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LITERATURE REVIEW

Gray mold caused by *Botrytis cinerea* limits grape production in Chile

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Abstract

B.A. Latorre, K. Elfar, and E.E. Ferrada. 2015. Gray mold caused by *Botrytis cinerea* limits grape production in Chile. Cien. Inv. Agr. 42(3): 305-330. Gray mold (GM) caused by *Botrytis cinerea* is a major disease of grapes (*Vitis vinifera*) that substantially reduces the yield and quality of grape production in temperate and humid regions of the world. *B. cinerea* is a necrotrophic fungus that attacks the non-lignified aerial organs of grapes; in particular, berries are highly susceptible during ripening. The polycyclic nature and exponential progress exhibited by GM at the beginning of the its epidemic, as well as the abundant inoculum production, the high dissemination efficiency, the wide host range and the high genetic variability of *B. cinerea*, explain the difficulties encountered in attempting to control GM. At present, integrated disease management, including cultural and chemical control, is the main control strategy. These control measures can be used to reduce the initial inoculum or to lower the disease infection rate. However, control measures that reduce the infection rate are the most effective means of controlling GM. Important progress toward understanding the complexity of the biology and epidemiology of this pathogen has occurred in recent decades. This has allowed the improvement and development of more effective and sustainable control strategies against *B. cinerea*. This review article provides a recent update regarding grape GM, with special emphasis on Chilean production conditions.

Key words: *Botryotinia fuckeliana*, bunch rot, epidemiology, fungicides, necrotroph, *Vitis vinifera.*

Introduction

Gray mold (GM) caused by *Botrytis cinerea* Pers. is the primary deteriorative factor affecting grapes (*Vitis vinifera* L.) in Chile and other grape-producing countries worldwide. GM reduces the yield and quality of wine and table grapes in geographical locations characterized by humid and temperate weather conditions during the spring and summer months.

Grape GM affects the aerial organs, i.e., the berries, which are highly susceptible from veraison (color change, berries with > 7% total soluble solids) to harvest. Grape GM is the most important cause of postharvest decay of table grapes during storage, transit to markets and commercialization. GM has limited table grape production in Chile, which is currently confined to relatively dry and temperate geographical

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locations extending from the Atacama (27°22' S) to the Maule (34°98' S) regions, along a north-south axis of approximately 1,000 km.

Notable textbooks (Coley-Smith *et al*., 1980; Elad *et al*., 2004) and review articles (Jacometti *et al*., 2010; Mundy, 2008; Romanazzi *et al*., 2012; Steel *et al*., 2013; van Kan *et al*., 2014; Williamson *et al*., 2007) have been published on the biology, epidemiology and control of the disease caused by *B. cinerea*. In this article, we reviewed numerous studies and discuss the factors that affect grape GM, as well as control strategies with special reference to the Chilean situation. This review further expands upon previously discussed information (Latorre, 2007).

Symptoms

Symptoms of GM are observed on berries, leaves, shoots, buds and canes, of which berries are the most affected grape organ.

Berry symptoms often appear prior to harvest or during cold storage, transit or commercialization (Figure 1). Berry symptoms commonly appear as reddish-brown discoloration that starts at the pedicel end and progresses toward the stylar end (Holz *et al*., 2003). Small (approximately 0.5 mm in diameter), round, reddish-brown necrotic spots often appear on the berry cheek after rainfall near harvest time, and reddish-brown decay at the stylar end has been observed in Chilean vineyards (Zoffoli *et al*., 2009). As the berry infection progresses, a loose skin ('slip-skin'), berry split and soft and watery decay are observed. Infected berries usually leak, which favors the colonization and sporulation of *B. cinerea* on the berry surface and promotes the dissemination of *B. cinerea* to neighboring berries, forming a nest of rotted berries (Figure 1B). Finally, the infected berries dehydrate and shrivel, remaining as berry mummies attached to the clusters or on the ground litter.

Leaf symptoms appear early in the growing season and are characterized by small brown necrotic spots around the veins and brown "U- or V-" shaped lesions at the margins of the leaf blade. Under humid conditions, abundant gray sporulation can be observed on the underside of the diseased leaf tissues. Occasionally, shoot blight, blossom blight and dark brown necrosis of the rachis are observed in spring. Shoot blight frequently occurs in grape nurseries. In Chile, *B. cinerea* can infect partially lignified canes after early frosts in autumn, resulting in the development of whitish necrotic lesions with large, irregular, black sclerotia (Latorre, 1986; Latorre and Váquez, 1996).

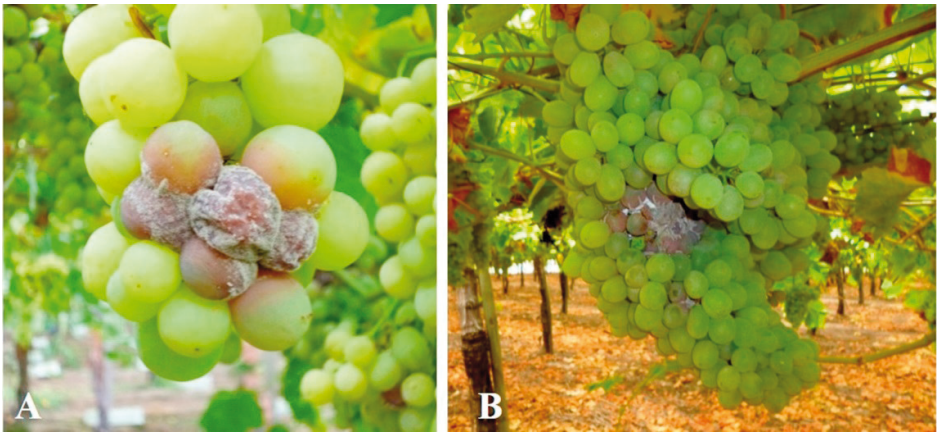

Figure 1. Gray mold symptoms on Thompson Seedless table grapes. A. Reddish brown discoloration, partially dehydrated berries and the presence of gray mold. B. Nest of diseased berries in a very compact cluster.

The pathogen

B. cinerea is a ubiquitous fungal pathogen; it is a necrotrophic, haploid, heterothallic ascomycete that attacks over 200 cultivated plant species and numerous wild plants, mostly dicotyledonous plants, worldwide (Elmer and Michailides, 2004). *B. cinerea* is a major pathogen of fruits, vegetables, ornamentals and forest trees in humid and temperate geographical zones across the world. In addition to grapes, this fungus has been reported on over 60 different cultivated host plants of different taxonomic categories in Chile (Acuña 2008, 2010; Mujica and Vergara, 1980). The capabilities of *B. cinerea* to colonize plants as an endophyte and its importance have been discussed extensively by van Kan *et al.* (2014).

