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Brown rot caused by *Monilinia* spp. on pome and stone fruits across Europe

Aneliya Raykova

Institute of Agriculture-Kyustendil, Department of Agrotechnology, Plant Protection and Economy on Crops, Kyustendil 2500, Agricultural Academy – Sofia Bulgaria;

*Corresponding author: aneliyaraykova@outlook.com

Aneliya Raykova ORCID ID: 0000-0003-1672-3345

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Abstract: The widespread cultivation of stone and pome fruits (*Rosaceae*) underlines their importance in the global market and their significant contribution to the fruit processing industry. However, destructive fungal pathogens from the genus *Monilinia* are causing Brown rot disease, threatening the stone and pome fruits around the world. Literature shows that the financial losses caused by *Monilinia* spp. have reached high levels, requiring appropriate management control strategies to limit the impact of the disease and reduce the developed resistance to fungicides. The latter is of the utmost importance for the wider concept of the Integrated Fruit Production (IFP) implemented in Europe as well as for developing alternative control measures for pests and diseases, while preserving the environment, organisms, and endangered ecosystems. Thus, the purpose of this review is to represent the framework of knowledge related to pathogen biology, host susceptibility, environmental factors, and known control measures to better understand the underlying processes related to Brown rot disease.

Key words: *Monilinia laxa*; *Monilinia fructigena*; *Monilinia fructicola*; brown rot; control strategies; PCR

INTRODUCTION

Brown rot (BR) caused by *Monilinia* species remains one of the most economically devastating diseases of stone and pome fruits globally (Larena et al., 2005; Poniatowska et al., 2016). Brown rot caused by *Monilinia laxa* (Aderhold & Ruhland) Honey, *Monilinia fructigena* (Aderhold & Ruhland) Honey, and *Monilinia fructicola* (G. Winter) Honey are fungal pathogens causing considerable damage and yield losses in important commercial crops (Martini and Mari, 2014), such as sweet (*Prunus. avium* L.) and sour cherry (*Prunus. cerasus* L.), plum (*Prunus. domestica* L.), apple (*Malus pumila* Mill.), and pear (*Pyrus communis* L.) (Batra, 1991). *M. laxa* affects the crops early in the season (flowering time), whereas *M. fructigena* is mainly observed during harvesting time. *M. fructicola* was also reported on

rot-infected fruits (Northover and Biggs 1990). In Europe, the predominant species include *M. laxa*, *M. fructigena*, and the recently established *M. fructicola*, all of which belong to the Ascomycota and share a necrotrophic lifestyle. These pathogens affect critical stages of crop development, manifesting as blossom blight, twig cankers, and fruit rot both in-field and postharvest. Losses can be significant, particularly under favourable environmental conditions that promote rapid disease development and secondary infection cycles.

The incidence and severity of brown rot are modulated by a complex interaction of factors, including cultivar susceptibility, canopy microclimate, orchard management, and regional weather patterns. Highly susceptible cultivars such as ‘Van’ are prone to early floral infections and severe fruit rot, whereas cultivars like ‘Regina’ exhibit partial resistance, likely associated

with morphological and biochemical traits. Disease outcomes are further compounded by the timing of pathogen activity relative to host phenological stages, with flowering and ripening periods representing critical windows of vulnerability. Thus, field diagnostics of *Monilinia* spp. may serve as a useful preliminary indication of infection; however, the laboratory based methods of identification remain essential (Martini and Mari, 2014). Literature outlines different methods for the identification of *Monilinia* spp., including morphological and cultural methods (Byrde and Willetts, 1977), and PCR identification (Holb, 2008b; Martini, 2013).

Control of the pathogens has heavily relied on synthetic fungicides. However, increasing public concern over fungicide residues, regulatory restrictions, and the emergence of fungicide-resistant pathogen populations necessitate the exploration of integrated and sustainable management strategies. These include cultural practices, biological control agents, and the adoption of predictive models informed by landscape epidemiology and climate data. Moreover, advances in molecular diagnostics are enabling rapid and accurate detection of *Monilinia* spp., facilitating timely interventions.

The present review summarises key information about (a) the pathogen and susceptible hosts, (b) the life cycle of *Monilinia* spp., (c) environmental determinants reported to impact the disease progression, and (d) management strategies to control the disease to gain deeper insights into the interactions and factors impacting disease progression.

