The brown rot fungi of fruit crops (*Monilinia* spp.): III. Important features of disease management

(Review paper)

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Summary In the third part of this review, important features of disease management are summarised for brown rot fungi of fruit crops (*Monilinia fructigena*, *Monilinia laxa*, *Monilinia fructicola* and *Monilia polystroma*). Several methods of brown rot disease management practices were collected and interpreted in five main chapters. In these chapters, details are given about the legislative control measures, the cultural, physical, biological and chemical control methods. Chemical control is divided into two parts: pre-harvest and post-harvest chemical control. In addition, host resistance and fungicide resistance statuses are also included in this part of the review. Finally, future aspects of brown rot disease control are discussed.

Key words: legislative control measures, cultural control, physical control, biological control, host resistance, fungicide resistance, disease warning, disease management

Introduction

There are at least three important phases in the life cycle of brown rot fungi which are strongly related to disease control. The first occurs during blossom, causing blossom blight and twig infection; the second is during fruit swelling and ripening, causing pre-harvest fruit rot, and the third is during storage, causing post-harvest fruit rot.

In most cases, disease control is not performed against *Monilinia fructigena*. This fungus is mainly a wounding pathogen; therefore, its occurrence is highly dependent upon the presence of wounding agents, such as mechanical injury and damage caused by insects and birds (*Moore*, 1950; *Croxall* et al., 1951; *Pauvert* et al., 1969; *Lack*, 1989; *Tobin*, 1989; *Van 't Westeinde*, 2001; *Xu* et al., 2001; *Holb*, 2003a,b, 2004). The most important wounding agents are insects, such as *Cydia pomonella* L. for apples, *Grapholita molesta* Busck for peach and apricot, and *Grapholita funebrana* Treitschke for plum (*Holb*, 2003a, 2004). The successive control of these pests significantly influences the incidence of fruit rot caused by *M. fructigena*. *M. fructigena* damage can be serious during storage in both pome and stone fruits. Most control measures are performed during blossom against *M. laxa* in stone fruits. The most endangered cultivated fruit species are cherries, plums and apricots, in order. *M. fructicola* causes severe yield losses, and is therefore controlled mainly during fruit development and ripening. However, control is also needed during blossom and storage in most stone fruits (*Ogawa & English*, 1991). There are only legislative control measures suggested against *M. polystroma* (*Van Leeuwen* et al., 2002).

Several control measures can be performed in order to minimise the losses caused by the brown rot fungi. Here, five disease control management practices (legislative control measures, cultural control, physical control, biological control and chemical control methods) are discussed.

Legislative measures

M. fructicola is a quarantine organism for Europe. In Europe, it has never been observed in the field so far. *M. fructigena* is a quarantine organism in the USA, although it has already been registered in the field (*Batra*, 1979). However, authorities in the USA state that the pathogen has been eradicated from the country.

M. fructicola has a wide range of hosts that are spread all over Europe, so an increase in fruit losses is expected. Recently, *Van Leeuwen* (2000) provided detailed pest risk analyses for the EU on *M. fructicola*. He concluded that the probability of the introduction of the fungus into EU countries is high. The import of stone fruits and of nursery stocks is the main possible source for introduction of the pathogen. With massive import of stone fruits from countries where the pathogen exists, infected fruits might slip through the inspection process at entry points. A more serious problem is the import of nursery stocks. Once the pathogen on nursery stock enters European countries, it will spread in the nursery sites and easily establish itself in orchards. The consequences of establishment would be that the direct losses by brown rot and the cost of control will increase, and control measures might become less effective because of the

development of fungicide resistance. Moreover, fruit export to regions where the pathogen would not have been established would decrease. In order to prevent the introduction of *M. fructicola*, phytosanitary measures have to be taken. In Europe, the responsible plant protection authorities are working to prevent the entrance of *M. fructicola*. However, it is very difficult to control all the imported goods coming from areas where the pathogen is established. Nevertheless, if the pathogen is introduced into the EU countries in the future, eradication or containment measures will provide the best means of minimising any economic impact.

M. polystroma is known to exist in Japan (*Van Leeuwen* at al., 2002). The occurrence of this fungus is possibly not restricted to Japan; however, it has not yet been reported from any other country. *M. polystroma* has not yet been declared a quarantine organism in any country, but attention should be paid to determine the risk of introduction of the fungus into areas outside Japan. In Europe, the same legislative control measures are suggested against *M. polystroma*, as discussed above, against *M. fructicola* (*Van Leeuwen* et al., 2002).