B. cinerea develops white to gray colonies on potato dextrose agar and other culture medium (Figure 2A), producing filamentous, hyaline, branched and septate mycelia with prominent cell walls. In contrast to other fungi, it contains a low proportion of chitin and a high proportion of neutral sugars and proteins (Cantu *et al.,* 2009). Most isolates produce abundant multinucleate (3-6 nuclei) conidia (macroconidia) which are unicellular, hyaline to slightly colored, smooth, ovoid to ellipsoid, and measure 10-12 × 8-10 µm. Conidia are produced on short sterigmata on the swollen tips of aerial, free, branched conidiophores (Holz *et al*., 2004) (Figure 2B, C). Espermatia (microconidia) rarely occur in nature; they are small, globose, unicellular, uninucleates that scarcely germinate and never infect plants (Urbasch, 1985). Chlamydospores have been described in *B. cinerea* and can also serve as a survival structure (Holz *et al.,* 2004; Urbasch, 1986).

Black, melanized, elongated or spherical sclerotia measuring 3 to 5 mm in length are produced under unfavorable conditions *in vitro* and *in planta*. Sclerotia play an important role in pathogen survival, dispersal and multiplication. They are commonly found on partially lignified grape shoots that are colonized by *B. cinerea* after early frosts in autumn (Elmer and Michailides, 2004).

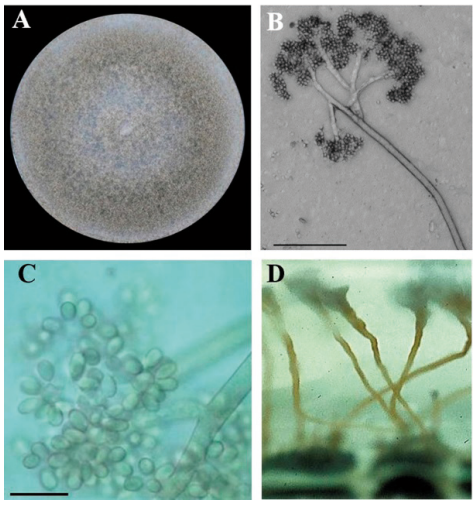

Figure 2. *Botrytis cinerea*. A. Colony morphology on potato dextrose agar. B. Conidiophore, bar = 100 µm. C. Conidia, bar = 10 µm. D. Apothecia.

B. cinerea produces macroscopic, stipitate apothecia that originate from sclerotia (Faretra and Antonacci, 1987) (Figure 2D). Nevertheless, the sexual stage rarely occurs in nature, but apothecia and ascospores can be obtained under controlled laboratory conditions (Faretra and Antonacci, 1987; Faretra *et al*., 1988).

Two mating types, *MAT1-1* and *MAT1-2*, have been described; they must both be present for ascospore production because most *B. cinerea* isolates are heterothallic. However, homothallic (self-fertile) strains and heterokariotic strains with *MAT1-1* and *MAT1-2* nuclei have been reported (Faretra *et al*., 1988; van Kan *et al*., 2010). The presence of sexually compatible strains has been demonstrated in *B. cinerea* in Chile (Faretra and Latorre, 2007). Therefore, environmental factors or factors other than the presence of sexual compatibility strains may explain the lack of ascospore production in Chile.

Although the sexual stage rarely occurs in nature, high genetic variability has been reported in populations of *B. cinerea* (Beever and Parkes, 1993; Beever and Weeds, 2004; Diolez *et al*., 1995¸ Giraud *et al*., 1997; Martinez *et al*., 2003; Muñoz *et al*., 1999, 2002; Thompson and Latorre, 1999; van der Vlugt-Bergmans *et al*., 1993). This

genetic diversity may be explained by (i) cellular aneuploidy (abnormal number of chromosomes in a cell) and heterokaryosis (presence of two or more genetically different nuclei in a cell), (ii) the presence of transposable elements such as *Boty* and *Flipper* in the genome, and (iii) spontaneous mutations (Büttner *et al*., 1994; van Der Vlugt-Bergmans *et al*., 1993).

Considerable genetic variability based on RAPD-PCR or PCR-RFLP analysis was found among Chilean isolates of different origins (Muñoz *et al*., 1999; Thompson and Latorre, 1999). This genetic variability was later associated with important differences in adhesion, the percent germination on tomato cutin and the ability to colonize tomato leaves, which were demonstrated between genetic groups of Chilean *B. cinerea* isolates (Cotoras and Silva, 2005).

Based on the presence or absence of the transposons *Boty* and *Flipper* in the genome, two sub-populations, *transposa* and *vacuma*, have been reported in *B. cinerea* (Giraud *et al*., 1997, 1999; Levis *et al*., 1997). The transposable elements *Boty* and *Flipper* were described for the first time by Diolez *et al*. (1995) and Levis *et al.* (1997), respectively. *Transposa* isolates carry *Boty* and *Flipper,* whereas both of these transposable elements are absent in *vacuma* isolates (Giraud *et al*., 1997). Isolates carrying either *Boty* or *Flipper* in the *B. cinerea* genome have also been described (De Miccolis Angelini *et al*., 2003; Giraud *et al*., 1999; Muñoz, *et al*., 2002). In addition, the presence of *Boty* and *Flipper* has been found in Chilean populations of *B. cinerea* that affect table grapes, and *transposa* is the most common isolate (Esterio *et al*., 2011). However, the presence of transposon elements in *B. cinerea* appears to be independent of the ability of the organism to cause infection (Ciliberti *et al*., 2015a).

Current evidence suggests that *B. cinerea* is a complex of species that comprises new phylogenetic species, some of which are recently described cryptic species (Fournier *et al*., 2005; Johnston *et al*., 2014; Lorenzini and Zapparoli, 2014; Walker *et al*., 2011; Zhou *et al.* 2014). Similarly, phylogenetic analysis suggests the presence of cryptic species in *B. cinerea* from stone fruits in Chile (Ferrada *et al.,* 2014).