1. Taxonomy, biology and molecular identification of *Monilinia* spp. causing BR disease

1.1. Taxonomy, morphology and molecular identification of *Monilinia* spp.

Monilinia spp. are found to differentiate in their geographic distributions, outlining that *M. laxa* and *M. fruticola* have been globally distributed, whereas *M. fructigena* is widely spread across South America, North Africa, Europe, and Asia (Zhu et al., 2016; Obi et al., 2018; Lyousfi et

al., 2024), but still a quarantine pest for Canada, the United States, Australia, and New Zealand (CABI, 2009). BR species were classified in the Discomycetes, a subdivision of the necrotrophic fungus Ascomycetes, belonging to the Helotiales (Leotiales) order, family Sclerotiniaceae, genus *Monilinia* (Obi et al., 2018). Thirty-five species of *Monilinia* Honey were described, and three were reported to significantly impact the commercial crops of stone and pome fruits: *M. laxa*, *M. fruticola*, and *M. fructigena* (Lino et al., 2016). As described by Martini and Mari (2014), the fruiting body of representatives from the class Discomycetes is found on the pseudosclerotial layer on the mummified fruits or litter containing debris on the orchard floor, where the sexual form (ascospores) is developed. In their study, Martini and Mari (2014), outlined the apothecium of *M. laxa* and *M. fruticola* as a cup or disk-shaped structure containing thousands of asci, with eight single-celled ascospores in each, combined with sterile hyphae. *M. laxa* and *M. fruticola* species are difficult to distinguish; therefore, in addition to the diagnostic protocols, the use of molecular techniques is recommended (Martini and Mari, 2014; Lino et al., 2016). A very characteristic sign of *M. fructigena* is the concentric rings of mycelium with conidia found to form on PDA agar, with cream to yellow colonies colour (Hrustić et al., 2020).

Literature suggests that the identification of *Monilinia* spp. could also be completed using different methods: PCR-based diagnostics (Ioos and Frey, 2000), real-time PCR and Quantification (Van Brouwershaven et al., 2010), Loop-Mediated Isothermal Amplification (LAMP) (Ortega et al., 2019), and Genomic and Metabarcoding Advances (De Miccolis Angelini et al., 2023). Due to the closeness of the species within the genus, especially *M. laxa* and *M. fruticola*, their cultural and morphological identification is the initial stage of the process, and then samples of the identified species are used to subsequently explore it using molecular approaches (Martini and Mari, 2014). The accurate detection and identification of *Monilinia* spp, particularly *M. laxa* and *M. fruticola*, are crucial for timely management and for

preventing the spread of regulated species. Conventional morphological identification remains useful in preliminary diagnosis but is limited by overlapping phenotypic traits and slow growth in culture. Therefore, molecular tools have become essential in modern plant pathology for both species-level resolution and epidemiological studies. As described by Martini and Mari (2014), prior to molecular identification of *Monilinia* spp., it is critical to identify the pathogen morphologically and culturally.

1.2. Disease cycle of *Monilinia* spp.

As polycyclic pathogens, *Monilinia* spp. could reproduce several secondary cycles per growing season of the host (Larena et al., 2021). *Monilinia* spp. coexist in the field orchards, and have similar life cycles (Holb, 2004), especially *M. laxa* and *M. fructicola*, which were reported to have similar epidemiological physiognomies (Bernat et al., 2017b). Moreover, studies reported that *M. fructicola* is displacing *M. laxa*, which could be attributed to the ability of *M. fructicola* to produce ascospores (Holtz et al., 1998; Obi et al., 2018), whereas the sexual phase of *M. laxa* is not known (de Cal et al., 2014). These pathogens have similar life cycles characterised by the three phases as described by Byrde and Willetts (1977), but still differentiate in aggressiveness, preferred infection stages, and symptom expression. Disease progression is closely regulated by host phenology and susceptibility (Guidarelli et al. 2014), environmental conditions, and management control (Holb, 2008b; Lino et al., 2016).

Monilinia laxa (Aderhold & Ruhland) Honey

M. laxa overwinters in mummified fruits, infected twigs, and blighted blossoms, where mycelia and conidia survive. Primary infections occur in early spring, when conidia are released from the mummified fruits or twig cankers, especially under moist conditions and temperatures between 15–20 °C (Holb, 2004; Obi et al., 2018). Symptoms caused by *M. laxa* are blossom blight, where infected flowers wilt, brown, and clings are visible. The fungus often progresses into twigs, causing cankers and shoot dieback, with gumming around lesions. Secondary infec-

tions can occur during the development stage of fruits via conidial dispersal from infected tissues. The survival mechanism of *M. laxa* consists of latent infections in mummified fruits, allowing the pathogen to survive and spread early in the following season (Mari et al., 2003).

