Management of Cercospora Leaf Spot in Conventional and Organic Table Beet Production

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Abstract

Cercospora leaf spot (CLS; *Cercospora beticola*) is the most important foliar disease affecting table beet. Epidemics occur annually and fungicides extend the survival of foliage to enable mechanized harvest. However, a high frequency of strobilurin-resistant *C. beticola* isolates necessitates the identification of fungicides with different modes of action for tactical rotation. There is also substantial demand for organically produced table beet, for which synthetic fungicides are prohibited. Five small-plot, replicated field trials were conducted over two years to evaluate conventional and Organic Materials Review Institute (OMRI)-listed products for CLS control in table beet cv. Ruby Queen at Geneva and Ithaca, New York. Benzovindiflupyr + difenoconazole significantly reduced temporal disease progress (measured by the area under the disease progress stairs; AUDPS) by 86.7 to 97.3% compared with nontreated plots, and mean survival time of leaves was significantly extended. The demethylation inhibitor, propiconazole, also provided significant disease control in two trials in 2016.

Disease severity in plots treated with succinate dehydrogenase inhibitors (boscalid, fluxapyroxad + pyraclostrobin, and penthiopyrad) was significantly decreased compared with nontreated plots but less than other fungicides. Efficacious fungicides significantly increased the dry weight of foliage but did not significantly affect the dry weight of roots, and root shoulder diameter. The enhanced longevity of leaves and increased dry weight of foliage may extend opportunities for mechanized harvesting without deleteriously affecting root yield parameters which are strictly regulated for the processing markets. In two trials, copper octanoate + *Bacillus amyloliquefaciens* strain D747 (as Cueva + Double Nickel LC) resulted in significantly improved disease control in comparison with application of either product alone and provided comparable and reproducible disease control equivalent to conventional fungicides at both locations. The implications of these findings for CLS control in conventional and organic table beet production systems are discussed.

New York is the second largest producer of table beet (*Beta vulgaris* ssp. *vulgaris*) in the United States (USDA 2012). The industry has historically been focused on the production of table beet for the processing industry, which currently uses exclusively conventional agronomic practices. However, table beet in New York is now produced in diverse production systems ranging from small, diversified farms to broad-acre fields (Stivers 2001). These systems may either be conventional or organic, meaning disease management recommendations need to be site-specific and responsive. The New York table beet industry is undergoing considerable growth from enhanced demand and opportunities for value-added products driven by the enhanced perception of health benefits of consumption (Clifford et al. 2015). The majority of this industry growth is for the fresh market and further opportunities have been identified for organic production.

One of the primary challenges in enhancing the profitability of table beet production is effective management of plant diseases (Abawi et al. 1986; Kikkert et al. 2010; Shah and Stivers-Young 2004). Cercospora leaf spot (CLS) is caused by the fungus *Cercospora beticola* Sacc. and is considered one of the most damaging diseases of table and sugar beet crops worldwide (Koike et al. 2010; Weiland and Koch 2004). The disease is significant to table beet production because it can cause defoliation (Weiland and Koch 2004). Early disease symptoms are discrete, necrotic lesions surrounded by a red to purple margin in a red table beet cultivar. The lesions have a gray center and the presence of pseudostromata is diagnostic (McKay and Pool 1918; Weiland and Koch 2004). In sugar beet, defoliation may result in reduced carbohydrate transport and abnormal root sizing, leading to reduced gross sugar and increases in impurities (Smith and Martin 1978; Smith and Ruppel 1973; Shane and Teng 1992).

For broad-acre table beet production, defoliation may lead to the inability to mechanically harvest, and consequently substantial crop loss. For the fresh market, roots may be sold with intact foliage or separately. The presence of lesions on the foliage is significant because it may lead to rejection for sale. CLS control in conventional table beet production in New York relies upon prophylactic applications of fungicides aimed at extending the survival of leaves to facilitate mechanized harvest. Fungicides are used prophylactically because of the high crop loss that may be incurred in an epidemic (Stivers 2001).

Control failures following repeated applications of the strobilurin fungicide, azoxystrobin (Quadris, Syngenta) were first reported from processing table beet fields in western New York in 2012 (Abawi et al. 2014). Later studies confirmed a high frequency of *C. beticola* isolates with the G143A mutation (41%) and a single isolate with the F129 L mutation (Vaghefi et al. 2016). The G143A and F129 L mutations are known to confer resistance to this class of fungicide (Bartlett et al. 2002). Resistance to multiple modes of action within populations of *C. beticola* from sugar beet is common as a result of repeated exposure (Campbell et al. 1998; Ioannidis and Karaoglanidis 2000; Secor et al. 2010). For example, resistance has been reported to multiple benzimidazoles (Bugbee 1996; Dovas et al. 1976; Georgopoulos and Dovas 1973; Rupel and Scott 1974), triphenyltin hydroxide (Giannopolitis 1978; Giannopolitis and Chrysai-Tokousbalides 1980), demethylation inhibitors (Bolton et al. 2012a, b; Karaoglanidis et al. 2000, 2001, 2002), and strobilurins (Bolton et al. 2013; Kirk et al. 2012). The high frequency of resistance to multiple fungicide modes of action is also a function of the high genotypic and genetic diversity within the *C. beticola* populations on sugar beet (Bolton et al. 2012c; Groenewald et al. 2007, 2008; Weiland et al. 2001) and table beet (Vaghefi et al. 2016, 2017a, b). The durability of current tactical approaches to CLS control in conventional table beet production is therefore critically threatened by the rapid development of resistance in pathogen populations to new modes of action.

