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Botrytis Gray Mold of Kiwifruit Caused by *Botrytis cinerea* in the United States and New Zealand

The kiwifruit (*Actinidia deliciosa* (A. Chev.) C.S. Liang et A.R. Ferguson) (syn. *Actinidia chinensis* Planch) is a member of the Actinidiaceae family and indigenous to Southeast Asia. It was relatively unknown several decades ago, but it has now become an important fruit crop in several countries worldwide. Kiwifruit was first grown in New Zealand at the turn of the century from seed from China, but the first commercial planting did not occur until 1930 at Te Puke in the Bay of Plenty region of the North Island (72). Large-scale planting of the Hayward cultivar did not occur until the 1970s, but New Zealand's success at marketing this cultivar worldwide encouraged other temperate-zone countries such as Australia, Chile, France, Greece, Italy, Japan, Portugal, South Africa, Spain, and the United States to develop commercial plantings (Table 1; 47). Robert Smith, based at the USDA's Plant Introduction Station in Chico, California, was the first to demonstrate that kiwifruit could be grown and produced successfully in Cali-

fornia. He provided seeds and cuttings for the establishment of the first commercial vineyards in the San Joaquin Valley in 1966 and Gridley in 1968. The main production areas in California are now concentrated in the northern Sacramento Valley.

Classification, Species, and Cultivars

The genus *Actinidia* is solely of Asian origin, found from northeast India through China to tropical Java, and in eastern Siberia. All kiwifruit cultivars grown commercially in California were introduced primarily from New Zealand by the USDA Plant Introduction Station at Chico in the 1930s. Commercial cultivars of kiwifruit are large-fruited selections of *A. deliciosa* (Fig. 1A), normally grafted onto *A. deliciosa* seedlings or onto the rootstock 'Kaimai', a New Zealand selection, but there are other species of interest because of their edible fruit. These are *A. chinensis*, *A. arguta*, *A. kolomikta*, *A. polygama*, and *A. eriantha*. For instance, *A. arguta* (cv. Issai) is winter hardy and able to tolerate severe subfreezing temperatures (6). Another species, *A. kolomikta* (Fig. 1B), a native of eastern Siberia, has small, berry-like fruit and thrives in the Central Valley region of California, where high temperatures are common. Cultivars of the small-fruited species differ widely, are often consumed at a more advanced stage of ripeness, and are characterized by a pleasant sweet flavor. Hayward, the most widely grown commercial cultivar, is usually considered the standard fruit prototype in

countries that grow kiwifruit. An early-maturing selection of Hayward (cv. Tomua) has been planted by some growers in New Zealand, since it has the advantage of maturing 2 to 3 weeks before the standard Hayward cultivar. More recently, a new hairless, yellow-fleshed cultivar (*A. chinensis* Hort 16A) with a sweet tropical taste has been introduced in New Zealand, and many growers are in the process of converting traditional Hayward vineyards to Hort 16A.

Because kiwifruit is dioecious, successful fruit production requires both the female cultivar and a male pollinator to supply the pollen. A number of male cultivars are being used by California and New Zealand growers (6).

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Table 1. Countries growing kiwifruit and respective hectareage (1990)^y

Country	Hectareage
New Zealand	17,325
United States (California)	1,810
Italy	19,150
Chile	9,120
Australia	1365
France	5,015
Greece	830
Japan	4,670
Portugal	NA ^z
Spain	985

^y Source (73).

^z Not available.

Kiwifruit Marketing

The area planted in the United States is about 1,810 ha, almost all of which is in California (Table 1), with a total production of approximately 30 million kg valued at 100 to 110 million U.S. dollars per year. More than 95% of the fruit is consumed fresh, while less than 5% is sold as a processed product. In 1998 in New Zealand, the total area in kiwifruit was 10,329 ha, with total production of 227 million kg valued at 197 million U.S. dollars. Nearly 98% is exported fresh, with the remaining 2% either exported as a processed product or sold fresh on the local market. Organic kiwifruit production New Zealand has increased steadily, and it has been further estimated that by 2000, organic production will expand to 2.5 million trays, representing approximately 4% of the industry volume (14). Organic production of kiwifruit in California is small and accounts for about 5 to 10% of the total hectareage planted.

Kiwifruit are harvested when they are unripe and firm (6.5 to 8% soluble solids and >6.35 kg/cm² firmness) and allowed to ripen during storage (80). Controlled atmospheres have extended the storage life of kiwifruit for up to 5 or 6 months in California and up to 8 months (April to December) in New Zealand (37,45,52). Extended storage periods help coordinate marketing of fruit, but it has caused some postharvest problems, including losses due to fruit decay caused by several fungal species. Although near-optimal conditions for quality maintenance can be achieved in commercial storage designed for the special needs of kiwifruit, conditions during transport to distant export markets may fluctuate and be far from optimal. Since transit times can be as long as 30 to 40 days, there is considerable potential for losses in fruit quality due to premature softening and fungal decay.

The most important postharvest rot is gray mold, caused by the fungus *Botrytis cinerea* Pers.:Fr. The first symptoms can appear as early as 1 month after the start of cold storage. Other fungi associated with fruit rots have also been identified, such as species of *Botryosphaeria*, *Cryptosporiopsis*, and *Phomopsis* (8,62,77,78), but these generally do not appear until fruit have become very soft and overripe at the end of their physiological life.

Botrytis Gray Mold

Initially, kiwifruit was regarded as a "disease-free" crop in New Zealand (39) and California (78). However, over the last 15 years, gray mold has become a potentially serious problem and has coincided with an increase in the area planted and longer storage times due to increased quantities of fruit in cold storage and marketing considerations. Inspection of Californian kiwifruit arriving at European markets indicated that the principal causes of fruit loss were desiccation and gray mold. Losses of up to 20% in California-grown kiwifruit were reported

Fig. 1. Fruit of (A) *Actinidia deliciosa* and (B) *A. kolomikta* (courtesy J. Kola).

Fig. 2. (A) Kiwifruit with typical gray mold at the stem end; (B) kiwifruit with gray mold symptoms on the side (side rot); (C) sectioned fruit with gray mold showing water-soaked symptoms in the fruit flesh; (D) sorting and repacking fruit done periodically during cold storage of kiwifruit.

Fig. 3. Cultures of *Botrytis cinerea* producing abundant (A) conidia or (B) sclerotia; (C) kiwifruit with abundant sclerotia developed during long-term cold storage (immature sclerotia are jellylike and white); (D) germination of *B. cinerea* sclerotium, producing a tuft of conidiophores.

(22,57,78), and losses of greater than 30% in New Zealand-grown kiwifruit have been recorded in extreme cases (68). However, direct fruit losses of 0.2 to 2% are more common. Indirect losses, such as premature fruit softening, mean that the total cost to the New Zealand industry some seasons may exceed 8 million U.S. dollars (34). Additional costs may also be incurred as a result of labor-intensive re-sorting and re-packing. Gray mold incidence in exported fruit from both countries has been unpredictable, and considerable variation occurs among growing seasons, regions, and individual orchards within a region (62,67,77).

Symptomatology. Gray mold symptoms on the fruit do not appear in the field but are usually visible at the stem end after 3 to 4 weeks of cold storage (35) at 0°C (Fig. 2A). In New Zealand, the long delay between initial fruit infection at the picking wound at harvest and first symptom expression is thought to occur, not necessarily as a result of a change in fruit susceptibility, but rather as a consequence of slow *B. cinerea* development through the fruit tissues at 0 to 1°C in cold storage. At these temperatures, fungal penetration of 0.2 mm/day was reported through the stem plug by Hallet and Sharrock (35). At this rate, the fungus was predicted to reach the fruit flesh in about 6 weeks, and this coincides with the time at which symptoms first appear on the fruit.

