PATHOGEN PROFILE

Current status and future prospects of grapevine anthracnose caused by *Elsinoe ampelina*: An important disease in humid grape-growing regions

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Abstract
Anthracnose, caused by *Elsinoe ampelina*, is one of the most destructive diseases of grapevines worldwide, especially in humid areas. *E. ampelina* mainly infects young tissues starting from shoots to berries and affects vine vigour and berry yield. The occurrence and the role of the sexual stage in the disease cycle and the grapevine–*E. ampelina* interaction remain poorly understood. However, the recent genome sequence data of *E. ampelina* provides the basis for further studies to understand its evolution, pathogenicity mechanisms, and effector repertoire. New studies on *E. ampelina* have been conducted in recent years. In this pathogen profile, we present a comprehensive literature review of *E. ampelina* to summarize the findings on its aetiology, infection mechanisms, genome, pathogenicity, and host resistance.

Taxonomy: *Elsinoe ampelina* Shear; Kingdom Fungi; Phylum Ascomycota; Subphylum Pezizomycotina; Class Dothideomycetes; Subclass Dothideomycetidae; Order Myriangiales Starbäck; Family Elsinoaceae Höhnel; Genus *Elsinoe* Racib.

Host range: *E. ampelina* only infects *Vitis* species and hybrids.

Distribution: The grapevine anthracnose is distributed worldwide but is most prevalent in Argentina, Australia, Brazil, Canada, China, India, Japan, Korea, New Zealand, South Africa, Thailand, USA, and Uruguay.

Disease symptoms: *E. ampelina* causes slightly abundant depressed spots on young leaves, petioles, stems, tendrils, rachises, and berries. Under severe infection conditions, early defoliation, berry dropping, and delayed berry development and ripening may occur.

Genome: The genomes of two *E. ampelina* isolates, YL-1 and CECT 20119, are publicly released with 8,057 and 10,207 predicted genes, respectively.

KEYWORDS
Elsinoe, fungal diseases, grapevine, host resistance, pathogen virulence
1 INTRODUCTION

Grape, one of the largest fruit crops, grown across 7.4 million hectares worldwide in 2019, is used for juice, wine, and raisin production and fresh consumption (OIV-International Organisation of Vine and Wine, 2019). Unfortunately, several diseases, including anthracnose, hinder the development of grapes. This disease, which is native to Europe, has spread worldwide and is responsible for serious epidemics during warm and humid seasons (Agrios, 2005; Thind et al., 2004). The grapevine anthracnose, also called bird’s eye rot and black spot, is caused by the ascomycete fungus E. ampelina, whose asexual stage is Sphaecoloma ampeliniun (Shear, 1929; Thind, 2015). However, the sexual stage is not found in many countries, which is possibly due to the absence of suitable mating types or the lack of harsh winter conditions that can promote ascocarp formation in grape tissues as an overwinter survival strategy (Magarey, Coffey, et al., 1993). Nevertheless, a recent study has shown that the asexual and the sexual stages have occurred concomitantly since the beginning of the epidemic under subtropical climates, but the role of ascospores in the epidemiology of the disease is unclear (Braga et al., 2020). Besides E. ampelina, a number of Colletotrichum species have been isolated from anthracnose lesions in the UK, Brazil, and India (Baroncelli et al., 2015; Santos et al., 2018a; Sawant et al., 2012a, 2012b). However, none of the seven Colletotrichum species identified in Brazil was pathogenic to the tested Vitis vinifera and V. labrusca cultivars (Santos et al., 2018a), whereas Colletotrichum capsici caused some tiny lesions, resembling a hypersensitive response reaction in V. vinifera ‘Thompson Seedless’ (Sawant et al., 2012b). These Colletotrichum species possibly survive endophytically or as quiescent appressoria in grape tissues, including anthracnose lesions, and favourable conditions can cause the ripe rot of berries (Santos et al., 2018a).

Although some studies have been conducted to elucidate the molecular interactions between grape and E. ampelina (Ahn, Kim, Jo, et al., 2014; Gao et al., 2020), the underlying mechanisms remain unclear. In the past, most of the studies focused on pathogen identification and pathogenicity (Santos et al., 2018a, 2018b), sporulation induction (Li et al., 2018; Santos et al., 2018b), characterization of the infection and colonization processes (Braga et al., 2019, 2020; Li et al., 2019), identification of resistant cultivars and hybrids (Hopkins & Harris, 2000; Poolsawat et al., 2012), development of molecular markers linked to disease resistance genes (Kim et al., 2008), and expression and screening of disease resistance-related genes (Gao et al., 2012; Seehalak et al., 2011; Vasanthaiah et al., 2010). Although many relevant studies have been published on grapevine anthracnose in recent years, in particular on the two genomes of E. ampelina recently published (Haridas et al., 2020; Li et al., 2020), a recent review of the literature for this pathosystem is not available. The last research overview of this important disease was published almost three decades ago (Magarey, Coffey, et al., 1993).