In organic table beet production, strategies to reduce disease risk such as rotation to nonhosts become more important for CLS control because of the inability to use synthetic fungicides. For example, given that *C. beticola* can survive on infested plant debris in soils for up to 3 years and initiate epidemics from splash dispersal of

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inoculum to the leaves (Solel 1970), rotation to nonhosts between table beet crops is highly recommended. Alternative hosts including weeds have also been reported as capable of acting as a green bridge between susceptible crops (Jacobsen et al. 2000; Lartey et al. 2005; McKay and Pool 1918). Optimal weed management to decrease the probability of inoculum perpetuation is an important disease management strategy in conventional table beet production but critical in organic production. Tactics for use within the season are limited to biorational pesticides or copper-based products that must be approved by the Organic Materials Review Institute (OMRI). These products contain either microorganisms that suppress the pathogen population through competition or contain naturally produced compounds derived from a microorganism that may induce a range of mechanisms (Thakore 2006). Preliminary studies examining a small number of biorational products for CLS control in table beet demonstrated limited efficacy. The use of resistant cultivars under low disease pressure was found to be more effective at reducing disease severity than the OMRI-listed products trialed (Lange et al. 2015). Some variation in susceptibility to CLS in table beet has also been further characterized (S. J. Pethybridge, unpublished data) but the strict produce size and appearance preferences imposed by the processing industry limits the ability to select cultivars based on this information. For the fresh market, even minor variation in CLS susceptibility may be a useful part of a disease management strategy.

The primary objective of this study was to evaluate fungicides with a broad range of modes of action and OMRI-approved biorational products for the control of CLS in table beet. A secondary objective was to quantify the effects of manipulating disease severity on yield components (dry weight of foliage and roots, and root size). Maintenance of healthy foliage is necessary to facilitate mechanized harvesting in broad-acre table beet production. The effect of these treatments on root yield components is important to characterize because of the requirement to conform with strict size grade standards established by the table beet processing industry. This information will be vital in the design of integrated management strategies for CLS at conventional and organic table beet farms in New York and neighboring production regions.

Materials and Methods

Field experiments. Five small-plot, replicated field trials were conducted in 2015 and 2016 at two locations in New York. Evaluation of the efficacy of conventional fungicides was conducted at Geneva in 2015 (trial 1), and Geneva and Ithaca in 2016 (trials 2 and 3, respectively) (Table 1). OMRI-listed products were evaluated in trials 4 and 5 at Geneva and Ithaca in 2016, respectively (Table 2). All fields were established from seed with cv. Ruby Queen (Seneca Foods, Dayton, WA) at an in-row plant density of 54 seeds/m using a vacuum Monosem planter (Monosem, Lenexa, KS). Trials at Geneva were planted on 22 May 2015 and 17 June 2016, and rows were separated

by 0.76 m. Trials at Ithaca were planted on 2 August 2016 and rows were separated by 0.38 m.

Fourteen days prior to planting, 336.3 kg/ha 10 N:10 P:10 K fertilizer (Phelps Supply Inc., Phelps, NY) was broadcast and shallowly incorporated using a tractor-mounted coulters mulcher. An additional 392.3 kg/ha of the same fertilizer was banded at planting. At 28 to 33 days after planting (DAP), 56.1 kg/ha nitrogen (22% as urea) was side-dressed by hand. Weeds were controlled by 0.78 liters/ha of the pre-emergent herbicide, s-metolachlor (as Dual Magnum; Syngenta Corporation, Greensboro, NC) + 2.2 liters/ha ethofumesate (as Nortron; Bayer CropScience, Research Triangle, NC); followed by 9.4 liters/ha demedipham (as Betanex; Bayer CropScience) + 280.2 g/ha clopyralid (as Stinger; Dow AgroSciences, Indianapolis, IN) + 1.1 liters/ha phenmedipham (as Spin-Aid; Dow AgroSciences) at 22 DAP when beet seedlings had reached two to four true leaves. At Ithaca (trials 3 and 5), the entire trial area received 0.6 liters/ha spinetoram (as Radiant; Dow AgroSciences) at 43 DAP for beet army worm control. The trials received supplementary irrigation (5 mm/h) using overhead sprinklers at 5- and 2-day intervals in 2015 and 2016, respectively.

Experimental design and treatments. In trial 1, each plot had four 6.1-m-long rows and were separated by two rows or 3.05 m within rows. In the 2016 trials, plots were 3.05 m long rows, and were separated by one row and 0.6 m within rows. Plots within trials in 2016 consisted of either two (Geneva) or four (Ithaca) rows. Rows and plants surrounding plots were noninoculated and untreated. Treatments were replicated five times within a completely randomized block design and applied using a carbon dioxide-pressurized backpack sprayer with four TeeJet 8002VS flat fan nozzles at 202.9 and 261.9 liters/ha in 2015 and 2016, respectively. Conventional fungicides were applied three times in trials 1 to 3 (Table 1). Some of the conventional fungicides initially included in trial 1 (e.g., fluazinam, cyprodinil, mancozeb) were not repeated the following year due to changes in preference crops by the registrants and/or extended pre-harvest intervals in table beet, making their use impractical. A subset of data from trial 1 has been published (Pethybridge et al. 2016). OMRI-listed products were applied four times in trials 4 and 5 (Table 2).

Inoculation with *C. beticola*. Eight isolates of *C. beticola* representative of the New York population (Tb14-047, Tb14-070, Tb14-081, Tb14-113, Tb14-116, Tb14-118, Tb14-146, and Tb14-154; Vaghefi et al. 2016) were included in mycelial suspensions that served as inoculum for the trials. Five *C. beticola* isolates included in the inoculum were resistant to strobilurin fungicides quantified under in vitro conditions with effective concentrations to reduce conidial germination by 50% ranging from 0.8 to 5.8 µg/ml. An individual liquid culture of each isolate was established by transferring mycelium into clarified V8 (CV8) juice broth (Secor and Rivera 2012) placed at room temperature on a shaker at 50 rpm under fluorescent light with a 12 h photoperiod for 14 days. Mycelia of each isolate were collected

Table 1. Fungicides and application timings (days after planting; DAP) used in each of three trials evaluating *Cercospora* leaf spot control in table beet at Geneva and Ithaca, NY