Fruit surface wounds caused by poor handling practices can also act as an entry point for *B. cinerea*, which then develops into a so-called “side rot” (Fig. 2B), but

Fig. 4. Life cycle of *Botrytis cinerea* in kiwifruit vineyard in California.

these losses are generally small. Gray mold usually begins at the stem end of unwounded fruit and advances evenly toward the distal end (Fig. 2C). Affected areas appear darker than the healthy part of the fruit, and white to gray mycelium and sporulation may be present (Fig. 2A). Internally, infected tissue turns dark green and is water-soaked (Fig. 2C). Conidiophores with conidia and sclerotia can develop on infected fruit. In advanced stages of disease, adjacent fruit become connected by mycelium, forming "nests" of disease.

The pathogen. *B. cinerea* is a cosmopolitan ascomycete of the fungal family Sclerotiniaceae, causing shoot blights and gray mold of fruit in numerous crops. Conidia are produced on dichotomously branched conidiophores, which have globose basal cells from mycelia and on sclerotia. Conidiophores are produced in the

vineyard on the surface of infected host tissues, on fruit in cold storage, and on the surface of internal flesh exposed to the open environment after fruit damage. The conidia are hyaline or pigmented, ellipsoid-obovoid, globose, without septa. Conidia are smooth and germinate in nutrient solutions to form one to five germ tubes, but in the absence of any stimulant (e.g., in distilled water), germination may not occur, or is much reduced. Conidia germination is stimulated by pollen (13). A wide range of isolates may be recovered from vineyards, ranging from isolates with sparse conidia production to isolates with abundant conidia or sclerotia (Fig. 3A and B). Sclerotia are small, hard, black survival structures and are often observed firmly attached to the outside of decayed kiwifruit (Fig. 3C). Sclerotia can also be produced on other host substrates in the vineyard

such as mulched prunings on the ground. In favorable conditions, germinating sclerotia produce conidiophores atop a small stromatic cushion formed on the surface of the sclerotium (Fig. 3D). Carpogenic germination can occur after a dormant period of 2 to 6 months. The rind of the sclerotium cracks open to reveal the elongating stipe of an apothecium, with two to six apothecia from one sclerotium. *B. cinerea* apothecia have not been reported in the field in California or New Zealand, but cupulate, brown apothecia have been produced in laboratory studies (82).

Epidemiology and Disease Cycle

Gray mold is a postharvest disease, but like many postharvest diseases, it has a preharvest component (Fig. 4). This section addresses our knowledge of *B. cinerea*

Fig. 5. Sources of sporulation of *Botrytis cinerea* in a kiwifruit vineyard floor. (A) Fruit eaten by rodents and/or birds; (B) a partially decomposed kiwifruit; (C) leaf of lemon with a distinct lesion and sporulation of *B. cinerea*.

Fig. 6. Progress of colonization by *Botrytis cinerea* of sepals and receptacles of kiwifruit in nine vineyards each in 1993 and 1994 in California. Data points represent means of nine vineyards per year with >300 sepals and 60 receptacles per vineyard in each sampling date.

Fig. 7. Seasonal pattern of *Botrytis cinerea* and relative contribution of each inoculum source to total potential spore production (TPSP) at several stages in a kiwifruit growing season in New Zealand. FFO = first female flower opening; FB = full bloom (100% of female flowers open); PF = petal fall; MF = mid-fruit development; and H = harvest. Values are back-transformed natural log values.

ecology in the vineyard from vine dormancy to fruit harvest and proposes the likely sequence of events that leads to fruit infection and subsequent gray mold in cold storage. In the next section, factors that affect the incidence of gray mold in both California and New Zealand are described.

California. In winter, *B. cinerea* can sporulate on infected fruit on the vineyard floor, or on fruit wounds caused by rodents, birds, or garden snails (Fig. 5A and B). In addition, the fungus can sporulate on senescing leaves of weeds (including grasses and broadleaf species), necrotic kiwifruit leaf and fruit remnants, or senescent leaves from nearby crops (e.g., *Citrus* spp. [Fig. 5C]). Overwintering sclerotia and mycelium produce abundant conidia in winter and spring (Fig. 4), which infect susceptible tissues if free moisture (rain, overhead irrigation, dew formation) is available. During bloom, petals and anthers become infected, and it is believed that these tissues provide the inoculum for sepal and receptacle infections, commencing 30 days after flowering (Fig. 4). The sepals are susceptible during the entire growing season until the fruit is harvested (21,22,54). *B. cinerea* isolation of surface-sterilized sepals collected at regular intervals during the growing season has demonstrated that most sepal infections occur between 30 to 60 days (April and May), then between 120 to 150 days (September to October) after fruit set (Fig. 6). There is a continuous increase in the incidence of *B. cinerea* in the tissues of the stem end region (sepals and receptacles), which suggests that the infections in this area are cumulative (Fig. 6). Infections of the sepals and receptacles are likely to be latent, since no apparent disease symptoms or pathogen signs are visible. Infections of the receptacle are probably less sensitive to environmental extremes, since they are embedded in the tissue, compared with sepal infections, which are more directly exposed to fluctuations of temperature and moisture.

External fruit contamination by *B. cinerea* propagules is a major source of inoculum for infections of sepals and receptacles in California. In coastal vineyards of Cali-

fornia, sporulation of *B. cinerea* is very common on flowers of male vines, and fruit harvested from female vines adjacent to male vines show greater incidence of gray mold, probably because of the greater incidence of sepal and receptacle infections compared with fruit from vines not adjacent to male vines (56). Since pollen is known to stimulate *B. cinerea* germination (13,71), abundant quantities available in the vineyard could enhance the aggressiveness of *B. cinerea* conidia, allowing for greater sepal and receptacle infection. *B. cinerea* can be found throughout the season, but conidial numbers are greatest in the spring and fall, when conditions tend to be more favorable for infection and sporulation. The presence of inoculum throughout the season in California vineyards may explain continuous infections of sepals and receptacles (Fig. 6) (54).

New Zealand. An extensive review of gray mold in kiwifruit conducted for the New Zealand kiwifruit industry in 1990 and published in 1992 (8) concluded that although abundant *B. cinerea* sporulation was frequently observed on senescent petals at the end of bloom and on dead vegetative tissue in the vineyard, there was a lack of quantitative information on inoculum sources for the entire growing season in New Zealand. To address this issue, a

method was devised by Elmer et al. (24) to measure total inoculum potential in vine plots. This method, based on incubation of host tissues, was similar to that used by Braun and Sutton for gray mold of strawberry (7). The area of sporulation on several different types of host tissue was estimated visually, and the number of conidia was determined using simple linear regression equations that were developed for each type of host tissue (25,26). The sum of potential conidia production from all tissue types was termed Total Potential Spore Production (TPSP).

The main sources of *B. cinerea* inoculum and their contribution to TPSP at different stages in the growing season are summarized in Figure 7. Early in the growing season (first female flowers opening), the majority of inoculum was produced on overwintering mycelium on prunings on the ground (Fig. 8). Occasionally, *B. cinerea* sclerotia on prunings were observed producing conidiophores. *B. cinerea* was not detected in leaf litter samples from beneath vine canopies from dormancy to harvest (24), although some researchers have reported sporulation in some growing regions in New Zealand. In contrast, sporulation of *B. cinerea* was found in overwintering leaf litter samples prior to flowering in California (P. A. G. Elmer and T. J. Michailides,

Fig. 8. Main source of sporulation of *Botrytis cinerea* prior to bloom and as the first female flowers open were prunings left on the ground in New Zealand.

