CHAPTER 14

EPIDEMIOLOGY OF *BOTRYTIS CINEREA* IN ORCHARD AND VINE CROPS

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Abstract. Substantial economic crop losses occur worldwide in tree fruits, nut crops, vines and small fruits as a result of infection by *Botrytis cinerea*. Fungicide-based management, once an accepted practice, is becoming increasingly restricted, a trend likely to continue in the future. Greater emphasis on alternative, non-chemical control will require improved knowledge of *B. cinerea* ecology and epidemiology in affected crops. Epidemics are often initiated in the spring from conidial inoculum produced on over-wintering structures on a very wide range of plant species. From floral infection in the spring, several infection pathways to fruit infection and crop loss at harvest are described. The majority of these pathways include a degree of symptomless latency, or quiescence, in the host tissue. In some crops (e.g. grapes) multiple pathways are described, each one dependant upon many complex host, pathogen and environmental factors. In other crops (e.g. berry fruits), a single dominant pathway is described. Latency, once poorly understood, has become the focus of research in the last decade. Several host defence mechanisms are described which may account for this period of enforced dormancy. Once pathogenic growth resumes and typical *B. cinerea* symptoms appear, many factors affect the subsequent rate of *B. cinerea* epidemics and we describe some of these in detail. The growth of organic production in the last decade has high-lighted the need for a greater understanding of the complexities of epidemic development in order to develop durable and sustainable disease control strategies.

1. Introduction

Epidemics caused by *Botrytis cinerea* can be severe and economically damaging to many agricultural and horticultural crops in conditions conducive to infection (Chapter 1). These losses continue to occur despite the availability of new botryticides (Chapter 12). However, the freedom to apply fungicides is becoming increasingly restricted and in some cases, forbidden. These restrictions are based upon public concerns about the adverse effects of synthetic pesticides on human health and the environment and zero chemical residue tolerance in many export markets. The growth of organic farming in the last decade has encouraged a re-

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orientation of thinking for plant pathologists as nil and significantly reduced fungicide programmes become common. To maintain yields in these systems, disease suppression strategies based on robust epidemiological knowledge will need to be devised. In some fruit crops (e.g. raspberries), many years of research has led to a greater understanding of the epidemiological processes which contribute to *Botrytis* outbreaks, while in other crops (e.g. grapes) our understanding of epidemiological processes is continually evolving. The objective of this chapter is to review our understanding of *B. cinerea* epidemiology in selected fruit crops. We focus on the grape system since this crop is one of the most economically important fruit crops with c. 8 million ha (Vivier and Pretorius, 2002) and estimated crop losses due to *B. cinerea* of 2 billion $US per annum.

2. Sources of primary inoculum for host infections

Conidia produced in late winter and early spring on over-wintering mycelium and/or sclerotia on host tissues and sclerotia on the surface of soil are considered the most important infective unit of *B. cinerea* for infection in the spring. Numerous alternative hosts exist in orchards and vineyards including herbicide-treated or senescing weeds and other infected crops in the vicinity (Figure 1). Over 200 host species were identified as sources of *B. cinerea* inoculum in one study (Sutton et al., 1991). Surveys from a wide range of fruit growing regions consistently rank conidia of *B. cinerea* as an important part of the aerial microflora (Bisiach et al., 1984). Insects in the spring and invertebrates may also act as casual vectors of *B. cinerea* inoculum (Sect. 7.2). Reports of apothecia in orchards and vineyards exist, but they are sporadic (Chapter 3). Although *B. cinerea* is readily detectable in the soil, it represents a small proportion of the soil fungal biomass (Dorado et al., 2001). In grapes, the most important primary inoculum source in the Hunter Valley region of New South Wales, Australia was sclerotia that initiated the disease cycle in the spring (Nair and Nadtotchei, 1987; Nair and Martin, 1987; Nair, 1990). In contrast, sclerotia were not detected at budburst in the relatively dry Marlborough wine growing region of New Zealand (NZ) (R. Balasubramaniam, HortResearch, NZ, unpubl.). A subsequent study identified the dominant sources of inoculum at 80% capfall as; old rachides on the ground, tendrils, leaf petioles and cane debris (Seyb et al., 2000b). In contrast, leaf petioles under vines were identified as the dominant source of over-wintering inoculum in Hawkes Bay, NZ (P. Wood, HortResearch, NZ, unpubl.). *B. cinerea* mycelium survived for up to 30 weeks in grape vine prunings, with weed control practices and temperature having a significant impact on mycelial longevity (Thomas et al., 1983). In California, conidial production on sclerotia and from over-wintering mycelium can be easily found on grape bunch clusters that remain on the vine and on canes, leaf petioles and laminae on the orchard floor (Figure 1). Significant positive correlations were found between over-wintered inoculum sources and flower infection in the Hunter Valley, New South Wales with carry-over inoculum accounting for 70% of the variation in flower infection in the canopy (Nair et al., 1995). In kiwifruit, *B. cinerea* sporulated profusely on infected fruit on the orchard floor, senescing leaves of weeds, necrotic

kiwifruit leaf and fruit remnants or senescent leaves from nearby crops at the end of winter and occasionally on remnants of floral tissues of male vines in California (Michailides and Elmer, 2000). The relative contribution of several inoculum sources during the kiwifruit growing season were quantified in NZ (Elmer et al., 1995). Early in the growing season as female flowers open, most of the inoculum was produced on over-wintering mycelium on prunings on the ground. Occasionally, sclerotia on prunings produced conidia. *B. cinerea* was rarely detected in leaf litter from beneath vine canopies from dormancy to harvest. As in California, profuse conidia production occurred on paraquat-treated perennial weeds (*Rumex* spp.) and senescent flowers of male vines.

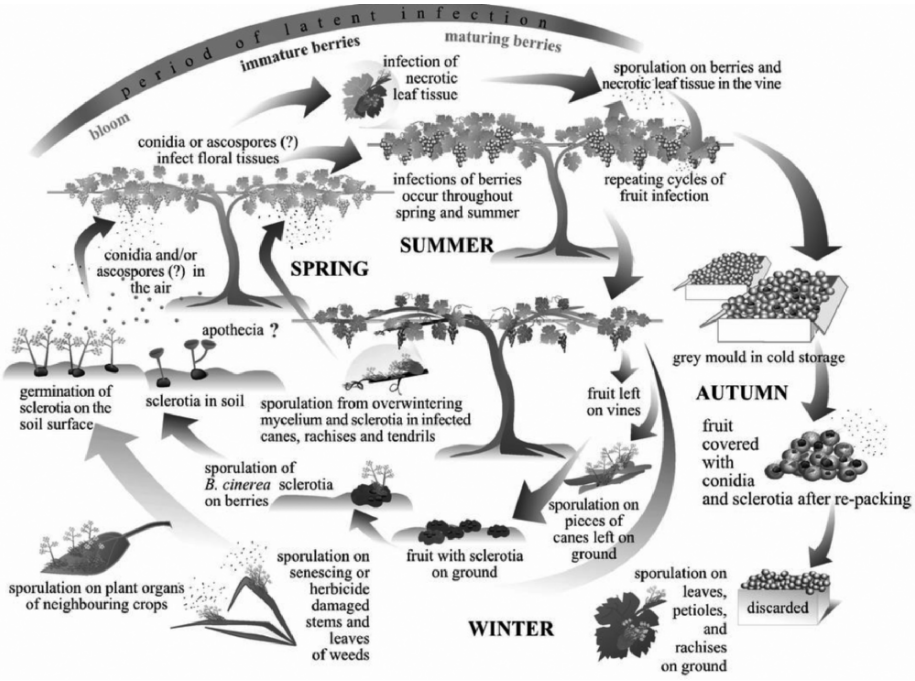

Figure 1. Proposed life cycle of *Botrytis cinerea* and disease cycle of grey mould in wine and table grape vineyards