Active ingredient (FRAC code) [†]	Product (rate/ha); manufacturer	Trial 1 (Geneva, 2015)	Trial 2 (Geneva, 2016)	Trial 3 (Ithaca, 2016)
		DAP	DAP	DAP
Azoxystrobin (11)	Quadris (0.6 liters); Syngenta	82, 89, and 94	59, 67, and 77	41, 48, and 57
Penthiopyrad (7)	Vertisan (1.1 liters); DuPont	82, 89, and 94	59, 67, and 77	41, 48, and 57
Penthiopyrad (7)	Vertisan (2.2 liters); DuPont	-	59, 67, and 77	41, 48, and 57
Benzovindiflupyr + difenoconazole (7 + 11)	Aprovia Top (0.9 liter); Syngenta	82, 89, and 94	59, 67, and 77	41, 48, and 57
Cyprodinil + difenoconazole (9 + 3)	Inspire Super (1.5 liters); Syngenta	82, 89, and 94	- [‡]	-
Fludioxonil (12)	Cannonball WP (490 g); Syngenta	82, 89, and 94	-	-
Mancozeb (M3)	Manzate FL (2.3 liters); United Phosphorus	82, 89, and 94	-	-
Fluazinam (29)	Omega (1 liter); Syngenta	82, 89, and 94	-	-
Boscalid (7)	Endura (0.8 liter); BASF	-	59, 67, and 77	41, 48, and 57
Propiconazole (3)	Tilt (0.3 liter); Bayer Crop Science	-	59, 67, and 77	41, 48, and 57
Fluxapyroxad + pyraclostrobin (7 + 11)	Merivon (0.4 liter); Bayer Crop Science	-	59, 67, and 77	41, 48, and 57

[†] Fungicide Resistance Action Committee code.

[‡] Not included in the respective trial.

and filtered through two layers of sterile cheesecloth and dispersed in sterile distilled water (SDW) in a Waring blender (VWR, Radnor, PA) at low speed for 30 s prior to additional filtration through sterile cheesecloth. The number of mycelial fragments per ml of inoculum was estimated using a hemocytometer. Inoculum was diluted in SDW prior to field application including 0.01% (v/v) Tween-20, with a backpack sprayer at a volume of 150 liters/ha. To estimate colony forming units (cfu) in the inoculum, 1 ml of the mycelial suspension was serially diluted (10^0 to 10^{-6}) in SDW, and 100 μ l was spread on CV8 plates. Three replicate plates were used for each concentration and incubated at room temperature exposed to a 12 h photoperiod of white light. After 3 days, the number of colonies was counted on each plate and quantified. In 2015 (trial 1), inoculum was applied to the two outer rows of each plot at 47 DAP followed by a second application of the entire trial area 7 days later at concentrations of 3×10^3 cfu/ml, and 1×10^4 cfu/ml, respectively. In 2016, inoculum was applied to the entire trial area at Geneva (trials 2 and 4) at concentrations of 3.6×10^3 cfu/ml at 52 DAP, followed by 1.6×10^4 cfu/ml on 61 DAP. Trials 3 and 5 (Ithaca, 2016) were inoculated at 42 and 56 DAP with inoculum at concentrations of 2×10^4 cfu/ml and 3.1×10^3 cfu/ml, respectively.

Disease and yield assessments. *CLS severity.* Disease severity was assessed 24 to 48 h prior to the first inoculations and at 4- to 12-day intervals thereafter until harvest. In trial 1, CLS severity was assessed at 80, 91, 96, 101, and 105 DAP. In trials 2 and 3, CLS severity was assessed at 56, 68, 75, 80, 84, and 94 DAP. In trials 4 and 5, CLS severity was assessed at 39, 50, 59, 71, and 87 DAP. At each time, the number of CLS lesions was counted on each of 20 arbitrarily selected leaves of similar age within each plot (10 in each of the two rows at approximately 0.5 m intervals) and used to calculate an average number of CLS lesions per leaf. Temporal disease progress within each plot was quantified by calculating the area under the disease progress stairs (AUDPS) (Simko and Piepho 2012).

Canopy reflectance. In trials 2 and 3, a handheld multispectral radiometer (MRS5; CropScan Inc., Rochester, MN) was used to measure canopy reflectance in five wavelength bands (485, 560, 660, 830, and 1,650 nm) on the same day as the disease severity assessments, between 12:00 and 14:00 (eastern time) when incoming radiation was at least 500 W/m². Relationships between canopy reflectance in the near infrared range (830 nm for the MRS5), growth, and yield have been well documented and used to assess plant health in many agricultural crops including sugar beet (Nilsson 1995; Steddom et al. 2005). The radiometer was placed 2 m above the soil surface and appropriately positioned using a spirit level and support pole allowing measurements of canopy reflectance from a 0.97-m² area in the middle of each plot. Canopy reflectance was calculated as a percentage of the voltage for the reflected radiation divided by the voltage for the incident radiation of each wavelength. Two readings were taken from each plot and averaged. Temporal changes in canopy reflectance within each plot were quantified by calculating the area under the canopy reflectance stairs (AUCRS) (Simko and Piepho 2012).

Yield. To assess table beet stand homogeneity as a potential covariate influencing foliar disease severity, the number of plants per row meter was counted in each of two 0.5 m lengths within each plot between 30 and 35 DAP. To evaluate yield components, plants were removed by hand from 1 m row lengths within each plot at 103 and 146 DAP (trial 1), 94 and 120 DAP (trial 2), and 94 DAP (trial 3). The

second harvests conducted in trials 1 and 2 examined treatment effects on regrowth following defoliation from CLS. In-ground storage into late autumn is often used to facilitate processing schedules and by fresh market growers to extend sales (Stivers 2001). Yield was not evaluated in trials 3 and 5 at Ithaca due to late season damage from deer. At each harvest, the total number of plants removed per unit row length was counted, and tops were removed from the roots and weighed separately following washing. The dry weights of the foliage samples were calculated by placing approximately 20% of the sample at 65°C for 48 h. The dry weights of roots were calculated after the subsample was diced and dried in the same conditions for 120 h. Twenty roots were also randomly selected from each plot and shoulder diameter was measured using digital calipers.