Monilinia fructicola (G. Winter) Honey

This species is considered the most virulent and invasive, and has recently expanded in Europe after originating in North America (EPSA, 2011). It affects a broad host range and is highly adaptable. *M. fructicola* occurs in both twig cankers and mummified fruits, with the capacity to form apothecia and release ascospores under conducive field conditions (EPPO, 2020). In Europe, *M. fructicola* was first detected in France (Lichou et al., 2002), followed by Austria, the Czech Republic, Italy, Switzerland, Germany, and Slovenia (EPPO Global Database, 2025). In Bulgaria, *M. fructicola* was first reported by Bobev et al. (2020). The primary inoculum of *M. fructicola* consists of conidia and ascospores capable of infecting blossoms, young fruits, and wounded tissues. Infections occur rapidly under warm, humid conditions (20–25 °C). Host symptoms caused by *M. fructicola* are similar to those caused by *M. laxa*, but progress more aggressively, including blossom blight, twig, and shoot dieback. Fruit rot begins as soft, sunken brown lesions that rapidly expand and are covered with dense, grayish-coloured pustules (EPPO, 2020). Rotted fruit may detach or remain on branches. Unlike the other species, *M. fructicola* readily infects through wounds, contributing to fruit-to-fruit transmission, especially when fruits are in close contact, a condition common during ripening and harvesting. Survival and spread mechanism of *M. fructicola* consists of its ability to infect through minor wounds and produce abundant conidia, which enhances epidemic spread, especially during harvest and in dense canopies (Holb and Schnabel, 2007; Burnett et al., 2010).

Monilinia fructigena (Aderhold & Ruhland) Honey

Predominantly, *M. fructigena* affects pome fruits (apple, pear), but it is also reported in stone fruits (Van Leeuwen, 2000). The pathogen over-

winters in the mummified fruits on trees or the orchard floor. Infection typically occurs during bloom and early fruit development stages, but symptoms often remain latent until fruit ripening or postharvest. Symptoms visible on the host are mainly fruit lesions appearing as firm, pale brown, concentric circles (EPPO, 2004), whereas isolates of *M. fructigena* cultured on PDA agar showed cream to yellow (Hrustić et al., 2020) to buff (EPPO, 2020) with concentric rings (Lino et al., 2020). Affected fruits by *M. fructigena* become mummified and often remain attached to the tree (Van Leeuwen et al., 2002). Similarly, the environmental conditions favourable for *M. fructigena* conidia during springtime were reported as 15–20 °C temperatures in combination with humid conditions (Van Leeuwen et al., 2002). Latent fruit infections contribute significantly to postharvest rot, making *M. fructigena* particularly important in storage and transport-related losses (Martini and Mari, 2014).

2. Host Range and Susceptibility

2.1. The host: stone and pome fruits

Brown rot (BR) is a major fungal disease affecting stone fruits (*Prunus* spp.) and some pome fruits (*Rosaceae*) (Martini and Mari, 2014). Based on literature, *M. laxa*, *M. fructigena*, and *M. fructicola* were reported to cause a significant threat to stone fruits (sweet cherries (*P. avium* L.), sour cherry (*P. cerasus* L.), plum (*P. domestica* L.), apricot (*P. armeniaca*), peaches (*P. persica*), and almond (*P. dulcis*) and pome fruits (apple (*M. domestica* Borkh.), pear (*Pyrus communis*), and quince (*Cydonia oblonga*)) (Batra, 1991). The latter were reported with a higher incidence of *M. fructigena*, in comparison to the rest of the Genus representatives (Martini and Mari, 2014).

2.2. Host Susceptibility

Literature describes host-level differences in susceptibility to *Monilinia* spp. (Lyousfi et al., 2024; Stancheva, 2021), along with cultivar-level variation in the susceptibility (Borovinova, 2007; Holb et al., 2011; Zhu et al., 2016), along with cultivar-rootstock susceptibility (Borovinova, 2007; Dziedzic and Błaszczyk, 2019; Mangararis et al.,

2022; Butac et al., 2023) and pathogenicity differences within the pathogens (Borovinova, 2007), where *M. laxa* infects flowering part and fruits (Papavasileiou et al., 2015), similarly *M. fructicola* has been reported to infect flowers and fruits (Martini and Mari, 2014), whereas *M. fructigena* has mostly been reported on ripening fruits (Xu and Robinson, 2000). Lastly, *M. laxa* and *M. fructicola* have been mostly reported on sweet and sour cherry, plum, and apricot (Martini and Mari, 2014), whereas *M. fructigena* is more prevalent on pome fruits (Xu and Robinson, 2000; Van Leeuwen et al., 2002; De Miccolis Angelini et al., 2022; Lyousfi et al., 2024).

