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Sushmita Kalika Singh
a) Department of Plant Pathology, North Dakota State University, Fargo, ND 58102, USA
b) Department of Biology, Faculty of Natural Science, University of Guyana, Berbice Campus, Tain, Corentyne, Guyana

Luis Del Rio Mendoza
Department of Plant Pathology, North Dakota State University, Fargo, ND 58102, USA

Mohamed Khan
Department of Plant Pathology, North Dakota State University, Fargo, ND 58102, USA

Correspondence Author:
Sushmita Kalika Singh
a) Department of Plant Pathology, North Dakota State University, Fargo, ND 58102, USA
b) Department of Biology, Faculty of Natural Science, University of Guyana, Berbice Campus, Tain, Corentyne, Guyana

Characterization of the sensitivity of *Cercospora beticola* to fungicides commonly used to manage cercospora leaf spot of sugar beet in the Red River Valley

Sushmita Kalika Singh, Luis Del Rio Mendoza and Mohamed Khan

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Abstract
Cercospora leaf spot (CLS), caused by *Cercospora beticola*, is a major foliar disease of sugar beet (*Beta vulgaris* L.) in the Red River Valley, often reducing yield and sucrose content by up to 50%. Current management relies on resistant cultivars and fungicides, though resistance has developed to some chemistries, including triphenyltin hydroxide (TPTH). This study assessed fungicide sensitivity in 40 *C. beticola* isolates collected from a commercial sugar beet field in Foxhome, Minnesota, during 2022. Isolates were cultured on CV-8 media amended with Headline (QoI), Proline (DMI), Champ® WG, Manzate Max, and Supertin (TPTH) at 0.01-800 ppm. After 14 days at 23 °C, EC_{50} values revealed low sensitivity to Manzate Max (8 ppm) and Supertin (2 ppm), but reduced sensitivity to Champ® WG (70 ppm), Headline (101 ppm), and especially Proline (496 ppm). Greenhouse assays confirmed poor performance of Proline in disease suppression. Together, the results highlight emerging fungicide resistance and stress integrated management strategies

Keywords: Cercospora leaf spot, *Cercospora beticola*, fungicide sensitivity, resistance management, sugar beet

Introduction
Sugar beet (*Beta vulgaris* L.) is a cornerstone of North Dakota's agricultural economy, with the Red River Valley ranking among the leading sugar beet-producing regions in North America (Farahmand *et al.*, 2013) [6]. Despite its economic importance, sugar beet production is threatened by Cercospora leaf spot (CLS), caused by the hemi-biotrophic fungus *Cercospora beticola*. This disease substantially reduces root yield and sucrose quality while increasing production costs due to intensive management requirements (Secor *et al.*, 2021) [24].

Management of CLS integrates the use of crop rotation, deployment of resistant cultivars, and fungicide treatments (Secor *et al.*, 2010) [21]. In the Red River Valley, CLS typically develops during the latter half of the growing season, often necessitating multiple fungicide applications to achieve adequate control (Khan *et al.*, 2007; Secor *et al.*, 2021) [16, 21]. Fungicides registered for sugar beet belong to several chemical groups classified by the Fungicide Resistance Action Committee (FRAC) according to their mode of action. These include thiophanate-methyl (methyl benzimidazole carbamates, FRAC 1), triphenyltin hydroxide and other organotin compounds (FRAC 30), quinone outside inhibitors (QoIs, FRAC 11, e.g., pyraclostrobin), and demethylation inhibitors (DMIs, FRAC 3, e.g., prothioconazole) (FRAC, 2020; Secor *et al.*, 2010) [7, 26]. To reduce the risk of resistance development, most DMI and QoI ungicides are applied alongside multi-site fungicides, for example, mancozeb (FRAC M03) or copper hydroxide (FRAC M01) (Secor *et al.*, 2020) [23].

Prolonged and extensive use of QoI and DMI fungicides has driven the emergence of resistant *C. beticola* populations in major sugar beet-growing areas including the United States (Kirk *et al.*, 2012) [17], Canada (Trueman *et al.*, 2017) [29], Serbia (Trkulja *et al.*, 2017) [28], and Greece (Karaoglanidis *et al.*, 2000; Nikou *et al.*, 2009) [14, 20]. Reduced sensitivity to organotin fungicides has also been reported (Hernandez *et al.*, 2023) [10] and isolates with dual or multiple fungicide resistance have been detected (Secor *et al.*, 2016; Trkulja *et al.*, 2017) [28].