Data analysis. *Relationships among multispectral canopy reflectance and other variables.* Linear regression analysis was used to model the relationships among canopy reflectance at 485, 560, 660, 830, 1,650 nm, selected wavelength ratios (660/560, 830/560, 1,650/560, 830/660, and 1,650/830 nm), and the normalized difference vegetative index (NDVI), and CLS severity, AUDPS, and the dry weight of foliage. Precision of the significant relationships was depicted by the coefficient of determination (R^2), and the standard error of the estimate of y (SE E_y). All analyses were conducted using the statistical software, Genstat Version 17.1 (Hemel Hempstead; U.K.).

Treatment evaluation. Exploratory data analysis detected significant differences among the means between datasets from trials with like treatments using the Anderson-Darling goodness-of-fit test (Anderson and Darling 1954), and nonhomogeneous variances using the Fisher F -test (Markowski and Markowski 1990). Data from the individual trials were therefore analyzed separately to evaluate treatments. The effect of treatment on CLS severity, AUDPS, canopy reflectance at 830 nm, and yield components (dry weight of foliage and roots at each harvest, and root shoulder diameter) was quantified using generalized linear models with treatment and replication analyzed as fixed and random effects, respectively. Means were separated with Fisher's protected least significant difference test ($P = 0.05$).

Effect of conventional fungicides on leaf survival. In trials 2 and 3, the effect of selected fungicides (benzovindiflupyr + difenoconazole, propiconazole, and fluxapyroxad + pyraclostrobin) was modeled on the time-to-death of leaves caused by CLS using survival analysis (Allison 1995). Propiconazole and fluxapyroxad + pyraclostrobin were selected as those currently registered for CLS control on table beet in New York. Benzovindiflupyr + difenoconazole was also included because of significant efficacy for disease control demonstrated in trial 1. In each of the select fungicide-treated and nontreated plots, nine leaves were tagged, and observed at intervals (5 to 9 days) and placed into a binary category (alive = 0 or dead = 1). Assessments began on 24 August and 21 September in trials 2 and 4, respectively. Therefore, within each trial, 180 individual leaves were assessed at each time. In trial 2, leaves were assessed eight times until 19 October. In trial 3, the final assessment was conducted on 31 October following six assessments. Dead leaves were defined as completely necrotic but not abscised from the plant. The time-to-death of leaves was estimated as the midpoint between two consecutive assessments between which the status of the leaf had changed from alive to dead.

Table 2. Organic Materials Review Institute-listed products and application timings (days after planting; DAP) in each of two trials evaluating the control of *Cercospora* leaf spot in table beet at Geneva and Ithaca, NY

Active ingredient (FRAC code) ^a	Product (rate/ha); manufacturer	Trial 4 (Geneva, 2016)	Trial 5 (Ithaca, 2016)
		DAP	DAP
Copper octanoate (M1)	Cueva (4.7 liters); Certis USA	59, 67, 77, and 84	41, 48, 57, and 64
<i>Bacillus amyloliquefaciens</i> strain D747 (44)	Double Nickel LC (2.3 liters); Certis USA	59, 67, 77, and 84	41, 48, 57, and 64
Copper octanoate (M1) + <i>B. amyloliquefaciens</i> strain D747 (44)	Cueva (4.7 liters) + Double Nickel LC (2.3 liters); Certis USA	59, 67, 77, and 84	41, 48, 57, and 64
<i>B. mycoides</i> isolate J (44)	Lifegard (3.5 g); DuPont	59, 67, 77, and 84	41, 48, 57, and 64
Polyoxin-D zinc salt (19)	Oso (0.5 liters); Certis USA	59, 67, 77, and 84	41, 48, 57, and 64

^a Fungicide Resistance Action Committee code.

Survival analysis was conducted by first calculating Kaplan-Meier estimates (Kaplan and Meier 1958) of the effect of fungicides on the time-to-death of leaves using PROC LIFETEST (SAS Version 9.4; Cary, NC). The Kaplan-Meier estimator defined the proportion of leaves surviving to a specific point in time. The effect of treatment on the survival curves for leaves were tested using the nonparametric log-rank and Wilcoxon tests and by visual inspection of plots of the survival functions (Allison 1995).

Accelerated failure time models were generated for the data and compared using the Weibull, exponential, logistic, log-logistic, and lognormal distributions using PROC LIFEREG (SAS Version 9.4). The fit of the models between distributions was assessed for goodness-of-fit using the Akaike information criterion (AIC) calculated by $-2(\log\text{-likelihood}) = 2k$; k = number of parameters for each distribution; Afifi et al. 2004). Accelerated failure time analysis modeling was conducted within PROC LIFEREG.

Results

Relationships among multispectral canopy reflectance and other variables. Assessment of the robustness of the relationship between AUDPS and canopy reflectance within trials 2 and 3 were used to identify 830 nm as having the highest predictive value for use as a means of discriminating between treatments (Supplementary Table S1). The relationships between the other collinear variables were explored using 830 nm (Supplementary Table S2). Variability in AUDPS explained by the number of CLS lesions per leaf at final assessment varied between 90.1 and 92.8%. Coefficients of determination indicated that between 55.6 and 70.7% of the variation in canopy reflectance could be explained by the number of CLS lesions per leaf at the final assessment indicating a moderate to good level of precision. Significant linear relationships were also described between canopy reflectance at 94 DAP and the dry weight of foliage in both trials with fair to moderate precision ($R^2 = 47.7$ to 52.1%).