In most temperate regions, by early spring there is ample inoculum in the environment for infection. Even newly emerged and partially expanded young green strawberry leaves were susceptible, the infection remaining in a latent stage until onset of leaf necrosis (Sutton et al., 1991). Latent infections of young green grape leaves have also been reported (Holz et al., 2003), but these early latent infections did not appear to play an important role as inoculum sources for flower infection, unless the host tissues senesced prematurely. To our knowledge latent infections on immature leaves of kiwifruit and berry fruits other than strawberries have not been reported. Apparently healthy kiwifruit sepals in California had latent infections early in the season, but did not sporulate and contribute to flower infection.

In blueberries, *B. cinerea* over-winters as dormant mycelium or sclerotia in or on plant debris. In raspberries it survives mainly as sclerotia and mycelium on over-wintering canes and on pruning debris (Williamson and McNicol, 1986). In the spring conidia produced on these tissues infect blossoms, twigs and fruit. After flower blight the fungus spreads down the peduncle to girdle the stem, killing all flowers and young green berries above the point of girdling (Bristow and Milholland, 1995). In NZ, leaf litter on the ground, herbicide desiccated primo-canes and receptacles left on the vines were sources of inoculum in boysenberry (Walter et al., 1997). The dominant source of inoculum in strawberries was over-wintering dead leaves (Braun and Sutton, 1988; Sutton et al., 1991).

Epidemiological studies show significant correlations between *B. cinerea* colonisation of senescent floral tissues in the spring with incidence and severity of grey mould at harvest and during cool storage. Flowering is therefore fundamentally important in studies of *B. cinerea* ecology and epidemiology and the current state of knowledge of infection pathways from flowering to fruit infection and crop loss are described (See also Chapter 2).

3. Flower to fruit infection pathways

Flowers of grape and many other fruit crops are highly susceptible to *B. cinerea* infection when they senesce (Jersch et al., 1989) and low resveratrol synthesis may contribute to the high susceptibility of grape flowers (Keller et al., 2003; Chapter 9). The abundance in pollen over the flowering period of most hosts increases severity of infection (Ogawa and English, 1960; Chou and Preece, 1968; Lehoczky, 1972; Michailides, 1991; Rose, 1996). In addition to stimulating conidia and pathogenicity, pollen favours many natural *B. cinerea* antagonists (e.g. *Sporobolomyces* spp.), thereby moderating *B. cinerea* epidemics (Lehoczky, 1972). Flower infections generally result in latent infections in immature fruit (Williamson, 1994), but not all fruit infections at harvest are the result of floral and latent infections at that time (e.g. grapes). Here we review current knowledge of infection pathways, with a special focus on grapes.

Pathway I - Conidial infection of the style and ovules - commences with conidia infecting stylar tissues, followed by slow systemic hyphal growth into the ovule where *B. cinerea* enters a latent phase. As host defences weaken, often coinciding with berry ripening, *B. cinerea* resumes growth and rots the fruit. This sequence of events has been postulated for grapes, kiwifruit, raspberry and blackcurrants. Infection of grape stigmas early in the season was proposed as the most likely pathway to fruit infection (McClellan and Hewitt, 1973; Nair and Parker, 1985; Nair and Hill, 1992). Berry rot resulted from latent infections at the 'stylar abscission' zone but in some wine growing regions, stylar infections were considered less important (Pezet and Pont, 1986; Holz et al., 2003), while in other areas they played a significant role in the ecology and epidemiology of *B. cinerea* (Keller et al., 2003). In raspberries, conidia of *B. cinerea* germinate in the stigmatic fluid; hyphae grow in the transmitting tissues of the style and continue in the stylar vascular tissues into the fruit carpel (McNicol et al., 1985; Williamson et al., 1987). In favourable conditions, reactivation of *B. cinerea* in the stylar tissues produces conidia, thereby

providing an additional source of inoculum for infection of other senescent and necrotic styles. Saprophytic survival within aborted carpels amongst swollen drupelets can also provide an inoculum source (Williamson et al., 1987). The role of floral organs other than the styles was investigated further by Dashwood and Fox (1988). Direct infection of individual raspberry drupes by *B. cinerea* conidia contributed minimally to fruit rot (Jarvis, 1962; Williamson and McNicol, 1986). The styles are the main infection pathway to fruit rot at harvest in boysenberries (*Rubus* hybrid) and these remained susceptible to infection up to harvest (Boyd-Wilson et al., 1996; Walter et al., 1997; Walter et al., 1999b). In kiwifruit stylar infections were rare (Bisiach et al., 1984) and were not associated with post-harvest storage rot (Fermaud and Gaunt, 1995). In blackcurrants, conidia germinated in the stigmatic fluid then spread symptomlessly, via the style, into the pericarp and ovules. These latent infections were an important inoculum source and contributed to premature abscission of fruits (McNicol and Williamson, 1989). Symptomless fruit infection from inoculations at flowering were not detected in another study (Pappas and Jordan, 1997), but *Botrytis* infected blackcurrant flowers frequently aborted.

Pathway IIa - Conidial infections of the stamens and or petals - followed by systemic hyphal growth through floral tissues to the receptacle, results in latent infections which become aggressive infections as the fruit ripens (e.g. grapes, kiwifruit and strawberries). In grapes, histological studies have shown that *B. cinerea* colonises the stamens, grows basipetally to infect the receptacle and then grows systemically to the pedicel and vascular tissues in the berries (Pezet and Pont, 1986). Seyb et al. (2000a) found no evidence for stamen infections. Cultivar host defence mechanisms could account for an apparent lack of growth in the stamen tissue. Alternatively, the dry climate of this region desiccated the delicate stamen tissues before *B. cinerea* reached the pedicel/receptacle tissue. In some cultivars, stamens detach rapidly rendering this pathway redundant.

A link between floral infection of petals and anthers by *B. cinerea* and post-harvest rot was proposed in kiwifruit (Sommer et al., 1983, 1984) and confirmed as an important source of inoculum for sepal and receptacle infections (Duncan, 1991; Michailides and Morgan, 1996a). The sepals remained receptive to *B. cinerea* infection for the entire growing season. Symptomless infections of sepals and receptacles in the stem-end region were cumulative. Artificial inoculation with a conidial suspension at full bloom resulted in the highest incidence of grey mould in cool storage, suggesting that conditions for infection and establishment of latent infections were favourable over the flowering period (Michailides et al., 1999). In kiwifruit, inoculation studies demonstrated a link between colonisation of senescent carpels in the spring and stem-end rot after cool storage in NZ (Beever et al., 1984). Receptacle infections at harvest ranged from 5 to 55% (1984-1986) from flowers with conspicuous infections on their petals. In contrast, infection frequency in the receptacles ranged from 0 to 10%, when fruits were sampled randomly, indicating a link between petal infection and subsequent receptacle infection (S.R. Pennycook, DSIR, NZ, unpubl.). Despite the presence of *B. cinerea* in the receptacle at harvest, many studies found no correlation with stem-end rot in cool storage (M. Manning, HortResearch, NZ pers. comm.) and the majority of fruit infections occurred at

harvest from conidia redistributed from the hairy fruit surface to the picking wound (Pennycook and Manning, 1992; Manning and Pak, 1993; Pak and Manning, 1994). In contrast, this was not found important in Californian and Italian kiwifruit vineyards (Bisiach et al., 1984). In strawberry, early studies suggested that senescent flower parts were important sources of inoculum for subsequent spread into ripening fruits (Powelson, 1960). However, systemic infection and growth of *Botrytis* through the filaments of infected stamens into the receptacle to form a symptomless infection is considered the primary infection pathway for fruit rot (Bristow et al., 1986). Conidia were not regarded as the primary inoculum source for fruit infection (Jarvis, 1962).