Fig. 9. (A) Profuse *Botrytis cinerea* sporulation on herbicide-treated weed (*Rumex* spp.) beneath kiwifruit canopy (Nelson region of New Zealand, 1992); (B) flower petals attached to kiwifruit fruitlets and infected by *B. cinerea*; (C) a leaf lesion resulting from contact with an infected petal; (D) wind-damaged canes are colonized by *B. cinerea* and are the major source of inoculum in the mid-fruit development stage of kiwifruit growth; (E) sporulation on leaves of wind-damaged canes.

unpublished). Profuse *B. cinerea* conidia production, similar to that observed in California, was observed in New Zealand on Paraquat-treated perennial weeds such as *Rumex* spp. (Fig. 9A). At full bloom, senescent male flowers and prunings were the main sources of *B. cinerea* inoculum (Fig. 7). There were no necrotic leaves in the vines at this growth stage. At petal fall, TPSP peaked (Fig. 7) and coincided with an abundance of senescing petals attached to fruitlets (Fig. 9B). Sepal infections were also detected, but their contribution to TPSP was negligible. Green leaves with necrosis were detected for the first time at petal fall, and *B. cinerea*-infected female petals adhering to the leaves (Fig. 9C) were responsible for many of the green leaf infections at that stage (24). There were significant ($P < 0.05$) positive correlations between the incidence of *B. cinerea* on senescent petals and incidence of green leaf infections and incidence of *B. cinerea* sporulation on leaf lesions in vines at that time. By mid-fruit development (January to February), strong winds damage rapidly developing vegetative shoots. These senescing "wind-damaged shoots" were the main source of *B. cinerea* inoculum at that time (Fig. 9D and E). It has been suggested (51) that these necrotic tissues do not produce large quantities of inoculum compared with other sources later in the season, but these tissues may function as an "inoculum bridge" for later infection of green, senescing, and dead leaves in the canopy in New Zealand (27,30). In contrast, leaves of wind-damaged shoots are not colonized by *B. cinerea* due to the hot and dry climate in California. By harvest in New Zealand, green leaves with necrosis (Fig. 10) become the main source of inoculum (Fig. 7). Another study in the South Auckland region

also identified dead leaves as the primary source of *B. cinerea* in kiwifruit vines (51). Analysis of the dynamics of *B. cinerea* populations in vineyards using spatial, temporal, and spatiotemporal autocorrelation analysis indicated that *B. cinerea* populations tended to be strongly aggregated at the individual vine level (25). Consequently, high *B. cinerea* populations in individual vines in several studies resulted in high levels of contamination of external fruit surfaces with *B. cinerea* inoculum.

Susceptibility of kiwifruit to *B. cinerea*. During the late 1980s and early 1990s, research in New Zealand focused on identifying the primary site of fruit infection. In 1991, Sharrock and Hallet (74) investigated whether *B. cinerea* gained entry through, rather than around, the "stem plug" region, including the remains of the calyx. Site-specific inoculation with *B. cinerea* onto the end of the long pedicel alone produced a high incidence (91%) of typical stem end gray mold in cold storage. Fruit inoculated only around the perimeter of the stem plug had only 5% incidence of typical rots. No rots developed in non-inoculated fruit samples. These and other studies (35,63) have demonstrated that the wounded stem end was the preferred entry point for *B. cinerea* in New Zealand kiwifruit (Fig. 11).

In California in 1997, however, spray-inoculations of kiwifruit with a spore suspension of 10^5 spores of *B. cinerea* per ml during late April to early October did not result in greater levels of gray mold than the water-sprayed control fruit after 5 months in cold storage. When fruit were evaluated after 7 months in cold storage, only those inoculated at full bloom had developed 14% gray mold, while 1.5 to

4.0% gray mold developed in fruit from inoculations at other growth stages (T. J. Michailides, unpublished data). Research in France tended to support that done in California, where two stages of fruit susceptibility were identified, the first during the bloom and fruit-set phase (75 to 80% of the infections) and the second at harvest (1).

Because the sepals remain on the fruit in cold storage even after mechanical prestorage brushing, they were selected as a possible entry site for *B. cinerea* and subsequent gray mold development in California. Periodic isolations from sepals indicated that sepals of kiwifruit were continuously infected by *B. cinerea*, with high levels of infection occurring early after fruit set and prior to harvest (Fig. 6). For example, sepal colonization by *B. cinerea* is typically 25% (from bloom to fruit set), then declines to 15% (about 3 months after fruit set) and tends to increase again up to 30% by harvest (Fig. 6; 55). *B. cinerea* infection of the receptacle area follows a different pattern from that of sepals, commencing at the same level as the sepals but then increases continuously until harvest to reach a relatively higher (35 to 40%) level of colonization compared with that of the sepals (Fig. 6). These infections do not cause any symptoms on fruit in the field and according to the generally accepted definitions are referred to as "latent infections."

Preharvest Factors

Role of environment. Environmental conditions during the growing season can influence the incidence of *B. cinerea* in cold-stored fruit in California. For example, greater postharvest gray mold losses occur when cool and wet spring conditions prevail during and after full bloom, even though no evidence of *B. cinerea* causing disease in the field in the fruit has been

Fig. 10. A well-developed lesion with profuse sporulation of *Botrytis cinerea* after prolonged leaf wetness (New Zealand)

Fig. 11. Infection of picking wound by *Botrytis cinerea* and abundant sporulation (New Zealand).

found (78). Environmental factors in New Zealand, such as rain at or near harvest, were considered by Brook (8) to be responsible for much of the variation between seasons and between regions.

Role of flower infection. Sommer et al. (77,78) suggested that floral infection and gray mold were linked. Inoculation experiments at full bloom in 1997 resulted in 0.6 to 2% and 1.2 to 3% gray mold after 3-month and 5-month cold storage, respectively, while the water-inoculated controls had no gray mold (T. J. Michailides, *unpublished*). However, the role of flower infection in the development of gray mold of California kiwifruit in cold storage needs further investigation.

In New Zealand, the role of *B. cinerea* at flowering and gray mold in cold storage has been the subject of some debate since Beever et al. (5) showed that *B. cinerea* applications to flowers resulted in a significant increase in gray mold incidence. This increase could occur by several mechanisms. The infected flowers may produce externally contaminated fruitlets, and the fungal propagules may survive on the fruit surface during the season. Alter-

natively, the flowers may provide inoculum for infection of other sites in the orchard, which later sporulate and contaminate the fruit nearer harvest. Research to date supports both possibilities. Studies by Elmer et al. (24) and subsequent studies (27, 29,30) showed that accumulation of *B. cinerea* propagules on the fruit surfaces can occur early in the growing season as a result of *B. cinerea* infections of senescing floral tissues. On developing fruitlets sampled at petal fall, fruitlets with *B. cinerea*-infected petals attached had 2.8×10^5 spores per fruit compared with fruitlets with apparently healthy petals attached (mean = 5.1×10^3 spores per fruit). There is evidence that long-term survival of *B. cinerea* on pear fruit (79) and kiwifruit (81) surfaces in the field occurs. Therefore, given the dense nature of the kiwifruit canopy, it is likely that *B. cinerea* inoculum could survive for long periods of time on the hairy external fruit surface, where *B. cinerea* spores would be protected from environmental extremes. Confirmation that a *B. cinerea* population could survive for up to 16 weeks of exposure to field conditions on the kiwifruit fruit surface was

recently reported (81). In separate studies, application of a *B. cinerea* population to fruitlets at petal fall to simulate high levels of external fruit contamination early in the growing season resulted in consistently high levels of external fruit contamination at harvest compared with the control fruit (Table 2). It was proposed that the combination of surviving *B. cinerea* inoculum and pollen on the external fruit surface could act together to enhance picking wound infections when fruit handling at harvest results in both being deposited onto the fresh picking wound (K. Sharrock, *personal communication*).