The persistence of fungicide resistance in *C. beticola* is affected by how well resistant strains perform. Fitness costs are studied in plant pathogens using both *in vitro* measures such as mycelial growth and sporulation, and *in vivo* traits such as pathogenicity and aggressiveness (Cox *et al.*, 2007; Dekker, 1976) [4, 5]. While fungicide resistance can confer a selective advantage in treated fields, resistance mutations may also disrupt key biological and chemical processes within the organism, reducing pathogen fitness (Anderson, 2005) [2]. Evaluating such fitness costs is therefore critical to predicting the stability of fungicide resistance and establishing sustainable disease management practices (Mikaberidze and McDonald, 2015) [19].

Given the economic significance of CLS and the increasing prevalence of fungicide resistance, assessing the sensitivity of *C. beticola* to frequently used fungicides is crucial. This study aimed to examine the responses of *C. beticola* isolates to prothioconazole, pyraclostrobin, triphenyltin hydroxide, copper hydroxide, and mancozeb. The findings offer valuable information on the current resistance patterns and contribute to creating more robust and durable strategies to control diseases.

Materials and Method

Sampling and Isolation

Following the severe CLS epidemic in 2016, symptomatic sugar beet leaves were collected annually from cultivated areas across North Dakota to provide *C. beticola* inoculum for field trials. This annual collection ensured that inoculum remained representative of diverse field populations statewide. Upon the completion of the 2022 growing season, symptomatic leaves were collected from a research trial in Foxhome, Minnesota, which evaluated 13 fungicide treatments. For each treatment, five symptomatic leaves were collected and dried. Approximately five lesions per leaf were excised with sterile scissors, placed in sterile Petri dishes, and treated with 50 µL of an isolation buffer (4 µL ampicillin, 1.2 µL Tween 20, and 1994.8 µL sterile distilled water). A suspension of spores was spread on water agar modified with ampicillin (2 mL/L) using the spread-plate technique and incubated at 22±2 °C in darkness for 48 h to promote single spore germination. Germinated spores were transferred to half-strength clarified V8 (CV8) agar, where pure cultures were established and maintained at 22±2 °C in darkness for 20 days.

Controlled Laboratory Assays for Fungicide Sensitivity

A total of 40 *C. beticola* isolates were evaluated for sensitivity to five fungicides: Proline® (Bayer CropScience; 41% prothioconazole, FRAC 3, DMI), Headline® (BASF; 23.6% pyraclostrobin, FRAC 11, QoI), Champ® WG (Nufarm; 37.5% copper hydroxide, FRAC M01), Manzate® Max (UPL; 37% mancozeb, FRAC M03), and Supertin® (UPL; 80% triphenyltin hydroxide, FRAC 30). Stock solutions were prepared based on active ingredient concentrations to generate final assay media concentrations ranging from 0.01 to 800 µg/mL.

Five-millimeter agar pieces were taken from the actively growing edges of 14-day-old cultures and placed upside down onto 100 × 15 mm Petri dishes containing CV8 agar supplemented with the designated fungicide levels. The plates were maintained in darkness at 22±2 °C. The experiment was structured as a randomized complete block design (RCBD) and conducted in three consecutive trials to calculate the effective concentration (EC_{50}) values.

1. **Experiment 1:** Concentrations of 0.01, 0.1, 1, and 10 µg/mL were tested. EC_{50} values were obtained only for triphenyltin hydroxide and mancozeb.
2. **Experiment 2:** Copper hydroxide and prothioconazole were evaluated at 0, 25, 50, 100, 200, and 500 µg/mL, and 0, 5, 10, 25, 50, and 75 µg/mL, respectively. EC_{50} values could only be calculated for copper hydroxide.
3. **Experiment 3:** Prothioconazole was further tested at 0, 50, 100, 200, 400, and 800 µg/mL. Additionally, nine isolates were tested against pyraclostrobin at 1-400 µg/mL.

To inhibit the fungal alternate respiration pathway (Shi *et al.*, 2020) [27], 100 µg/mL salicylhydroxamic acid (SHAM) dissolved in methanol was added to all fungicide treatments except controls for pyraclostrobin. Colony growth was measured by diameter following a 14-day incubation period, once growth reached approximately two-thirds of the Petri dish. Two perpendicular measurements were taken per colony using a Sangabery six-inch caliper. Each experiment was performed twice, with two replicates per treatment plus an untreated control.

