alpha = 0.05$) for main effect (seed-generation) was conducted separately for each cultivar. The general linear model procedure (Proc GLM) in SAS (SAS 9.1; SAS Institute, Inc., Cary, NC) was used.

To determine which inoculum source, seed- or soilborne, is more dominant in black dot development and severity, naturally infected certified potato seed generations differing in incidence of initial inoculum were exposed to soil naturally infested with *C. coccodes*. Assays to quantify inoculum levels of soilborne pathogens, in which soil extracts were plated on agar media, were found to be inaccurate (22); and molecular-based techniques, such as real-time PCR, although sensitive (5), are unable to distinguish between viable and nonviable inoculum. Furthermore, in the case of black dot, the relationship between *C. coccodes* soilborne inoculum threshold and disease severity is still unknown. Therefore, in order to overcome the soilborne inoculum obstacle, we decided to expose the seed tubers to fields with a history of severe black dot that had similar crop rotations (potato was grown every fourth year). *C. coccodes* sclerotia were reported as viable up to 8 years (8); therefore, we assumed similar viable inoculum levels in the soil. Lower yields, higher disease severity, and a higher incidence of infected progeny tubers were expected on generation 3 plants than generation 1 plants due to the higher initial disease incidence in the mother tubers.

Differences between the seed generations were recorded for Norkotah Russet in 2003 and 2004. Generation 1 Norkotah Russet plants, which had lower seedborne infection than generation 3, had higher yields under a similar soilborne inoculum level, pointing to the additive effect of seedborne inoculum, which is in agreement with the results of Dashwood et al. (6). The incidence of infected progeny tubers was significantly higher for Norkotah Russet generation 1 than for generation 3 in 2003 and 2004, which suggested that progeny tubers developing from less-infected mother tubers (generation 1) were more prone to infection from soilborne inoculum than those developing from more-infected mother tubers (generation 3). These results are in agreement with Read and Hide (19,20) and Danner (7), who pointed out that soilborne inoculum may be more dominant than seedborne. However, the results recorded for Russet Burbank did not indicate a different effect of inoculum source on black dot severity. These results were in agreement with those of Dashwood et al. (6), who did not record a difference in disease severity when plants of cv. Maris Piper were infected from either seed- or soilborne inoculum. The inconsistencies obtained among the different studies may be due to a different sampling methodology; however, it also is probable that the effect of inoculum source on black dot severity is cultivar specific.

Black dot studies to date evaluated seedborne inoculum based on external skin blemishes (7,19,20), overlooking internal infection in the vascular tissues at the stolon end. The russet-skin potato cultivars used in the present study were the most commonly produced by the potato industry in the Columbia basin of Washington State and infrequently demonstrate external skin blemishes. Therefore, seedborne infection in this study was determined by isolations from the vascular tissue at the stolon end. Isolation of *C. coccodes* from the stolon end is a valid way to evaluate the incidence of infected seed tubers. However, it is possible that latent (14,24) microscopic skin blemishes, for which no test was done, were present on mother and progeny tubers. Further research should look into the importance and contribution of the two types of seedborne inoculum to black dot severity.

In conclusion, azoxystrobin-based fungicides are a promising means for black dot management. In the future, azoxystrobin-based fungicides should be used in combination with practices that minimize plant stress and possibly reduce high inoculum levels in soil. Strategies should be employed that reduce the risk of selecting for and increasing strains of *C. coccodes* resistant to strobilurin fungicides. The novel findings of the present study in relation to seed- and soilborne inocula should be explored further. Efforts should be made to evaluate the effect of inoculum sources on different potato cultivars and in relation to inoculum threshold in seed and soil.