Cultural control

Brown rot fungi show a great variability in the degree of incidence from year to year and they should not be forgotten even in years with insignificant disease incidence. There are several examples of severe incidences caused by these pathogens that occurred following years with very low incidence. Therefore, the importance of sanitary measures as a way to remove the sources of infection is clear. Primary infections always start by spore development in fungus fructifications on twigs, leaves, fruits, spurs and branches which had become infected in the previous year. Within a short time, numerous *Monilia* fructifications may be formed and they begin a cycle of secondary infection that will continue through the whole season. Thus, the sooner the infected parts are detected and removed, the more efficient sanitary measures can be (*Wormald*, 1954; *Byrde & Willetts*, 1977; *Batra*, 1991).

Cutting out of infected spurs, twigs and branches should be performed, if possible, when the disease is recognised. During winter, when normal pruning is performed, some infected twigs and spurs are not easily recognised, consequently, there is a high probability that they are left behind. Mummified fruits are visible at this time and these should be removed and destroyed, especially in areas where *M. fructicola* is present, to avoid the formation of the sexual stage. Wild hosts near the orchard should be removed and ornamental bushes should be under surveillance to prevent introduction of inoculum from outside.

Insect control should be performed due to the importance of these agents in increasing infections of fruits (*Agrios*, 1997). It is known that covering sweet cherry trees with rain shields made of polyethylene or other waterproof, light-transmitting material prior to harvest to prevent fruit cracking will reduce fruit decay by various fungi. *Borve & Stensvand* (2003) demonstrated that fungicide applications were not needed when cherry fruits were covered during rainy periods from bloom until the end of harvest. They concluded that rain shields can be used both as a supplement and a replacement for fungicide applications to reduce fruit decay in sweet cherry.

Physical control

Several physical methods are used to reduce mainly the post-harvest decay of brown rot fungi, such as anoxia, heat, hydro-cooling, hydrair-cooling, CO, CO_2, UV-light, sub-atmospheric pressure, electrolyzed oxidizing water and hot water brushing treatments (*Table 1*).

Anoxia (oxygen-free circumstances) or heat treatments and their combination were tested by *Bussel* et al. (1969, 1971) and *Sommer & Fortlage* (1970). Their studies showed that freshly harvested spores of *M. fructicola* were unable to germinate in anoxia. When they were incubated at 25 °C in air before exposure to anoxia, young colonies were suppressed by anoxia. They concluded that there was a synergic effect of heat treatment combined with anoxia, when heated conidia (45 °C, 4 minutes, or 50 °C, 30 seconds)

Table 1 Physical control methods against brown rot disease of fruits caused by *Monilinia* spp.

Target organism	Exposure system	Control method	Reference
M. fructicola	*In vitro*, agar plate	anoxia, heat treatment	*Bussel* et al., 1969, 1971; *Sommer & Fortlage*, 1970
M. fructicola	peach fruit	cooling of heated and non-heated fruits	*Smith & Redit*, 1966
M. fructicola, *M. laxa*	peach fruit	hydro-cooling, hydrair-cooling	*Wells & Bennett*, 1976
M. fructicola	agar plate	carbon monoxide	*El-Goorani & Sommer*, 1979
M. fructicola	nectarine fruit	carbon dioxide	*Ahmadi* et al., 1999
M. fructicola	sweet cherry fruit	carbon dioxide	*Tian* et al., 2001
M. fructicola	peach fruit	ultraviolet light	*Stevens* et al., 1996
M. fructicola	peach fruit	ultraviolet light with yeast antagonist	*Stevens* et al., 1997, 1998
M. fructigena	*in vitro*, agar plate	ultraviolet light, heat treatment	*Marquenie* et al., 2002
M. laxa	sweet cherry fruit	sub-atmospheric pressure	*Romanazzi* et al. 2001
M. fructicola	peach fruit	electrolyzed oxidizing water	*Al-Haq* et al., 2001
M. fructicola	peach and nectarine fruits	hot water brushing	*Karabulut* et al., 2002

were held for 24 hours under nitrogenous air before the heat treatment sensitized spores were subjected to anoxia.