***B. cinerea* and invertebrate interactions in kiwifruit.** In California, the brown garden snail (*Helix aspersa* Müller) (Fig. 12A) consumes sepal tissue around the receptacle area of the stem end (Fig. 12B), resulting in significantly more gray mold compared with fruit without snail damage (55). In addition, slime secreted by garden snails (Fig. 12C) stimulated the germination of *B. cinerea* conidia. Fruit with snail damage prior to harvest developed six times greater incidence of gray mold after 5 months of cold storage than did fruit without snail damage. To prevent snails from climbing onto the vines, growers attach to the vine trunk an apron made of a copper band (Fig. 12D).

In New Zealand, adults of the flower thrips (*Thrips obscuratus* (Crawford)) often occur in large numbers on both male and female flowers of kiwifruit (58) and act as vectors of *B. cinerea* (31; Table 3; Fig. 12E). A highly ($P < 0.01$) significant thrips $\times$ *B. cinerea* interaction indicated that the presence of thrips infestation, in association with the presence of *B. cinerea* inoculum, would contribute to an increase

Table 2. Number of *Botrytis cinerea* CFU per fruit after inoculation at petal fall, midfruit, and prior to harvest (Nelson region, New Zealand, 1994) (71)

Spores per fruitlet (no.)	Growth stage		
	Petal fall 10 December	Midfruit 15 January	Mature fruit 8 April
Control (0)	3.7×10^{2z}	4.2×10^3	3.3×10^3
2×10^3	1.9×10^3	8.4×10^3	7.7×10^3
2×10^5	3.5×10^4	2.4×10^4	1.3×10^4

^z Statistical analysis was performed on the natural log transformed values.

Fig. 12. Vectors of *Botrytis cinerea* in kiwifruit. (A) Common brown garden snail (*Helix aspersa*) suspended on kiwifruit; (B) upper row damaged by eating of sepals around fruit peduncle, lower row undamaged; (C) slime deposited by snail (California); (D) copper band apron attached to trunk to prevent snails from climbing onto vine; (E) scanning electron micrograph of a thrips (*Thrips obscuratus*) with conidia of *B. cinerea* attached to its surface; (F) honeybee (*Apis mellifera*) visiting staminate kiwifruit flower (New Zealand).

in *B. cinerea* inoculum on the fruit surface, thereby placing fruit samples at risk of gray mold (32).

Honey bees in New Zealand are by far the most important pollinators of kiwifruit, and growers place up to eight hives per hectare to ensure adequate pollination (Fig. 12F) (9). One study found that up to 87% of honey bees foraging on kiwifruit flowers carried viable *B. cinerea* propagules (71). When flowers were artificially contaminated with spores of *B. cinerea*, foraging honey bees picked up 1.4×10^4 *B. cinerea* CFU per bee and transferred *B. cinerea* inoculum to all visited flowers, thereby facilitating rapid and broad spread of the fungus. There have been no studies examining the role of vectors in Californian kiwifruit vineyards.

Some researchers in New Zealand have argued that flower infection is not important since *B. cinerea* epidemics usually commence about 6 weeks prior to harvest and are associated with a buildup of necrotic and senescent leaf tissues in the canopy at that time (51). However, the evidence gathered to date suggests that a buildup of *B. cinerea* on senescing flowers is important epidemiologically (27,29,30). Ignoring the flower-infection phase of the disease cycle early in the season may underestimate the inoculum load on the fruit surface at harvest and therefore the potential for contamination of picking wounds and subsequent gray mold development.

Type and availability of necrotic tissues. Hoyte et al. (41) identified four different types of necrotic leaf tissues in kiwifruit canopies: necrotic, senescent, dead, and those with red-brown lesions. *B. cinerea* colonization was determined by incubating these tissues in high-humidity chambers. There were greater numbers of dead leaves in a dense (mean number of leaves per $m^2 = 216$) canopy, and these contributed to a greater proportion of potential spore production compared with a heavily pruned kiwifruit canopy (mean number of leaves per $m^2 = 133$). Subsequent studies by Pak and Manning (60) during the preharvest period in 18 orchards showed a clear association between the number of necrotic leaves per square meter of canopy and the area under the disease progress curve. In a separate study, the size of *B. cinerea* populations in an experimental orchard was manipulated by artificial inoculation of green leaves (high inoculum potential) or by complete removal of all known *B. cinerea* inoculum sources in the canopy (low inoculum potential). There were significantly ($P < 0.05$) fewer viable *B. cinerea* propagules on the fruit surface at harvest (27,30) and significantly ($P < 0.05$) less gray mold in low-inoculum plots compared with high-inoculum plots (Table 4). These findings validated the practices of some kiwifruit growers who consider removal of necrotic tissues in canopies an important part of gray mold management.

Canopy management. Findings by several New Zealand researchers (40,41,60,75) of significant positive correlations between canopy density, extent of necrosis, and gray mold incidence in cold storage led to recommendations that emphasize adequate summer pruning to create more open vine canopies. This has resulted in a reduction of necrotic and senescent tissues and improved vine ventilation. Canopy and shelter management in New Zealand are now regarded as the most important strategies for increasing light penetration and improving vine ventilation, thereby reducing *B. cinerea* buildup prior to harvest.

External contamination of the fruit surface prior to harvest. In New Zealand, infection of fruit is initiated during harvest, grading, and packing by direct contamination of the stem wound made when fruit is snapped from its pedicel (61). Evidence of a link between *B. cinerea* inoculum on the fruit surface at harvest and gray mold was

found in a survey of orchards in 1992 (24). Fruit from vines immediately adjacent to male vines had significantly more viable *B. cinerea* propagules on the external fruit surfaces at petal fall compared with fruit adjacent to other female vines (27), thereby potentially placing them at greater risk of gray mold. There were significant ($P < 0.05$) correlations between TPSP and the number of viable *B. cinerea* propagules on the fruit surface at harvest in experimental research blocks. When fruit at harvest were sampled adjacent to inoculum sources (e.g., green leaves with necrosis, and senescent and necrotic leaves), there was a significantly greater number of *B. cinerea* propagules contaminating the fruit surface compared with fruit sampled at least 1 m away from an inoculum source (27). A significantly greater number of *B. cinerea* propagules contaminated the fruit surface at harvest from plots with high compared with low inoculum potential (24). This

Table 3. Effects of *Thrips obscuratus* and treatments on contamination of kiwifruit floral parts by *Botrytis cinerea* at complete petal fall (Nelson region, 1991) (32)

Treatment	Petal infection ^w	Mean number of <i>B. cinerea</i> propagules per fruit ^x	Fruit with gray mold symptoms
No thrips ^y	21.3	5,848	2
Control	26.4	7,886	1
<i>B. cinerea</i> only	36.1	11,619	1
Thrips only	36.6	17,962	2
Thrips and <i>B. cinerea</i>	78.8	68,190	7
LSD ($P = 0.05$)	8.4	...	
Orthogonal contrasts ^z			
<i>B. cinerea</i>	**	**	
Thrips	**	**	
Interaction	**	**	

^w Incidence (%) of *B. cinerea* sporulation after 6 to 7 days of incubation at 10°C in moist chambers.

^x Calculated from mean number of *B. cinerea* colonies per plate (43) using a suspension–dilution method. ANOVA originally performed on the square roots of colony numbers per plate.

^y The no thrips treatment involved insecticide application to kill any thrips present in flowers (<1 per flower) from natural infestation. The control treatment involved enclosing flowers in bags for the same duration as other treatments.

^z Main effect of *B. cinerea* inoculation and thrips infestation and the interactions using orthogonal contrasts based on the binomial distribution. ** = significant at $P < 0.01$.