In vivo Fungicide Sensitivity Assays

Greenhouse trials were conducted at the Jack Dalrymple Agricultural Research Complex, North Dakota State University, under a 14-h photoperiod and 25±2 °C. A CLS-susceptible variety, Crystal 912, was planted in 10 × 10 × 12 cm pots filled with Sunshine Mix 1 (Sun Gro Horticulture Ltd., Alberta, Canada). Each pot was sown with three seeds, and after germination, only the most robust seedling was retained.

Based on *in vitro* EC_{50} values, isolates were grouped by fungicide sensitivity: two categories (most vs. least sensitive) for triphenyltin hydroxide and mancozeb, and three categories (most sensitive, moderately sensitive, least sensitive) for copper hydroxide, prothioconazole, and pyraclostrobin. For inoculum preparation, four mycelial plugs from 14 days of growth, cultures were placed into 400 mL CV8 broth and incubated on a shaker. After incubation, propagules were blended, filtered through sieve cloth, and placed in spray bottles for plant inoculation.

When plants reached the six-leaf stage, they were treated with fungicides at multiple concentrations, including recommended field rates. Treatments included: triphenyltin hydroxide (1, 10, 1472 µg/mL), mancozeb (1, 10, 8708 µg/mL), copper hydroxide (100, 500, 5512 µg/mL), prothioconazole (1, 400, 1074 µg/mL), and pyraclostrobin (1, 400, 977 µg/mL). Fungicides were applied using a calibrated sprayer (De Vries Manufacturing, Hollandale, MN, USA) at 138 kPa with a TeeJet 8001E nozzle.

Plants treated with triphenyltin hydroxide, mancozeb, and copper hydroxide were inoculated 24 h after fungicide application, as these fungicides act primarily preventively. In contrast, plants treated with prothioconazole and pyraclostrobin were inoculated immediately after application to test curative activity, as both fungicides are systemic. Inoculation involved spraying leaves until uniformly saturated. Following inoculation, plants were incubated in mist chambers for 48 h at 95-100% RH, 25±2 °C, and a 14-h photoperiod. Following incubation, the

plants were relocated to the greenhouse and watered at the base to prevent cross contamination.

The level of infection was visually evaluated at 5, 10, 15, and 21 days post-inoculation using the 1-10 scale of Jones and Windels (1991), where ratings correspond to increasing lesion counts and percent leaf area affected (1 = 1-5 spots/leaf, 10 = >200 spots/leaf or 50% severity). The experiment followed an RCBD with three replicates per treatment and was repeated twice.

Data Analyses

For each fungicide, concentration, trial, isolate, and replication; colony diameters were averaged and used to calculate percent growth suppression relative to growth on non-amended media. Nonlinear regression analysis was performed to estimate the concentration needed to suppress 50% of colony growth (EC_{50}) compared with the untreated control. Levene's test was used to assess homogeneity of variances within each fungicide group to find out if data from separate trials could be combined. Homogenous variances were pooled and analyzed using analysis of variance (ANOVA) under the Generalized Linear Mixed Model (GLIMMIX) procedure in SAS 9.4 (SAS Institute, Cary, NC, USA). Differences between means were evaluated using the Tukey-Kramer post hoc test with a significance level of $P = 0.05$.

In the greenhouse-based experiments, disease severity ratings (1-10 scale) collected at 5, 10, 15, and 21 days post-inoculation served to determine the area under the disease progress curve (AUDPC). Homogeneity of variances was confirmed with Levene's test prior to analysis. ANOVA was performed using PROC GLIMMIX in SAS 9.4 to evaluate the main effects of fungicide concentration, isolate sensitivity category (sensitive vs. insensitive), and their interaction on AUDPC values. Treatment means, including significant interactions, were evaluated with the Tukey-Kramer post hoc test at $P = 0.05$.

Results

Laboratory Evaluation of *Cercospora beticola* Responses to Various Fungicide Classes

Levene's test for homogeneity of variances indicated difference between the trials for triphenyltin hydroxide ($P = 0.11$), mancozeb ($P = 0.70$), copper hydroxide ($P = 0.46$), prothioconazole ($P = 0.67$), and pyraclostrobin (0.25) were not significant. Therefore, a combined analysis of trials for each fungicide was conducted. Sensitivity to all compounds was initially evaluated using concentrations from 0.01 to 10 µg/mL. At a concentration of 10 µg/mL, triphenyltin hydroxide suppressed the colony growth of all 40 isolates, with a mean EC_{50} of 2 µg/mL. The most sensitive isolate had an EC_{50} of 0.7 µg/mL, while the least sensitive isolate showed an EC_{50} of 6 µg/mL. Mancozeb had a mean EC_{50} value of 8 µg/mL (Table 1), with the most and least sensitive isolates displaying EC_{50} values of 5 µg/mL and 10 µg/mL, respectively. For the other fungicides, copper hydroxide and prothioconazole, EC_{50} values could not be determined in this experiment due to insufficient inhibition within the tested concentrations.