Sweet cherry cultivars' susceptibility

Although there are no known sweet cherry cultivars resistant to *M. laxa*, some cultivars were reported to be less susceptible than others (Holb et al., 2011; Kappel et al., 2012; Žďárská et al., 2017). As described by Žďárská et al. (2017), cultivars 'Starking Hardy Giant' and 'Regina' were reported with the least observed damage, whereas 'Van' and 'Felicitá' were reported as the highest damage. Similar findings for a lower degree of susceptibility in the sweet cherry cultivar 'Regina' were reported by Quero-García et al. (2017), while higher susceptibility was observed in 'Van' (Borovinova, 2007). Additionally, in parallel with the general susceptibility of 'Van' to *Monilinia* spp, further research on the impact of the rootstock identified different degrees of susceptibility to cracking (Popatanasova and Sotirov, 2005) and fruit rot (Borovinova, 2007). Furthermore, Borovinova (2007) reported an association between fruit cracking and *Monilinia* spp, highlighting the significant role of rain quantity and duration in the development of fungal pathogens and cracking factors. Popatanasova and Sotirov (2005), and Borovinova (2007) showed that rootstocks Hibrid-2 and P-7 were susceptible to cracking and fruit rot (*Monilinia* spp, *A. alternata*, *B. cinerea*, and *Gl. cingulata*), with prevalence on *Monilinia* spp. The latter could also be attributed to the early flowering time of the cultivars, which coincides with the favourable meteorological conditions that play a critical role in the development of *Monilinia* spp, and are mostly

characterised by greater rainfall quantity and duration (mm/day), along with temperature conditions ranging 15-20°C (Holb, 2004). Once provided with the opportunity to develop, the pathogen progresses and affects the fruits later in the season. An additional factor for the increased infection of *Monilinia* spp., the cracking of the fruits was found to be associated with rainy periods, during which the effectiveness of the fungicides is reduced (Borovinova, 2007).

Sour cherry cultivars susceptibility

The sour cherry cultivars reported with different levels of susceptibility were 'Heimanns Rubinveichsel' (Borovinova, 2015; Stancheva, 2006), 'Chachanski rubin' (Stancheva, 2006), whereas 'Dryanovska ranna vishna' and 'Tsiganska Vishna' were reported as less susceptible (Stancheva, 2006). Low susceptibility sour cherry cultivars, such as 'Latviszkaja Nizkaja', 'Nagy Angol', 'Mocanesti', 'Ljubszkaja', 'Sirpotreb', 'Oblacsinszkaja', 'Cigánymeggy 3', 'Maraska Savena', 'Mettar', and 'Elegija' were also described by Holb (2008a), where partly tolerant to *M. laxa* were reported to be 'Csengödi', 'Akasztói' and 'Cigánymeggy 59'.

Plum cultivars susceptibility

Plum cultivars reported with high susceptibility to *M. laxa* were 'Stanley' (Holb et al., 2011; Mitre jr et al., 2015; Stoev et al., 2017; Todorova and Marinova, 2024) and 'Matilda' (39.5%), 'Elena' (33.1%), and 'Jubileu 50' (31.9%) were found with lower infection by Moldovan et al. (2023), while lower susceptibility was reported on 'Kystendilska sinya sliva' (Stancheva, 2006; Stoev et al., 2017), 'Silvia' (Holb et al., 2011), and 'Zamfira' (6.9%), 'Anna Späth' (7.0%), and 'Doina' (7.7%) (Moldovan et al., 2023). Moreover, in their study, Mitre jr et al. (2015) described plum cultivars 'Topend', 'Jojo', 'Tophit', and 'Nectarina rosie' as resistant to *M. laxa*.

Apple cultivars susceptibility

Several studies have outlined the importance of *M. fructigena* on apple fruits and its significant role in the economic impact, including reduced yield production (Xu and Robinson, 2000; Konstantinou et al., 2011; Chițulescu and Cristea, 2018). According to Sharma and Kaul (1988)

and Xu and Robinson (2000), apple cultivars are susceptible to *M. fructigena* with associated incidence of brown rot with wounds on the fruits. Similar association between *Monilinia* spp. and fruit cracking was observed on sweet cherry cultivars with different ripening periods (Borovinova, 2007; Popatanasova et al., 2005). In the study by Konstantinou et al. (2011) and the experimental cultivars were found to have higher variability in disease incidence across the cultivars and study years, they showed that the frequency of recovery of *M. fructigena* on natural apple fruits was the highest for 'Golden Delicious' (in 2008 (5.3%)), and the lowest for 'Granny Smith' (in 2009, 0.00%) and 'Fuji' (in 2008, 0.00%). Another study by Chițulescu and Cristea (2018) reported that reduced incidence of *M. fructigena* was achieved on 'Florina' variety using the seven fungicide treatment managements.