B. cinerea sensu stricto (Group II) is predominant in vineyards and includes the *transposa* and *vacuma* isolates, whereas Group I occurs in low frequency and includes only *vacuma* isolates (Albertini *et al.,* 2002; Fournier *et al*., 2003, 2005; Vercesi *et al*., 2014). The latter isolates are naturally resistant to fenhexamid and are highly susceptible to fenpropidin and edifenphos (Leroux *et al*., 2002). However, there is no consensus regarding the relationship between sensitivity to fenhexamid and the presence of *Boty* and/or *Flipper* elements in the *B. cinerea* genome (Ma and Michailides, 2005). Recently, Vercesi *et al.* (2014) demonstrated that fungicide applications (fenhexamid or cyprodinil+fludioxonil) did not influence the transposon distribution patterns. Isolates belonging to Group I have been identified as *B. pseudocinerea* sp. nov. (Walker *et al*., 2011), and these isolates appeared to play a minor role in the gray mold epidemiology of grapevines (Vercesi *et al.,* 2014).

Regarding the functionality and molecular studies of genes, different research groups have extensively studied the genetic and molecular aspects of *B. cinerea.* The genome sequences of *B. cinerea* strains B05.10 and T4 have been published (Amselem *et al*., 2011). Additionally, the functionality of the genes involved in the pathogen-host interactions that affect both pathogenicity and virulence have been reported (Baldwin *et al*., 2006).

Survival, inoculum sources and dispersal

B. cinerea is a necrotrophic, facultative saprophyte that can survive as mycelia and/or sclerotia on grapes (tendrils, cane debris, senescent clusters, and senescent leaf petioles), in numerous alternative hosts and plant residues in the vineyards,

on other neighboring crops and on some wild plants (Mundy *et al*., 2012). Depending on the temperature, *B. cinerea* mycelia can survive for up to 30 weeks in grape vine prunings (Elmer and Michailides, 2004).

Conidia can contaminate grape clusters; however, they survive for a short period on grape berry surfaces, particularly if they are exposed to sunlight (Coertze and Holz, 2002; Rotem and Aust, 1991). Nevertheless, long-term (up to 16 weeks) conidial survival has been reported on kiwifruits (Walter *et al*., 1999).

Therefore, abundant conidia are produced in multiple and very diverse overwintering structures. Conidia are the primary inoculum during flowering and ripening, and it is likely that from the beginning of spring, there is always an abundant and constant availability of viable conidia in the air. The highest conidial concentrations occur on warm humid days after rain (Diaz, *et al*., 1998; Leyronas and Nicot, 2013; Mundy *et al*., 2012; Rodríguez-Rajo *et al*., 2002, 2010; Stepalska, and Wolek, 2005). The conidia are produced in multiple and very diverse substrates (Coertze and Holz, 1999; Corbaz, 1972; Holz *et al*., 2003; Nair and Nadtotchei, 1987; Williamson *et al*., 2007).

In Chile, conidia are disseminated locally by wind and water droplets but can also be disseminated by some insects in countries where important insect pests infest grape clusters (Holz *et al.,* 2004). For instance, the larvae of *Lobesia botrana* can disseminate viable conidia of *B. cinerea* within vineyards (Fermaud and Le Menn, 1989; Pavan *et al*., 2014). Therefore, the recent detection of *L. botrana* in Chile poses an important epidemiological risk for GM development.

Grape GM can also be disseminated by berry to berry contact due to mycelial growth from a diseased to a healthy berry, which is very frequently observed at harvest and in stored table grapes in Chile. Berry to berry dissemination is favored on highly compact clusters and thin skin cultivars and usually explains the presence of nests of rotted berries (Figure 1B).

Infection

B. cinerea can infect grape berries during seasonal grape berry development. Immature berries are considered relatively resistant to *B. cinerea*. However, immature berries rapidly increase in susceptibility as the berries mature (Comménil *et al*., 1997; Deytieux-Belleau *et al*., 2009; Hill *et al*., 1981; Mlikota Gabler *et al*., 2003; Mundy and Beresford, 2007).

Free moisture and temperate are required for infection. Under these conditions, conidia germinate after deposition, hydration and attachment (first stage) to the host surface (Cotoras and Silva, 2005; Doss *et al*., 1995), forming a single or branched germtube, often with an appressorium at the distal end (Viret *et al*., 2004). The appressorium facilitates attachment (second stage) and active (direct) penetration of intact host surfaces by means of penetrating hyphae. Current knowledge indicates that a complex enzymatic process, rather than the pressure caused by hyphae penetration, is primarily responsible for active penetration (Tenberge, 2004). Passive (indirect) penetration can occur through natural openings (stomata and lenticels) and wounds (Holz *et al*., 2003; Kars and van Kan, 2004).

The infection level of GM at maturity is typically the result of (i) latent infections that occur at flowering or during the early stages of berry development and (ii) direct infections during ripining or even later during storage and transit (Mundy *et al*., 2012; Pezet *et al*., 2003).

Infection pathways

At least three infection pathways have been described:

a. Pedicel end infection through the cap scar or receptacle area at the insertion of the pedicel into the berry. Symptoms start at the berry base as a reddish-brown discoloration and progresses toward the stylar end of the grape berry. This appears to be the most frequent *B. cinerea* infection pathway during blooming (Holz *et al*., 2003; Keller *et al*., 2003b; Pezet and Pont, 1986; Viret *et al*., 2004; Zoffoli *et al*., 2009).

b. Stylar end infection through the stigmata at bloom. Symptoms appear at the stylar end of mature berries. This pathway appears to be the least frequently observed, and it is almost non-existent in most grape-growing regions (Holz *et al*., 2003; Pezet and Pont, 1986, Pezet *et al*., 2003; Viret *et al.,* 2004). Nevertheless, it has been observed in table grapes in Chile (Zoffoli *et al*., 2009).

c. Lateral infection through berry cheeks. Symptoms begin as small, round, reddish-brown necrotic spots and skin split. This infection pathway is relatively common after rainfall during harvest and is associated with conidia deposition on the surface of the berry. This is the most frequent infection pathway detected during storage of table grapes in Chile, particularly after rainfall events near harvest time (Zoffoli *et al.,* 2009).

Latent infection

McClellan and Hewitt (1973) were the first to demonstrate the infection of *B. cinerea* through the stigma at the stylar end of the flower. *B. cinerea* remained latent at the stylar end of the grapes, restarting to grow after veraison, causing early rotting at berry maturity. Since then, various reports and further studies have corroborated this finding (Nair and Paker, 1985; Nair *et al*., 1995; Pezet and Pont, 1986; Pezet *et al.* 2003; Viret *et al*., 2004). At present, it is accepted that *B. cinerea* can infect berries at the capfall stage and remain latent in immature grapes. Nevertheless, latent infections can occur during other growth stages of berry development. The final GM incidence results from the cumulative latent infections that occur throughout the growing season (Hill *et al*., 2014).