Pathway IIb - Fruit infection via the fruit pedicel - A 6-year research programme in the wine growing region of the Western Cape province of South Africa (SA) has contributed significantly to our knowledge of the ecology of *B. cinerea* in grapes (Holz et al., 1997, 2000, 2003). Inoculation confirmed that the pedicel, lateral, and to a lesser extent the rachii, were susceptible to *B. cinerea* at the early stage of grape berry development, but with the exception of the pedicel, their resistance to infection increased as the season progressed (Holz, 2001). At the point where the berry is attached to the pedicel up to 30% of berries yielded *B. cinerea* following surface sterilisation and freezing. The rachises and laterals yielded c. 20% *B. cinerea* and berry cheek infections were infrequent at 5%. The stomata and lenticels were proposed as entry point for infection of the rachii and pedicel (Holz et al., 2003).

A high proportion (76 points to 90%) of fruit infections were initiated from the stem-end of table grape berries in California (Michailides et al., 2000b). In a separate study, table grape clusters of Red Globe were periodically inoculated from bloom through to harvest. Results indicated that the greatest incidence of infection post-harvest was derived from inoculations of bunches during early flowering and full bloom (Michailides et al., 2000b, c). Pedicel infections occur frequently in French vineyards (Dubos and Roudet, 2000), and fungicide timing studies in many vineyards world-wide have confirmed the importance of flowering infection. For example, a single botryticide application at the late flowering stage reduced bunch rot at harvest by 78% compared to the untreated controls (Viret and Keller, 2000). However, as described in Figure 1, multiple infection pathways occur, and conidial infections of ripening berries later in the growing season are as important as latent infections of green structural tissues earlier in the growing season (Wilcox, 2002).

Pathway III - Conidial infection and extensive colonisation of floral debris-in grapes (e.g. aborted fruitlets and calyptras) this is followed by a saprophytic over-summering phase in these tissues trapped in the developing bunch, but little or no fruit infection occurs due to preformed host barriers. At veraison, host defences weaken as the berries ripen; berry exudates reactivate saprophytic mycelium, provide sufficient inoculum potential to overcome remaining preformed barriers and direct fruit infection occurs. At bloom, the calyptra separates along a preformed abscission zone at the base of the ovule. This zone consists of a localised band of necrotic tissue ('cap scar') and is considered to be the initial site of *B. cinerea* colonisation leading to the establishment of latent infections (Keller et al., 2003). EM studies of the cap scar supported this hypothesis based upon *B. cinerea* germ tube growth and penetration into this tissue. The proportion of rapidly senescing and

necrotic tissues in grape bunches in Australian vineyards consisted of calyptras (67%) and stamens (22%), but at veraison, the calyptras were the more prevalent necrotic tissue type in the bunch (Nair and Hill, 1992). Up to 10% of the flowers did not set fruit ('aborted fruitlets') within an inflorescence and when infected were considered to be a major source of inoculum within developing bunches (Nair and Parker, 1985). Infected floral tissues potentially provide a large base of saprophytic mycelium, the extent of which varies significantly between cultivars. Collectively, the necrotic tissues within bunches in NZ are referred to as 'bunch trash' (Seyb et al., 2000a) and consist of stamens, aborted flowers, aborted berries, calyptras ('caps'), tendrils and leaf pieces. *B. cinerea* infection of bunch trash ranged from 13 to 72%, indicating that prior to bunch closure there was considerable *B. cinerea* inoculum potential within the bunch, immediately adjacent to berries. Aborted fruitlets, damaged berries and calyptras were considered the most important contributors to bunch rot at vintage, being the more numerous in terms of biomass and the most frequently infected. These components were also significantly positively correlated with the incidence of infected berries at harvest, but not in all seasons (Seyb et al., 2000a). Stamen infections were not detected for three consecutive years (1998-2000).

Pathway IV - Conidial accumulation within the developing bunch - Similar to Pathway III with extensive floral tissue colonisation *B. cinerea* enters a saprophytic over-summering phase in the bunch and during conducive conditions crops of conidia are produced resulting in contamination of the external surfaces of the ripening berry with conidia inside the bunch itself. Preformed host barriers prevent the majority of *B. cinerea* latent infections from becoming established until host defences weaken as berries ripen; leakage of exudates from the most mature berries in the bunch provide sufficient stimuli for conidial fruit infection and a rot focus commences. Inoculum is readily dispersed within bunches between flowering and veraison to pedicels, rachii and laterals (Holz et al., 2003), but also to immature fruit surfaces. Active sporulation on aborted fruitlets and calyptras in immature bunches is frequently observed (P.N. Wood and P.A.G. Elmer, unpubl.) resulting in conidial contamination of the berry surfaces within the bunch. Conidia of *B. cinerea* were detected in the receptacle/cap scar zone, and represented a resting state as opposed to a latent infection. These trapped conidia are in an ideal position at the berry base to infect the berry when host and environmental conditions favour infection (Sarig et al., 1998). Microscopic examination of the receptacle/berry attachment area and cap scar has confirmed that conidia are present (M. Cole, Monash University, Australia, pers. comm.),

Pathway V - Conidial infection of ripening fruit - As host defences weaken (e.g. in a small number of physiologically more mature berries), symptoms appear and a new crop of conidia are dispersed to new infection sites and the "classical" pre-harvest polycyclic epidemic commences. In vineyards world-wide, peaks of airborne conidia were consistently found at flowering, veraison and harvest (Vercesi and Bisiach, 1982). Therefore, when ripening commences there is ample inoculum for both canopy and fruit infections. The importance of fruit infection post veraison was well illustrated in NZ (P.N. Wood, HortResearch, NZ, unpubl.). All experimental plots were treated with repeated applications of a selected botryticide

(cyprodinil/fludioxonil) to suppress *B. cinerea* populations early in the growing season. Control plots ('unsprayed', Figure 2), received no botryticides, then at véraison, different treatments were applied. In the control plots without botryticide pre- and post-veraison, the grey mould epidemic developed as predicted and continued after bunches were incubated in high humidity chambers. In the absence of sprays applied post-veraison ('ver. & pre-harvest omitted'), the epidemic continued and was not significantly different to that in the unsprayed plots (Figure 2). Plots that received botryticides post-veraison ('industry full season', Figure 2) had low *Botrytis* infection at vintage, which did not change even after bunches were incubated for up to 10 days at high relative humidity (Figure 2). Therefore, post-veraison infections were not likely to have come from the bunch trash or latent infections of the green structural components (e.g. pedicels) since these were suppressed with botryticides over flowering and bunch closure ('ver. and pre-harvest omitted'). The importance of post-veraison conidial infections have been described in several wine growing regions (Latorre et al., 2001; Wilcox, 2002). An increase in latent infection frequency from veraison (20%) to harvest (100%) was believed to be the result of secondary infections of the ripening berries from air-borne conidia (Keller et al., 2003).