In this work, we provide a comprehensive review of the literature on the grapevine anthracnose. This review summarizes the disease symptoms, distribution, cycle, genomics of E. ampelina, and the genetics of host resistance. We also provide perspectives for future studies to understand the pathogen biology and develop effective disease management strategies.

2 IMPORTANCE AND DISEASE SYMPTOMS

Anthracnose is a major threat to grapevine production in humid and warm areas (Hemanth et al., 2010; Thind, 2015). For instance, in the USA, anthracnose occurs in states with a humid climate, such as Florida, but does not occur on the dry west coast (Mirica, 1988). In South Africa, the disease occurs in districts with high rainfall during the growing season (Boelema, 1968). In Brazil severe anthracnose epidemics have been reported in southern and south-eastern regions, where the weather is characterized by temperatures around 25–30 °C and frequent rain during the grapevine growth cycle (Santos et al., 2018a).

E. ampelina infects young and tender tissues of grapevine, including leaves, petioles, stems, tendrils, racines, and berries (Figure 1). The first symptoms appear as isolated small red-brown to black-brown spots on young shoots. The spots enlarge rapidly in wet weather, becoming lightly sunken with grey-white centres and dark brown or purple edges, round or irregular shaped (Magarey, Coffey, et al., 1993; Thind et al., 2004). As the leaf matures, the necrotic centres of older lesions usually fall off, forming a “shot-hole” under a dry environment (Gao et al., 2012; Hemanth et al., 2010; Thind, 2015). Similarly, on leaf veins, lesions are depressed, grey or greyish brown with a dark brown edge, and cause distortion of the leaf blade in young leaves.

The first symptoms on berries are characterized by dark-brown spots that enlarge and become sunken lesions with a grey-white centre and dark-brown to purple-brown margins, resembling a bird’s eye (Magarey, Coffey, et al., 1993). Multiple lesions may coalesce into large lesions and often cause berry cracking, favouring infection by opportunistic microorganisms that cause sour rot. Infection on petioles, tendrils, and young stems is recognizable by light-brown spots, which are initially circular but later elongate into elliptical, sunken, necrotic cankers. However, the lesion size varies between resistant and susceptible grape genotypes. For instance, big and circular lesions on leaves are formed on susceptible cultivars, whereas small and irregular lesions are formed on resistant cultivars (Gao et al., 2012; Kono et al., 2012). Ontogenic (age-related) resistance to E. ampelina is observed in grapevines, where young tissues are susceptible to pathogen infection and disease development, whereas old tissues are highly resistant (Santos et al., 2020; Thind, 2015). In addition, the lesion size on young leaves is greater than that on old leaves (Brook, 1973; Suhag & Grover, 1972).

At high disease severity, anthracnose causes early defoliation, berry drop, stem breakage, and delayed development and ripening of berries (Santos et al., 2018b; Thind et al., 2004). In the past, anthracnose caused worldwide damage in important grapevine production regions, such as Australia and India, in the late 18th
century (Magarey, Coffey, et al., 1993). Yield losses due to anthracnose are reported to be around 10%–15%, but severe infection can cause up to 100% yield losses on highly susceptible cultivars (Anderson, 1956; Bedi et al., 1969; de Castella & Brittlebank, 1918; Refatti, 1950). In Brazil, *E. ampelina* caused reduction in shoot dry weight by up to 80% in cv. Niagara Rosada plants and 56% in cv. Moscato Giallo kept in a growth room with relative humidity above 95% (Santos et al., 2018a). In addition to direct yield losses, the disease can alter the biochemical constituents of berries and affect the fruit quality (Thind et al., 1998). Sugars and total phenols are reduced in diseased leaf tissue, but the levels of phospholipids increase (Daulta & Chauhan, 1981; Kansal & Lal, 1979). Murria et al. (2018) found that the contents of chlorophyll, carotenoids, and ascorbic acid in infected leaves are lower than those in healthy leaves, but the malondialdehyde content is significantly higher in most susceptible hosts.