Table 4. Effect of *Botrytis cinerea* population size in the canopy on number of viable *B. cinerea* propagules on the fruit surface at harvest and gray mold incidence (Orchard M, Nelson region, 1994) (27)

Potential population size in canopy	Harvest and postharvest measurements		
	TPSP ^w per m^2 canopy	Number of viable <i>B. cinerea</i> propagules per fruit ^x	Gray mold (%) ^y
Low	1×10^2 a ^z	408 a	2.4 a
High	8.4×10^6 b	2,864 b	11.1 b
Control (untreated)	6.3×10^5 b	4,188 b	5.5 b

^w TPSP = total potential spore production based on incubation tests.

^x Calculated from mean number of *B. cinerea* colonies per plate on a *Botrytis* selective medium (43) using a suspension–dilution method.

^y Visually assessed after 16 weeks in cold storage at 0°C.

^z Statistical analysis was performed after transforming the data to natural log values; means, representing back-transformed values, within a column with a common letter do not differ significantly ($P < 0.05$, LSD).

finding was supported by later studies examining the relationship between canopy necrosis, *B. cinerea* in necrotic tissue, and external fruit contamination (64). Detailed studies with a carbendazim-resistant isolate of *B. cinerea* (BC48) found highly significant positive correlations ($r = 0.87$; $P < 0.001$) between the number of viable BC48 propagules on the fruit surface at harvest and the number of BC48 propagules contaminating the picking wound and subsequent gray mold caused by BC48 in cold storage (30; $r = 0.83$, $P < 0.001$). These and other studies have allowed us to construct a likely sequence of events that leads to picking wound contamination in New Zealand (Fig. 13).

A similar relationship between proximity to male vines and gray mold was identified in California by Morgan and Michailides (57). Gray mold in kiwifruit

harvested from vines next to male vines whose flowers were removed had significantly lower (5%) gray mold incidence in comparison with the incidence (12%) of gray mold in fruit from vines next to male vines whose flowers were not removed (56).

Fruit maturity at harvest. Commercial and research experience in New Zealand has shown that gray mold incidence tends to decline as fruit maturity, measured as soluble solids content, increased (68,69,84,85). The precise mechanism for this reduction in susceptibility is not known, but some isolates of *B. cinerea* have been found with reduced growth rates on media amended with an osmotic regulator (3). This indicated an abnormal sensitivity to high osmotic pressures, as previously reported by Beever (3). As kiwifruit become more mature on the vine, an in-

crease in osmotic pressure may slow *B. cinerea* growth and allow for host defense mechanisms to become activated.

Postharvest Factors

Curing of kiwifruit after harvest and before cold storage. When fruit are held at ambient temperatures between harvest and cold storage, for up to 4 days, there is a marked reduction in gray mold incidence and no adverse affect on fruit quality (2,4,35,36,63,65). More detailed studies have confirmed these findings but identified treatment variability, suggesting that this may be due to inadequate relative humidity (RH) control during the curing process (48). Low and medium humidity during the curing period did not reduce gray mold, and in some cases gray mold increased. There was usually a marked decrease in infection in fruit exposed to >95% humidity during curing (48), a finding that was consistent with studies in Italy (42).

High activities of pathogenesis-related enzymes in fruit on the vine suggested that their expression was governed by environmental or maturity factors rather than by postharvest *B. cinerea* infection. The change occurring in the early postharvest period appeared to be more related to the early postharvest decline in susceptibility to infection (53). There are still many unanswered questions with regard to the underlying biological mechanism(s) involved in the curing process, but it is now standard practice at New Zealand packing-houses to hold fruit in wooden bins in a suitable containment facility for a minimum of 48 h to reduce the potential for gray mold development.

Electron microscopic studies showed that there was little germination of *B. cinerea* spores on the picking wounds of cured kiwifruit compared with those on uncured fruit (36). Similar studies found that germ tubes of spores placed on stem scars of boiled kiwifruit (to inactivate physiological defense mechanisms) grew faster than those from spores on untreated fruit. The cellular constituents of the spores on untreated fruit showed evidence of organelle deterioration indicative of the presence of antifungal materials (48). The effectiveness of curing seems to depend on pathogenesis-related (PR) proteins that are produced during curing in kiwifruit (84,86). In cured fruit, more suberin and phenolics develop close to the stem wound, and the quantities of phenylalanine ammonia-lyase (PAL) and polyphenoloxidase (PPO) and endogenous phenolics are about 10 times greater in cured than in noncured fruit (42).

In California, curing as a postharvest strategy to reduce gray mold has been investigated for the last 5 years (16). A 48-h delayed cooling at 15°C and high air velocity (ethylene free) inhibited decay of wound-inoculated kiwifruit and slowed naturally occurring gray mold (16). After

Fig. 13. Sequence of events leading to infection of the kiwifruit picking wound by *Botrytis cinerea* in New Zealand vineyards. Solid lines represent strong relationships between the variables. Dotted lines represent relationships that are not clearly defined.

such treatment and a 4-month cold storage, fruit quality attributes (fruit firmness, soluble solids concentration, and fruit shrivel) did not change significantly. The best treatment for reduction of gray mold was 48 h delayed cooling at 15°C, high (2 m/s) air velocity, and 95% relative humidity (16).

Cold storage conditions. Fruit rapidly cooled to 0°C (forced-air cooling) developed more gray mold during cold storage than those passively cooled, a pattern that was not affected by the initial holding temperature (46). The formation of condensation at the picking wound as fruit at field temperatures were cooled rapidly is believed to be the factor responsible for this difference in New Zealand.

In California, it is believed that gray mold develops in cold storage as kiwifruit start ripening, and emphasis therefore is placed on avoiding exposure of fruit to ethylene gas to prevent rapid fruit ripening and softening (76,77). The use of controlled atmospheres (CA) of 2.5% oxygen and 5% CO₂ slow ripening considerably. Since exhausts of forklift trucks with inter-

nal combustion engines are common sources of ethylene, only electric forklifts are used in kiwifruit packing facilities. Fruit infected by *B. cinerea* may also produce small amounts of ethylene in cold storage, which may lead to premature softening and reduced storage life of healthy fruit within the same tray (59,61,69). When increasing numbers of *B. cinerea*-decayed fruit were enclosed among healthy kiwifruit, gray mold incidence of healthy fruit increased significantly after 45 days in cold storage (56).

Disease Monitoring and Prediction

In California. In order to develop more efficient methods for controlling gray mold, it is necessary to understand the relationship between *B. cinerea* latent infections in the field and the incidence of gray mold in storage. Growers routinely apply vinclozolin during bloom and preharvest, regardless of anticipated levels of gray mold. When kiwifruit are removed from storage and are ready to be sent to market, shippers do not have a measure of disease risk and therefore cannot identify which fruit lines need to be sold first and which to keep for longer storage. An indirect criterion used by shippers is the historical background of fields in relationship to gray mold. Sommer and Fortlage (76) and Sommer et al. (77) showed that fruit from plots having the highest levels of blossom infection also had the highest fruit rot after 6.5 months of storage. Furthermore, fruit that had low levels of sepal or receptacle (stem end) colonization by *B. cinerea* also had low incidence of postharvest gray mold kept in storage for up to 5 months (54).

Botrytis monitoring (BOTMON) involves collection of fruit samples and analysis of collected samples (Fig. 14). One month before harvest is considered the best time for sampling fruit for *B. cinerea* monitoring and gray mold prediction, since

the correlation coefficients of *B. cinerea* colonization of sepals and receptacles and gray mold in cold storage is high ($r = 0.92$ to 0.98). After obtaining the results, growers can use Table 5 to predict the levels of gray mold in cold storage for a specific lot of fruit. In California, more and more kiwifruit growers have been using BOTMON every year to make decisions on the need for preharvest vinclozolin spray(s). Similarly, packinghouse operators and shippers now have the necessary criteria to decide on the need for fruit sorting and repacking (Fig. 2D) to minimize secondary spread of the disease in storage and to time the marketing of these fruit. By using BOTMON, growers can possibly (i) send to market first the fruit expected to have higher incidence of gray mold and keep in storage longer those lots expected to have lower incidence of gray mold; (ii) spray with vinclozolin only those fields expected to produce fruit with high potential for disease; (iii) reduce the number of fungicide applications in fields with medium disease potential; or (iv) not spray at all in fields with low or negligible disease potential.

