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Cercospora leaf spot of sugar beet: A review of biology, epidemiology, and management strategies

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Abstract
Cercospora leaf spot (CLS), caused by *Cercospora beticola*, is the most destructive foliar disease of sugar beet (*Beta vulgaris* L.) worldwide and one of the leading production challenge in the Red River Valley of the United States. Epidemics of CLS result in severe defoliation, root yield losses of up to 40%, and reduced sucrose quality. For decades, CLS management has relied on fungicides, particularly quinone outside inhibitors (QoIs) and demethylation inhibitors (DMIs). However, the intensive use of these single-site fungicides has led to the emergence of resistant *C. beticola* populations, causing a decline in fungicide efficacy and complicating management decisions. The spread of resistance across major sugar beet-growing regions underscores the urgent need for integrated disease management strategies that combine cultural practices, resistant cultivars, and strategic fungicide use. This review synthesizes current knowledge on the biology, epidemiology, host range, and disease cycle of *C. beticola*, with particular emphasis on major fungicide resistance mechanisms and their implications for CLS management to provide a comprehensive framework that informs sustainable CLS management and supports the development of effective resistance management programs.

Keywords: *Cercospora beticola*, pathogen, sugar beet, fungicides, management

Introduction
Sugar beet (*Beta vulgaris* L.) is a globally important crop, contributing nearly one-third of the world's sugar supply. Situated in eastern North Dakota and western Minnesota, the Red River Valley produces nearly 54% of the nation's sugar beet crop, underscoring its critical role in U.S. sugar production (Secor *et al*., 2013) [40]. However, *Cercospora* leaf spot (CLS), caused by *Cercospora beticola*, poses the greatest threat to production. The disease, first identified on sugar beet in the mid-20th century, has since become endemic in nearly all sugar beet-growing regions (Duffus and Ruppel, 1993) [10]. Under warm, humid conditions, CLS can cause up to 40% yield loss, increase storage losses, and reduce sucrose purity, resulting in significant economic and processing challenges for growers and the sugar industry (Weiland and Koch, 2004; Khan, 2018) [43, 22].
The biology and epidemiology of *C. beticola* contribute to its destructive potential. The pathogen overwinters in infected debris, produces large quantities of conidia that spread rapidly by wind and rain splash, and secretes non-host-specific toxins such as cercosporin that facilitate necrosis (Fajola, 1978; Khan, 2018) [11, 22]. As a polycyclic pathogen, *C. beticola* completes multiple infection cycles during a growing season, leading to rapid epidemic buildup under conducive conditions. Its broad host range further complicates management by maintaining inoculum reservoirs (Harveson, 2013) [17].
Management of CLS generally includes crop rotation, residue incorporation, and resistant cultivars, but fungicides remain the cornerstone of control (Secor *et al*., 2010) [38]. Since the 1970s, growers in North Dakota and Minnesota have relied on multiple fungicide applications per season, often exceeding six sprays in high-pressure areas. However, intensive use of single-site fungicides has imposed strong selection pressure on *C. beticola* populations (Kirk *et al*., 2012) [25]. Widespread resistance to benzimidazoles, QoIs, DMIs, and organotins has been reported which reduce fungicide efficacy, limit grower options, and elevate the risk of management failure (Bolton *et al*., 2013) [3].
With the continued reduction of fungicide efficacy, sustainable CLS management requires a deeper understanding of *C. beticola* biology, epidemiology, and resistance evolution, along with a critical evaluation of integrated control strategies. While numerous studies have addressed specific aspects of CLS, a comprehensive synthesis that connects pathogen

biology, fungicide resistance, and integrated management is needed to support coordinated and sustainable practices. This review aims to synthesize current knowledge of *C. beticola* biology, epidemiology, and host-pathogen interactions; evaluate the mechanisms and global status of fungicide resistance in *C. beticola* populations; and assess integrated management strategies that combine cultural practices, host resistance, and strategic fungicide use to mitigate resistance risks.