### 3 | CHARACTERIZATION OF *E. AMPELINA*

Acervuli containing short, cylindrical conidiophores bearing conidia are formed on necrotic lesions once the disease is established (Li et al., 2019). Conidia are cylindrical to oblong with rounded ends (Figure 2d), hyaline, and aseptate, 3.4–7.5 × 2.0–3.5 µm (Li et al., 2018; Magarey, Coffey, et al., 1993; Santos et al., 2018c; Sompong et al., 2012). When the sexual stage occurs, ascospores are produced in asci (12.9–34.3 × 12.6–29.6 µm), which contain eight ascospores each. Ascii are globose or elliptical bitunicate and distributed irregularly in the upper part of the ascoma (Braga et al., 2020; Magarey, Coffey, et al., 1993). Ascospores are hyaline, one- to three-septate and measure 15–16 × 4–5 µm (Braga et al., 2020; Shear, 1929).

The colonies of *E. ampelina* (Figure 2a,b) show slow growth on artificial media and high variability in colour, including red, yellow, brown, black, white, coral, and dark-brick hues on potato dextrose
agar (PDA) (Li et al., 2018; Santos et al., 2018c; Sompong et al., 2012). On PDA, the colony growth rate of Australian and Brazilian isolates ranged from 0.05 to 0.06 mm/day, reaching 1.9–2.5 cm in diameter at 28 days, whereas the colonies of Thai isolates had a size of 3.7–3.8 cm at 35 days (Santos et al., 2018c; Sompong et al., 2012). Colonies are generally round to irregular in shape with wrinkled texture and sometimes have cottony white aerial mycelium on the surface (Figure 2c). The cultural characterization of *E. ampelina* on different media shows that the colony growth, colour, and presence/absence of aerial mycelium are affected by the medium (Poolsawat et al., 2009). In that study, the cereal agar was the best medium for colony growth, and the formation of aerial mycelium was commonly observed on PDA.

As *E. ampelina* rarely sporulates on artificial media, conidia were collected from the lesions of field-collected grape tissues and used for artificial inoculations in the past (Brook, 1973; Gao et al., 2012; Sosnowski et al., 2007). Santos et al. (2018c) have reported that no conidium was found on cultures grown on PDA, corn agar, yeast extract agar, oatmeal agar, Sabouraud’s agar, Czapek’s agar, or Richard’s agar for 30 days, but a few conidia were produced on Fries’s liquid medium. Yun et al. (2006) have developed a method to induce conidial production based on culturing the colonies in Fries’s liquid medium and V8 juice agar under ultraviolet light to improve conidia yield. Kono et al. (2009) reported that *E. ampelina* conidia can be easily obtained by managing the colony density and shaking in water in the dark. Later on, an efficient method for sporulation was developed using rainwater or distilled water and shaking in darkness for 7 days (Santos et al., 2018b). Additionally, a considerable amount of conidia was reported on 25-day-old colonies grown in PDA bottles kept at 21 °C for 24 hr in the dark (Li et al., 2018). These studies indicate that unfavourable culture conditions (relatively high colony density or liquid culture) and treatments (low temperature or ultraviolet light) may induce the conidia formation of *E. ampelina* as a stress response.

**FIGURE 2** Morphological characterization and infection process of *Elsinoe ampelina*. Colony morphology of a 25-day-old *E. ampelina* isolate grown on potato dextrose agar (PDA) (a, b). Aerial mycelium on PDA (c). Scanning electron micrograph of a conidium (d). Conidial germination, forming several germ tubes on grape leaves after trypan blue staining (e). Conidial germination and appressoria formation on grape leaves (f). Transmission electron micrograph of infection hyphae in leaves of *Vitis vinifera* ‘Red Globe’ (g). *E. ampelina* infection hyphae in Red Globe leaves (h). Scale bars: (a) 0.5 cm, (b) 100 µm, (c, e, f, h) 10 µm, (d, g) 1 µm.