In New Zealand. The potential size of *B. cinerea* populations in the canopy might be useful as a predictive tool for disease management (51,60). A system was developed based on sampling disks of necrotic leaf tissue from kiwifruit canopies 6 to 8 weeks prior to harvest. Incubation of leaf disks in humid chambers for 24 h is used to determine the incidence of *B. cinerea* in the canopy, and blocks are assigned a disease risk rating (low, medium, and high). Since no preharvest fungicide is permissible in New Zealand, blocks that are identified as medium or high risk are then given priority for packing, transit, and off-shore sale to minimize the expression of gray mold. It may be argued that there is a more direct link between spores present on the fruit surface and gray mold than between canopy spore production and disease. The relationship between *B. cinerea* in the leaf

Fig. 14. (A) Schematic of kiwifruit as dissected for plating of sepals and receptacles to determine colonization by *Botrytis cinerea*; (B) five sepals plated onto acidified potato dextrose; (C) plates with sepals (perimeter) and stem ends (center) showing well-developed *B. cinerea* colonies after 6 days at 7°C and 3 more days at 23°C incubation.

Table 5. Categories of kiwifruit sepal and receptacle colonization by *Botrytis cinerea* 4 months after fruit set and vineyards with fruit in low, moderate, and high levels of postharvest gray mold decay after 5 months in storage (-0.5°C and ≤ 8 ng of ethylene per ml) during 1993 and 1994 in California

Sampled plant part	Colonization level	Colonization (%) ^z	No. of vineyards with fruit showing different levels of gray mold decay ^y		
			Low (<2%)	Moderate (2 to 6%)	High (>6%)
Sepals	Low	0 to 15	6 2	0 2	0 0
	Medium	16 to 50	0 0	3 3	0 0
	High	>50	0 0	0 0	0 1
Receptacles	Low	0 to 15	4 1	0 1	0 0
	Medium	16 to 50	2 1	2 3	0 0
	High	>50	0 0	0 1	1 1

^y First column represents nine vineyards in 1993, and second column represents eight vineyards in 1994.

^z Percentage of colonization was determined by plating >300 sepals and 60 receptacles per sampling in each orchard.

canopy and on the fruit surface was found to be highly correlated with gray mold in cold storage, and Pennycook et al. (64) suggested that either the leaf disk prediction system of canopy inoculum or measurements of external fruit contamination would be satisfactory for prediction of gray mold in New Zealand. The simplicity of the leaf disk system has led to this tool being used throughout the New Zealand kiwifruit industry. Measuring external fruit contamination is also used for research purposes since it is a direct measure of inoculum load and therefore potential for picking wound contamination.

Old and New Technologies in Disease Control

Chemical control. In California, Sommer et al. (78) and Suadi-Hasbun (80) demonstrated that two sprays of vinclozolin at bloom and two more preharvest sprays were the most effective treatments for controlling gray mold in storage. Michailides and Morgan (54) showed that one application of vinclozolin before harvest reduced significantly the incidence of gray mold in a 1991 trial but not in 1992. However, when disease incidence was 3% or less, one bloom and/or bloom and preharvest, or only preharvest sprays did not significantly reduce gray mold. Sprays 1 week, or 2 and 1 weeks, before harvest reduced significantly the total incidence of gray mold decay in vineyards with high disease risk (Table 6). But these sprays

were not effective in vineyards where disease levels were less than 2.0% (Table 6). Since the development of the kiwifruit BOTMON system, a number of California growers send immature fruit samples to private laboratories for BOTMON analyses. After paying a nominal fee for each analysis, a grower can have the results within 10 days after submission of a sample. After receiving the results of a sample from his field as percentage of sepal or receptacle colonization by *B. cinerea*, a grower then uses Table 5 to predict expected levels of gray mold decay in storage and makes a decision on preharvest vinclozolin sprays (54). In a similar way, a processor can use the same results to make decisions on marketing of fruit.

Up to four applications of vinclozolin, a dicarboximide fungicide, in Californian vineyards may increase the risk of resistant strains of *B. cinerea* emerging. Resistance to dicarboximides has been detected in kiwifruit *B. cinerea* populations in Europe (49), in New Zealand kiwifruit orchards (50), and California (P. A. G. Elmer and T. J. Michailides, unpublished data). The ease with which *B. cinerea* resistance can be selected in populations, combined with environmental issues associated with pesticides and ever-increasing public concern over pesticide residues in food, has prompted research on alternatives to pesticides or, at the least, strategies to reduce pesticide use in kiwifruit vineyards. In California, when conditions are not conducive to the disease, gray mold control can

be achieved with fewer fungicide applications. However, two sprays may be sufficient when one is applied at full bloom and the other at 7 to 10 days before harvest. Good coverage is needed for the fungicide sprays to be successful. In vineyards on a T-bar system, good coverage is particularly difficult to achieve. Chemical control should be accompanied by good cultural and careful storage and handling practices for the control of gray mold in cold storage.

In New Zealand, dicarboximide fungicides reduced the incidence of gray mold when applied as a preharvest spray (61). However, the mechanism by which this reduction occurred is not known, since the likely infection site (picking wound) cannot be protected because it is not directly accessible at the time of preharvest spraying. The widespread emergence of dicarboximide resistance in New Zealand kiwifruit orchards (50) and the development of an industry-wide integrated pest and disease management system (KiwiGreen) has meant that no preharvest dicarboximide fungicides are applied to kiwifruit in New Zealand. However, iprodione and benomyl are the only two fungicides allowed in the KiwiGreen system for control of *Sclerotinia* flower and fruit rot, but only if applied at bloom. Control of postharvest gray mold is now aimed at reducing the size of preharvest populations of *B. cinerea* in vines. Since preharvest fungicides are not permitted, disease management, as described earlier, involves removal of internal shelter belts and extensive summer pruning to improve ventilation. The severity of summer pruning required to achieve open canopies has caused some kiwifruit physiologists in New Zealand to question whether the vine can sustain the removal of large quantities of leaf material without negative consequences for fruit and vine development.

New Technologies

Organic production. Packinghouses in New Zealand have repeatedly reported that organic kiwifruit have a lower incidence of gray mold in cold storage compared with fruit from conventional KiwiGreen systems. A recent study found that even after one growing season, so-called transitional organic blocks had significantly ($P < 0.05$) less gray mold in cold storage than blocks that received the conventional KiwiGreen program (P. A. G. Elmer, M. Spiers, and R. A. Hill, unpublished; Table 7). The biological basis for this apparent difference is not fully understood since canopy density and area of necrosis were not significantly ($P < 0.05$) different between the two systems. In contrast, both incidence and size of *B. cinerea* populations in the kiwifruit canopies were significantly different ($P < 0.05$; Table 7). The mechanism(s) responsible for these differences is currently under investigation.