It is a common method that harvested fruits are shipped in refrigerated carriers in order to delay ripening and to control decay. Usually fruits are hydro-cooled at 0 to 3°C before shipment to remove field heat. Additionally, hot water treatments can supplement hydro-cooling and refrigerating by reducing decay by post-harvest organisms. Another method for cooling fruits before shipment is hydrair-cooling, when fruits are pre-cooled with air and not with cold water. *Smith & Redit* (1966) and *Wells & Bennett* (1976) demonstrated that hydrair-cooling treatments generally reduced lesion development as effectively as hydro-cooling. Water used for hydro-cooling can be contaminated by spores of post-harvest decay organisms, so it can increase post-harvest decay during storage. Therefore, they suggested that hydrair-cooling could be substituted for hydro-cooling and it might prevent contamination.

Increasing the concentration of carbon monoxide (CO) and carbon dioxide (CO_2) in controlled atmosphere are other options to suppress post-harvest decay caused by brown rot fungi. *El-Goorani & Sommer* (1979) showed that if the atmosphere in the storage was enriched with carbon monoxide, disease caused by *M. fructicola* was successfully suppressed during storage. The authors noted that the use of CO in storage or transit atmospheres requires special safety precautions. Therefore, the highest safe concentration for commercial use is probably less than 10%. Brown rot decay can also be controlled with high CO_2 concentration in the storage atmosphere. *Ahmadi* et al. (1999) demonstrated that the brown rot of the nectarine cv. 'Summer Red' decreased when the air in the storage was enriched with 15% CO_2 at 5 °C. Two years later, *Tian* et al. (2001) showed that growth of *M. fructicola* significantly declined with an increase in CO_2 concentration and by decreasing temperature on sweet cherry fruits both *in vivo* and *in vitro*. CO_2 concentrations of 15–25% resulted in a significant reduction in lesion size and a 30% concentration completely prevented lesion formation of the fungus.

Stevens et al. (1996) have studied the possibilities of using ultraviolet light-C for control of post-harvest rots of peaches, among which *M. fructicola* was included. In these experiments, low doses of UV were used, and they reduced disease incidence by 50 to 90% (when compared to non-radiated control). However, these results were not satisfactory when compared to the effectiveness of pesticides. A promising method seems to be the combination of UV irradiation with the application of a yeast antagonist – *Debaryomyces hansenii* (*Stevens* et al., 1997, 1998). This combination was very effective in reducing storage rot incidence. It seems that UV light is effective in controlling latent infections while the yeast controlled only superficial infections appearing in recent wounds. In a further study, *Marquenie* et al. (2002) used combinations of UV-C light and heat treatments in order to inactivate conidia of *M. fructigena*. They demonstrated that most inactivation was achieved when the heat treatment was preceded by an UV-C irradiation.

Sub-atmospheric pressures can also suppress disease development during storage. *Romanazzi* et al. (2001) demonstrated that on sweet cherries exposed to sub-atmospheric pressure (0.5 atm) for 4 hours the incidence of brown rot (*M. laxa*) was significantly decreased compared to fruits held under normal conditions.

Other methods, such as electrolyzed oxidizing water and hot water brushing, were also evaluated for reducing post-harvest decay of *Monilinia* species. *Al-Haq* et al. (2001) revealed that electrolyzed oxidizing water is an effective surface saniter on ripe peach, but it only delayed disease development of *M. fructicola*. *Karabulut* et al. (2002) used hot water brushing (HWB) against post-harvest rot of *M. fructicola* on peaches and nectarines. If the fruits were inoculated with *M. fructicola* followed by HWB at 55 or 60 °C for 20 seconds, decay inhibition was 70 and 80%, respectively, compared to the control.

Host resistance

As brown rot fungi are facultative saprophytes with a wide range of hosts and great variability, *Batra* (1991) noted that it is impractical to detect cultivars that are resistant to these three species. However, *Byrde & Willetts* (1977) stated that many cultivars of fruit trees have proved to be more or less resistant to one or more brown rot fungi and this feature is directly related to their fruit characteristics. These characteristics may conflict with commercial requirements, particularly in dessert fruits. For example, thick skin is not popular with consumers and high acidity and phenolic content do not result in good flavour. Susceptibility of fruit species and cultivars to brown rot fungi under East European climate conditions was discussed by *Soltész* (1997), therefore, only some examples are mentioned here on this subject (*Table 2*).