In summary, the susceptibility to *Monilinia* spp. differ based on species, cultivars, rootstocks, and cultivar-rootstock combinations (Borovinova, 2007; Holb et al., 2011; Manganaris et al., 2022). Additionally, studies demonstrated evident associations between *Monilinia* spp. and fruits with wounds and cracking in years with increased amount of rainfall (Borovinova, 2007; Byrde and Willetts, 1977; Popatanasova et al., 2005). In their study, Popatanasova et al. (2005) investigated 22 cultivars and elites for their susceptibility to fruit cracking and rot disease, outlining that the most susceptible cultivars to both factors were early ripening cultivars 'Bigarreau Jaboulay', 'Patriotka Krima', and mid-season elite 11 W-15-37. According to Popatanasova et al. (2005), fruit cracking and rot diseases observed during ripening and harvesting times were reported at higher frequencies in years with increased rainfall. As explained by the authors, the fruit cracking is not only associated with the biological characteristics of the cultivar and rootstock, but critical factors also include the soil and weather conditions, along with the shape, density of the crowns, and the selected growing systems. According to Popatanasova et al. (2005), cracking occurs if the raindrop remains on the fruit for an extended period, where part of the moisture is absorbed through the stro-

ma on the skin, causing longitudinal cracks on the top of the fruit. Thus, based on the literature, it is reasonable to conclude that the higher susceptibility observed in species could be attributed to some extent to the early flowering time, which coincides/coincidence with meteorological conditions favourable for the *M. laxa* development, including higher frequency of rainfall and optimal temperature (Borovinova, 2007; Todorova and Marinova, 2024). That said, the host susceptibility, as described by Holb (2008a), could be used as another means to achieve more appropriate disease management. In addition, the observed range of susceptibility is a useful tool for a better selection of cultivars for conventional and organic fruit production (Holb et al., 2011), highlighting the importance of good growing conditions, horticultural practices, and monitoring, ensuring that optimal fungicide sprays are applied to control the disease (Iliev et al., 2011).

3. Landscape epidemiology and environmental factors, impacting *Monilinia* spp.

The development of fungal pathogens is governed by a complex interplay between host, pathogen, environmental conditions, and spatial-temporal dynamics (Plantegenest et al., 2007; Manlove et al., 2022). That said, with the expansion of the airborne pathogens *Monilinia* spp. across continents, and the increased incidence of *M. fructicola*, the importance of a wider understanding of disease dynamics and factors such as landscape epidemiology is needed (Radici et al., 2024).

3.1. Landscape epidemiology

Landscape epidemiology, as described by Manlove et al. (2022), emphasizes how the spatial configuration of host plants, microclimatic heterogeneity, and dispersal corridors influence pathogen dynamics at multiple scales. In the context of stone and pome fruits, according to Rungjindamai et al. (2014), the proximity of infected fruits, abandoned host trees, and their susceptibility, inoculum source (mummified fruits on trees and floor) can serve as pathogen reservoirs, enhancing primary inoculum pressure and

facilitating long-distance spore dispersal through wind and insect vectors. Also, the severity and timing of brown rot epidemics caused by *Monilinia* spp. are strongly influenced by specific meteorological conditions (Petróczy et al., 2012; Martini and Mari, 2014; Obi et al., 2018) and the phenological stages (Rungjindamai et al., 2014) of the host plant. These parameters modulate both the life cycle of the pathogen (Holb, 2004; Villarino et al., 2010) and host susceptibility through gene expression, which is known to be influenced by environmental factors (Guidarelli et al., 2014), ultimately shaping epidemic dynamics. Additionally, a metapopulation study of *M. fructicola* by Radici et al. (2024) using landscape heterogeneity parameters (climate, host density, and airborne connectivity) has revealed the significant role of the wind-driven transport of *M. fructicola* in disease propagation, offering a framework for national-level plant protection strategies.

3.2. Environmental characteristics influencing Brown Rot

The severity and timing of brown rot epidemics caused by *Monilinia* spp. are strongly influenced by specific weather parameters, particularly temperature, humidity, and precipitation, as well as their interaction with the phenological stages of the host plant (Van Leeuwen, 2000). These parameters modulate both the pathogen's life cycle and host susceptibility, ultimately shaping epidemic dynamics (Martini and Mari, 2014).

Temperature

As an important parameter, temperature was found to be critical for *Monilinia* spp. development (Martini and Mari, 2014). Studies consistently report that *Monilinia* spp. favour moderate temperatures (15–25 °C) and high relative humidity (>85%), particularly in the presence of free moisture (Holb, 2008a; Martini and Mari, 2014; Rungjindamai et al., 2014). Higher temperatures (>25°C) were found, in laboratory conditions, to reduce the viability of conidia (Martini and Mari, 2014). Primary infections occur in early spring, when conidia are released and infect blossoms, especially under moist conditions and temperatures between 15–25 °C (Angeli et al.,

2017). As described by Martini and Mari (2014), under moist conditions, visible symptoms of *Monilinia* spp. infection on peach fruits could be observed within 48-72 h of infection at a temperature range of 20-25°C. Notably, a study by Xu et al. (2001) investigating the effect of temperature and relative humidity on *M. fructigena* conidia reported that conidia only germinate under saturation humidity ($\geq 97\%$ RH) with a germination rate increasing with the temperature to a maximum of 23-25 °C, then following a decreasing rate. *M. fructigena* conidia were found to germinate within 2 h at 20- 25 °C, while on detached fruits, sporulation was not observed at 5 or 25°C (Xu et al., 2001). Based on the study conducted by Xu et al. (2001), conidia viability was found to be more dependent on temperature than on relative humidity. The latter was in agreement with the results reported by Hall (1963), where sporulation and maturation of spores were influenced by temperature.