Table 6. Effect of vinclozolin sprays in reducing gray mold decay of kiwifruit caused by *Botrytis cinerea* in four vineyards with different levels of expected disease in 1994 in California (54)

Vineyard ^v (date of harvest)	Treatment ^w	Time of applic. (weeks before harvest) ^x	Incidence of gray mold decay (%) ^y	
			3 months	5 months
1 (San Luis Obispo Co.) 7 November	Untreated	...	5.6 a ^z	9.1 a
	Vinclozolin	2	4.4 a	6.5 a
	Vinclozolin	1	2.4 b	6.3 a
	Vinclozolin	2 and 1	1.2 b	1.7 b
2 (Kern County) 13 October	Untreated	...	1.2 a	1.9 a
	Vinclozolin	2	0.4 a	0.7 a
	Vinclozolin	1	0.1 a	0.8 a
	Vinclozolin	2 and 1	0.6 a	1.3 a
3 (Kern County) 13 October	Untreated	...	0.7 a	1.3 a
	Vinclozolin	2	0.0 a	0.2 a
	Vinclozolin	1	0.0 a	0.5 a
	Vinclozolin	2 and 1	0.3 a	0.8 a
4 (Butte County) 18 October	Untreated	...	0.5 a	1.0 a
	Vinclozolin	2	0.1 a	0.2 a
	Vinclozolin	1	0.1 a	1.0 a
	Vinclozolin	2 and 1	0.0 a	0.1 a

^v Orchards were selected based on both previous history of levels of gray mold decay and on isolations of *B. cinerea* in 1993 and 1994.

^w Vinclozolin was applied at 1.2 g a.i./liter.

^x Time of application was selected based on the time of commercial harvest.

^y Fruit was stored in a commercial controlled-atmosphere cold storage (-0.5°C and 8 ng of ethylene per ml) for 5 months.

^z Numbers followed by different letters are significantly different using Fisher's protected LSD at $P < 0.05$.

Biological control. Alternative disease control strategies such as biological control show great promise, since this method of disease suppression has the advantage of greater public acceptance and reduced environmental contamination (21). Biological control of gray mold in kiwifruit commenced in 1990 in both California (21,22) and New Zealand (10,33,38). In California, postharvest dip treatments in suspensions of *Bacillus subtilis* reduced gray mold infections by 78% (21). However, dipping in tap water alone produced reductions similar to postharvest dips in *Bacillus subtilis*. *Cryptococcus laurentii* survived well and multiplied from an initial density of 2.6×10^4 to 4×10^7 CFU per wound in about 10 days at 0.5°C. These high levels of *C. laurentii* may explain why it was more effective, whereas *Bacillus subtilis* declined and did not become well-established in wounds. This study was extended in 1990-91 (21). Three rifampicin-susceptible and one rifampicin-resistant strains of *Bacillus subtilis*, an isolate of *Pseudomonas syringae* pv. *lachrymans*, a *Corynebacterium* sp., *Acremonium breve*, the yeasts *C. laurentii* and *C. flavus* (Isolates 89-160 and 89-211), and *Exophiala jeanselmei* were evaluated in laboratory and field studies. These antagonists reduced germination of conidia, inhibited mycelial growth of *B. cinerea*, and reduced gray mold incidence by 14 to 68%. Disease control was variable with dicarboximide fungicides, since vinclozolin reduced disease by 100% and iprodione by only 23% (21).

Three selected antagonists were applied in three kiwifruit vineyards at bloom and preharvest or used as a postharvest treatment. A yeast antagonist survived well on the kiwifruit in the field and also in storage. Despite low levels of disease in untreated fruit, field application of *Bacillus subtilis* and *C. laurentii* as a mixture re-

duced ($P = 0.10$) incidence of gray mold storage decay only in one field trial. However, sprays of vinclozolin at bloom or preharvest were the most effective treatments for reducing gray mold incidence (21).

Significant suppression of gray mold incidence was reported in screening tests in New Zealand in 1990 using isolates of *Trichoderma viride* and *Cladosporium cladosporioides* (38). In separate screening experiments, Franicevic (33) identified *Epicoecum purpurascens* and a *Pseudomonas* sp. as effective antagonists. Over 120 yeast isolates were tested by Cheah and Hunt (10) on kiwifruit picking wounds, and two isolates of *Kluyveromyces* spp. reduced gray mold from 33 to 6%. More recently, an additive effect against gray mold of kiwifruit was reported when a yeast biocontrol agent was combined with fruit curing (15).

The kiwifruit industry in New Zealand does not allow postharvest treatments to export fruit at present, so biological control emphasis has shifted to preharvest suppression of *B. cinerea*. The first practical demonstration that a biological control agent could reduce *B. cinerea* colonization of senescent floral tissues and contamination of fruit surfaces was reported by Pyke et al. (66) using preharvest applications of an isolate of *T. viride* (R. Hill strain). However, survival studies indicated that this isolate might not persist in the canopy for long periods of time (R. A. Hill, unpublished). A new strategy for *Botrytis* spp. suppression was outlined by Köhl et al. (44), and in collaboration with IPO-DLO Research Institute for Plant Protection, Wageningen, The Netherlands, a new screening program was set up by Elmer et al. (28). Newly selected antagonists were evaluated in 11 repeat field bioassays in two kiwifruit-growing regions of New Zealand. One *Alternaria* sp. isolate, four

isolates of *E. purpurascens*, and three isolates of *Ulocladium* sp. consistently and repeatedly reduced *B. cinerea* spore production by 90 to 100% on necrotic leaf disks preinoculated with a *B. cinerea* suspension (Fig. 15). All these antagonists were more effective than the standard fungicide, iprodione, as shown in a representative field bioassay (Tauranga site, Bay of Plenty Region, NZ) (Fig. 16).

The antifungal metabolite 6-pentyl-2H-pyran-2-one (6PAP). The antifungal activity of the *Trichoderma* metabolite 6PAP was first described in 1983, and its practical potential was outlined by Cutler et al. (18). An isolate of *T. hamatum* from New Zealand was found to be a potent 6PAP producer with potential for application to kiwifruit picking wounds. In a large-scale test in 1994, aliquots of 6PAP were applied to the picking wounds of kiwifruit 51 h after aliquots of *B. cinerea* were applied to the freshly harvested fruit. There was no gray mold in the 6PAP-treated fruit, compared with 75% in the control fruit and 55% in the fungicide-treated fruit (19,20). Residue studies show a rapid loss of 6PAP from the picking wound with no detectable residue in internal tissues even at low temperature storage (65). In 1997, 6PAP was evaluated in California kiwifruit and reduced decay significantly (Table 8). The naturally derived 6PAP extracted from *Trichoderma* spp. is approved by the United States Food and Drug Administration as a food additive, but as a postharvest treatment to kiwifruit it does not yet have international approval.

Experimental control methods. Experimental control methods include hot water dips, application of natural coatings, and UV radiation. Hot water dips may cause fruit injury (12). Application of chitosan, a natural polymer derived from the exoskeletons of crustaceans, prolonged shelf life by reducing weight loss and respiration and inducing host resistance (23), but was not effective in reducing gray mold in kiwifruit stem ends in New Zealand (83). UV irradiation reduced gray

Table 7. Comparison of canopy variables and postharvest gray mold incidence in transitional organic (average of six blocks) and conventional KiwiGreen orchards (average of six blocks) in the Bay of Plenty Region of New Zealand (1997)

Measurements	Orchard system			LSD ($P < 0.05$)
	Conventional KiwiGreen	Transitional organic		
At harvest				
Canopy density ^v	261	231	ns ^w	31.1
Degree of canopy necrosis ^x	1,088	760	ns	336.3
<i>B. cinerea</i> incidence (%) in leaves with necrosis	10	4	* ^y	4.6
<i>B. cinerea</i> potential ^z	84	18	*	54.2
Postharvest				
% cumulative gray mold after 31 weeks	2.1	0.8	*	0.83

^v Number of leaves per m² of canopy based on quadrant counts.

^w Not significant ($P > 0.05$).

^x Determined using methods described by Hoyte et al. (41).

^y Significant at $P < 0.05$.

^z Area of *B. cinerea* conidiophore production in cm² per m² of canopy, after incubation of senescent and necrotic leaf tissues in *B. cinerea* conducive conditions (27).