The latency of *B. cinerea* in grape berries has been related to constitutive and inducible defense mechanisms (Keller *et al*., 2003b). Among the latter, the accumulation of phytoalexins (*e.g.*, resveratrol, a stilbene derivate), which are self-defense secondary metabolites produced in immature grapes (Bavaresco *et al*., 1997; Flamini *et al*., 2013; Goetz *et al*., 1999; van Baarlen *et al*., 2004; Wang *et al*., 2013), and the synthesis of PR-proteins have been extensively studied (Elmer and Michailides, 2004; Williamson *et al*., 2007). The activation of latent infection in mature grape berries has been associated with the weakening of the natural defense barriers and the decline in phytoalexins during repining in susceptible grape cultivars (Bais *et al*., 2000; Goetz *et al*., 1999; Jeandet *et al*., 1991; Keller *et al*., 2003b; Pezet *et al*., 2003; Prusky and Lichter, 2007).

Colonization of senescent tissues

B. cinerea colonizes floral debris, such as senescent stamens, caliptra and aborted berries, between bloom and fruit set, remaining quiescent until later in the growing season. This infected floral debris is often retained in the grape clusters and provides inoculum for berry infection during ripening or even later during storage and transit (Calvo-Garrido *et al*., 2014a; Jacometti *et al*., 2010; Keller *et al*., 2003; Latorre and Vásquez, 1996; Latorre *et al*., 2001; Nair, *et al*., 1988, 1995; Pezet and Pont, 1986; Viret *et al*., 2004; Wolf *et al*., 1997). It is possible that the abundance of pollen and stigma exudates during flowering increases the level of colonization of floral debris (McClellan and Hewitt, 1973).

Predisposing factors

Predisposing factors are genetic (physiological and anatomical), physical (wounds), environmental

(climate and weather conditions) and agronomic (cultural practices), all of which can render grapes more susceptible to or favor the development of the pathogen by enhancing GM severity. Knowledge of these factors has been very important in establishing appropriate control strategies.

Genetic factors

In addition to cluster compactness, several genetic factors, *i.e.,* morphological, anatomical, and chemical features of the berry skin, predispose berries to *B. cinerea* infection in nature. These factors include thin berry cuticles (Comménil *et al.*, 1997; Zoffoli *et al.*, 2009), low epicuticular wax content (Marois *et al.*, 1986, 1987; Percival *et al.*, 1993), high berry porosity (Blaich *et al.*, 1984; Mlikota Gabler *et al.*, 2003), and the number and thickness of the skin cell layers of the berry (Mlikota Gabler *et al.*, 2003). A sensor that enables the measurement of the electrical impedance of the grape berry cuticles and their epicuticular waxes has been developed as a reliable method to estimate the degree of berry susceptibility to GM and could be a valuable tool for genetic analysis in breeding programs (Herzog *et al*., 2015).

Physical factors

The nature of physical factors can be biotic (*e.g.*, insects, birds, snails, other plant pathogens) and abiotic (*e.g.*, rain, hail, frost, sunburn, rapid water intake) (Becker and Knoche, 2012a, b; Coertze and Holz, 1999; Fermaud and Le Menn, 1989; Nair, *et al*., 1988). These factors induce fresh wounds in the skin that are very often microscopic cracks, which prevent the action of the cuticle as a physical barrier to penetration. For instance, on stored table grapes, the overuse of sulfur dioxide can induce hairline microcracks which facilitate *B. cinerea* infection of grapes (Zoffoli *et al*., 2008). Suberized berry injuries do not serve as infection sites (Coertze and Holz, 2002; Elad and Evensen, 1995).

Environmental factors

It is widely accepted that microclimate conditions within the grape canopy, specifically temperature and humidity (relative humidity and free moisture), are key factors for *B. cinerea* infection (Broome *et al*., 1995; English *et al.*, 1989; Nair and Allen, 1993; Steel *et al*., 2011; Thomas *et al*., 1988; Valdés-Gómez *et al*., 2008).

Since the early work of Nelson (1951), several studies have been published regarding the relationship between GM infection and ambient parameters. Currently, it is accepted that conidia germinate between 0 and 30 °C with optimum temperatures between 20 and 25 °C. At 30 °C, conidia are not produced, and conidial germination is arrested. At optimal temperatures, germination occurs within 3 h. Numerous studies have demonstrated that free moisture is essential for conidial germination and berry infection (Broome *et al*., 1995; Ciliberti *et al*., 2015b; Coertze and Holz, 2002; Latorre and Rioja, 2002; Latorre *et al*., 2002; Nair and Allen, 1993; Steel *et al*., 2011; Thomas *et al*., 1988).

At optimum temperature, the estimated incubation period for flower infection on Cabernet Sauvignon grapes was considerably shorter (< 1.3 h) than the incubation period estimated for berry infection (13.9 h) (Nair and Allen, 1993). Latorre *et al.* (2002) worked with Thompson Seedless table grapes and determined an incubation period of less than 24 h at 20 ºC for flower infection and 48 h at 20 ºC for berry infection. The incubation period was shorter for berry infection of Thompson Seedless than Flame Seedless table grapes (Latorre *et al*., 2002). Therefore, berry infection appears to be dependent on the grape cultivar.

In addition to temperature and relative humidity, wind speed affects aerial mycelia and conidia production. These three factors determine the evaporative potential within the grape canopy, which reflects the capacity of the air to evaporate water (English *et al*., 1990; Thomas *et al*., 1988). Therefore, maximum production of conidia oc-

curs when the evaporative potential fluctuates between 0.05 and 0.15 $g \cdot h^{-1}$ of water, and aerial mycelia are produced below 0.25 $g \cdot h^{-1}$ of water. At 21 °C, abundant conidia were produced at a 94% relative humidity and 0.6 $m \cdot sec^{-1}$ wind speed (Thomas *et al.*, 1988). Aerial mycelia were produced at 21 °C, 94% relative humidity and 0 $m \cdot sec^{-1}$ wind speed, but no aerial mycelia were developed at a 69% relative humidity and wind speed > 0 $m \cdot sec^{-1}$ (Thomas *et al.*, 1988).

Berries that are exposed to sunlight, particularly UV-B light, produce thicker wax layers than berries under shaded conditions (Keller *et al*., 2003a). Hence, berries exposed to sunlight usually have a thicker cuticle, which reduces GM incidence and severity (Percival *et al*., 1993). Furthermore, UV light has a strong germicidal effect against the conidia of *B. cinerea* (Latorre *et al.,* 2012).