Efficacy of conventional fungicides. Trial 1 – Geneva (2015). Prior to the application of fungicides, in-row plant density ($P = 0.226$) and the number of lesions per leaf ($P = 0.86$) were not significantly different across the trial area. Within-row plant density varied between 31.4 and 35 plants/m. All fungicides significantly reduced the number of CLS lesions per leaf and AUDPS (Fig. 1A; Table 3). Azoxystrobin, benzovindiflupyr + difenoconazole, and cyprodinil + difenoconazole reduced the number of CLS lesions per leaf by 88.1, 92.3, and 89.9%, respectively, compared with the nontreated plots at the final assessment. These products also significantly reduced AUDPS between 83.8 and 88.1% compared with nontreated plots. Penthiopyrad provided relatively poor disease control reducing the number of CLS lesions per leaf and AUDPS by 34.3 and 28.1% compared with nontreated plots, respectively. AUDPS was not significantly different between the plots treated with fludioxonil and fluazinam. Mancozeb was also efficacious and reduced the number of CLS lesions per leaf by 76%, which was not significantly different from plots receiving azoxystrobin, benzovindiflupyr + difenoconazole, and cyprodinil + difenoconazole (Fig. 1A). Fungicides also had a significant effect on the dry weight of foliage at first harvest ($P = 0.009$) and regrowth following defoliation at 146 DAP ($P < 0.001$; Table 3). In plots receiving benzovindiflupyr + difenoconazole, the dry weight of foliage was increased by 128 and 141% at first and second harvests, respectively. The only other fungicide that increased the dry weight of foliage upon regrowth was azoxystrobin, with an increase of 92% compared with nontreated plots. Regrowth at 146 DAP in plots receiving penthiopyrad, cyprodinil + difenoconazole, fludioxonil, mancozeb, and fluazinam was not significantly different from nontreated plots. Fungicide application had no significant effect on in-row plant density (number of beets/row meter) at 104 DAP ($P = 0.467$) and 146 DAP ($P = 0.672$). Moreover, fungicides had no significant effect on the dry weight of roots ($P = 0.905$) and root shoulder diameter ($P = 0.593$) at 104 DAP (Table 3).

Trial 2 – Geneva (2016). All fungicides significantly reduced disease severity and AUDPS, and increased AUCRS compared with nontreated plots (Fig. 1B; Table 4). Benzovindiflupyr + difenoconazole was the most efficacious fungicide decreasing AUDPS and increasing

AUCRS by 86.7 and 19.3%, compared with nontreated plots, respectively. AUDPS in plots treated with azoxystrobin was not significantly different from boscalid and penthiopyrad. AUDPS was significantly lower in plots treated with fluxapyroxad + pyraclostrobin compared with boscalid, penthiopyrad, and azoxystrobin, with AUDPS reduced by 34.2% compared with nontreated plots. Propiconazole was also highly efficacious, reducing AUDPS by 64% compared with nontreated plots but was significantly lower than in plots receiving benzovindiflupyr + difenoconazole (Fig. 1B). AUCRS was significantly higher in plots treated with boscalid, fluxapyroxad + pyraclostrobin, penthiopyrad, and propiconazole, ranging from 3.5 to 7.4% over nontreated plots. AUCRS was not significantly different between azoxystrobin and nontreated plots (Table 4). The dry weight of foliage was also significantly

Fig. 1. Effect of conventional fungicides on temporal progress of *Cercospora* leaf spot (CLS) epidemics in small plot replicated trials at **A**, Geneva in 2015 (trial 1); **B**, 2016 (trial 2); and **C**, Ithaca, NY, in 2016 (trial 3). Values are the mean number of CLS lesions per leaf across treatments and the bars represent the standard error.

increased from fungicide application at both harvest dates. At the first harvest (94 DAP), the dry weight of foliage was significantly increased only from application of benzovindiflupyr + difenoconazole (108.7%) and fluxapyroxad + pyraclostrobin (60.6%) compared with nontreated plots. At 120 DAP, the dry weight of foliage was significantly increased from all fungicides compared with nontreated plots, but again was significantly more in plots treated with benzovindiflupyr + difenoconazole (209.8%). Increases in foliar dry weight associated with the remaining fungicides ranged from 96.2% (propiconazole) to 135.9% (fluxapyroxad + pyraclostrobin) compared with nontreated plots. Fungicides had no significant effect on the dry weight of roots, number of plants/m, and root shoulder diameter at first harvest (Table 4).

Trial 3 – Ithaca (2016). Similar results were obtained within trial 3 to those at Geneva (trial 2; Fig. 1C). The average number of CLS lesions per leaf at the final assessment ranged from 2.1 (benzovindiflupyr + difenoconazole) to 69.8 (nontreated plots) (Fig. 1C). AUDPS was significantly ($P < 0.001$) reduced by all fungicides, but benzovindiflupyr + difenoconazole was the most efficacious, resulting in a reduction of 97.3% compared with nontreated plots (Fig. 2A). Application of azoxystrobin resulted in a 27.1% reduction in AUDPS compared with nontreated plots, which was significantly higher than in plots treated receiving boscalid or penthiopyrad. Fluxapyroxad +

pyraclostrobin provided moderate disease control, reducing AUDPS by 67.4% compared with nontreated plots and significantly higher than plots treated with propiconazole (Fig. 2A).

Effect of conventional fungicides on time-to-death of leaves. Across the entire area of trials 2 and 3, 26 and 14.4% of leaves, respectively, had died by the final assessment. In trials 2 and 3, the mean survival time of leaves was 42.3 ± 2.1 and 25.3 ± 0.73 days, respectively. The effect of fungicide on survival curves of leaves in both years was significant ($P < 0.0001$; Wilcoxon [test statistics = -99.12 and -80.24 in trials 2 and 3, respectively] and log-rank tests [test statistics = -126.8 and -95.78 in trials 2 and 3, respectively]).

Accelerated failure time modeling provided a good fit to the data quantifying the effect of fungicides on time-to-death of leaves in both trials. In trial 2, comparisons of each model with variable distributions identified the Weibull distribution as providing the best fit (AIC = 160.9), whereas the AIC for all other distributions tested varied between 166.9 (logistic) and 353.6 (exponential). The Weibull distribution was also identified as providing the best fit (AIC = 127.56) compared with the other distributions. Visual inspection of the plots of the Kaplan-Meier survival curves and accelerated failure time models with a Weibull distribution indicated similar probabilities with both approaches and similar parameter estimates (Fig. 3).