Rainfall

Rainfall events play a triple role: they not only disperse spores (via splash or air currents) (Byrde and Willetts, 1977; Bernat et al., 2018), but also, when the period with rain is longer, it creates wounds through cracking or bruising, facilitating infection (Gibert et al., 2009). Longer rain periods, near flowering and harvest, strongly correlate with increased BR disease incidence (Michailides et al., 2007). In the study by Holb (2008b), the aerial spore density and meteorological variables were found to correlate, suggesting that dry conditions enable spore dispersal, whereas longer rain periods affect the number of trapped conidia. It was also reported that spores of *M. fructigena* were found to germinate and infect fruits at a very high saturation humidity ($>97\%$). Rain is also related to the leaf wetness duration, and an experiment by Brun et al. (2023) demonstrated that rain shelters significantly reduced leaf wetness, thereby reducing the *Monilinia* pathogens.

Leaf wetness duration (LWD)

Described as one of the critical environmental factors (Michailides et al., 2007), leaf wetness is

used in many control systems (Brun et al., 2023; Casals et al., 2023) and is a known factor to impact spore germination and infection efficiency in *Monilinia* spp. (Martini, 2013). As described in the literature, spores of *Monilinia* spp. require free water on the plant surface to initiate germination and penetrate host tissue (Petróczy et al., 2012). Several studies demonstrate that infection by *M. laxa* and *M. fructicola* occurs when wetness duration exceeds 6-8 h under favourable temperatures (Byrde and Willetts, 1977; Biggs and Northover, 1988). In field conditions, prolonged rain events create microclimatic conditions in the orchards conducive to infection, especially during flowering and pre-harvest stages (Kreidl et al., 2015).

Humidity

Relative humidity (RH) exerts a dual influence: (i) promoting sporulation at high levels ($>95\%$) (Kreidl et al., 2015) and modulating host tissue susceptibility (Mustafa et al., 2021). The increased level of RH supports conidial production on infected tissues and enhances the viability of airborne inoculum (Holb et al., 2004). While spore germination is not entirely RH-dependent, optimal germ tube elongation generally requires RH above 90% (Xu et al., 2001; Garcia-Benitez et al., 2017), which is a morphological identification factor (Van Leeuwen, 2000). Moreover, high RH contributes to micro-wetness — the thin films of moisture on floral structures or fruit cuticles that may not be macroscopically visible but still facilitate infection (Xu et al., 2001).

In conclusion, the environmental parameters were reported as critical in the effective forecasting and integrated BR disease management. Their combined effect influences not only the viability of spore germination and infection initiation but also shapes the seasonal dynamics of inoculum availability and latent infection.

4. Management strategies to control Brown rot

4.1. Agricultural practices improving growing conditions

The effective management of *Monilinia* spp. in orchards begins with the implementation of

cultural practices that enhance tree health and reduce pathogen pressure (Dowling et al., 2019). The establishment of new orchards should prioritise well-ventilated sites and an appropriate planting layout that facilitates air circulation and minimises humidity within the canopy, key factors in limiting the infection of *Monilinia* spp. and sporulation (Holb, 2004). Additionally, cultivating less susceptible cultivars, along with a rigorous canopy management through pruning and thinning, further reduces the incidence of disease by improving light penetration and reducing microclimate conditions favourable for fungal growth (Barba et al., 2015; Stancheva, 2021). As suggested, all desiccated branches and mummified fruits must be removed during dormant season pruning to eliminate overwintering inoculum sources. Additionally, applying horticultural pastes and sealants to cover large wounds to prevent colonisation by wound-invading pathogens (Stancheva, 2021). These agronomic interventions collectively enhance tree growing conditions, which reflect tree vitality (Dobbertin, 2005) and could be used as additional means to the management strategies in withstanding fungal pathogens from the phylum Ascomycota (Raykova et al., 2025).