Cercospora Leaf Spot

Cercospora beticola, the causal agent of *Cercospora* Leaf Spot (CLS), was first described by Saccardo in Italy in 1876 and later reported as a sugar beet pathogen by Chupp in 1953. CLS is regarded as one of the most destructive foliar diseases of sugar beet worldwide, contributing to substantial yield and quality losses (Duffus and Ruppel, 1993; Weiland and Koch, 2004) [10, 43]. In the Red River Valley of eastern North Dakota and western Minnesota, an area responsible for more than half of U.S. sugar beet production, CLS remains a chronic challenge that continues to limit productivity (Windels *et al*., 1998; Secor *et al*., 2013) [45, 40]. The pathogen favors warm, humid conditions, under which epidemics can develop rapidly, and losses of up to 40% have been documented (Whitney and Duffus, 1986; Rossi *et al*., 2000; Saito, 1966; Khan, 2018) [44, 36, 37, 22]. Beyond yield reduction, the disease reduces sugar quality and complicates postharvest storage, thereby increasing processing costs. Affected plants also redirect sugar reserves from the taproot to support new leaf growth, further diminishing recoverable sugar yields (Shane and Teng, 1992; Holtschulte *et al*., 2010) [39, 18].

Taxonomic Classification of *Cercospora beticola*

- Kingdom: Fungi
- Phylum: Ascomycota
- Class: Dothideomycetes
- Order: Capnodiales
- Family: Mycosphaerellaceae
- Genus: *Cercospora*
- Species: *Cercospora beticola* Sacc. (Rangel *et al*., 2020) [34]

Biology and Epidemiology of *Cercospora beticola*

The conidia of *C. beticola* are slender, multiseptate, and needle-shaped, typically ranging from 2-3 × 36-107 µm. They arise on light brown, septate conidiophores that emerge in clusters from stromatic tissue (Weiland and Koch, 2004; Skaracis *et al*., 2010) [43, 41]. Disease development is favored by warm, humid, and rainy weather, conditions that promote both sporulation and dispersal. Conidia are disseminated primarily by wind, rain splash, and insects, allowing rapid spread during the growing season. Sporulation occurs optimally at 20-26 °C with relative humidity between 90-100%, while successful germination and infection require slightly warmer daytime temperatures (25-35 °C) and nighttime temperatures above 15 °C, combined with high humidity (Khan, 2018) [22]. Upon reaching the leaf surface, conidia germinate, producing germ tubes and appressoria that penetrate through stomata. Once inside, the fungus colonizes intercellular spaces and secretes phytotoxic metabolites such as cercosporin and beticolin, as well as lytic enzymes, which disrupt host tissues and lead to necrosis of sugar beet leaves (Daub and Ehrenshaft, 2000; Fajola, 1978) [9, 11].

The pathogen survives between growing seasons as pseudostromata on infected plant residues, which represent the primary inoculum source for local epidemics (Khan *et al*., 2008) [23]. These structures are resilient, persisting for more than three years on sugar beet or alternate host debris if left on the soil surface (Knight *et al*., 2019) [26]. Although no sexual stage has been observed, population studies suggest panmixia and potential recombination, highlighting the adaptive capacity of *C. beticola* (Groenewald *et al*., 2006; Bolton *et al*., 2012a) [15, 4]. CLS is polycyclic, with multiple infection cycles occurring within a single growing season. Inoculum arises from both carryover debris and active infections within the crop (Harveson, 2013; Khan, 2018) [17, 22]. Symptoms generally initiate on the lower canopy and progress upward, with the incubation period varying from 5 to 21 days depending on temperature, moisture, inoculum pressure, and host resistance (Saito, 1966; Rossi *et al*., 2000; Holtschulte *et al*., 2010) [18, 36, 37].

Symptoms

Cercospora leaf spot (CLS) typically begins as small, circular lesions that appear dark brown to purplish with a lighter brown or tan center, measuring about 2-5 mm in diameter at full expansion (Figure 1a). With disease progression, individual lesions enlarge and coalesce, producing extensive necrotic areas that can compromise the entire leaf surface (Figure 1b). Under warm and humid conditions, pseudostromata within the lesions give rise to conidiophores and conidia, resulting in a characteristic gray to steel-blue, felt-like growth on the spot surface (Asher and Hanson, 2006) [1]. A distinctive diagnostic trait is the presence of minute black pseudostromata embedded in the center of the grayish-tan lesions. In advanced infections, the merging of multiple spots leads to blighting, extensive leaf necrosis, and ultimately, premature leaf death (Harveson, 2013) [17].