Table 3. Efficacy of conventional fungicides for *Cercospora* leaf spot (CLS) control in table beet on severity and temporal epidemic progress, dry weight of foliage, and shoulder diameter of roots in a small plot, replicated trial at Geneva, NY, in 2015 (trial 1)

Treatment ^v	CLS lesions per leaf		Dry weight of foliage (g/m)		Root shoulder diameter (mm)
	104 DAP ^w	AUDPS ^x	104 DAP	146 DAP	104 DAP
Azoxystrobin	27.6 e	278 de	1,027 ab	371 a	48.0
Penthiopyrad	152.5 b	1,235 b	780 bc	233 b	57.2
Benzovindiflupyr + difenoconazole	18.0 e	204 e	1,252 a	465 a	46.0
Cyprodinil + difenoconazole	23.4 e	229 e	1,074 ab	251 b	46.0
Fludioxonil	111.6 c	897 bc	933 ab	235 b	44.6
Mancozeb	55.7 de	429 de	791 bc	193 b	42.9
Fluazinam	65.9 d	650 cd	879 bc	255 b	45.1
Nontreated	232.1 a	1,717 a	548 c	190 b	49.6
Statistics					
LSD ^y	38.1	387.7	339.4	125.7	na
P-value	<0.001	<0.001	0.009	<0.001	0.593 (ns)
CV (%) ^z	34	43	34	38.2	23.6

^v Means followed by the same letter are not significantly different ($P > 0.05$).

^w Days after planting.

^x Area under the disease progress stairs.

^y Least significant difference ($P = 0.05$); na = not applicable when $P > 0.05$.

^z Coefficient of variation.

Table 4. Efficacy of conventional fungicides for *Cercospora* leaf spot (CLS) control in table beet on temporal epidemic progress (area under the disease progress stairs; AUDPS) and canopy reflectance at 830 nm (area under the canopy reflectance stairs; AUCRS), the dry weight of foliage at two harvest timings, and dry weight of roots, number of plants/m, and root shoulder diameter at one harvest in a small plot, replicated trial at Geneva, NY, in 2016 (trial 2)

Treatment ^w	AUDPS	AUCRS	Dry weight of foliage (g/m)		Dry weight of roots (g/m)	Number of plants/m	Root shoulder diameter (mm)
			94 DAP ^x	120 DAP	94 DAP	94 DAP	94 DAP
Benzovindiflupyr + Difenoconazole	253 e	2,656 a	599 a	202 a	253	50	42.4
Boscalid	1,561 b	2,386 b	379 b	152.7 b	213	54	42.5
Fluxapyroxad + pyraclostrobin	1,250 c	2,392 b	461 ab	153.8 b	234	57.6	41.2
Azoxystrobin	1,707 b	2,307 bc	279 b	139.8 b	205	56.6	39.9
Propiconazole	683 d	2,371 b	368 b	127.9 b	268	45.4	43.6
Penthiopyrad	1,617 b	2,340 b	419 ab	146.3 b	176	58	39.0
Nontreated	1,899 a	2,227 c	287 b	65.2 c	236	48.6	42.9
Statistics							
LSD ^y	151.4	102.9	198.1	45.8	na	na	na
P-value	<0.001	<0.001	0.049	<0.001	0.362	0.831	0.451
CV (%) ^z	8.7	1.3	10.9	8.8	28.4	30.7	13.5

^w Means followed by the same letter are not significantly different ($P > 0.05$).

^x Days after planting.

^y Least significant difference ($P = 0.05$); na = not applicable when $P > 0.05$.

^z Coefficient of variation.

The effect of treatment was significant in both trials (Fig. 3; Table 5). In trial 2, estimated survival times ranged from 12.7 ± 0.53 to 78.1 ± 2.34 days in nontreated and benzovindiflupyr + difenoconazole-treated plots, respectively. In trial 4, the mean leaf survival time in plots treated with benzovindiflupyr + difenoconazole was estimated as 33.2 ± 0.39 days but was 11.3 ± 0.99 days in nontreated plots. In trial 2, the estimated leaf survival time was slightly longer in plots receiving propiconazole compared with fluxapyroxad + pyraclostrobin (Fig. 3; Table 5).

The magnitude of the Weibull scale parameters in both trials was greater than 0.3, indicating that the hazard rate of leaf death increased over the duration of the assessments. Based on the accelerated failure time models, application of benzovindiflupyr + difenoconazole reduced the time-to-death of leaves in trials 2 and 4 by 588.9 and 194%, respectively (calculated using the formula: $[e^{\beta} - 1] \times 100$; β = parameter estimate for benzovindiflupyr + difenoconazole; Table 5). Similarly, propiconazole reduced the time-to-death by 97 to 278%; and fluxapyroxad + pyraclostrobin by 109.6 to 141%, respectively, compared with the nontreated control.

Efficacy of OMRI-listed products. *Trial 4 – Geneva (2016).* All products with the exception of *B. mycoides* strain J and polyoxin-D salt significantly reduced disease severity when compared with the nontreated plots (Fig. 4A; Table 6). Disease severity in plots receiving applications of *B. amyloliquefaciens* D747 was significantly reduced by 27.9% compared with the nontreated plots. Disease severity in plots receiving copper octanoate was not significantly different than those receiving *B. amyloliquefaciens* D747. However, when copper octanoate and *B. amyloliquefaciens* D747 were applied together, disease severity was significantly reduced compared with plots treated with either product alone, and reduced by 50.9% compared with nontreated plots (Fig. 4A). Similar reductions in disease severity were obtained when evaluating AUDPS (Table 6).