In the case of the sweet cherry, fruit rot is more important than blossom or twig blights. Most cultivars are susceptible to brown rot. However, the cracking feature of a fruit is more important for the infection caused by *M. laxa* than cultivar susceptibility. *Ubrizsy* (1965) supposed that on brown rot resistant sweet sherry cultivars, the stigma and ovary of a flower produce an antibiotic-like material which prevents infection. This prevention is lost if the weather is rainy for a long period and wetting of flowers deactivates the antibiotic effect. *Bargioni* (1982) demonstrated that sweet cherry cultivars with thin fruit skins are more susceptible to brown rot than those with thicker fruit skins.

In the case of the sour cherry, blossom and twig blights are the most important symptoms of brown rot decay. There are some sour cherry cultivars with low susceptibility or disease tolerance, such as 'Lativiszkaja Nizkaja', 'Nagy Angol', 'Mocanesti', 'Ljubszkaja', 'Sirpotreb', 'Oblacsinszkaja', 'Cigánymeggy 3', 'Maraska Savena', 'Mettar' and 'Elegija' (*Soltész*, 1997). Moreover, *Apostol* (1990), *Apostol & Véghelyi* (1992) and *Véghelyi* et al., (1996) revealed that

Table 2 Examples of resistant and susceptible fruit cultivars to brown rot caused by *Monilinia* spp.

Fruit	Host resistance	Plant organ	Cultivar	Reference
almond	high susceptibility	blossom, twig	Drake, Jordanolo	*Ogawa* et al., 1985, 1986
almond	moderate susceptibility	blossom, twig	Ne Plus Ultra, Texas	*Ogawa* et al., 1985, 1986
apricot	high susceptibility	fruit	Royal, Bleinheim, Perfection, Derby Royal	*Hesse*, 1938
apricot	tolerant	blossom, twig	Neptun, Mamaia, Silvana, Sulina, Sirena	*Cociu* cit. *Soltész*, 1997
apricot	high susceptibility	blossom, twig	Budapest, Mandulakajszi	*Szabó*, 1997a
apricot	moderate susceptibility	blossom, twig	Ceglédi óriás, Liget óriás, Polonais	*Szabó*, 1997a
apricot	low susceptibility	blossom, twig	Borsi-féle kései rózsa, Piroska, Pannónia, Ceglédi bíborkajszi, Magyar kajszi, Rakovszky	*Szabó*, 1997a
peach	high susceptibility	fruit	Early, Lord Napier, Michigan, Triumph	*Mohácsy* et al., 1963
peach	high susceptibility	fruit	Shipley	*Koroknai*, 1971
peach	moderate susceptibility	fruit	Alexander, Amsden, Champion, Ford, Győztes, Mayflower	*Koroknai*, 1971
peach	low susceptibility	fruit	Canada, Carman, Elberta, J. H. Hale, Incrotio Pieri, Magyar arany duránci	*Koroknai*, 1971
peach	tolerant	blossom	Bolinha	*Feliciano* et al., 1987; *Ogawa & English*, 1991
plum	high susceptibility	blossom	Santa Rosa, Wickson, Imperial, French	*Ogawa & English*, 1991
peach	high susceptibility	fruit	J.H. Hale, Champion	*Soltész*, 1997
plum	high susceptibility	fruit	Bluefre, President, Stanley	*Szabó*, 1997b
plum	moderate susceptibility	fruit	Cacanska najbolja, President	*Szabó*, 1997b
plum	low susceptibility	fruit	Besztercei, Silvia, Tuleu gras	*Szabó*, 1997b
sour cherry	partial resistance	blossom, twig	Csengõdi, Akasztói, Cigánymeggy 59	*Apostol*, 1990; *Apostol & Véghelyi*, 1992; *Véghelyi* et al., 1996
sour cherry	low susceptibility	blossom, twig	Lativiszkaja Nizkaja, Nagy Angol, Mocanesti, Ljubszkaja, Sirpotreb, Oblacsinszkaja, Cigánymeggy 3, Maraska Savena, Mettar, Elegija	*Soltész*, 1997

cvs. 'Csengődi', 'Akasztói' and 'Cigánymeggy 59' were partly resistant to *M. laxa*.