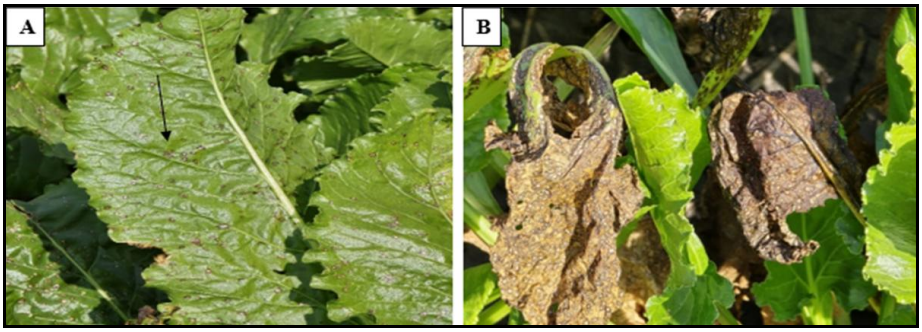

Fig 1: *Cercospora* leaf spot symptoms. A) Small, circular dark brown to purple lesions with a light brown or tan center, and B) individual spots merging to form larger necrotic areas, eventually leading to necrosis of the entire leaf.

Host Range

The pathogen *C. beticola*, is not restricted to sugar beet but infects a broad spectrum of hosts across the Chenopodiaceae and Amaranthaceae families (Weiland and Koch, 2004) [43]. Susceptible hosts include several *Beta* species and several weeds such as *Amaranthus*, *Chenopodium*, *Atriplex*, *Cyclamen*, *Plantago*, *Malva*, and *Limonium* (Groenewald *et al*., 2006; Lartey *et al*., 2010) [15, 28]. In addition, cultivated crops closely related to sugar beet such as table beet, Swiss chard, and spinach along with other plants like mallow, pigweed, bindweed, and wild *Beta* species, can also act as reservoirs for the pathogen (Weiland and Koch, 2004) [43].

Disease Cycle

As a polycyclic disease, CLS completes several successive infection cycles during one sugar beet season. The pathogen overwinters as pseudostromata within infected leaf debris, where it can persist for one to two years on or just below the surface of the soil. This survival strategy provides a major source of primary inoculum, particularly in fields with continuous beet cultivation and limited rotation (Khan *et al*., 2008) [23]. Additional inoculum reservoirs include infected weed hosts and, in some cases, beet seeds. Disease symptoms generally appear 5-21 days after infection, with onset strongly dependent on favorable environmental conditions (Windels *et al*., 1998) [45]. Lesions serve as centers for conidial production, enabling repeated secondary infection cycles throughout the season.

Conidia are disseminated by multiple agents such as rain splash, wind, irrigation water, insects, and mites with wind considered the dominant vector (Carlson, 1967) [7]. Under conducive field conditions, the disease cycle can be completed in as little as 12 days (Rossi *et al*., 2000; Weiland and Koch, 2004) [36, 43]. Infection begins when conidia settle on leaf surfaces and, given sufficient moisture, germinate and penetrate primarily through stomata. Fungal hyphae initially spread intercellularly in the apoplast, occupying spaces between plant cells and occasionally adhering to host cell walls, before progressing to an intracellular phase. Whether *C. beticola* functions mainly as an intercellular or intracellular pathogen remains unresolved (Daub and Ehrenshaft, 2000) [9].

During colonization, the fungus secretes several effectors, including the toxins cercosporin and beticolin. Cercosporin acts as a photosensitizer, producing reactive oxygen species under light, which results in oxidative stress and eventual cell death (Daub and Ehrenshaft, 2000) [9]. Beticolin, a non-protein toxin, disrupts cellular integrity by impairing energy production and damaging membranes, further contributing to symptom development (Blein *et al*., 1988) [2].

Management of *Cercospora* Leaf Spot

Effective management of *C. beticola* in sugar beet production relies on an integrated strategy that combines cultural practices, the use of resistant cultivars, and fungicide applications (Miller *et al*., 1994; Lamey *et al*., 1996; Secor *et al*., 2010) [30, 27, 38]. Among these, timely fungicide treatments remain a critical component for controlling CLS throughout the growing season (Khan *et al*., 2008; Secor *et al*., 2010) [23, 38].