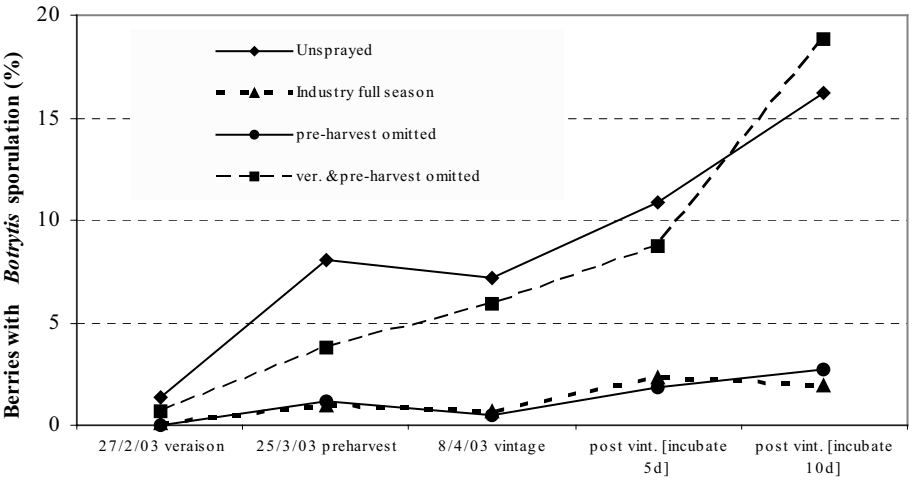

Figure 2. Effect of post-veraison berry infections on bunch rot at vintage and after bunches were incubated in *Botrytis* conducive conditions in 2002 (cv. Sauvignon Blanc, Hawke's Bay, NZ). 'Post vint.' data points refer to the number of days that harvested bunches were incubated in *Botrytis*-conducive conditions (From P.N. Wood, HortResearch, NZ, unpubl.)

In stone fruit orchards, primary inoculum was identified as fruit 'mummies' (desiccated old fruits) with *B. cinerea* sclerotia and mycelium (Hong and Michailides, 2000). *Botrytis*-infected petals of early blooming cultivars are difficult

to distinguish from those caused by *Monilinia* spp. when conditions are non-conducive for sporulation. When conditions are conducive, however, blossom blight occurs as infected by *M. fructicola.* In the Western Cape province of SA, epidemiological investigations found that flower and early fruit infections were less important compared to direct conidial penetration of the fruit during the growing season (Fourie and Holz, 1987, 1994, 1995). The importance of the late season stone fruit infection pathway was confirmed in later studies (Fourie et al., 2002) using surface sterilisation and paraquat (Gindrat and Pezet, 1994). The spatial pattern of *B. cinerea* that emerged from the fruit surface was random and not associated with specific floral tissues. Inoculum for fruit infections within the orchard included herbicide-treated weeds and conidia from early-maturing cultivars (Fourie et al., 2002). *B. cinerea* was unable to directly infect the cuticle of the fruit, but entered the sub-stomatal cavity where further growth ceased due to host defence responses. Close to harvest, conidial infections directly penetrated the fruit cuticle. In addition to stomatal infections, cuticular micro-cracks (Fogle and Faust 1975) have been suggested as possible infection pathways, as in grapes (Pucheu Plante and Mercier, 1983; Commenil et al., 1997) and sweet cherries (*Prunus avium*; Sekse, 1998). These cracks originate from microscopic fractures on the cherry surface which rapidly enlarge as the cherry swells after rainfall near harvest (Glenn and Poovaiah, 1989), thereby facilitating infection (Børve et al., 1998, 2000).

Pathway VI - Conidial accumulation on fruit and dispersal to picking wounds - If conditions are dry after flowering in kiwifruit plantations, *B. cinerea* enters a resting over-summering phase in the canopy. Wet periods during the growing season result in the production of successive crops of conidia which are then dispersed into the canopy where contamination of the external hairy surface of the fruit occurs, but not infection. These conidia are then re-dispersed to the picking wound at harvest. Post-harvest rots occur after a period of cool-storage and this pathway is believed to be specific to kiwifruit in NZ. Accumulation of *B. cinerea* on the fruit surfaces occurs early in the growing season as a result of infections of floral tissues in NZ (Elmer et al., 1995, 1997). Under conditions favourable for the pathogen, profuse sporulation was observed on attached petals. On developing fruitlets, inoculum loads were 100-fold higher on fruitlets with attached petals compared to those where petals had abscised (Elmer et al., 1995). Flowering is therefore epidemiologically important in the ecology of *B. cinerea* in NZ kiwifruit plantations and new, more efficient sampling strategies have been devised, based upon knowledge of the spatial pattern of *B. cinerea* on flowers at full bloom (Elmer et al., 1993). Spatial autocorrelation analysis (Cliff and Ord, 1981; Gaunt and Cole, 1992; Elmer et al., 1998) was used to provide information on the size and orientation of *B. cinerea* foci in kiwifruit plantations.

Kiwifruit shoots and leaves are susceptible to physical damage, providing ample opportunity for *B. cinerea* infection from air-borne conidia (Greaves et al., 2001). Wind-blown senescent shoots, green leaves with necrosis (Hoyte et al., 1994; Elmer et al., 1995, 1997) and senescent leaves in the canopy (Manning and Pak, 1993; Pak and Manning, 1994) were identified as important inoculum sources for external fruit contamination in the pre-harvest period. Methods to quantify the relative contribution of each inoculum source to 'total potential inoculum production' (TPIP)

within vine plots were devised (Hoyte et al., 1994; Elmer et al., 1995). Significant linear relationships between TPIP per plot and the level of external fruit contamination at harvest were identified (Elmer et al., 1997) and also between the level of external contamination at harvest and stem-end rot in storage (Michailides and Elmer, 2000). Conidia have been considered 'short lived propagules' when exposed to direct sunlight (Rotem and Aust, 1991) or high temperatures and dryness (Yunis and Elad, 1989), but long-term survival of *B. cinerea* conidia on fruit surfaces has been reported (Spotts, 1985; Walter et al., 1999a). *B. cinerea* applied to kiwifruit fruits early in the growing season survived exposure to field conditions for up to 16 weeks (Walter et al., 1999a). In contrast, conidia did not survive for 'extended periods' on grape berry surfaces (Coertze and Holz, 2002).

4. The phenomenon of latency in *B. cinerea* epidemiology

Latency, once described as an 'enigmatic aspect of *Botrytis* ecology' (Coley-Smith, 1980), has been the focus of many research studies in order to define its epidemiological role and relationship to crop loss. Several methods have been used to detect latent infections of fungi (Sinclair and Cerkauskas, 2000), including the paraquat induced senescence technique (Cerkauskas and Sinclair, 1980; Gindrat and Pezet, 1994) and freezing of green immature tissues, a method initially designed for *M. fructicola* (Michailides et al., 2000a). Both methods break down any preformed and constitutive host barriers allowing for rapid *B. cinerea* re-growth and sporulation. Many host and pathogen factors could account for the inhibition and apparent suspended state of *B. cinerea* and we examine several host defence mechanisms that may be responsible for latency. Three important stages are often recognised; 1) establishment, 2) containment/arrested phase and 3) resumption of active growth, sometimes referred to as 'the escape phase' (Wade and Cruickshank, 1992a; Latunde-Dada, 2001).