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LITERATURE CITED

1. Ammerman, E., Lorenz, G., Schelberger, K., Wenderoth, B., Sauter, H., and Rentzea, C. 1992. BAS 490 F-A broad-spectrum fungicide with a new mode of action. Pages 403-410 in: Brighton Crop Protection Conference—Pests and Diseases—1992, vol. 1.Lavenham Press, Lavenham, Suffolk, UK.
2. Anke, T. 1995. The antifungal strobilurins and their possible ecological role. Can. J. Bot. 73(Suppl. 1): S940-945.
3. Baldwin, B. C., Clough, J. M., Godfrey, C. R. A., Godwin, J. R., and Wiggins, T.E. 1996. The discovery and mode of action of ICIA5504. Pages 69-78 in: Modern Fungicides and Antifungal Compounds. H. Lyr, P. E. Russel, and H. D. Sisler, eds. Intercept, Andover, Hants, UK.
4. Barkdoll, A. W., and Davis, J. R. 1992. Distribution of *Colletotrichum coccodes* in Idaho and variation in pathogenicity on potato. Plant Dis. 76:131-135.
5. Cullen, D. W., Lees, A. K., Toth, I. K., and Duncan, J. M. 2002. Detection of *Colletotrichum coccodes* from soil and potato tubers by conventional and quantitative real-time PCR. Plant Pathol. 51:281-292.
6. Dashwood, E. P., Fox, R. A., and Perry, D. A. 1992. Effect of inoculum source on root and tuber infection by potato blemish disease fungi. Plant Pathol. 41:215-223.
7. Denner, F. D. N., Millard, C. P., and Wehner, F. C. 1998. The effect of seed- and soil-borne inoculum of *Colletotrichum coccodes* on the incidence of black dot on potato. Potato Res. 41:51-56.
8. Dillard, H. R., and Cobb, A. C. 1998. Survival of *Colletotrichum coccodes* in infected tomato tissue and soil. Plant Dis. 82:235-238.
9. Geary, B. D. 1999. Pages 87-104 in: Epidemiology of silver scurf and black dot, two tuber diseases of potato. Ph.D. thesis, Washington State University, Pullman.
10. Gold, R. E., Ammerman, E., Koehl, H., Leinhos, G. M. E., Lorenz, G., Speakman, J. B., Stark-Urnau, M., and Sauter, H. 1996. The synthetic strobilurin BAS 490 F: Profile of a modern fungicide. Pages 79-92 in: Modern Fungicides and Antifungal Compounds. H. Lyr, P. E. Russel, and H. D. Sisler, eds. Intercept, Andover, Hants, UK.
11. Heaney, S. P., and Knight, S. C. 1994. ICIA5504: A novel broad-spectrum synthetic fungicide for use on fruit, nut and horticultural crops. Pages 509-516 in: Brighton Crop Prot. Conf.—Pests and Diseases—1994, vol. 2. Major Print, Nottingham, UK.
12. Hewitt, H. G. 1998. Fungicides in Crop Protection. CAB International, Oxon, UK.
13. Jeger, M. J., Hide, G. A. Van den Boogert, P. H. J. F., Termorshuizen, A. J., and Van Baarlen. P. 1996. Pathology and control of soil-borne fungal pathogens of potato. Potato Res. 39:437-469
14. Johnson, D. A. Rowe, R. C., and Cummings, T. F. 1997. Incidence of *Colletotrichum coccodes* certified potato seed tubers planted in Washington State. Plant Dis. 81:1199-1202.
15. Jordan, D. B., Livingston, R. S., Bisaha, J. J., Duncan, K. E., Pember, S. O., Picollelli, M. A., Schwartz, R. S., Stermberg, J. A., and Xiao-Song, T. 1999. Mode of action of famoxadone. Pestic. Sci. 55:105-188.
16. Komm, D. A., and Stevenson, W. R. 1978. Tuber-borne infection of *Solanum tuberosum* 'Superior' by *Colletotrichum coccodes*. Plant Dis. Rep. 62:682-687.
17. Link, T. A., Haase, U., Brandt, U., and Von Jagow, G. 1993. What information do inhibitors provide about the structure of the hydroquinone oxidation site of ubihydroquinone: cytochrome c oxidoreductase? J. Bioenerg. Biomembr. 25:221-232.
18. Mitsuhiro, I., Michio, M., Hideyuki, T., Hasagawa, R., Ichiba, T, Yoshio, H., and Takeda, R. 1999. Structural and fungicidal activities of methoxy-iminophenylacetamide derivatives. Pestic. Sci. 55:347-349.
19. Read, P. J., and Hide, G. A. 1988. Effect of inoculum source and irrigation on black dot disease of potato (*Colletotrichum coccodes* (Wallr.) Hughes) and its development during storage. Potato Res. 31:493-500.
20. Read, P. J., and Hide, G. A. 1995. Development of black dot disease (*Colletotrichum coccodes* (Wallr.) Hughes) and its effect on the growth and yield of potato plants. Ann. Appl. Biol. 127:57-72.
21. Scholte, K., Veenbaas-Rijks, J. W., and Labruyere, R. E. 1985. Potato growing in short rotations and the effect of *Streptomyces* spp., *Colletotrichum coccodes*, *Fusarium tabacium* and *Verticillium dahliae* on plant growth and tuber yield. Potato Res. 28:331-348.
22. Termorshuizen, A. J., Davis, J. R., Gort, G., Harris, D. C., Huisman, O. C., Lazarovits, G., Locke, T., Melero Vara, J. M., Mol, L., Paplomatas, E. J., Platt, H. W., Powelson, M., Rouse, D. I., Rowe, R. C., and Tsror, L. 1998. Interlaboratory comparison of methods to quantify microsclerotia of *Verticillium dahliae* in soil. Appl. Environ. Microbiol. 64:3846-3853.
23. Tsror (Lahkim), L. 2004. Effect of light duration on severity of black dot caused by Colletotrichum coccodes on potato. Plant Pathol. 53:288-293.
24. Tsror (Lahkim), L., Erlich, O., and

Hazanovsky, M. 1999. Effect of *Colletotrichum coccodes* on potato yield, tuber quality, and stem colonization during spring and autumn. Plant Dis. 83:561-565.
25. Tsror (Lahkim), L., and Johnson, D. A. 2000. *Colletotrichum coccodes* on potato. Pages 362-373 in: *Colletotrichum*—Host Specificity, Pathology and Host–Pathogen Interaction. D. Prusky, S. Freeman, and M. B. Dickman. American Phytopathological Society Press, St. Paul, MN.
26. Vincelli, P., and Dixon, E. 2002. Resistance to QoI (Strobilurin-like) fungicide in isolates of *Pyricularia grisea* from perennial ryegrass. Plant Dis. 86:235-240.
27. Wong, F. P., and Wilcox, W. F. 2001. Comparative physical modes of action of Azoxystrobin, Mancozeb, and Metalaxyl against *Plasmopara viticola* (grape vine downy mildew). Plant Dis. 85:649-656.
28. Ypema, H. L., and Gold, R. E. 1999. Kresoxim-methyl: Modification of a naturally occurring compound to produce a new fungicide. Plant Dis. 83:4-19.