4 | DISEASE CYCLE, EPIDEMIOLOGY, AND MANAGEMENT

The sexual stage of *E. ampelina* is not clearly understood, but a model for the disease cycle has been proposed (Pirrello et al., 2019). Sclerotia are formed on diseased grape tissue from late summer to winter (du Plessis, 1940). Although the factors responsible for this change are unknown, hot dry weather and cane hardening may be the main factors (Magarey, Coffey, et al., 1993). Sclerotia may overwinter on infected berries, leaves, live canes, and pruned canes on the ground with an incubation period of 2–5 years and have a higher viability on live canes than those on pruned canes on the ground (Brook, 1992; Paufilova, 1950; Suhag & Grover, 1972). Primary conidial sporulation can occur from overwintering sclerotia even at low temperatures (Anderson, 1956; Brook, 1973). Anderson (1956) has reported that aside from conidia, the ascospores of *E. ampelina* also cause primary infection; however, little is known about the ascospore dispersal distance. Conidia are spread within 7 m from the source of inoculum during rainfall or overhead irrigation (Brook, 1973; Mirica, 1988).
The conidial germination and infection of *E. ampelina* in China (Li et al., 2019) are the same as that described by Braga et al. (2019) in Brazil based on histopathological observation. Every conidium forms one to five germ tubes during germination on grape leaves (Figure 2e,f) (Braga et al., 2019; Li et al., 2019). Cuticle degradation is observed on the surface of grape leaves underneath germ tubes, and direct penetration of *E. ampelina* occurs on the leaf surface with or without appressorium formation (Figure 2g) (Braga et al., 2019). Afterwards, moniliform hyphae are formed (Figure 2h) and enlarged in intercellular and intracellular spaces of grape tissues during the colonization (Li et al., 2019). The collapse of epidermal and parenchyma cells is observed in the centre of lesions, where infected tissues become necrotic (Braga et al., 2020). As colonization develops, acervuli are formed on the lesions and new conidia are produced, which are dispersed by rain splash and infect other healthy grape tissues (Braga et al., 2020; Li et al., 2019). Suitable environmental conditions, such as high humidity and temperature, are required for intense conidia production, while light has little effect on sporulation (Kore & Gurme, 1978). Little information is known about the environmental conditions required for ascospore production. Overall, sclerotia carry the fungus in diseased grape tissues through winter and produce conidia in the spring. The conidia and the ascospores infect the healthy and young leaves, stems, and berries, thereby causing lesions, forming acervuli, and producing new conidia to spread the disease during the growing season (Figure 3).

The infection and establishment of *E. ampelina* are affected by agroclimatic conditions. Several models have been developed to predict the infection and incidence of anthracnose. A multiple linear regression model and correlation coefficient are used to predict the disease incidence of grape anthracnose by monitoring relative humidity, rainfall, and air temperature (Thind et al., 2001). The epidemiological study of Carisse and Lefebvre (2011) with a nonlinear sigmoid model revealed an overlap in the availability of primary and secondary inocula during the period of rapid leaf growth. Carisse and Morissette-Thomas (2013) have established survival analysis to investigate the factors influencing defoliation using the proportion

![Diagram](image-url)
of leaf area diseased. Ji et al. (2020) developed a mechanistic model based on published literature for the entire life cycle of *E. ampelina* and provided highly accurate predictions of infection occurrence. The influence of leaf wetness duration, temperature, and age-related susceptibility on infection of *E. ampelina* was studied to estimate the risk of anthracnose (Carisse et al., 2020, 2021). Forecast models are necessary to schedule fungicide application and improve management strategy, but more information is needed to fully understand the epidemiology of grapevine anthracnose.

High humidity and precipitation are important factors for the disease development of grape anthracnose. Humidity is required for most of the processes during anthracnose development in primary sporulation, conidial spread, and infection (Magarey, Coffey, et al., 1993). Sporulation from acervuli on the lesions does not require free water but increases with humidity. Free water for 12 hr as a film is required for conidial germination (Dubos, 2000). A similar study shows that conidia are germinated after 12 hr in susceptible grape leaves under high relative humidity (Li et al., 2019). The incidence of anthracnose is high when humidity and rainfall are high at moderate temperature. Brook’s (1992) study shows that the first infection may occur with rainfall of 1–2 mm in New Zealand and Rao and Dakshinamurty (1964) reported that a minimum rainfall of 50 mm over 3 days is sufficient to cause severe infection on the vine foliage. In India, March–April and August–September are the two peaks of grape anthracnose severity, thereby showing correlation with rainfall and temperature (Thind et al., 1992). Similarly, the development of grape anthracnose is favoured by rainy seasons in south Brazil (Barros et al., 2015; Sônego et al., 2005). Therefore, the disease causes increased damage during rainy seasons or areas under high humidity.

Temperature is generally not a limiting factor for grape anthracnose but influences infection and disease development. First, conidia are formed and released from sclerotia when temperatures reach 2 °C with at least 24 hr of wetness in the spring (Mírica, 1988; du Plessis, 1940). Second, conidial germination and infection can occur at temperatures ranging from 2 to 32 °C with the optimal temperature between 24 and 26 °C (Carisse et al., 2020; Dubos, 2000; Li et al., 2018; Thind, 1995). On the surface of grape leaves, the conidial germination rate reaches 95% at 26 °C after 24 hr (Malakhova, 1977) and 85% at 25 °C after 3 days (Li et al., 2019), respectively. Under controlled conditions with optimal wetness, the infection of *E. ampelina* needs 7–10, 4–7, 2–4, and 1.5–2 hr at 12, 16.5, 21, and 30 °C, respectively. The incubation period of *E. ampelina* needs 7–12 days at 12 °C and 3–4 days at 21 °C. Conidial production requires 14 and 5 days at 12 °C and 21 °C, respectively (Brook, 1973).