The number of plants (in-row plant density) surviving until harvest was not significantly affected by treatment ($P = 0.661$). Reductions in disease severity obtained by all treatments translated to significant increases in the dry weight of foliage at 94 DAP. For example, application of copper octanoate + *B. amyloliquefaciens* D747 resulted in a 112.8% increase in the dry weight of foliage compared with

Fig. 2. Effect of **A**, conventional fungicides (trial 3) and **B**, OMRI-listed products (trial 5) on area under the disease progress stairs representing temporal disease progress of *Cercospora* leaf spot epidemics in small plot replicated trials at Ithaca, NY, in 2016. Line within the box indicates the median. Box boundaries represent the 25th and 75th percentiles. Abbreviations: B + D = benzovindiflupyr + difenoconazole; B = boscalid; F + P = fluxapyroxad + pyraclostrobin; A = azoxystrobin; Pr = propiconazole; Pe = penthiopyrad; CO + Ba = copper octanoate + *Bacillus amyloliquefaciens* D747; Bm = *B. mycoides* isolate J; Ba = *B. amyloliquefaciens* D747; PDS = polyoxin-D zinc salt; Non = nontreated.

Fig. 3. Survival functions of table beet leaves affected by *Cercospora* leaf spot in small plots at **A**, Geneva (trial 2) and **B**, Ithaca (trial 3), NY, treated with benzovindiflupyr + difenoconazole (solid); propiconazole (· · ·); fluxapyroxad + pyraclostrobin (· - · - ·); compared with nontreated (dashed). Kaplan-Meier estimates are represented in the step-diagrams. Fitted values using the accelerated failure time model with a Weibull distribution are represented by the smooth lines.

Table 5. Estimates of parameters and associated test statistics for the accelerated failure time models quantifying the effect of fungicides on the time-to-death of table beet leaves affected by *Cercospora* leaf spot at Geneva (trial 2) and Ithaca (trial 3), NY, in 2016

Parameter	df	Estimate (95% confidence limits)	Wald χ^2	$P > \chi^2$	T (days), mean $\pm$ standard error ^z
Trial 2					
Intercept	1	2.62 (2.53 to 2.72)	3,110.22	<0.0001	-
Benzovindiflupyr + difenoconazole	1	1.93 (1.72 to 2.15)	303.85	<0.0001	78.1 $\pm$ 2.34
Propiconazole	1	1.33 (1.18 to 1.47)	340.04	<0.0001	34.9 $\pm$ 1.22
Fluxapyroxad + pyraclostrobin	1	0.88 (0.76 to 1.01)	181.58	<0.0001	30.6 $\pm$ 1.37
Nontreated	0	0			12.7 $\pm$ 0.53
Scale parameter	1	0.31 (0.27 to 0.36)	-	-	
Weibull shape parameter	1	3.21 (2.81 to 3.68)	-	-	
Trial 3					
Intercept	1	2.71 (2.60 to 2.82)	2,458.05	<0.0001	-
Benzovindiflupyr + difenoconazole	1	1.08 (0.90 to 1.25)	145.09	<0.0001	33.2 $\pm$ 0.39
Propiconazole	1	0.68 (0.53 to 0.82)	83.35	<0.0001	27.1 $\pm$ 0.85
Fluxapyroxad + pyraclostrobin	1	0.74 (0.59 to 0.89)	100.10	<0.0001	29.6 $\pm$ 0.75
Nontreated	0	0			11.3 $\pm$ 0.99
Scale parameter	1	0.33 (0.29 to 0.37)			
Weibull shape parameter	1	3.02 (2.67 to 3.42)			

^z Means and standard errors of median survival times.

Fig. 4. Effect of OMRI-listed products on temporal progress of *Cercospora* leaf spot (CLS) epidemics in small plot replicated trials at **A**, Geneva (trial 4), and **B**, Ithaca, NY (trial 5), in 2016. Values are the mean number of CLS lesions per leaf across treatments and standard error.

Table 6. Efficacy of Organic Materials Review Institute-listed products for the control of *Cercospora* leaf spot (CLS) in table beet on disease severity and temporal epidemic progress, the dry weight of foliage and roots, number of plants/m, and root shoulder diameter at harvest in a small plot, replicated trial at Geneva, NY, in 2016 (trial 4)

Treatment ^v	CLS lesions per leaf		Number of plants/m	Dry weight of foliage (g/m)	Dry weight of roots (g/m)	Root shoulder diameter (mm)
	87 DAP ^w	AUDPS ^x	94 DAP	94 DAP	94 DAP	94 DAP
Copper octanoate + <i>Bacillus amyloliquefaciens</i> D747	40.5 d	733 e	35.8	334.9 a	295	42.2
Copper octanoate	66.6 bc	1,319 cd	46.2	294.7 ab	294.7	38.5
<i>B. mycoides</i> isolate J	74.1 ab	1,457 c	46.2	244.4 c	244.4	39.8
<i>B. amyloliquefaciens</i> D747	59.4 c	1,193 d	42.6	250.5 bc	250.5	39.2
Polyoxin-D zinc salt	84.4 a	1,786 a	47.8	251.6 bc	251.6	38.9
Nontreated	82.4 a	1,806 a	35.2	157.4 d	237.5	42.9
Statistics						
LSD ^y	10.5	196.7	na	48.9	na	na
P -value	<0.001	<0.001	0.661 (ns)	<0.001	0.142 (ns)	0.768 (ns)
CV (%) ^z	4.7	11	22.3	14.5	16	14.5

^v Means followed by the same letter are not significantly different ($P > 0.05$).

^w Days after planting.

^x Area under the disease progress stairs.

^y Least significant difference ($P = 0.05$); na = not applicable when $P > 0.05$.

^z Coefficient of variation.

nontreated plots. Despite the lack of disease control from application of polyoxin D salt and *B. mycoides* strain J, these treatments resulted in increases in the dry weight of foliage of 55.2 and 59.9% compared with nontreated plots, respectively. The dry weight of roots ($P = 0.142$) and root shoulder diameter ($P = 0.768$) were not significantly affected by treatment (Table 6).

Trial 5 – Ithaca (2016). The efficacy of OMRI-listed products demonstrated at Geneva (trial 4) was also similar as measured by AUDPS within the trial at Ithaca (Figs. 3B and 4B). Polyoxin-D salt provided only moderate disease control reducing AUDPS by 15.7% compared with nontreated plots. Application of copper octanoate or *B. amyloliquifaciens* D747 alone significantly reduced AUDPS by 51.1 and 57.9% compared with nontreated plots, and were not significantly different from each other. Application of *B. amyloliquifaciens* D747 + copper octanoate significantly reduced AUDPS compared with the use of either single product, and was 85.3% less compared with nontreated plots (Fig. 3B). Similar effects between treatments were reflected in CLS lesions per leaf at the final assessment (Fig. 4B).