A subsequent experiment was performed to evaluate copper hydroxide and prothioconazole, using concentrations ranging between 25 and 500 µg/mL for copper hydroxide and from 5 to 75 µg/mL for prothioconazole. At 100 µg/mL, copper hydroxide effectively suppressed mycelial growth, resulting in a mean EC_{50} of 70 µg/mL (Table 1). The most sensitive isolate had an EC_{50} of 33 µg/mL, while the least sensitive isolate had an EC_{50} of 89 µg/mL. The EC_{50} for prothioconazole, however, could not be calculated. In other words, the tested concentrations were insufficient to reduce mycelial growth by 50%.

To determine the EC_{50} for prothioconazole, a third experiment was conducted using concentrations from 50 to 800 µg/mL. Growth suppression was observed at 800 µg/mL, with a mean EC_{50} of 496 µg/mL (Table 1). The most sensitive isolate had an EC_{50} of 124 µg/mL, while the least sensitive isolate had an EC_{50} of 797 µg/mL.

Table 1. Fungicide sensitivity of *Cercospora beticola* isolates based on Effective Concentration at 50% inhibition (EC_{50}) in µg/mL.

Fungicide	FRAC Code	Recommended Field Rate (µg/mL)	Sensitivity to the Fungicide (EC_{50} (µg/mL)	
			Mean	Range
triphenyltin hydroxide	30	1472	2	0.7 - 6
mancozeb	M03	8708	8	5 - 10
copper hydroxide	M01	5512	70	33 - 89
prothioconazole	3	1074	496	124 - 797
pyraclostrobin	11	977	101	8 - 256

For pyraclostrobin, Levene's test confirmed homogeneity of variances for both EC_{50} values ($P = 0.25$) and trials ($P = 0.73$). Supplementation with SHAM (100 µg/mL) did not significantly affect growth compared with non-amended media ($P = 0.07$), suggesting no measurable impact of alternative respiratory pathway inhibition. Pyraclostrobin EC_{50} values varied widely, with an overall mean of 101 µg/mL. At 200 µg/mL, however, pyraclostrobin consistently inhibited >50% of mycelial growth across all isolates.

The GLIMMIX ANOVA revealed significant differences in fungicide sensitivity ($P<0.05$). Tukey-Kramer post hoc tests separated isolates into two groups: six isolates with lower EC_{50} values (greater sensitivity) and three isolates with elevated EC_{50} values (reduced sensitivity) (Table 2). These results highlight heterogeneity in pyraclostrobin sensitivity among *C. beticola* populations.

Table 2. Effective Concentration at 50% inhibition (EC_{50}) of *Cercospora beticola* isolates in responses to pyraclostrobin.

Isolate	[a]EC_{50} (µg/mL)
108B	8 b
110A	23 b
211C	24 b
207B	29 b
112	45 b
102	54 b
412	219 a
110A	252 a
407A	256 a

aEC50 alues assigned the same letter did not differ significantly at $P = 0.05$ using Tukey Kramer test.

***In vivo* Assessment *C. beticola* Sensitivity to Different Fungicide Classes**

Levene's test was performed to evaluate variance homogeneity across trials, with results showing no significant differences ($P>0.05$ for all fungicides), indicating consistent variances. Consequently, data from all trials were combined for further analyses. The test yielded the following P-values: triphenyltin hydroxide (0.64), mancozeb (0.19), copper hydroxide (0.13), prothioconazole (0.07), and pyraclostrobin (0.59).

A combined ANOVA assessed the treatment effects

Where prothioconazole was the only fungicide where the status of the isolates was statistically significant (P = 0.04). Fungicide concentration was not significant (P = 0.18), but the interaction between status and concentration was significant (P = 0.02). For all other fungicides, status was not significant ($P>0.19$), concentration had a significant effect ($P<0.001$), and the interaction was not significant ($P>0.11$). The study evaluated the efficacy of these fungicides against *C. beticola* isolates using Area under Disease Progress Curve (AUDPC) values to quantify cumulative fungal growth and disease intensity.