Cultural Practices

In areas such as North Dakota and Minnesota, where CLS pressure is high, cultural methods form an essential part of disease management. Crop rotation is widely recommended to reduce the carryover inoculum of *C. beticola*. By alternating sugar beet with non-host crops such as small grains, corn, or beans over two to three years, growers can interrupt the disease cycle and lower the soil-borne pathogen load (Jacobsen and Franc, 2009; Khan *et al*., 2008) [19, 23]. Additionally, fall cultivation practices that incorporate infected leaf debris into the soil accelerate residue decomposition, thereby decreasing the survival of pseudostromata and limiting their impact on subsequent crops (Khan *et al*., 2008) [23].

Resistant Cultivars

The deployment of resistant sugar beet varieties represents another key pillar of CLS management. Through backcrossing with wild sea beets (Beta vulgaris subsp. maritima), breeders have introduced resistance genes into commercial cultivars. While these varieties are not completely immune, they effectively reduce infection rates and slow disease progression, offering growers a valuable tool for disease suppression (Rossi *et al*., 2000) [36]. The selection of cultivars is guided by extensive field trials and standardized assessment scales, such as the Klein Wanzlebener Saatzucht (KWS) scale, to ensure consistent performance under field conditions (Jones and Windels, 1991) [20]. Since 2021, improved sugar beet cultivars carrying the BvCR4 gene (CR+) that confers enhanced CLS tolerance have become available to producers, further strengthening integrated management options (Törjék *et al*., 2020) [42].

Fungicide Application

Fungicides remain a cornerstone of CLS management in sugar beet, particularly in regions experiencing high disease pressure. Since the 1970s, growers in North Dakota and Minnesota have relied on targeted fungicide programs to protect crops during periods of peak pathogen activity. Fungicides are categorized according to their mode of action (MOA), which defines the specific biochemical pathways they disrupt in the fungus. The primary classes used against CLS include methyl benzimidazole carbamates (MBC, FRAC group 1), quinone outside inhibitors (QoI, FRAC group 11), and demethylation inhibitors (DMI, FRAC group 3), along with multi-site fungicides such as organotins (FRAC group 30), inorganic copper compounds (FRAC group M01), and dithiocarbamates (FRAC group M03) (FRAC, 2020; Khan, 2018; Rangel *et al*., 2020) [14, 22, 34].

Effective control of CLS depends on the timely application of fungicides, generally initiated at the first signs of disease or under environmental conditions favorable for infection, often around late June to early July after row closure. Follow-up applications are typically made at 10-14 day intervals, with adjustments based on rainfall, disease progression, and field conditions (*Hakk et al*., 2016) [16]. However, frequent applications of single-site fungicides heighten the risk of resistance development in *C. beticola* populations.

Classified as a medium-risk pathogen for fungicide resistance (FRAC, 2019), *C. beticola* possesses high genetic variability, annual proliferation, and is exposed to repeated fungicide treatments averaging six to eight applications per season in high-pressure areas. These factors collectively increase the likelihood of resistance evolution. Indeed, reduced sensitivity and resistance to multiple fungicide

classes have been documented (Bolton *et al*., 2012b, 2013) [5, 3]. The continual use of single-site fungicides imposes strong selective pressure on the pathogen, accelerating the emergence of resistant populations (Eckert and Ogawa, 1988).

Managing Fungicide Resistance

Fungicide resistance represents a significant challenge in controlling CLS, as *C. beticola* produces numerous conidia over multiple infection cycles per season, increasing the probability of resistance mutations (FRAC, 2019). Resistance has been documented across several major fungicide classes, including benzimidazoles, quinone outside inhibitors (QoIs), demethylation inhibitors (DMIs), and organotins (Bugbee, 1996; Bolton *et al*., 2013; Secor *et al*., 2010) [6, 3, 38]. The repeated use of these fungicides, particularly single-site products without rotation or tank-mixing with complementary chemistries, has accelerated the evolution of resistant populations. Historical CLS epidemics in North Dakota and Minnesota occurring in 1981, 1998, and 2016 were associated with the failure of benzimidazoles, organotins, and QoIs, respectively (Khan *et al*., 2018; Secor *et al*., 2010) [22, 38]. Ongoing surveillance of *C. beticola* for shifts in fungicide sensitivity is essential to inform adaptive management strategies.