Grapevine anthracnose is still a serious fungal disease if not controlled properly. Recent reports on disease control are not available, but several measures have been suggested to control the disease. Fungicide treatment is the most effective measure to protect plants from infection by *E. ampelina*. Many fungicides, such as chlorothalonil, captan/captanol, quinone outside inhibitors (QoIs), and demethylation inhibitors (DMI), have been reported to provide effective disease control (Coffey et al., 1991; Emmett & Nair, 1991; Magarey et al., 1977; Shetty et al., 2014). Dithiocarbamate fungicides, such as ziram and thiram, reduce the severity and occurrence of grape anthracnose (Coombe, 1953; Taylor, 1954). Deokate et al. (2002) have reported that carbendazim, a methyl benzimidazole carbamate fungicide, controls the pathogen by inhibiting microtubule assembly during mitosis. Magarey, Emmett, et al. (1993) have evaluated the use of preventive fungicides in field and greenhouse, and the results show that dichlofluanid, captan, chlorothalonil, and fluazinam are effective against *E. ampelina*. However, other fungicides, such as copper, metalaxyl, lime sulphur, vinclozolin, and procymidone, used for the control of other grapevine diseases are ineffective for anthracnose (Coffey et al., 1991; Emmett et al., 1981; Magarey & Emmett, 1992; Moore & Schroeder, 1983). In Brazil, four fungicides (thiophanate-methyl, captan, chlorothalonil, and mancozeb) have been applied for disease control (Barros et al., 2015). The disease severity should be maintained below 25% leaf area diseased to avoid premature leaf drop (Carisse & Morissette-Thomas, 2013).

A fungicide spray programme should be initiated early in the growth (budburst) and dormancy stages on susceptible cultivars to prevent the infection of young leaves and reduce the overwintering inoculum, especially when a history of anthracnose in the vineyard is present (Carisse & Lefebvre, 2011; Magarey, Coffey, et al., 1993; Santos et al., 2020). Copper sulphate, lime sulphur, and Bordeaux mixture have been used as dormant sprays (du Plessis, 1940). However, copper-based fungicides can cause phytotoxicity to young tissues and should be replaced by the effective dithiocarbamate fungicides (Coome, 1953; Taylor, 1954). In addition to fungicide applications, biocontrols are explored to inhibit grape anthracnose. Mycelial inhibition of 78% and 72% was recorded in treatments of onion and garlic extracts, respectively (Patil et al., 2018). Li et al. (2020a) found that an endophytic fungus, *Albifimbria verrucaria*, from the leaves of the Amur grape has a biocontrol activity against *E. ampelina*. Additionally, cultivar selection, removal of canes with cankers, and pruning techniques can reduce the occurrence of grape anthracnose in climates or areas prone to the disease.

### 5 | GENOMICS AND PATHOGENICITY OF *E. AMPELINA*

Genome sequencing is effective in revealing the underlying virulence or pathogenicity. Thus far, two assembled *E. ampelina* genomes have been deposited in the NCBI genome database (Table 1). The first genome sequence of *E. ampelina* (28.29 Mb) was obtained from the Chinese isolate YL-1 using a combination of Illumina short-read and PacBio long-read sequencing technologies (Li et al., 2020b). Another genome was sequenced from the *E. ampelina* strain CECT 20119 using only the Illumina sequencing data, and its assembly comprises 28.27 Mb (Haridas et al., 2020). Although similar genome sizes were assembled for the two isolates, a high number of genes was predicted for the *E. ampelina* CECT 20119. In addition to genome size, the GC contents of the two genomes are similar. The comparative genome analysis shows that *E. ampelina* from the order Myriangiales
and *Aureobasidium* sp. from the order Dothideales are clustered in the same clade (Haridas et al., 2020). This result is also supported by the phylogenetic analysis, indicating a close genetic relationship among fungi belonging to the orders Myriangiales and Dothideales (Schoch et al., 2006).

Different aggressiveness levels have been observed among *E. ampelina* isolates. Pathogenicity tests on cv. Black Queen leaves revealed that seven out of 11 isolates showed various degrees of virulence (Sompong et al., 2012). Poolsawat et al. (2012) found that the disease severity on leaf pieces from 10 grapevine genotypes with the Nk4-1 isolate sampled from cv. Nakho Ratchasima was higher than that with the Rc2-1 isolate sampled from cv. Ratchaburi grapes. Although five isolates were isolated from different regions in Thailand, Rc2-1 and Cr1-1 are the most virulent, and Nk5-1 is the least virulent (Poolsawat et al., 2010). Ten *E. ampelina* isolates from Brazil caused anthracnose symptoms on Moscato Giallo and Niagara Rosada with high conservation in the internal transcribed spacer (ITS) region and the elongation factor 1-α (TEF) regions, but not in the histone H3 (HIS3) gene (Santos et al., 2018a). Therefore, screening the virulence of different isolates can be useful for the identification of resistant hosts, and representative isolates should be used in grape-*E. ampelina* disease system studies.