In the case of apricot and peach, both fruit rot and blossom blights caused by *M. fructigena* or *M. laxa* can be important. Susceptibility of cultivars is high if they are late blooming and if the fruit can be injured easily. *Hesse* (1938) demonstrated that apricot cvs. 'Royal', 'Bleinheim', 'Perfection', and 'Derby Royal' were highly susceptible to blossom infection, whereas 'Tilton' was noticeably less susceptible. In the 1980s, the level of cultivar susceptibility was characterized by 9 grades according to *Guerriero & Watkins* (1984). In Romania, several apricot cultivars tolerant to brown rot were bred such as 'Neptun', 'Mamaia', 'Silvana', 'Sulina' and 'Sirena' (*Cociu* cit. *Soltész*, 1997). Recently, *Szabó* (1997a) classified several apricot cultivars into brown rot susceptibility groups. He evaluated that cvs. 'Budapest' and 'Mandulakajszi' are highly, 'Ceglédi óriás', 'Liget óriás' and 'Polonais' are moderately and 'Borsi-féle kései rózsa', 'Piroska', 'Pannónia', 'Ceglédi bíborkajszi', 'Magyar kajszi' and 'Rakovszky' are lowly susceptible to blossom and twig blights caused by *M. laxa*.

In an early Hungarian study, *Mohácsy* et al. (1963) noted that peach cvs. 'Early', 'Lord Napier', 'Michigan' and 'Triumph' were very susceptible to infection caused by brown rot fungi. Some years later, *Koroknai* (1971) demonstrated that peach cv. 'Shipley' was highly susceptible, cvs. 'Alexander', 'Amsden', 'Champion', 'Ford', 'Győztes' and 'Mayflower' were moderately susceptible while 'Canada', 'Carman', 'Elberta', 'J.H. Hale', 'Incrotio Pieri', 'Magyar arany duránci' were lowly susceptible to brown rot. In contrast, *Soltész* (1997) noted that cvs. 'J.H. Hale' and 'Champion' were highly susceptible to infection caused by *Monilinia* spp. Both authors mentioned that most nectarine species are highly susceptible to brown rot and the reason for this is that peels of nectarine species can be injured very easily; therefore, ice and insects can cause multiple wounds on the fruit surface (*Soltész*, 1997). *Adaskaveg* et al., (1991, 1992) noted that thicker fruit skin and higher phenolic content were found in resistant peach genotypes.

In the case of plum, fruit rot is the most important damage, but flower infection can also occur. Such features as vulnerable fruit peeling, long, wet weather periods during fruit maturity and clustering of fruits are the main factors responsible for susceptibility to brown rot infection (*Soltész*,

Table 3 Bacteria, fungi and yeasts as biological control agents against brown rot diseases caused by *Monilinia* spp.

Target organism	Biological control agent	Exposure system	Reference
M. laxa, M. fructigena	*Trichoderma viridae*	*in vitro*, agar plate	*Ale-Agha* et al., 1974
M. laxa	*Aspergillus flavus*, *Epicoccum nigrum*, *Penicillium chrysogenum*, *P. frequentans*, *P. purpurogenum*	peach twigs and flowers, laboratory examination	*Melgarejo* et al., 1985; 1986
M. laxa	*Penicillium frequentans*	*in vitro*, agar plate, laboratory study	*De Cal* et al., 1988; *Melgarejo* et al., 1989; *De Cal & Melgarejo*, 1994; *Larena & Melgarejo*, 1996; *Pascual* et al., 2000; *De Cal* et al., 2002
M. laxa	*Penicillium frequentans*	field study, peach twigs	*De Cal* et al., 1990
M. laxa	*Epicoccum nigrum*	field study, peach twigs	*Madrigal* et al., 1991
M. laxa	*Epicoccum nigrum*	*in vitro*, agar plate, laboratory study	*Madrigal & Melgarejo*, 1994; *Pascual* et al., 1999; *Larena* et al., 2003
M. laxa	*Metschnikowia pulcherrima* (yeast)	peach fruits	*De Curtis* et al., 1996
M. fructicola	*Bacillus subtilis* B-3 strain	post-harvest brown rot of fruits	*Pusey* et al., 1984
M. fructicola	*Bacillus subtilis* B-3 strain	laboratory study	*McKeen* et al., 1986; *Gueldner* et al., 1988
M. fructicola	*Bacillus subtilis* B-3 strain	commercialisation test on stored fruits	*Pusey* et al., 1988
M. fructicola	*Bacillus subtilis* B-192	post-harvest rot of peach and nectarine	*Fan* et al., 2000
M. fructicola	*Pseudomonas corrugate* and *P. capacia*	post-harvest brown rot of fruits	*Smilanick* et al., 1993
M. fructicola	*Aureobasidium pulans*, *Gliocladium roseum*, *Epicoccum nigrum*	cherry blossom	*Wittig* et al., 1997