The AUDPC varied among isolates classified as least sensitive (LS), most sensitive (MS), or moderately sensitive (MoS) to different fungicides (active ingredients). For triphenyltin hydroxide, AUDPC values did not differ significantly between LS (1.6) and MS (1.9) isolates. Similarly, for mancozeb, LS isolates had an AUDPC of 1.4, while MS isolates had an AUDPC of 2.1, however, these differences were not statistically significant. Copper hydroxide exhibited minimal variation in AUDPC between LS (1.3) and MS (1.4) isolates. In contrast, prothioconazole-treated isolates showed a significant difference, with LS isolates exhibiting a lower AUDPC (1.3) compared to MoS isolates (2.4). Pyraclostrobin-treated isolates had variable responses, with LS isolates displaying an AUDPC of 0.9, MoS isolates at 1.1, and MS isolates at 1.1, indicating no significant effect of status on disease progression.

Copper hydroxide showed a less pronounced effect, with AUDPC values remaining similar at 0 µg/mL (1.8) and 100 µg/mL (1.8), decreasing to 1.2 at 500 µg/mL and 1.1 at field concentration. Isolates treated with prothioconazole did not exhibit a concentration dependent response, as AUDPC remained relatively stable across concentrations (1.5 at 0 µg/mL, 1.4 at 100 µg/mL, 1.8 at 400 µg/mL, and 1.8 at field concentration). However, pyraclostrobin showed a marked decline in AUDPC with increasing concentrations, from 1.5 at 0 µg/mL to 1.2 at 100 µg/mL, 0.7 at 400 µg/mL, and 0.8 at field concentration (Table 3.).

Table 3. Effect of concentration on Area under Disease Progress Curve (AUDPC) for the different fungicides.

Active Ingredient	Concentration (µg/mL)	[a]AUDPC
triphenyltin hydroxide	0	3.0 a
	1	2.0 b
	10	1.1 c
	1472 (Field Rate)	0.8 c
mancozeb	0	3.0 a
	1	1.7 b
	10	1.5 bc
	8708 (Field Rate)	1.0 c
copper hydroxide	0	1.8 a
	100	1.8 b
	500	1.2 b
	5512 (Field Rate)	1.1 b
prothioconazole	0	1.5 a
	100	1.4 a
	400	1.8 a
	1074 (Field Rate)	1.8 a
pyraclostrobin	0	1.5 a
	100	1.2 a
	400	0.7 b
	977 (Field Rate)	0.8 b

[a]AUDPC values that share a common letter for each active ingredient were not significantly different at $P = 0.05$ using Tukey Kramer test.

Discussion

C. beticola represents a persistent threat in sugar beet-producing regions, particularly under conditions favorable for CLS outbreaks on sensitive varieties. Repeated fungicide treatmenrs are typically required for efficacious management, but this enhances the likelihood of emerging resistance. Over the past two decades, the extensive use of QoI and DMI fungicides has led to widespread resistance in *C. beticola* populations in several countries, including Serbia (Trkulja *et al.*, 2017) [28], and Greece (Karaoglanidis *et al.*, 2000; Nikou *et al.*, 2009) [14, 20], Canada (Trueman *et al.*, 2014) [29], and the United States (Kirk *et al.*, 2012; Secor *et al.*, 2017) [17, 22].

QoI fungicides block mitochondrial respiration by attaching to the quinol oxidation (Qo) site of the cytochrome bc1 complex, disrupting electron transport and ATP production in sensitive isolates. The target, cytochrome b (cytb), is encoded by the mitochondrial cytb gene, and resistance has been most frequently associated with point mutations such as G143A, which prevent fungicide binding and confer varying levels of resistance (Fisher and Meunier, 2008) [8]. DMI fungicides, by contrast, target CYP51, is a crucial enzyme for ergosterol production. Resistance in *C. beticola* has been linked to mutations in this gene as well as increased expression of CbCyp51 in the CYP51 protein that reduces binding affinity (Bolton *et al.*, 2012) [3]. Reports of isolates harboring multiple resistance mechanisms (Secor 2017; Trkulja *et al.*, 2017) [22, 28] further complicate management.

In the Red River Valley, QoI fungicide failure combined with declining DMI efficacy contributed to a severe CLS epidemic in 2016 (Khan, 2018) [15]. Monitoring programs in subsequent years revealed that QoI resistance remained highly prevalent (up to 90%), while sensitivity to DMIs proceeded to decline from 2016 to 2021 (Secor *et al.*, 2024) [25]. These trends reinforce the urgency of diversifying disease management strategies.