Agronomic factors

Agronomic factors predispose grapes to GM infection because they essentially alter the microclimate conditions within the grape canopy. These factors include canopy density and leaf removal after flowering (English *et al*., 1989; Gubler *et al*., 1987; Molitor *et al*., 2011; Valdés-Gómez *et al*., 2008; Zoecklein *et al*., 1992), cluster compactness and thinning (Barbetti, 1980; Ferree *et al*., 2003; Hed *et al*., 2009; Marois *et al*., 1986; Molitor *et al*., 2011; Percival *et al*., 1994; Smithyman *et al*., 1998; Vail and Marois, 1991), nutrition and fertilization (Mundy, 2008), grape training systems (de Bem *et al*., 2015) and winter pruning (Savage and Sall, 1984).

Forecasting system

The use of forecasting systems to predict GM infection conditions can be a useful decision support tool to complement integrated disease management strategies. Forecasting systems allow the reduction of unnecessary fungicide applications, restricting them to periods that are conducive to disease development. Models to predict conditions for *B. cinerea* infection in grapes, which are based on environmental conditions (Broome *et al*., 1995; González-Domínguez *et. al*., 2015; Kim *et al*., 2007; Latorre *et al*., 2002; Nair and Allen, 1993; Shtienberg, 2004; Shtienberg and Elad, 1997) and inoculum concentration and their dynamics (Carisse *et al*., 2014; Fernández-González *et al*., 2012), have been developed and proposed to predict the risk of GM infection. For example, Broome *et al.* (1995) developed a predictive model to determine the risk of *B. cinerea* infection on grapes. This model estimates the infection risk as a function of the temperature and duration of wetness. Thus, farmers have a useful tool to establish the need for a fungicide treatment. This model was successfully validated on a commercial basis in Chile, and it has been used by farmers as a guide in control decisions.

Pathogen detection

The detection and quantification of *B. cinerea* is highly desirable for research and disease management purposes. Based on this information, it would be possible to estimate the potential risk of GM in lots of table grapes, saving only lots with a low infection risk for long storage periods. Similarly, it would aid in the discrimination of wine grape lots by price penalizing or rejecting lots above a threshold value of GM prevalence and severity. Several detection methods have been tested (Dewey and Yohalem, 2004), including the following: (i) Direct plating on selective or semi-selective agar media (Kerssies, 1990) or agar media amended with 1% (v/v) Igepal (Alltech Associates, Inc. Illinois) used as a colony growth restrictor (Latorre *et al*., 2001, 2011); (ii) induction of tissue senescence using paraquat or freezing as a means of promoting the development of *B. cinerea* on the culture medium (Holz *et al*., 2003); (iii) enzyme-linked immunosorbent assays (ELISAs) employing specific monoclonal antibodies (Fernández-Baldo *et al*., 2011; Obanor *et al*., 2004;

Ricker *et al*., 1991; Ruiz and Ruffner, 2002); (iv) methods based on nucleic acid detection, such as polymerase chain reaction (PCR) (Gindro *et al*., 2014), reverse transcription PCR (RT-PCR) (Choquer *et al*., 2003) and quantitative real-time PCR (qPCR) (Diguta *et al*., 2010; Sanzani *et al*., 2012); and (v) autofluorescence response (Belanger *et al*., 2011). Other detection techniques, such as the incubation of berry samples under conditions conducive for GM development, adapted from Harvey (1955), have been used by Chilean table grape export companies to estimate the potential risk of GM during storage (Zoffoli *et al*., 2009).

A disease assessment key and assessment training software (Bunch Rot Assessment Trainer, BRAT) were developed based on disease incidence and severity relationships to improve the accuracy, repeatability and speed of visual GM assessment, which is commonly conducted when wine grape lots are received by the wineries (Hill *et al*., 2010).

Integrated disease management

The considerable increase in the understanding of *B. cinerea* biology and GM epidemiology has resulted in important improvements in grape GM control. It is generally accepted that integrated disease management is the only possible strategy to achieve the GM threshold tolerance at harvest, which is below 0.5% and 2.0% for table and wine grapes, respectively. At present, integrated disease management mainly includes cultural and chemical control, but the use of biological control (Montealegre and Pérez, 2014) and the search for resistant cultivars (Rubio *et al*., 2015) is gaining importance in conventional and organic grape production.

The progression of the grape GM epidemic over time follows an exponential relationship $y = x_0 e^{rt}$, where y = final disease level; x_0 = initial disease level (which is associated with the inoculum load); t = time and r = disease infection rate (Madden, 1980).

The difficulties associated with the control of GM can be explained by the exponential nature of GM progression during the beginning of the epidemic (Beresford *et al*., 2006; Madden, 1980) and by the relatively high infection rates that usually characterize this disease under optimal environmental conditions. For example, in New Zealand vineyards, it has been estimated that GM severity can increase at a rate of 1-2% per day near harvest time (Evans, 2010).

Integrated disease management against grape GM includes the use of several control measures (Evans, 2010), some of which reduce the initial inoculum (x_0), whereas others are oriented toward lowering the disease infection rate (r). Regarding the pathogen, the epidemiological factors and the exponential nature of the disease progression curve that characterizes grape GM, the major contribution is usually achieved when control measures are oriented to reduce r rather than x_0. Therefore, all control measures contribute to final GM control, but in different proportions. Consequently, the selection of a control measure can vary among different geographical areas according to the purpose of the grape production and the cost benefit relationship (Evans, 2010).

Cultural control

Canopy management. Canopy management practices are viticultural techniques that modify the canopy characteristics with the aim of improving yield, quality and vigor by facilitating mechanization, increasing the efficiency of pesticides and other agricultural chemicals, or preventing grape diseases (Smart *et al*., 1990). Among the cultural practices, the removal of senescent debris and infected pruned wood reduces the initial inoculum (x_0). In addition, avoiding berry wounds, cluster elongation, cluster thinning, cluster removal, heading, leaf removal and shoot thinning can affect the canopy microclimate and limit the conditions that are conducive to GM in the cluster zone, significantly

reducing the infection rate (*r*), often as much as fungicide treatments.

Leaf removal. Numerous studies have demonstrated the effect of leaf removal on GM control. Typically, one to three basal leaves per shoot in the fruit zone are manually or mechanically removed (Gubler *et al*., 1991). At present, leaf removal is used worldwide to manage high-density canopies by enhancing light exposure and airflow through and around the cluster zones and by increasing wind speed, evaporative potential and UV exposure (Bettiga *et al*., 1986; English *et al*., 1989,1990,1993; Ferree *et al*., 2003; Gubler *et al*., 1987,1991; Hed *et al*., 2015; Latorre, 1986; Molitor *et al*., 2011; Poni *et al*. 2006; Thomas *et al*., 1988; Zoecklein *et al*., 1992). The efficiency of leaf removal on GM control depends, among other factors, on the seasonal weather conditions. Leaf removal is highly efficient in relatively dry seasons but is insufficient to obtain a good degree of GM control in very wet seasons. However, in very wet seasons, leaf removal enhances the efficacy of fungicide treatment (English *et al*., 1993).