QoI Fungicides

Quinone outside inhibitors, including pyraclostrobin and trifloxystrobin, act by disrupting mitochondrial respiration through binding to the cytochrome bc1 complex and are considered high-risk for resistance development (FRAC, 2022) [13]. Pyraclostrobin initially offered effective control of CLS and enhanced sucrose yields (Khan and Smith, 2005; Secor *et al*., 2010) [24], but resistance emerged rapidly after its commercial introduction in 2002, with reduced sensitivity observed by 2004 (Secor *et al*., 2010) [38].

QoI Resistance Menchanism

The primary mechanism conferring resistance to QoI fungicides is the G143A mutation in the cytb gene, which encodes the cytochrome b protein, a critical component of the mitochondrial electron transport chain. This mutation leads to high levels of resistance and has been detected in over 90% of *C. beticola* isolates from Michigan (Rosenzweig *et al*., 2015) [35] and the Red River Valley (Rangel *et al*., 2020) [34], resulting in widespread QoI failures in CLS management programs.

DMI Fungicides

Demethylation inhibitors, such as tetraconazole, difenoconazole, prothioconazole, and propiconazole, act by inhibiting ergosterol biosynthesis, a key component of fungal cell membranes. DMIs are widely used due to their systemic activity and are classified as medium to high risk for resistance development (FRAC, 2022) [13]. Resistance evolution in this group is influenced by factors including local fungicide use patterns, pathogen population dynamics, and environmental conditions.

DMI Fungicide Resistance

Resistance to demethylation inhibitors (DMIs) has been documented in *C. beticola*, primarily through overexpression of the cyp51 gene (Bolton *et al*., 2012b) [5]. Additional mechanisms, including specific point mutations in the target enzyme and the action of drug efflux pumps, also contribute to reduced sensitivity (Nakaune *et al*., 1998) [32]. Unlike resistance to QoI fungicides, which often leads to complete loss of efficacy due to mutations in the cytochrome b gene that prevent fungicide binding, DMI resistance generally manifests as a gradual decline in sensitivity. This is because DMIs inhibit ergosterol biosynthesis, a critical component of fungal cell membranes, and mutations in the target enzyme reduce binding affinity, resulting in a slower erosion of control over time (Bolton *et al*., 2012b; Rangel *et al*., 2020) [5, 34].

Among the DMI fungicides, tetraconazole and difenoconazole tend to provide superior disease control compared to older compounds like propiconazole; however, the development of resistance remains a concern (Secor *et al*., 2010) [38]. The persistence of resistant *C. beticola* populations is influenced by the fitness of resistant isolates. Some studies indicate fitness costs associated with DMI resistance, including decreased virulence, spore production, and mycelial growth (Karaoglanidis *et al*., 2001; Moretti *et al*., 2003) [21, 31]. Conversely, other research reports no significant differences in competitive ability, germination, or disease severity between resistant and sensitive isolates (Bolton *et al*., 2012b; Nikou *et al*., 2009) [5, 33]. These mixed findings suggest that, although resistance may impose constraints in some instances, resistant populations can persist and remain competitive under field conditions.

To slow the development of resistance, implementing an integrated disease management approach is essential. Recommended strategies include rotating fungicides with differing modes of action, combining DMIs with multi-site fungicides, reducing overall application frequency, and applying cultural practices to minimize inoculum levels. Such a holistic approach is critical for sustaining fungicide efficacy and mitigating resistance risk in *C. beticola* populations (Corkley *et al*., 2022; van den Bosch *et al*., 2014) [8, 46].

Conclusion

This disease and pathogen remain a major constraint on sugar beet production due to its rapid, recurring infection cycles, broad host range, and ability to persist in crop debris. Although the use of resistant cultivars and cultural practices can reduce disease severity, fungicides continue to play a pivotal role in management. The increasing prevalence of resistance to single-site fungicides, including QoIs and DMIs, poses a significant threat to long-term disease control. Effective and sustainable management of CLS therefore depends on an integrated approach that combines crop rotation, resistant varieties, and strategic fungicide applications, such as rotating modes of action and combining single-site products with multi-site fungicides. Continuous monitoring of pathogen populations and adaptive management strategies are essential to preserve fungicide effectiveness while maintaining both yield and sugar quality in sugar beet production.

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