The collapse of epidermal, parenchyma, and collenchyma cells has been observed in infected grape tissues by *E. ampelina* (Braga et al., 2019), indicating that plant cell wall-degrading enzymes and cutinases are produced by *E. ampelina*. Similar findings are also reported in citrus leaves infected by *E. fawcettii* (Paudyal & Hyun, 2015). Additionally, a total of 158 plant cell wall-degrading enzymes were identified in the *E. ampelina* genome (Li et al., 2020b), and these enzymes may be related to infection and colonization events. A recent study also revealed that carbohydrate-active enzymes, pathogen–host interaction genes, and secreted proteins are upregulated during infection (Li et al., 2021). Fungal metabolites can act as important virulence factors during the infection of plant cells (Möbius & Hertweck, 2009). The culture filtrate of *E. ampelina* has been used to evaluate the resistance of different grape genotypes (Kim et al., 2008; Louime et al., 2011; Yun et al., 2004). Results from these studies indicate that the metabolites of *E. ampelina* produce similar symptoms to those caused by the conidial infection. Although the key virulence metabolites for *E. ampelina* are still unknown, many studies have shown that the elsinochromes produced by *Elsinoe* spp. are vital to the pathogenic process (Jiao et al., 2019; Liao & Chung, 2008; Wang et al., 2009). Elsinochromes, which are light-dependent and nonhost-selective phytotoxins, are structurally similar to the cercosporin phytotoxin (Yang & Chung, 2010). The colour of elsinochromes is red or orange, which is similar to the colour of *E. ampelina* colonies (Li et al., 2018; Santos et al., 2018c). Elsinochromes may be potential virulence factors associated with the development of grape anthracnose and further studies are needed to address this gap.

## 6 | GENETICS AND HOST RESISTANCE

Although the colony morphology varied in colour and shape among isolates, this morphological diversity did not reflect genetic diversity (Poolsawat et al., 2010; Santos et al., 2018a, 2018c). The genetic variability among *E. ampelina* isolates has been assessed using random amplified polymorphic DNA (RAPD) markers and phylogenetic analysis based on ITS, HIS3, and TEF sequences (Poolsawat et al., 2010; Santos et al., 2018a). A high degree of genetic variation among 19 isolates from different geographical regions of Thailand was found using RAPD (Poolsawat et al., 2010). However, phylogenetic analysis based on ITS and TEF sequence data revealed low genetic variability among Australian, Brazilian, and North American isolates (Santos et al., 2018a, 2018c). HIS3 sequence analysis revealed 54 polymorphic sites, including four base substitutions and the absence of the intron region, only detected in a few Brazilian isolates (Santos et al., 2018c).

Disease incidence and disease severity assessments are fundamental for identifying the resistance among grape cultivars or comparing the virulence among the fungal isolates. The disease intensity of anthracnose on leaves is estimated based on the lesion score, lesion numbers, and disease severity using standard area diagrams and image analysis software. Recently, two standard area diagrams were developed to evaluate the grapevine anthracnose on leaves, fruit, and shoots (Modesto et al., 2020; Santos & Spósito, 2018). Lesion score can be evaluated based on lesion numbers on the basis of each inoculated droplet using a scale from 1 to 5, and then the score is transformed using the \( x + 1 \) formula for disease assessment (Poolsawat et al., 2010). The lesion number also can be quantified in the part or whole leaf area (Louime et al., 2011; Santos et al., 2018a, 2018c). In addition to lesion numbers, lesion diameter and lesion area have been also used for grape anthracnose evaluation (Kono et al., 2012; Murria et al., 2018; Poolsawat et al., 2012). The incidence of anthracnose

### TABLE 1 Summary of Elsinoe ampelina isolates that have fully sequenced genomes

| Elsinoe ampelina isolates | YL-1 | CECT 2019 |
|---------------------------|------|-----------|
| Genome size (Mb)          | 28.29| 28.27     |
| Sequencing technology     | PacBio+Illumina | Illumina |
| Sequencing depth          | 253.6×| 100.2×    |
| Contigs                   | 13   | 503       |
| GC content (%)            | 49.50| 49.60     |
| Number of genes           | 8,057| 10,207    |
| Average gene length (bp)  | 1,484| 1,693     |
| CAZymes                   | 407  | 528       |
| Secondary metabolite      | 20   | 19        |
| clusters                  |      |           |
| NCBI accession            | SWYM000000000 | JAAEiW000000000 |
| Reference                  | Li et al. (2020) | Haridas et al. (2020) |

Note: CAZymes, carbohydrate-active enzymes.
on berries can be estimated based on percentage lesions over the entire berry surface with a scale from 0 to 7 (Li et al., 2008). For grape seedlings, fewer than 10 lesions per seedling are indicated as resistant and more than 20 lesions are susceptible (Hopkins & Harris, 2000).