Leaf removal decreases *B. cinerea* and other epiphytic fungi commonly found on grape berries (Duncan *et al*., 1995). Additionally, it can stimulate phytoalexin, as well as epicuticular wax and cuticle production, in exposed berries and consequently prevent GM infections (Percival *et al*., 1993).

It has been suggested that the evaporative potential within the vine canopy can provide a simple means of determining the canopy opening and the drying conditions resulting from leaf removal (English *et al*., 1993). The evaporative potential within a grape canopy is inversely related to canopy density and 1 $mL \cdot h^{-1}$ has been suggested to be the minimum evaporative potential to diminish GM (English *et al.,* 1993). However, further research is needed to develop this technique and validate it under different grape training systems and different agro-ecological conditions.

Heading and shoot removal. Heading entails pruning off the over-hanging current season growth (Bettiga *et al*., 1986; Savage and Sall, 1982), and shoot removal entails the removal of an excessive number of shoots per vine to modify microclimate conditions within the grape canopy, which reduces the conditions conducive to GM. These viticultural practices increase airflow and sunlight penetration within the grape canopy and are especially important for table grapes trained as Pergola, which often have a very dense foliage canopy (Figure 3) (Zoecklein *et al*., 1992).

Cluster removal and cluster thinning. In most table grape cultivars, cluster removal and cluster thinning are essential for obtaining quality fruit to satisfy market demand. These viticultural practices are also of paramount importance to

Figure 3. Thompson Seedless table grapes trained as Pergola. A. A general view at harvest time. B. A dense canopy interfering light penetration and air flow around clusters.

prevent severe GM. Cluster removal prevents over cropping and bunch crowding at harvest, preventing a delay in cluster maturation and assuring high quality berries. Cluster thinning prevents compactness, improves airflow within clusters, and limits berry to berry contact, which restricts cuticle development at the contact points and prevents berry split in the interior of the clusters. Furthermore, fungicide spray cover becomes imperfect as cluster compactness increases (Hed *et al*., 2009; Marois *et al*., 1986; Tardaguila *et al*., 2008; Vail and Marois, 1991; Zoecklein *et al*., 1992). Additionally, it has been demonstrated that cluster thinning increases the total resveratrol level (Prajitna *et al*., 2007).

Cluster elongation. In some wine grapes, cluster elongation, which can be achieved with the use of growth regulators (*e.g*., GA3 gibberellin, prohexadione-Ca), can reduce cluster compactness, improve airflow and considerably reduce microclimate conditions within the grape canopy that favor GM development (Ferree *et al*., 2003; Hed *et al*., 2011, 2015; Molitor *et al*., 2011; Pearson and Riegel, 1983). However, this technique has not been used commercially in Chile, and its use appears to be quite limited in other countries.

Removal of senescent debris. It has been demonstrated that the physical removal of senescent tissues (*e.g*., floral debris, aborted berries, leaves) retained on the clusters can partially reduce GM incidence and severity by approximately 30%. However, the GM reduction can vary with other epidemiological factors. Physical removal of senescent debris can be achieved with high air speeds using air blast sprayers or backpack blowers (Jermini *et al*., 1986; Wolf *et al*., 1997).

Nutrition and fertilization

Plant nutrition and fertilization are important predisposing factors that affect the susceptibility of grapes to GM infection and consequently affect the disease infection rate (*r*) rather than the initial inoculum (x_0). Nitrogen and calcium are the best documented nutrients in connection with their effects on grape GM.

Several studies have reported that high nitrogen nutrition predisposes berries to GM infections (Keller *et al*., 2001; Mundy, 2008; Valdés-Gómez *et al*., 2008). High nitrogen promotes excessive vegetative growth and vigor, which enhance canopy density, thus generating a microclimate within the grape canopy that favors GM. In addition, high nitrogen levels delay berry ripening, increase cluster compactness and reduce the thickness of the berry cuticle (Valdés-Gómez *et al*., 2008; Keller *et al*., 2001).

Calcium is an essential mineral element that plays an important role in enzymatic and hormonal processes, preserving the integrity of membranes and cell walls, improving the storage quality of table grapes and other fruits and acting in plant cell signaling (Sanders *et al*., 1999). In general, increasing the calcium content of grape berries, particularly the skin, lowers berry susceptibility to *B. cinerea* infection (Chardonnet and Donèche, 1995; Miceli *et al*., 1999). Furthermore, it has been reported that some calcium salts exhibit inhibitory activity against *B. cinerea in vitro* and/or *in vivo* by inhibiting the polygalacturonase enzyme, spore germination and germ tube elongation (Al-Qurashi and Awad, 2013; Chervin *et al*., 2009; Nigro *et al*., 2006).

Grapevine training system

Training systems that promote a high canopy density tend to create favorable microclimate conditions around clusters, exposing grapes to longer periods of wetness, and thus fostering GM (Elmer and Michailides, 2004; Phillips *et al*., 1990). Therefore, to reduce GM severity, training systems that favor airflow around clusters should be preferred. However, the Pergola system (Figure 3), which is a high trellis system with a 2-m high horizontal plane of vegetation

that favors GM severity, is widely used for table grape production in Chile because it promotes high yield and quality.

Irrigation

Most grape production in Chile is under irrigation, which is essential to fulfill the water requirements of grapes during the spring and summer months. The oldest vineyards are surface irrigated (flood and furrow irrigation systems), whereas most of the new vineyards use drip irrigation systems. Regardless of the system used, irrigation has been considered to be predisposing factor for GM development because it increases humidity around the clusters and, in combination with nitrogen fertilization, promotes shoot vigor, foliage growth, cluster compactness, and delays fruit maturity (Valdés-Gómez *et al*., 2008).

Biological control

Biological control (biocontrol) has been defined as the use of an antagonistic microorganism to control a plant disease. Following this approach, a number of microorganisms have been reported as antagonists of *B. cinerea in vitro*; they have been suggested as potential agents to control GM on grapes and other crops (Elad and Stewart, 2004; Elmer and Reglinski, 2006; Jacometti *et al*., 2010).