Containment of latent infections is achieved in grapevines by many pre-formed constitutive and inducible defence mechanisms that may account for the high relative resistance of green immature berries to *B. cinerea* (Nair and Hill, 1992; Barnavon et al., 2001; Keller et al., 2003). Preformed morphological features act as a physical barrier, while cuticular waxes are capable of preventing infection due to their antimicrobial and hydrophobic properties (Padgett and Morrison, 1990). Cuticle thickness (Prudet et al., 1992) and chemical composition change as berries ripen, often at a time coinciding with increased susceptibility to *B. cinerea* (Rosenquist and Morrison, 1989; Nair and Hill, 1992; Commenil et al., 1997). If the cuticle is breached, uninfected cells adjacent to the wounded cells commence suberisation, thereby completely isolating invading mycelium (Hill, 1985; Nair and Hill, 1992; Forbes-Smith, 1999). In the *M. fructicola*/apricot system, suberisation and periderm formation was greatest during the early stage of fruit development and was absent from infections on mature fruit. Suberisation around latent infections may also impede nutrient diffusion to the pathogen, thereby ensuring its containment (Wade and Cruickshank, 1992a, b) and blocking fruit ripening signals (Cruickshank and Wade, 1992). Other mechanisms of pathogen inhibition include

polygalacturonase inhibitor proteins and proanthocyanins (Nair and Hill, 1992; Pezet and Pont, 1992; Bezier et al., 2002), and their activity tends to decrease towards harvest (Nair and Hill, 1992; Pezet et al., 2003).

The best characterised inducible defence response of grapevines to fungal infections is the accumulation of phytoalexins and the synthesis of PR-proteins (Mauch et al., 1988; Renault et al., 1996; Chapters 9 and 20). The accumulation of resveratrol and grapevine resistance to *B. cinerea* are well correlated (Jeandet et al., 1995; Sbaghi et al., 1995; Adrian et al., 1997; Chapter 9), and decline in berries to low levels prior to harvest (Bais et al., 2000) associated with an increase in susceptibility (Jeandet et al., 1991; Nair and Hill, 1992). Some PR proteins are capable of causing death of conidia (Pezet, 1988) by breaking down chitin in the cell wall (Mauch et al., 1988; Giannakis et al., 1998), a mechanism that may account for the observed decline in latent infection frequency. Berry phenolics (e.g. catechin) may play a role in resistance of young berries to *B. cinerea* (Goetz et al, 1999), since these compounds were inhibitors of stilbene oxidase, an enzyme implicated in *B. cinerea* pathogenesis (Sbaghi et al., 1996). Unlike grape berries, leaves tend to become more resistant to *B. cinerea* as they mature, the result of cell wall-bound phenolics being released during attempted infection (Weber et al., 1995).

Only a small proportion of early season latent infections became invasive when fruits ripened. In contrast, latent infections derived from inoculations made approximately 30 days before harvest resulted in high levels of fruit rot (Wade and Cruickshank, 1992a, b). These findings suggest that the majority of early season latent infections may never become active, whereas post-veraison latent infections are more likely to become pathogenic when host defences decline.

5. Factors predisposing host tissues to *B. cinerea*

Any factor which causes damage to tissues will facilitate *B. cinerea* infection. Grape berries can be wounded by a range of edaphic, e.g. frost, hail, wind-blown sand, sun (Nair and Hill, 1992), and biotic factors, e.g. insects, snails, birds, excessive fruit swelling (usually in wet soils) and fruit split, berry compression and lesions caused by other pathogens (e.g. powdery mildew, caused by *Uncinula necator*; Jarvis, 1977). In California for instance, pistachio leaves and young green stone fruit wounded by hail were severely infected by *B. cinerea* (Michailides, 2002). Insects are known to be vectors of several plant pathogens, affect disease development (Michailides and Spotts, 1990) and create wounds by which pathogens can enter the host tissue, thereby by-passing several biophysical and biochemical host barriers (Webber and Givvs, 1989).

5.1. Cuticle integrity

The structure and thickness of the cuticle and the epidermal layers have long been regarded as major factors of resistance against *B. cinerea* infection (Commenil et al., 1997). Berry-to-berry contact, where the cuticle is absent or very thin, increases the susceptibility of grape berries to *B. cinerea* (Marois et al., 1986; Rosenquist and

Morrison, 1989) and clones within the same cultivar (e.g. Chardonnay) characterized by tight clusters also develop more severe bunch rot (Vail and Marois, 1991; Vail et al., 1998).

In general, stomata number (Bernard and Dallas, 1981) or natural openings (Pucheu Plante and Mercier, 1983) were independent of susceptibility to *B. cinerea*. However, a recent study found that the number of stomata in the berry epidermis was negatively correlated, while the number and thickness of epidermal and hypodermal cell layers and cuticle and wax contents were positively correlated with resistance to *B. cinerea* in a wide range of table grape cultivars (Mlikota Gabler et al., 2003). Vines grown under UV screens had less cuticular wax and lower lipid oxidase (an indicator of membrane damage) than those grown under ambient light, suggesting that an increase in UV light could lead to thicker wax on the fruit and leaf tissues, which may reduce susceptibility to *B. cinerea* (Steel, 2001).

Developing vegetative and floral tissues are highly susceptible to frost damage, but the role of freezing injury and early season build-up of *B. cinerea* epidemics have not been well studied in orchard crops, though profuse *B. cinerea* sporulation was visually observed on terminal grape shoots after frost injury prior to flowering in Chardonnay grapes (P.A.G. Elmer, HortResearch, NZ, unpubl.).

5.2. Association with insects, invertebrates and vectors of *B. cinerea* inoculum

Botrytis outbreaks in grapes have been shown repeatedly to be associated with the grape berry moth, *Lobesia botrana*. The first generation of the pest attacks flowers, the second feeds on immature berries promoting green berry rot (Fermaud and Giboulot, 1992) and the third generation damages ripe berries. (Bulit and Verdu, 1973; Fermaud and Le Menn, 1992). SEM studies revealed that numerous conidia contaminated the ornamentations of the cuticle segments of the larva and ingested conidia remained viable after passing through the insect's digestive system (Fermaud and Menn, 1989). Supplying viable conidia to second-generation larvae resulted in an increase in the proportion of injuries infected by *B. cinerea* in grape berries (Fermaud et al., 1992). The first instar larvae were attracted by *B. cinerea* infection on grape berries, possibly due to volatile kairomones produced by *Botrytis* (Mondy et al., 1998) and a mutualistic relationship between the two partners was proposed. The presence of *B. cinerea* infected grapes consistently increased insect fecundity and attracted females to oviposit (Mondy and Corio-Costet, 2000).