Mortensen (1981) has proposed a hypothesis that the inheritance of resistance to grapevine anthracnose is controlled by three independent genes in V. labrusca (American) and V. vinifera (European) cultivars. An$_1$ and An$_2$ appear to be dominant genes for susceptibility, whereas An$_3$ is probably a single dominant gene for resistance. The resistant trait of grape anthracnose is highly heritable on the basis of segregating populations of grape F$_1$ hybrids (Poolsawat et al., 2013; Wang et al., 1998). Additionally, additive genetic effects are predominant over other types of gene action, and male parents contribute more additive genetic effects than female parents for the inheritance of grape anthracnose resistance (Poolsawat et al., 2013). Wang et al. (2000) have developed a RAPD marker OP13-300, which is linked to an anthracnose resistance gene, using V. quinquangulalis ‘Shang-24’ as a resistance source to provide help for molecular marker-assisted breeding. However, no significant association between the RAPD marker and anthracnose resistance in seven cross combinations or grapevine in Thailand was observed (Poolsawat, 2010). The SCAR marker OPB 151247 was linked to the anthracnose resistance locus in grape (Kim et al., 2008). Three resistance gene analog-single-strand conformation polymorphism (RGA–SSCP) markers were subsequently found with significant correlation with anthracnose resistance using the interspecific hybrid NY65.0550.04 as a resistance source (Tantasawat et al., 2012). The markers from different resistance sources seem to have limitations of application to some extent.

Evaluating the resistance to E. ampelina is an important foundation for resistance breeding to grape anthracnose. Screening for such resistance among grape genotypes has been performed in the field, greenhouse, or laboratory. Laboratory and greenhouse experiments for screening resistance to anthracnose saves labour and are economical and consistent with the field evaluation results (Hopkins & Harris, 2000; Poolsawat et al., 2012). More than 26 Vitis species are resistant to grape anthracnose. Mortensen (1981) and Patil et al. (1990) have reported sources of resistance to anthracnose in V. aestivalis, V. champini, V. labrusca, V. munsoniana, V. rotundifolia, V. rupestris, V. shuttleworthii, V. simpsoni, V. smalliana, V. tiliaefolia, and V. vulpina. Genetic resources for anthracnose resistance are also found in several Asiatic and Chinese Vitis species, including V. adstricta, V. amurensis, V. bashanica, V. coignetiae, V. davidii, V. flexuosa, V. hancockii, V. liubanensis, V. piaezkii, V. pseudoreticulata, V. qinlingensis, V. quinquangulalis, V. romanetii, V. thunbergii, and V. yeshanensis (Ahn, Kim, Jo et al., 2014; Li et al., 2008; Lu, 1997; Tian et al., 2008; Wang et al., 1998). However, some genotypes are moderately resistant and anthracnose epidemics can occur under high disease pressure and/or favourable weather conditions. For instance, V. labrusca cultivars are highly susceptible to anthracnose in Brazil under high humidity conditions (Barros et al., 2015; Santos et al., 2018a). Muscadinia grapes (V. rotundifolia) are partly resistant to E. ampelina (Mortensen, 1981; Yun et al., 2007), whereas V. vinifera, such as cultivars Chardonnay, Carignane, Red Globe, and Thompson Seedless, are highly susceptible (Louime et al., 2011). Grape anthracnose is a worldwide disease because V. vinifera is one of the finest table and wine grapes in the world. Therefore, evaluating and exploiting resistant genetic resources is useful for grape production and decreased fungicide applications.