For example, there are a large number of filamentous fungi and Oomycetes, such as species of *Epicoccum*, *Gliocladium (Clonostachy) Trichoderma*, *Pythium* and *Ulocladium*; yeasts, such as species of the genera *Acremonium*, *Aerobasidium*, *Candida, Cryptococcus, Debaryomyces, Hanseniaspora, Issatchenkia, Pichia, Saccharomyces, Schizosaccharomyces,* and *Torulaspora*; and some bacterial species belonging to *Bacillus, Brevibacillus, Pseudomonas* and *Serratia* (Elmer and Reglinski, 2006; Vargas *et al*., 2012) that have been identified as potential control agents. However, very few antagonists have demonstrated activity under field conditions and even fewer have been formulated and used commercially against grape GM (Elmer and Reglinski, 2006; Nally *et al*., 2012).

There is a long history of studies on *Trichoderma* spp. against *B. cinerea*, both in the laboratory and in the vineyard. Isolate T39 of *T. harzianum* was the first to be formulated as Trichodex (Makhteshim-Agan, Israel) (Elmer and Reglinski, 2006). The use of isolate T39 resulted in partial control, which was significantly different ($p < 0.05$) from untreated controls and equal to or less than the control achieved using vinclozolin (Ronilan 50 WP, 1.5 kg ha^{-1}) but similar to the control achieved using captan (Captan 80 WP, 4 kg ha^{-1}) on table grapes infected with *B. cinerea* (*Harman et al., 1996;* Latorre *et al*., 1997). The *Trichoderma* population on table grape flowers and clusters decreased relatively rapidly, suggesting a short survival in the grape canopy (Latorre *et al*., 1997). Based on these and several other reports, the degree of control provided under field conditions seems to be adequate only under low to moderate disease pressure (Latorre, 2013; Montealegre and Perez, 2014).

The use of species of *Bacillus*, including *B. circulans*, *B. brevis* and *B. subtilis* as antagonists against *B. cinerea* has been documented under laboratory and field conditions (Ben Maachia *et al*., 2015; Elmer and Reglinski, 2006). *B. subtilis* strain QST-713, which is formulated as Serenade (Agra Quest, USA), provided good GM control under field conditions in Chile (Esterio *et al*., 2000). In *B. subtilis*, cyclic lipopeptides (surfactins, iturins and fengycins) have been involved in activating plant defenses; some of these cyclic lipopeptides can act directly against *B. cinerea* (Farace *et al*., 2015).

Biocontrol agents exert their antagonistic action as a result of several biological mechanisms acting alone or combined, which may include competition for nutrients and space, the production of inhibitory metabolites, the induction of biological processes and parasitism (El Ghaouth *et al*., 2003; Elmer and Reglinski, 2006). Biocontrol agents

have been used to protect grape berries, *i.e.*, as a replacement for fungicide treatments; therefore, they are used to reduce the infection rate (*r*). However, there is no consensus on whether this is the best approach.

At present, biological control is recognized as a complex process that should take into consideration the pathogen, the host and the environment, as well as their interactions (Droby *et al.,* 2009). Following this concept, several new approaches to strengthen biological control are being studied. These approaches include the combination of two or more antagonistic strains, the induction of natural biological processes (*e.g.*, inducing systemic resistance) (El Ghaouth *et al*., 2003; Reglinski *et al*., 2005), the use of natural antimicrobial products (*e.g.*, chitosan, lysozyme) and plant resistance (Calvo-Garrido *et al*., 2013, 2014b; Droby *et al*., 2009). In addition, the identification and use of yeasts, which are normal components on the surface of grape berries, have been studied for their capacity as biocontrol agents (Nally *et al*., 2012; Parafati *et al*., 2015; Vargas *et al*., 2012). A better understanding of the microbial ecology of grape berries will help to improve studies to determine the best biocontrol alternative (Barata *et al.,* 2012). Overall, it is expected that these new approaches in the study of biological control can improve the efficacy and consistency of new products, making biocontrol a more reliable strategy to prevent GM in grapes and other crops.

Finally, the presence of dsRNA mycoviruses has been described in *B. cinerea* in Chile and other countries. Considering that some of these mycoviruses are associated with hypovirulence, there is considerable interest in their study and eventual use as biocontrol agents (Castro *et al*., 2003; Howitt *et al*., 1995; Vilches and Castillo, 1997).

Chemical control

Chemical treatment is still a very important control strategy against grape GM; its use is often required for a high degree of grape GM control. The foliar application of fungicides is used to protect the grape cluster and reduce the infection rate (*r*) as much as possible during the growing season. Nevertheless, the use of fungicides is increasingly restricted because of major environmental and human health concerns (Komárek *et al*., 2010) and because of the frequent development of resistant strains of *B. cinerea* (De Miccolis Angelini *et al*., 2014; Latorre and Torres, 2012; Leroux, 2004; Leroux *et al.,* 2002).

Fungicide groups

At present, highly effective synthetic fungicides with different biochemical modes of action are available against grape GM (FRAC, 2015). Most of the recently developed fungicides are site-specific compounds with a single-site mode of action while older fungicides are compounds with multi-site modes of action.

Currently, chemical control is mainly based on the use of fungicides with a single-site mode of action. These fungicides belong to the following groups (FRAC, 2015; Leroux, 2004): i. Anilinopyrimidines (*e.g.*, cyprodinil, mepanipyrim, pyrimethanil); ii. Succinate dehydrogenase inhibitors (SDHI) (*e.g.*, boscalid, penthiopyrad); iii. Demethylation inhibitors (DMI), Class I (*e.g.*, tebuconazole); iv. DMI, Class III, hydroxyanilides (*e.g.*, fenhexamid); v. Dicarboximides (*e.g.*, iprodione); vi. 2,6-Dinitroanilines (*e.g.*, fluazinam); vii. Phenylpyrroles (*e.g.*, fludioxonil); and viii. Quinone outside inhibitors (QoI) (strobilurines) (*e.g.*, azoxystrobin). Benzimidazoles are still available, but because of resistance problems, they are no longer used against grape GM.

Fungicides with multi-site modes of action, such as phthalimide derivatives (captan and folpet), sulfamide derivatives (dichlofluanid and tolyfluanid) and chloronitrile derivatives (chlorothalonil), are still used against *B. cinerea* (Leroux, 2004). These fungicides have protective action, and with few

exceptions, resistance rarely occurs (Pollastro *et al*., 1996).

Fungicide timing

Fungicides against *B. cinerea* applied as pre-infection (protective) treatments allow better control efficiency than post-infection (curative) treatments. The post-infection activity of most fungicides is short, usually less than 24 to 48 h (Serey *et al*., 2007; Smilanick *et al*., 2010).