Discussion

This study expanded our knowledge of the tactics available to growers for the control of CLS in conventional and organic production of table beet. Efficacious OMRI-listed products may also have a place in conventional table beet production because of the tendency of *C. beticola* to develop resistance to single site modes of action (e.g., strobilurins; Kirk et al. 2012; Vaghefi et al. 2016). Applications of azoxystrobin resulted in variable disease control across the trials. For example, in trial 1, azoxystrobin was one of the most efficacious products, decreasing disease severity by 88.1% compared with nontreated plots. However, in trials 2 and 3, disease severity in plots receiving azoxystrobin was significantly less than in nontreated plots but overall provided poor disease control, reducing disease severity by 10 to 15%. The variable disease control with a consistent mixture of isolates within the inoculum suggests the potential for a deleterious pleiotropic effect (i.e., fitness cost) associated with this mutation (Jeger et al. 2008). As defined by Shaner et al. (1992), fitness can be defined as the survival and reproductive success of a specific fungal isolate and as such, reduced virulence may be associated with strobilurin resistance in *C. beticola*. This phenomenon has been reported in some fungi (e.g., Fisher and Meunier 2008; Karaoglanidis et al. 2011) but may be species and isolate-specific (Avila-Adame and Köller 2003; Fernández-Ortuno et al. 2008). Different environmental conditions between the two years may have also had a substantial effect on the virulence of specific *C. beticola* isolates. In table beet, azoxystrobin continues to be essential at planting and establishment for the control of pocket rot caused by *Rhizoctonia solani* (Gugino et al. 2008). To reduce the exposure and continued selection of strobilurin-resistant *C. beticola* isolates, identification of alternative products for pocket rot control to reduce the usage of strobilurin fungicides in the cropping system should be a priority. Information on the fitness of local strobilurin-resistant *C. beticola* isolates is also important to understand the persistence of isolates with this mutation in the population over time and inform future disease management strategies.

Apart from benzovindiflupyr + difenoconazole, the efficacy of FRAC group 7 fungicides, applied either alone or in combination with active ingredients from other FRAC groups, was generally poor. The suboptimal disease control from penthiopyrad was surprising considering its recent registration for CLS control in sugar beet in the United States. In Canada, penthiopyrad also failed to provide control of CLS in sugar beet (Trueman 2014). The relatively poor disease control resulting from other FRAC group 7 products (e.g., boscalid and fluxapyroxad + pyraclostrobin) is also concerning. There are no reports of resistance to succinate dehydrogenase inhibitors within *C. beticola*. To investigate the poor field performance of FRAC 7 fungicides, further work should concentrate on identifying mutations within the *sdh* genes associated with resistance (Sierotzki and Scalliet 2013). The availability of the *C. beticola* genome will facilitate this research (Vaghefi et al. 2017a). Benzovindiflupyr + difenoconazole was highly and consistently effective at significantly reducing disease severity and temporal epidemic progress, resulting in extended

survival of leaves and increased dry weight of foliage, prolonging opportunities for mechanized harvesting into autumn. Benzovindiflupyr + difenoconazole (as Aprovia Top) was registered within the United States in 2015 and is undergoing approval for use in New York. To facilitate further assessments of the utility of this product in table beet disease management, research should focus on quantifying the in vitro baseline sensitivity of local *C. beticola* populations and field performance to either active ingredient. The absence of significant effects from benzovindiflupyr + difenoconazole on root yield and size is highly encouraging for the table beet processing industry, which requires longevity of foliage in the absence of significant changes in root yield parameters due to strict size regulations for inclusion in cans and jars (USDA 2017). Other agronomic factors, such as within row planting density and between row spacing, have been shown to strongly influence root yield and size gradings (Kikkert et al. 2010).

Differential demethylation inhibitor efficacy within *C. beticola* has been reported, and resistant strains were found to have lower competitive or saprophytic survival ability (Karaoglanidis et al. 2001, 2002). A demethylation inhibitor, propiconazole, is also registered for CLS control in table beet in New York so careful rotational guidelines are required to avoid overuse of demethylation inhibitors as well as resistance development.

The consistent and likely synergistic disease control obtained from combining the OMRI-listed products, copper octanoate + *B. amyloliquifaciens* D747, was encouraging for use in conventional and organic table beet production (Flemming and Trevors 1989). The inclusion of microbial biopesticides in rotational integrated pest management programs, which was not tested in this study, may also be beneficial (Jacobsen et al. 2004). For example, *B. mycoides* isolate J was demonstrated to provide control of CLS in sugar beet due to the induction of systemic resistance (Bargabus et al. 2002).

Considering the biorational product efficacy trials were conducted under high and homogenous disease severity due to inoculation of the trial areas with *C. beticola*, the reproducibility of these results for CLS control is highly encouraging as these types of products may be highly variable in field performance between locations. One potential reason for the reproducible disease control of the biorational products in this study is their well-timed applications. In the current trials, the products were applied as protectants prior to inoculations with relatively short intervals between applications (Table 2). In-field performance of these (and other) products may be reduced if applied after symptoms occur and with longer application intervals (McGrath 2004). The inclusion of biorational products in rotation with conventional fungicides as a resistance management strategy could reduce the probability of resistance to single-site mode of action fungicides (van den Bosch et al. 2014).

Further research should examine the potential for application scheduling through forecasting systems to promote use of conventional and OMRI-listed products when not economically warranted (e.g., Windels et al. 1998). In conventional table beet production, this would reduce exposure of the *C. beticola* population to single site mode of action fungicides (Metz and Khan 2017). In organic table beet production, this approach would potentially reduce the use of copper-based products and decrease buildup in soil and the potential for phytotoxicity.

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