The mechanism of resistance to anthracnose remains unclear, but various studies have investigated the role of biochemical compounds and related genes in disease resistance. Berries become highly resistant to E. ampelina when they reach a soluble solid content of 5%–7% (Brook, 1973). High levels of total phenols, free amino acids, proline, α-tocopherol, total soluble proteins, and soluble sugars and an increase in the activity of peroxidase and polyphenol oxidase are found in the resistant variety H516 (Murria et al., 2018). Braga et al. (2019) have also found that phenolic compounds accumulate in infected areas compared with healthy areas, indicating the accumulation of total phenols in resistance response. The hyphal growth of E. ampelina was suppressed on leaves of resistant V. quinquangulalis when compared with susceptible V. vinifera (Han et al., 2020). Exogenous active elicitors, such as chitosan and benzo-(1,2,3)-thiadiazole-7-carboxothioic acid S-methyl ester, induce resistance to E. ampelina and involve the salicylic acid (SA) pathway (Prakongkha et al., 2013a, 2013b). Several antifungal genes, such as genes encoding chitinase, stilbene synthase, chalcone synthase, polygalacturonase-inhibiting protein, and lipid transfer-protein in Muscadine grapes (V. rotundifolia), are rapidly expressed in tolerant cultivars after E. ampelina infection (Louime et al., 2011). Proteomic analysis between tolerant and susceptible genotypes shows that the gene expression of chitinase, stilbene synthase, glutamine synthetase, mitochondrial ATPase, and ribulose-1,5-bisphosphatecarboxylase are upregulated in tolerant genotypes (Lake Emerald and Blue Lake) compared with susceptible genotypes (Blanc du Bois and Suwannee) (Vasanthaiah et al., 2009, 2010), suggesting that these genes may be partly responsible for anthracnose tolerance. Of the 33 genes encoding thiamatin-like proteins, 23 are responsive to E. ampelina infection (Yan et al., 2017). Jayasankar et al. (2003) have reported that the spore germination and the hyphal growth of E. ampelina are significantly inhibited by a V. vinifera thiamatin-like protein. Several genes encoding β-1,3-glucanase (Ahn, Kim, Yun et al., 2014), nucleotide-binding site domain proteins (Islam et al., 2015a; Seehalak et al., 2011), receptor-like protein kinase (Islam et al., 2015b), glutathione S-transferase (Ahn et al., 2016), Mlo-like proteins (Islam & Yun, 2016a), carboxylesterase proteins (Islam & Yun, 2016b), and EDS1-like proteins (Islam & Yun, 2017) are responsive genes to E. ampelina infection, indicating that these genes may participate in the disease-resistance response. The gene expression profile was analysed in grape associated with resistance to E. ampelina based on suppressive subtraction hybridization (Gao et al., 2012) and de novo transcriptome (Ahn, Kim, Jo et al., 2014), and the results showed that secondary metabolic and biosynthetic processes are the main biological processes in response to E. ampelina. Uprogulated pathogenesis-related protein 1 and 10, chitinase, WRKY transcription factor, and jasmonate ZIM-domain
protein seem to be involved in *V. quinquangularis* resistance against *E. ampelina* (Gao et al., 2012).

7 | FUTURE PROSPECTS

The grape anthracnose is a global fungal disease that causes a significant reduction in yield and quality. This disease is a threat for grape production. In recent decades, studies have increased our knowledge about the understanding of biological characteristics of *E. ampelina*, infection behaviour, epidemiology, and management of anthracnose. The recent availability of genomic data of *E. ampelina* has provided important clues to reveal grape–*E. ampelina* interaction and develop methods for rapid detection. Although some areas of the disease remain unclear, we believe that future research should address the following areas.

First, although the sexual morph of *E. ampelina* is known, future studies are needed to reveal the occurrence of sexual reproduction, ascospore production, and its infection process. Such results clarify the role of ascospore in the disease cycle of the grape anthracnose. Knowledge regarding the population structure and the diversity of *E. ampelina* is still scarce. Understanding of the sexual recombination and mating type is necessary.

Second, the *Vitis* germplasm resistant to *E. ampelina* has been identified in recent decades. DNA markers or quantitative trait loci linked to the resistance genes should be developed for breeding programmes. Resistance breeding is fundamental to control grape anthracnose. Grape breeders should use identified resistant genotypes, particularly wild grapes, and transfer the resistant traits to current cultivars.

Third, further research should be conducted on host-pathogen interactions to improve our knowledge of resistance mechanisms. Genome sequences can serve as a powerful tool to identify fungal genes underlying virulence, especially the effectors. Further functional studies are needed to confirm the essential genes of *E. ampelina* and regulate the immune response of grapevines. Additionally, many fungal metabolites are identified as important virulence factors. The key phytotoxins of *E. ampelina* and its pathway genes should be identified, especially the synthesis and the regulation of elsinochromes.

Lastly, effective control measures should be investigated to manage the disease. Several areas, including sources of inoculum, survival and spread of *E. ampelina*, environmental factors, fungicide efficacy, effective pathogen detection and identification, and disease prediction models, should be conducted on a wide global cooperation. New control strategies should be developed to reduce the application of fungicides and environmental risks using bioactive molecules and biocontrol strategies.

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DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created or analysed in this study.

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