Field studies have delimited the grape growth stages at which fungicides should be applied to obtain a high degree of GM control. Farmers commonly use specific and very effective fungicides only at the most critical grape growth stages. In Chile and other countries, the fungicide application programs most commonly consist of three to four preventive fungicide applications, which include one fungicide spray at flowering, bunch closure, veraison and pre-harvest (Calvo-Garrido *et al.,* 2014a; Edder *et al*., 2009; Latorre *et al*., 2001; Petit *et al*., 2010). However, the relative importance of these stages can vary under different agro-ecological conditions and with grape management and cultivars.

In contrast to reports that question the value of flowering applications (De Kock and Holz, 1994), fungicides sprayed at flowering significantly reduced GM incidence and severity on Thompson Seedless and Red Globe table grapes at harvest in Chile, but fungicide efficacy was lower at flowering than between veraison and harvest (Latorre *et al.,* 2001). Recently, Calvo-Garrido *et al.* (2014a) concluded that the most effective fungicide treatment consisted of applications at flowering, with and without pre-bunch closure, or after veraison applications. Nevertheless, additional sprays at pre-bunch closure or during the late season were needed when conditions were highly conducive to GM infection.

During flowering, fungicide treatments are aimed at reducing the latent infection and colonization of senescent tissues; however, fungicide treatments applied after veraison protect berries against late GM infections and often provide post-harvest protection for table grapes (Franck *et al*., 2005; Smilanick *et al*., 2010).

Fungicide resistance

B. cinerea is considered to be a high resistance risk pathogen because it produces abundant conidia as a primary inoculum, which is then efficiently disseminated. In addition, this fungus has a high genetic variability and wide host range; thus, a high number of fungicide applications are commonly required for control because of the polycyclic nature of GM (Brent and Hollomon, 2007a, b; Latorre and Torres, 2012; Myresiotis *et al*., 2007).

To control GM, fungicides with single- and multi-site modes of action are available; these correlate with a moderate to high and a low risk of the development of resistant strains of *B. cinerea*, respectively. Cross-resistance to fungicides with the same mode of biochemical action has been extensively described (Leroux, 2004). Therefore, to avoid resistance, fungicides with different biochemical modes of action should be alternated or combined each growing season. Furthermore, the use of fungicides with a single-site mode of action is frequently limited to one or two applications per season (Brent and Hollomon, 2007a).

Resistance to fungicides with a single-site mode of action has been found in Chilean vineyards (Latorre *et al*., 1994, 2002; Carreño and Alvarez, 1990; Esterio *et al*., 2007, 2015; Latorre *et al*., 2002; Piqueras *et al*., 2014; Thompson and Latorre, 1999). Recently, multiple resistance has been reported in *B. cinerea* in grapes in Chile (Latorre and Torres, 2012) and other countries (De Miccolis Angelini *et al*., 2014; Leroch *et al*., 2011). This reflects the danger of the intensive use of fungicides against GM and reinforces the fact that fungicide resistance is a serious threat to the grape industry.

Postharvest gray mold control on table grapes

B. cinerea causes substantial postharvest decay on table grapes. Even a single infected berry within a table grape package can cause severe losses if control strategies are not taken. Considering that the primary inoculum always comes from the vineyard, a thorough control strategy should always start in the field. Furthermore, fungicide residues of certain fungicides applied just before harvest can also protect grapes during storage and transportation (Smilanick *et al*., 2010). In stored grapes, refrigeration (-0.5 to 0.5 °C) and the use of sulfur dioxide are the strategies currently used to prevent GM in stored table grapes (Franck *et al*., 2005).

Conclusions

Grape GM caused by *B. cinerea* is a major fungal disease affecting grapes and other crops worldwide. The polycyclic nature of the GM epidemic, the abundant production of *B. cinerea* inoculum and the efficient dissemination mechanisms, as well as the wide host range and high genetic variability of *B. cinerea*, explain the difficulties encountered in attempting to control GM. In Chile, GM has limited table grape production to geographical zones that are less prone to infection. It is frequently the cause of important losses at destination markets in the United States, Europe or Asia. Similarly, GM has caused considerable yield losses and has reduced the quality of wine grapes worldwide. The level of GM infection in vineyards results from the interaction of various factors, such as the host, pathogen and environmental conditions. In the last two decades, tremendous progress has been made toward understanding pathogen biology and epidemiology, as well as toward improving control strategies. At the same time, the incorporation of molecular tools to study the pathogen has resulted in important genetic contributions and the development of new control opportunities. Today, there is considerable concern over the rapid development of *B. cinerea* strains that are resistant to fungicides, as well as the use of fungicides in general, because of environmental and toxicological considerations. Therefore, new knowledge is essential to establish novel sustainable control strategies that allow more effective control and reduced use of fungicides.

Resumen

B.A. Latorre, K. Elfar y E.E. Ferrada. 2015. Pudrición gris, causada por *Botrytis cinerea*, limita la producción de vid en Chile. Cien. Inv. Agr. 42(3): 305-330. La pudrición gris (PG) causada por *Botrytis cinerea,* es una de las principales enfermedades de la vid (*Vitis vinifera*) que limita la producción y reduce los rendimientos y la calidad de la fruta en zonas templadas y húmedas a nivel mundial. *B. cinerea* es un hongo necrótrofo que ataca órganos aéreos no lignificados de la vid, siendo las bayas altamente susceptibles durante la maduración. La naturaleza policíclica y el desarrollo exponencial de las epidemias de PG, junto con la abundancia de inóculo, la eficiente dispersión más el amplio rango de hospederos y gran variabilidad genética que presenta *B. cinerea,* explican las dificultades para lograr un control satisfactorio. Ante lo cual se hace necesario realizar una estrategia de control integrado que combine medias de control cultural y químico. Estas medidas pueden estar orientadas a reducir el inoculo inicial o la tasa de progreso de la enfermedad, siendo las medidas de control destinadas a reducir la tasa de progreso las que más aporta al control de PG. En las últimas décadas se han producido importantes progresos en el conocimiento de la compleja biología de este patógeno y de los aspectos epidemiológicos de la PG. Esto ha permitido mejorar las estrategias de control logrando alternativas más efectivas y sustentables. En este artículo se revisan los aportes científicos recientes realizados en relación con la PG de la vid, teniendo especial énfasis en la situación del viñedo chileno.

Palabras clave: *Botryotinia fuckeliana*, pudrición gris, epidemiología, fungicidas, necrótrofo, *Vitis vinifera*.

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