In NZ, adult thrips of *Thrips obscuratus* (Walker, 1985), often occur in large numbers on both male and female flowers of kiwifruit (Mound and Walker, 1982), feed on pistillate flower petals, act as vectors of *B. cinerea*, increase *Botrytis* inoculum on the fruit surface (Fermaud et al., 1994) and infection of berries (Fermaud and Gaunt, 1995). Use of a marker strain of *B. cinerea* deficient in nitrate reductase to track *B. cinerea* from flowering to vintage (Weeds et al., 1998; Beever and Parkes, 2003; Parkes et al., 2003) showed that the presence of thrips increased berry infection in cv. Riesling, but not in cv. Pinot Noir (Marroni et al., 2003).

Severe outbreaks of *Botrytis* in wine growing regions of Australia have been associated with infestation by light-brown apple moth, *Epiphyas postvittana* (Bailey et al., 1997). The larvae vectored conidia on their cuticles and viable propagules were recovered from larval faecal pellets. Larvae remained resident within a bunch with little bunch-to-bunch movement, increasing the severity of an existing infection by distributing *B. cinerea* to uninfected berries in a bunch. *Drosophila* spp. have long been known as disseminators of microorganisms especially in vineyards (Capy et al., 1987) and tree fruit orchards (Michailides and Spotts, 1990). Conidia of *B. cinerea* were carried externally on the cuticle of *D. melanogaster* and may be carried internally through the digestive tract. The explorative behaviour of these flies on grapes suggests that they spread conidia to ripening fruit and to surfaces where sugary exudates exist, although their precise role as potential *B. cinerea* vectors requires further study. See also Chapter 2.

To ensure adequate pollination of kiwifruit in NZ eight bee hives per hectare are recommended (Bryant, 1986). When foraging, 87% of honey bees were found to carry viable *B. cinerea* propagules (Rose, 1996). Foraging honey bees effectively transferred up to 1.4×10^4 *B. cinerea* CFU per bee from artificially inoculated flowers to control flowers, thereby effectively spreading the pathogen within the canopy (Rose, 1996). However, honey bees are cost effective pollinators and the benefits of good pollination outweighs their potential to vector *B. cinerea*. Another possible vector of *B. cinerea* is the raspberry beetle, *Byturus tomentosus* (Woodford et al., 2000; Woodford et al., 2002). Larvae of raspberry beetle feed on the receptacle and the druplets often causing significant physical damage to the basal druplets (Taylor and Gordon, 1975), resulting in greater post-harvest storage losses. However, the precise role of the beetle in *B. cinerea* epidemiology in red raspberries is not clear.

In California, the brown garden snail (*Helix aspersa*) consumes sepal tissue around the receptacle area of the stem-end of kiwifruits resulting in more post-harvest grey mould than in un-damaged fruits. Snail slime also enhanced the germination of *B. cinerea* conidia (Michailides and Morgan, 1996b). Wounds cause by birds ('bird pecks'), especially on the shoulder of the bunch (Chambers, 1993) caused by the birds claws provide an ideal infection site for *B. cinerea*. Birds are attracted to maturing fruit and in order to protect crops, bird control is routinely carried out to reduce berry damage. Infection by *B. cinerea* of fleshy fruits damaged by birds was also observed in apples, pears, late cultivars of stone fruits, citrus, pomegranates, persimmons, quince and kiwifruit.

6. Effect of plant nutrition on *B. cinerea* epidemics

The effects of specific nutritionally relevant ions on host susceptibility and development of *B. cinerea* epidemics has been well documented (Jarvis, 1980; Goodman et al., 1986). Nitrogen (N) and calcium (Ca^{2+}) have been the two most studied (Elad and Shtienberg, 1995), but often with conflicting results (Elad, 1997).

6.1. Nitrogen nutrition

Low N nutrition is a significant problem in viticulture, associated with 'stuck' fermentations (Tromp, 1984; Conradie and Saayman, 1989) and deterioration of wine aroma (Marangoni et al., 2001). An over-supply of N leads to excessive growth in terms of vine vigour, berry number, bunch compaction and cuticle thinning - factors all known to increase grey mould (Delas et al., 1984, 1991; Keller et al., 2001). Other studies report no adverse effect of N on wine quality (Conradie and Saayman, 1989) and no increase in grey mould (Chambers et al., 1993).

Excessive N fertilization in kiwifruit in Italy did not increase plant growth or leaf number, but *B. cinerea* incidence in cool-stored fruits was higher (Pertot and Perin, 1999). In NZ *Botrytis* incidence in cool storage was strongly linked to excessive N (Prasad et al., 1990; Prasad and Spiers, 1991), but a later study found no evidence of a link (Smith and Buwalda, 1994). *B. cinerea* populations pre-harvest were not measured in the studies described above and we suggest that the relationship between N and post-harvest *Botrytis* is indirect, perhaps leading to an increase in the susceptibility of leaves and shoots to physical damage, reducing disease resistance of leaves, as reported in related host-pathogen systems (Daane et al., 1995), thereby increasing inoculum potential in the canopy.

6.2. Calcium

Ca^{2+} plays a vital role in fruits and vines (Ferguson, 1984), increases resistance to disease (Volpin and Elad, 1991; Conway et al., 1991), reduces leakage of exudates to the host surface thus, reducing their availability to the pathogen (Volpin and Elad, 1991) and modulates various cell functions (Conway, 1982; Elad et al., 1992). In contrast, Ca^{2+} deficiency increases susceptibility to *B. cinerea* (Schwab et al., 1993). Grape cultivars differ in their response to Ca^{2+} and enzymatic degradation by *B. cinerea*, indicating that the relationship between Ca^{2+} and *B. cinerea* is complex (Chardonnet and Doneche, 1995; Chardonnet et al., 1997). When Ca^{2+} was applied before veraison to a range of grape cultivars, infection was reduced. In contrast, Ca^{2+} applied after veraison had no effect on epidemic development (Doneche and Chardonnet, 1996). When Ca^{2+} applications were made to table grapes in the field, resistance to *B. cinerea* was increased and correlated with increased levels of cellulose and of both oxalate and alkali-soluble pectins (Miceli et al., 1999). Incubating *B. cinerea* conidia in increasing concentrations of $CaCl_2$ decreased conidial germination and germ tube length (Chardonnet et al., 2000). However, the inhibitory effect of $CaCl_2$ on *B. cinerea* could be overcome by the addition of glucose to the medium (Wisniewski et al., 1995). This has important implications since sugar leakage from grape berries increases post-veraison (Kosuge and Hewitt, 1964), potentially neutralising the beneficial effects of Ca^{2+}. The inhibitory activity of Ca^{2+} on *B. cinerea* was also dependant on isolate (Chardonnet et al., 2000). In strawberries, variable results with Ca^{2+} have been reported. In one study, vinclozolin and captan were replaced with $CaCl_2$ to protect strawberries from *B. cinerea*, but the $CaCl_2$ treatments did not

reduce the epidemic (Erincik et al., 1998). In contrast, increasing fruit calcium content reduced the incidence of *Botrytis* (Cheour et al., 1990; Karp and Starast, 2002; Wojcik and Lewandowski, 2003).