4.2. Conventional and integrated plant protection control

The fungicide treatments of *Monilinia* spp., responsible for BR in stone and pome fruits, are highly dependent on host species and development phenophases (Table 1). Synthetic fungicides are widely applied both pre- and post-harvest to reduce infection incidence and disease severity (Holb and Schnabel, 2005; 2007; Lyousfi et al., 2024; Martini and Mari, 2014). However, repeated and prolonged use of specific active ingredients such as benomyl, dicarboximides, and demethylation-inhibiting (DMI) fungicides—including cyproconazole, difenoconazole, fenbuconazole, and tebuconazole—has led to the development of resistance in *Monilinia* populations in several regions (Lyousfi et al., 2024). Notably, *M. fructicola* isolates demonstrated high resistance to benzimidazole fungicides, with some also exhibiting resistance to dicarboximides (Tshikhudo

et al., 2022). To mitigate this issue, the application of fungicides with diverse modes of action is essential. Anti-resistance strategies such as alternating or combining fungicides from different FRAC (Fungicide Resistance Action Committee) groups are strongly recommended (Feliciano et al., 1987; Holb and Schnabel, 2005; Miessner and Stammler, 2010; Rungjindamai et al., 2014; Tshikhudo et al., 2022; Wan et al., 2021). For example, fungicides like thiophanate-methyl, which retain efficacy against many *Monilinia* spp., can be strategically employed, especially where low resistance levels are observed in *M. laxa* (Burnett et al., 2010). Field studies, such as those conducted by Petrova et al. (2015), highlight the impact of fungicide treatment management intensity. Their findings showed significantly higher brown rot damage in conventionally managed orchards (cultivar ‘Erdi Biötermö’) during 2011, which the authors attributed to reduced fungicide application treatments in the previous year, highlighting the cumulative importance of year-on-year control measures.

4.3. Biological control options and microbial antagonists

Although synthetic fungicides remain the main approach to control *Monilinia* spp. in stone and pome fruits due to their efficacy and reliability (Holb and Schnabel, 2005; Martini and Mari, 2014), increasing public concern over food safety, pesticide residues, and environmental sustainability, coupled with the growing incidence of fungicide-resistant pathogen strains, has intensified the search for effective alternatives (Wan et al., 2021; Borisova and Petrova, 2023; Lyousfi et al., 2024). Biological control strategies, particularly those employing microbial antagonists, have emerged as promising alternatives to chemical control methods. These biocontrol agents (BCAs) often function through multiple mechanisms, including nutrient and space competition, the discharge of antifungal compounds, the induction of host plant resistance, and the disruption of pathogen communication (mycoparasitism and antibiosis) (Lahlali et al., 2020; Casals et al., 2022). However, despite the abundance of experimental

Table 1. Chemical control to *Monilinia* spp.

Species	Host	Active ingredient	Mode of Action/Frac group	Reference
<i>M. laxa</i>	Sour cherry	Thiram	Multi-site action (M3)	Petrova et al. (2015)
<i>M. fructicola</i>	Peach	Tebuconazole	DMI fungicide (G1)	Martini (2013), Martini and Mari, (2014)
<i>M. fructigena</i> <i>M. fructicola</i>	Peach	Thiophanate Methyl	Benzimidazole (B1)	Martini (2013)
<i>M. fructicola</i>	Nectarine	Azoxystrobin	QoI fungicide (C3)	Dowling (2015)
<i>M. laxa</i>	Stone fruits apple	Fenhexamid	Hydroxylanilide (E1)	Malandrakis et al. (2013)
<i>M. laxa</i>	Plum	Chlorothalonil (Bravo SC), Boscalid + Pyraclostrobin (Signum), Captan (Merpan)	Multi-site, SDHI + QoI, Phthalimide	Popa et al. (2013)
<i>M. laxa</i> <i>M. fructicola</i>	Sweet cherry	QoI fungicides (pyraclostrobin and azoxystrobin)	C3	Miessner and Stammeler (2010)
<i>M. fructicola</i>	Peach	QoI fungicides (azoxystrobin, trifloxystrobin, and pyraclostrobin + boscalid)	C3 + SDHI	Burnett et al. (2010)
<i>M. fructigena</i>	Apple	Copper hydroxide Copper sulfate Methyl thiophanate	Multi-site + B1	Florian et al. (2018)
<i>M. fructicola</i>	Peach	Fenbuconazole	DMI fungicide (G1)	Holb and Schnabel, (2007)
<i>M. laxa</i> <i>M. fructigena</i>	Sweet Cherry	Copper hydroxide Tebuconazole Dodine Myclobutanil	Multi-site, G1	Borovinova (2004)
<i>M. laxa</i> <i>M. fructigena</i>	Cherry	Bordeaux mix, Cuproxat, Dithianon (Delan), Difenconazole (Difcor), Embrelia, Captan	Various modes	Stancheva, (2021)

* Note: Resistance to tebuconazole has been confirmed in *M. fructigena* and *M. laxa*, while *M. laxa* has shown low resistance to thiophanate-methyl. Sensitivity to thiophanate-methyl remains high across most isolates tested (Burnett et al., 2010; Miessner and Stammeler, 2010)

evidence, commercial application remains limited due to inconsistent field performance, formulation challenges, and regulatory hurdles (Mari et al., 2012; Zhang et al., 2019). As described in Table 2, *Bacillus* spp. (including *B. subtilis* and *B. amyloliquefaciens*) are among the most widely studied BCAs against *Monilinia* spp. These bacteria produce a range of lipopeptides that inhibit fungal growth, sporulation, and germ tube elongation (McKeen et al., 1986). For instance, the