Fig. 15. Suppression of *Botrytis cinerea* sporulation on a necrotic kiwifruit leaf disk that was first inoculated with the pathogen and then treated with a spore suspension of a selected *Ulocladium* sp. (left disk, note the sparse conidiophore production) or water control (right disk, note profuse conidiophores of *B. cinerea*).

mold (17), but the mechanism for this is not known. Fumigation of fruit with SO₂ reduced gray mold, and complete control was achieved at 1,600 to 3,200 µl/g without any fruit injury (11).

Genetic and induced resistance. Resistance of kiwifruit to *B. cinerea* was recently reviewed by Wurms et al. (85), and to our knowledge there have been no published reports of inherent resistance to *B. cinerea* in other *Actinidia* spp. Stimulation of kiwifruit's own defense mechanisms by pretreatment with elicitors has been reported (70). Kiwifruit leaves treated with salicylic acid and other chemically related derivatives reduced *Sclerotinia sclerotiorum* lesion expansion by 85%, and biochemical assays indicated that this was likely due to induction of the host's own defense mechanisms (70). In a more recent study, significant differences in *S. sclerotiorum* lesion expansion rate on leaves of different species of *Actinidia* were measured (T. Reglinski, *personal communication*). The effect of selected elicitors applied to vines on kiwifruit susceptibility to *B. cinerea* at harvest is currently being investigated.

Industry Difficulties, Prospects, and Future Directions

In the last several years, the California Kiwifruit Commission has had major marketing difficulties because of competition

from other kiwifruit exporting countries such as Chile, Italy, and New Zealand. Although the California Kiwifruit Industry is relatively small (Table 1), its hectareage is steady, and there are no plans for increasing the hectareage in the near future. Research emphasis is now placed on curing to reduce Botrytis gray mold, correct storage to reduce water loss, nondestructive sampling for determining maturity, and disease monitoring and prediction. After the development of the BOTMON prediction method, about 5% of growers have samples of immature fruit analyzed yearly and make spray, storage, and marketing decisions based on the results of the BOTMON analyses.

In New Zealand, gray mold management is based upon summer pruning to create more open canopies, preharvest prediction to identify at risk blocks, and postharvest curing. Arguably, the *Botrytis* management program based on these nonchemical strategies has been an industry-wide success, since losses due to gray mold have been substantially reduced compared with the losses experienced prior to 1996. Consequently, the perception in the industry is that gray mold is no longer a problem, and that funds should be spent on other research areas. This may change in the near future if conditions conducive for *B. cinerea* epidemics, absent for the last four seasons, return to kiwifruit-growing regions in

New Zealand. Evidence also suggests that the new smooth-skinned cultivar (Hort 16A) being planted by kiwifruit growers is less prone to gray mold, but the factors responsible for this are only just being understood. Field observations suggest that necrotic tissues of Hort 16A do not appear to be as readily colonized by *B. cinerea* in comparison to the Hayward cultivar, a finding that will be further investigated by New Zealand scientists in 2000. Preliminary studies by P. A. G. Elmer and N. B. Pyke (*unpublished*) found that there were substantially lower numbers of viable *B. cinerea* propagules on the smooth-skinned Hort 16A fruit surface compared with the hairy fruit surface of Hayward. This finding would indicate that fewer propagules would be transferred to the picking wound during harvest operations, thereby reducing the risk of gray mold.

Summary and Conclusions

Both Californian and New Zealand kiwifruit industries are based primarily on the same cultivar (Hayward) and have similar problems with regard to gray mold in cold storage. There are, however, major differences in the epidemiology of *B. cinerea* between the two countries. In California, research evidence collected to date shows that colonization of fruit sepals and receptacles by *B. cinerea* results in a latent infection of these tissues (Figs. 4 and 6). As fruit ripen in cold storage, these infections become active and slowly rot the fruit. Therefore, the greater the level of sepal and receptacle infection prior to harvest, the greater the incidence of gray mold in cold storage. Although *B. cinerea* inoculum from the fruit surface may infect picking wounds during harvest operations, as is the case in the New Zealand system, it is believed that the majority of gray mold decay in storage in California is caused by

Fig. 16. Effect of selected antagonistic biological control agents (BCA) and iprodione on the percent area of necrotic kiwifruit leaf disks covered with *Botrytis cinerea* conidiophores. Kiwifruit leaf disks were preinoculated with a *B. cinerea* suspension, suspended overnight in a kiwifruit canopy, and then BCA suspensions were applied. The area of the leaf disk covered with *B. cinerea* conidiophores was estimated visually after exposure of inoculated leaf disks to field conditions for 7 days in another kiwifruit canopy, followed by incubation at 18°C in the dark for 10 days. (BCA 1 and 2 = *Trichoderma* spp.; BCA 3 through 7 = *Alternaria* spp.; BCA 8 through 12 = *E. purpurascens* isolates; and BCA 13 through 17 = *Ulocladium* spp. Values were arcsine transformed prior to ANOVA.

Table 8. Effect of *Cryptococcus laurentii*, *Trichoderma*, and 6PAP on the natural incidence (noninoculated) of gray mold caused by *Botrytis cinerea* on fruit after 2.5 months in controlled atmosphere storage in California during 1997

Treatment ^w	Gray mold (%)
Control	8.2 ab ^x
<i>Trichoderma</i> (Th 1)	11.3 a
<i>Cryptococcus laurentii</i>	3.9 bc
<i>Trichoderma</i> (Th 2) ^y	3.5 bc
6PAP ^z	0.4 c

^w 40 µl of a 1 × 10⁶ spores per ml of each *Trichoderma* or 5 × 10⁵ CFU/ml of *Cryptococcus* was applied on the stem of the fruit.

^x Numbers followed by the same letter are not significantly different according to an LSD at *P* = 0.05.

^y Isolate Th 2 is *T. harzianum*.

^z 5 µl of 6PAP was applied per fruit stem wound.

latent infections of fruit sepals and receptacles (Fig. 6).

In contrast to the findings in California, studies of the main site of infection (1990 to 1993) and epidemiological studies (1991 to 1995) in New Zealand have concluded that *B. cinerea* inoculum from necrotic tissues in the canopy collects on the hairy external surfaces of kiwifruit and that this process begins as early as petal fall on developing fruitlets (Fig. 13). During harvest operations, *B. cinerea* inoculum on the fruit surface is transferred to the picking wounds, and if the fruit is in a susceptible physiological state, fruit infection will occur, causing gray mold (Fig. 13). Studies of the primary infection site using artificial inoculation procedures found that 91% of the gray mold in cold storage could be directly attributed to the picking wound (Fig. 11). Fruit inoculated around the perimeter of the receptacle stem scar exhibited a much lower (5%) incidence of gray mold, suggesting that the majority of the infections are caused by *B. cinerea* contamination of the picking wound. These findings also suggest that receptacle infections that give rise to gray mold can also occur, but that their incidence is low. Receptacle infections early in the season have been detected in the past, but there was no correlation with gray mold in cold storage (P. A. G. Elmer and K. S. H. Boyd-Wilson, unpublished). Low levels of latent infections have been detected in New Zealand kiwifruit by other researchers but do not appear to cause the distinctive gray mold symptoms that begin at the stem end of the fruit during cold storage and transit.

In conclusion, the main site of infection pathway in California and New Zealand appears to differ, and this emphasizes the need for more research. Tests that identified the picking wound as the main entry point in New Zealand could be repeated in California, and similarly, tests that identified sepals and stem ends as the main entry point in California could be repeated in New Zealand. New research tools, such as fluorescent marker genes, have the potential to be inserted into *B. cinerea* isolates and could greatly improve our understanding of infection processes and epidemiology of *B. cinerea* in both California and New Zealand systems.

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