7. Host management factors and *B. cinerea* epidemics

7.1. Rootstocks and rooting depth

The effect of rootstocks on *Botrytis* bunch rot of grapes has been well studied (Egger et al., 1979; Delas et al., 1984; Ferreira and Marais, 1987; Cristinzio et al., 2000) and generally is indirect in its nature, primarily affecting scion vigour and bunch compactness (Ferreira and Marais, 1987). The rootstock may impart a 'resistance factor' to the scion, for example leaves of cv. Falanghina produced smaller lesions on rootstock SO4, compared to three other rootstocks (Cristinzio et al., 2000). However, inoculum production from such lesions and the nature of the resistance mechanism requires further investigation. Extensive research in France on Mèdoc and Graves soils on the impact of rooting depth and water up-take on skin splitting and grey mould have been made (Ribèreau-Gayon et al., 1980); deep rooted vines were much less susceptible to splitting and grey mould than shallow-rooted vines. Rootstocks also had a significant impact on the extent of fruit micro-cracking in sweet cherries and differences in soil moisture uptake by rootstocks were believed to be responsible (Cline et al., 1995).

7.2. Cultivars

In grapes, cultivar is one of the most important variables affecting grey mould epidemics. The morphological, anatomical and chemical characteristics of 42 cultivars with a range of resistance to *B. cinerea* were measured to establish whether resistance was linked to specific characteristics. Eleven were classed as highly resistant and the number of naturally occurring pores was negatively correlated with resistance to *B. cinerea* (Mlikota Gabler et al., 2003), thus confirming earlier studies (e.g. Eibach, 1994). Cultivar resistance was attributed to higher cuticle and wax contents and certain anatomical features, rather than induced or constitutive antifungal host defence mechanisms (Mlikota Gabler et al., 2003). In some raspberry cultivars, the stigmatic fluid was inhibitory to *B. cinerea* thereby avoiding latent infections (Williamson et al., 1987; Williamson and Jennings, 1992). These findings suggest that cultivar selection will play a major role in future *Botrytis* management strategies.

7.3. Canopy management

There have been continued global efforts to find the best combination of optimal canopy type, training and pruning systems and *Botrytis* suppressiveness to optimise fruit quality in grapes.

7.3.1. Vine training and pruning systems

Grapes grown in dense canopies are exposed to greater periods of wetness after rainfall, resulting in increased susceptibility to *B. cinerea* (Steel, 2001). A range of different vine training systems were evaluated in Italy on several grape cultivars to identify systems that were non-conducive to pathogen development. The highest incidence of *B. cinerea* was reported in the 'Pergola' system, while vines pruned to the 'Guyot' system had the lowest disease development (Cargnello et al., 1991). Vines trained in the horizontal bilateral cordon ('traditional Moser system') had improved exposure to light and lower incidence of *Botrytis* and powdery mildew (*Uncinula necator*), higher yields and better quality grapes than the 'high' cordon system supported by a one-wire trellis (Redl, 1988). Also, the practice of leaving 60 rather than 40 nodes per vine in vigorously grown Chenin Blanc grapes reduced bunch rot in spur- or cane-pruned systems, and the *Botrytis* reduction was attributable to less compact clusters (Christensen, 1981).

In Australia, the practice of 'lighter pruning' the vine canopy reduced berry-to berry-contact within the bunch and *B. cinerea* development (Martin, 1990). Non-contact Riesling berries had 15.7 and 35% more epicuticular wax and cuticle compared to the contact samples, explaining the lower incidence of bunch rot (Percival et al., 1993). Similarly, reducing epicuticular waxes in grapes by spraying an adjuvant can increase bunch rot (Marois et al., 1987). Along with the training system itself, the bunch architecture can also affect development of the pathogen. Infection of Cabernet Sauvignon clusters after veraison by *B. cinerea* was significantly influenced mainly by cluster compactness (Vail and Marois, 1991; Fermaud et al., 2001a, b); reduced *Botrytis* was correlated with less compact clusters, associated with lower berry number and reduced cluster weight. Thus, training and pruning systems adopted to reduce the risk of *Botrytis* at vintage may be cultivar-specific and dependant upon a range of other factors.

Vine 'hedging' is the practice of pruning off the over-hanging current season growth at veraison. Vines trained on a two-wire trellis, sprayed and hedged, had a 39% reduction in bunch rot as compared to vines sprayed and not hedged. Hedging improved air circulation in the bunch zone, reduced relative humidity in the canopy and exposed more fruit bunches to light (Savage and Sall, 1982). This practice has now been widely adopted in Australasian (Clingeleffer, 1984; Sommer et al., 1995) and in North American vineyards (Reynolds and Wardle, 1993) as a cost-effective alternative to hand pruning and as a cultural operation aiming to reduce bunch rot. A better practice was proposed subsequently, based on careful selection of node number at winter pruning, providing better shoot spacing and thus creating a canopy with optimal density (Smithyman et al., 1997).

Different cultivars respond to pruning regimes quite differently. In seasons conducive to infection, the practice of removing or thinning 'distal' clusters just before veraison reduced infection in northern Italian vineyards. The level of cluster thinning depended on the particular cultivar, e.g., bunch rot incidence at harvest was 21% for no thinning, 10% for the 20% cluster thinning and 7% for the 40% cluster thinning level. In contrast, cluster thinning in Cabernet Sauvignon had no significant effect on bunch rot at vintage (Palliotti et al., 2000).

7.3.2. Leaf removal

Leaf removal from the fruiting zone of vines ('leaf plucking') has significantly reduced epidemics thereby improving *Botrytis* control in grapes in European (Zoecklein et al., 1992), Californian and Australian vineyards (Gubler et al., 1987; Percival et al., 1994). Leaf removal affects the microclimate (temperature, vapour pressure deficit, wind speed and wetness) in and around the receptive bunch, often reducing bunch rot at vintage. Increased wind speed after leaf plucking (English et al., 1989) increased the evaporative potential on the berry surface, thereby significantly reducing *B. cinerea* infection and development (Thomas et al., 1988; English et al., 1993). In addition, stimulation of phytoalexin production by increased UV light has been reported as a result of leaf removal (Langcake, 1981). Following leaf removal, exposed berries of Riesling grapes had 19 and 35% more epicuticular wax and cuticle, respectively, compared to the shaded bunches resulting in significantly less grey mould (Percival et al., 1993). Leaf removal has been adopted globally as an effective non-chemical practice to manage *B. cinerea* in vineyards.

7.3.3. Removal of potential substrates for the pathogen

A less well-adopted practice to manage *Botrytis* is the removal of potential substrates to reduce inoculum potential in the bunch early in the season. Removal of senescent floral tissues and aborted berries ('bunch trash') reduces *B. cinerea* by c. 30% in Merlot grapes (Jermini et al., 1986). The relationship between senescent floral debris retained in fruit clusters of Chardonnay and *Botrytis* bunch rot was investigated for three seasons in California. Compressed air was used to remove bunch trash at early or late fruit set. Removal of inoculum in bunch trash significantly reduced bunch rot in some, but not all vineyards, indicating that other factors besides bunch trash biomass may contribute to subsequent bunch rot at harvest (Wolf et al., 1997). In addition, in a Californian kiwifruit plantation, removal of flowers from male vines, a potent source of *B. cinerea*, reduced stem-end rot by 60% in neighboring female vines compared to fruit from vines where the male flowers were retained (Michailides and Elmer, 2000).