Bacillus amyloliquefaciens strain CPA-8 showed strong effectiveness in reducing the incidence of BR in peaches and nectarines under both laboratory and semi-commercial conditions (Casals et al., 2021). Additionally, antagonistic activity has been observed in yeasts such as *Pichia membranifaciens* and *Kloeckera apiculata*. This activity primarily occurs through competition and secretion of killer toxins that disrupt the membranes of fungal cells (Zhang et al., 2019). The epiphytic

Table 2. Biological control to *Monilinia* spp.

Species	Host	Active ingredient	Reference
<i>M. laxa</i> , <i>M. fructigena</i>	Plum	<i>Serenade</i> ® ASO (<i>Bacillus amyloliquefaciens</i> QST 713) <i>Ecosist-Arbanasi</i> (<i>Bacillus subtilis</i>)-soil and foliar application	Borisova and Petrova (2023)
<i>M. laxa</i> , <i>M. fructigena</i> <i>M. fructicola</i>	Stone fruits	<i>Bacillus</i> and <i>Pseudomonas</i> spp	Wan et al. (2021)
<i>M. fructicola</i>	stone fruit	<i>Bacillus subtilis</i>	McKeen et al. (1986)
<i>M. laxa</i> , <i>M. fructigena</i> <i>M. fructicola</i>	Peaches Nectarine Cherries	<i>Bacillus amyloliquefaciens</i> CPA-8 (CPA-8) <i>Penicillium frequentans</i> 909 (Pf909)	Casals et al. (2021)
<i>M. laxa</i> , <i>M. fructigena</i>	Apple	<i>Alcaligenes faecalis</i> ACBC1, <i>Bacillus amyloliquefaciens</i> SF14 and SP10, <i>Pantoea agglomerans</i> ACBP1	Lahlali et al. (2020)
<i>M. fructicola</i>	Plum	<i>Pichia membranaefaciens</i> <i>Kloeckera apiculata</i>	Zhang et al. (2019)
<i>M. laxa</i> , <i>M. fructicola</i> , <i>M. fructigena</i>	Stone fruits	<i>Aureobasidium pullulans</i>	Mari et al. (2012)

fungus *Aureobasidium pullulans* is well-known for its ability to adapt to phyllosphere conditions and its broad-spectrum antagonistic activity against various pathogens, including *Monilinia* spp. The latter makes it a strong candidate for commercial formulations (Mari et al., 2012). Despite these advancements, the practical deployment of microbial antagonists is hindered by variability in performance under fluctuating environmental conditions. For future success, integrated use of BCAs within broader disease management frameworks, combined with resistant cultivars and predictive models, may enhance their efficacy and commercial viability (Lahlali et al., 2020; Wan et al., 2021).

CONCLUSIONS

The successful management of *Monilinia*-induced brown rot in stone and pome fruits requires a comprehensive understanding of pathogen biology, disease epidemiology, host and cultivar susceptibility. The three main species, *M. laxa*, *M. fructigena*, and *M. fructicola*, mainly cause the progression of BR disease, with *M. laxa* and *M.*

fructicola primarily affecting blossoms, twigs, and fruits, whereas *M. fructigena* predominantly affects pome fruit. The literature review summarised the critical meteorological parameters reported to create favourable conditions, including temperatures (15–25 °C), high relative humidity, and prolonged wetness, particularly during flowering and fruit ripening leading to spore development and BR disease progression. These weather parameters, combined with other environmental factors (e.g., landscape epidemiology), host and cultivar susceptibility, and pathogen inoculum pressure from previous years, determine the epidemic potential each season. The latter highlights the key role of disease modelling and forecasting as another means of adapting to changing weather conditions and mitigating the risk of disease progression, while utilising the control management strategies at the appropriate time.

Chemical control remains the main approach to control BR disease. However, resistance to benzimidazoles, dicarboximides, and demethylation-inhibiting fungicides has been widely reported, requiring the application of other approaches and rotational use of fungicides with different modes of action. As an alternative, literature suggests

that biological control agents, such as *Bacillus* spp., *Penicillium frequentans*, and *Aureobasidium pullulans*, have demonstrated promising results in controlling *Monilinia* spp. under both pre- and postharvest conditions. The deployment of resistant cultivars, such as 'Regina', in combination with appropriate agricultural practices, will provide another means of BR management through reducing the inoculum pressure. Future research should continue to improve the early detection forecasting, understand the determinants of resistance, and develop IPM strategies that are both economically and environmentally sustainable.

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