The activity of various fungicides against *C. beticola* both laboratory and greenhouse conditions. A limitation of this work is the relatively small sample size (40 isolates), which may not capture the full spectrum of genetic diversity and resistance variability across field populations. Nonetheless, mycelial inhibition *in vitro*, measured as EC_{50} values, largely corresponded with disease suppression *in vivo*, assessed by AUDPC, suggesting that laboratory assays can serve as reliable predictors of field performance. However, certain fungicides exhibited high variability across isolates, underscoring the importance of sensitivity testing before large-scale applications.

Triphenyltin hydroxide was among the most effective fungicides tested, suppressing growth across all isolates *in vitro* and producing strong *in vivo* disease control. Yet, resistance risk remains a concern. Although the mean EC_{50} observed here was 2 µg/mL, consistent with strong activity, Secor *et al.*, (2024) [25] reported resistance frequencies as high as 98.9% at 1 µg/mL in 2021. In this study, 45% of isolates had EC_{50} values exceeding 1 µg/mL, aligning with this trend. Resistance management is therefore critical to prolong efficacy. Previous work suggests that reduced fitness resulting from resistance may decrease the stability of resistant strains gradually, without exposure to fungicide (Ishii, 2015) [12], but proactive management is essential. Reduced use of tin-based fungicides, in combination with rotation strategies, could help lower resistance frequencies (Secor *et al.*, 2024) [25].

Mancozeb showed relatively consistent activity, with EC_{50} values from 5 to 10 µg/mL (mean 8 µg/mL). AUDPC values did not differ significantly among isolates, indicating uniform efficacy *in vivo*. However, isolates capable of growing at 5 µg/mL are considered tolerant (Weiland, 2001) [32], suggesting that reduced sensitivity may already be present. Similar patterns have been reported in Turkey (Tümbek *et al.*, 2011) [31]. Given its multi-site mode of action, mancozeb is less vulnerable to resistance (Hollomon 2015) [11] and remains a valuable tool in integrated management (FRAC 2020) [7], though reliance on single modes of action should be avoided.

Copper hydroxide required considerably higher concentrations for inhibition, with EC_{50} values between 33 and 89 µg/mL (mean 70 µg/mL), far exceeding the <10 µg/mL values reported by Malandrakis *et al.*, (2019) [18]. Similarly, Aiming *et al.*, (2025) [1] found EC_{50} values of 2-8 µg/mL in isolates collected in 2017, suggesting the isolates in this study are significantly less sensitive (nearly 14-fold on average) than those previously reported. While copper-based fungicides remain important in resistance management programs, especially where QoI and DMI efficacy is declining (Friskop *et al.*, 2020) [9], the elevated EC_{50} values observed here indicate emerging insensitivity that warrants continued monitoring.

Prothioconazole displayed high variability *in vitro*, with mean EC_{50} values approaching 500 µg/mL, and *in vivo* trials confirmed poor efficacy, with no significant differences between untreated and treated plants at field concentrations. These results are consistent with earlier reports of declining DMI performance in the region (Karaoglanidis *et al.*, 2000; Secor *et al.*, 2010; Secor *et al.*, 2024) [14, 21, 25]. Moreover, recent monitoring in Michigan detected rising resistance factors across multiple DMIs, including difenoconazole, tetraconazole, and mefentrifluconazole (Hernandez *et al.*, 2023) [10]. These data suggest that reduced sensitivity to prothioconazole is already widespread, making reliance on DMIs increasingly risky.

Pyraclostrobin showed variable results among isolates. Although disease severity was reduced by nearly 50% at higher concentrations, differential performance across isolates reflects diminished efficacy and phenotypic evidence of QoI resistance. Similar resistance trends have been observed globally, frequently linked to the G143A alteration in the cytochrome b gene (Bolton *et al.*, 2012; Karadimos and Karaoglanidis, 2006) [3, 13]. Although this study did not conduct molecular screening, the patterns observed are consistent with resistance mechanisms documented elsewhere.

Conclusions: Collectively, these findings underscore the necessity of integrated management strategies. Fungicide programs should emphasize rotation among different modes of action, tank mixing with protectants such as mancozeb or triphenyltin hydroxide, and application timing at early disease onset, ideally before canopy closure, to optimize control and extend fungicide utility. Non-chemical strategies, including crop rotation and residue management, should complement chemical approaches to reduce early inoculum pressure. Regular sensitivity screening and adaptive fungicide programs tailored to local resistance

trends will be critical for sustaining effective CLS control while mitigating resistance development.

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