The impact of removal of necrotic tissue on epidemics was also demonstrated in The Netherlands (Köhl et al., 1992). In this study, removal of up to 30-50% of necrotic tissues by hand reduced the number of *Botrytis* spp. conidia in the air by 34% and subsequently delayed the *Botrytis* epidemic. This finding was used as the rationale for developing a new biocontrol strategy, based upon saprophytic colonization of necrotic tissues by selected antagonists (Köhl et al., 1995; Chapter 13). Use of compressed air to remove necrotic canopy and bunch tissue in grapes in NZ reduced the *B. cinerea* epidemic and at harvest, bunch rot incidence was reduced by c. 50% (P.N. Wood, HortResearch NZ, unpubl.). These and other studies demonstrate the importance of necrotic tissue substrates for *B. cinerea* epidemics.

7.3.4. Harvest practices to limit *B. cinerea* losses

Harvesting earlier than scheduled is the commonest cultural practice used to limit losses of mature grapes by *Botrytis* (Nair, 1985). For premium grade Chardonnay wines, grapes are harvested between 23 and 25º Brix (soluble solids content). If conditions favour *Botrytis* development, the crop will be harvested at 18º Brix to limit losses in NZ.

In kiwifruit, a different relationship between º Brix and *B. cinerea* infection of the picking scar was established (Pennycook and Manning, 1992; Pyke et al., 1993). The majority of kiwifruits are harvested at 6.2º Brix to optimise storage keeping quality, but a common practice in organic kiwifruit plantations is to harvest at a more advanced maturity (e.g. 7.5º Brix). Field and experimental data support that more mature fruit at harvest have increased levels of resistance to *B. cinerea* than less mature fruit (Pyke et al., 1993).

8. Effect of growing system

Despite the strong increase in production of organic food, driven by consumer concern for food safety and the environment, only eight papers have been published since 1979 on *B. cinerea* in organic fruit systems, thus indicating clear needs for future research. Organic grapes are perceived to sustain more frequent and severe *Botrytis* epidemics than those under conventional production systems. However, *B. cinerea* was reported to be less in an organic system in Germany when compared to the conventional integrated system (Hoffman et al., 1997), but the factors responsible for this reduction of grey mould were not identified.

Greater uptake of organic viticulture may be achieved if naturally occurring genetic resistance could be identified that produced wines with high quality attributes. Some cultivars are regarded as highly resistant and possess a wide range of active and preformed defence mechanisms (Stein and Blaich, 1985; Stein, 1985; Nair and Hill, 1992). Efforts to combine *B. cinerea* resistance and grape quality have progressed slowly, for example, 28 years were required to produce a new grape cultivar (cv. Regent) with resistance to *B. cinerea*, downy mildew (*Plasmopara viticola*) and powdery mildew (*Uncinula necator*), suitable for use in organic wine production. The yield and sensory characteristics of the new cultivar were reported to be similar to other grape cultivars (Topfer and Eibach, 2002).

In organic Italian kiwifruits, *B. cinerea* is a significant cause of crop loss and summer pruning is the only strategy recommended to increase air flow in the canopy (Giusti and Rossi, 2002). Organic kiwifruit plantations in NZ generally have low *Botrytis* rots in cold storage, compared to conventional systems, a finding supported in one comparative study (Michailides and Elmer, 2000). The epidemiological basis for the differences is not yet fully understood since canopy density and area of necrosis were not significantly different between these two systems, whereas the incidence and severity of *B. cinerea* populations in necrotic canopy tissues were significantly different.

Necrotic strawberry leaves were the primary source of *B. cinerea* inoculum in strawberries (Braun and Sutton, 1987). An obvious strategy to reduce *B. cinerea*

epidemics is to remove these tissues, but this practice has not slowed epidemic development (Daugaard, 1999). Increasing aeration around the plants by single row planting has reduced grey mould incidence (Schmid, 1996) compared to double and triple rows (Dijkstra and Van Oosten, 1985; Strik et al., 1997). Plant age affected the severity of *B. cinerea* over growing seasons, indicating perhaps an inoculum build-up; short cropping cycles were recommended to delay the build-up of inoculum (Daugaard, 2000). A significant positive relationship between N and *B. cinerea* fruit rot has led to low N use (Kopanski and Kawecki, 1994; Wilcox et al., 1994; Cooley et al., 1996). Finally, although no specific genetic sources of resistance have been found (Hortynski et al., 1991; Maas and Galetta, 1997), differences in *B. cinerea* susceptibility in strawberry cultivars have been identified (Daugaard, 1999; Barth et al., 2002; Rhainds et al., 2002).

9. Conclusions

When environmental conditions favour *B. cinerea* in the spring, epidemics are common and associated with an abundance of senescing host tissues. Removing the dominant sources of *B. cinerea* substrates reduces inoculum loading in orchards, but does not eliminate it, since conidia are so common in the air. During the summer months it is often perceived that *B. cinerea* 'virtually disappears' and this period of relative inactivity is frequently referred to as 'over-summering'. At the end of the growing season, coinciding with fruit ripening, a sudden resurgence of *B. cinerea* can occur especially in disease favourable conditions.

Considerable research has focused on the dominant infection pathways from flowering to fruit rot with a view disrupting this part of the *B. cinerea* disease cycle, thereby reducing epidemic development later in the season. There is an even greater imperative in the future for acquiring this new knowledge because it will form the foundation for sustainable disease control strategies for the 'reduced pesticide' and organics era. Most of the infection pathways described included a latent or inactive period. Several host mechanisms were described to account for this enforced state of inactivity (Chapter 9); however, the trigger that reactivates *B. cinerea* growth in latent infections remains a mystery. The epidemiological importance of the infection pathways described differs by location and cultivar. Saprophytic development of mycelium in floral debris, aborted fruitlets and calyptras in grapes, calyces (shucks) in stone fruit and almond, bud and bud scales in pistachio and flower sepals in kiwifruit are important in disease epidemics. Further research on the 'within bunch conidial accumulation' pathway to fruit rot, and the 'conidial infection of the external fruit surfaces pathway', proposed for grapes, kiwifruit (California), stone fruits, pome fruits, blueberries, blackcurrants, pomegranates, and figs are required.

Significant knowledge gaps exist for the host and microclimate conditions required for latent infection to occur during the host's phenology. If these parameters were more precisely defined, then predictive models for *B. cinerea* would be more accurate. Latent infection detection is based upon surface sterilants and there are variations in the type, number, duration and sequence of these. There is a need for faster and more efficient methods of latent infection detection and the use

of real time polymerase chain reaction (RT PCR) will provide the basis for efficient, accurate and more rapid detection of *B. cinerea* inoculum.

Factors predisposing host tissue to infection by *B. cinerea* included biotic (insects, invertebrates, and humans) and abiotic (nutrition, chemical and cultural practices). Controlling insects (e.g. grape berry moth), modifying microclimate (e.g. leaf removal, special pruning) and reducing substrate availability for *B. cinerea* has significant impacts on *B. cinerea* survival and epidemic development.

Breeding for resistance has not been as successful as hoped for but significant *B. cinerea*/cultivar interactions indicate that greater emphasis should be placed on cultivars with morphological, anatomical and inherent host defence mechanisms which reduce susceptibility (Mlikota Gabler et al., 2003). Finally, organic producers frequently observe *B. cinerea* epidemics declining over time, yet the host, pathogen, environmental and human variables responsible have not been resolved and represent an exciting challenge for the future.

10. Dedication

We dedicate our review to the memory of the late Professor Roy Edward Gaunt, an internationally respected epidemiologist and a close colleague.

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