1997). Recently, *Szabó* (1997b) classified several European plum cultivars into brown rot susceptibility groups. He found that 'Bluefre', 'President' and 'Stanley' are highly, 'Cacanska najbolja' and 'President' are moderately, and 'Besztercei', 'Silvia' and 'Tuleu gras' are lowly susceptible to fruit rot caused by *M. laxa*.

In the case of apple and pear, fruit rot is mainly dependent upon the presence of biotic and abiotic wounding agents as it has been noted previously.

Some of the earlier literature from the USA also mentioned host resistance and susceptibility to brown rot fungi. In the case of *M. laxa*, *Ogawa* et al. (1985) and *Ogawa* et al. (1986) noted that among almond cultivars, 'Drake' and 'Jordanolo' were highly susceptible to blossom infection, and 'Ne Plus Ultra' and 'Texas' were moderately susceptible. Severe blossom infection was uncommon in 'Nonpareil', 'Peerless' and 'Davey'. *Crossa-Raynaud* (1969) evaluated resistance based on the rate of canker development in young branches of apricot and almond cultivars and showed some differences. *Ogawa & English* (1991) demonstrated cultivar differences in various regions in the USA. In California, e.g. plum cvs. 'Santa Rosa', 'Wickson', 'Imperial' and 'French' suffered severe blossom infection by *M. laxa*. In Oregon, Italian prune was susceptible to sporadic blossom infection by both *M. laxa* and *M. fructicola*. In a few nectarine and peach orchards in California, severe blossom blight and fruit rot by *M. laxa* had occurred, but in most orchards only *M. fructicola* was isolated. No peach cultivar has been known to be highly resistant to blossom brown rot caused by *M. fructicola*. Only cv. 'Bolinha' showed moderate resistance (*Feliciano*, et al., 1987; *Ogawa & English*, 1991).

Biological control

Biological control of *Monilinia* spp. might be an alternative method to replace pesticides in the future, especially during storage. No biological method has been developed yet which is as effective as chemical control, but less expensive. Nevertheless, research has been focused on trying to discover alternative methods of disease control. Some studies and their results related to brown rot fungi are presented here (*Table 3*).

One of the first reports on the antagonistic effect between brown rot fungi and other micro-organisms was made in the 1970s. *Ale-Agha* et al. (1974) reported that the heat-killed spores from *Trichoderma viridae* inhibited the mycelial growth of *M. laxa* and *M. fructigena*. In 1980 and 1981, microflora of peach twigs and flowers was assessed. The most frequent genera were *Penicillium*, *Alternaria*, *Aspergillus* and *Cladosporium* spp. (*Melgarejo* et al., 1985). The authors found that five species (*Aspergillus flavus*, *Epicoccum nigrum*, *Penicillium chrysogenum*, *P. frequentans* and *P. purpurogenum*) inhibited the growth of *M. laxa*. These substances were apparently active against spore germination and hyphal growth. In a similar work, *Melgarejo* et al. (1986) studied the potential of *A. flavus*, *E. nigrum*, *P. frequentans* and *P. purpurogenum* for the biocontrol of *M. laxa*. The experiments were conducted in spring and early autumn in the field, in Spain (Zaragoza). In spring, *E. nigrum*, *P. frequentans* and *P. purpurogenum* significantly reduced infection when introduced before inoculation with the pathogen. However, in autumn, only the treatments with *E. nigrum* resulted in a reduction of the *M. laxa* infection. *De Cal* et al. (1988) showed that *Penicillium frequentans* produces antifungal compounds that are active

against *M. laxa*: This production started after 10 days of incubation of *P. frequentans* in potato dextrose broth, and continued for approximately 20 days, when inhibition reached a maximum. Two antibiotic compounds were isolated. They inhibited the germination of spores of *M. laxa* on peach twigs. In further studies, the effects of *P. frequentans* and its antibiotics were studied on production of stromata (*Melgarejo* et al., 1989) and on unmelanized hyphae of *M. laxa* (*De Cal & Melgarejo*, 1994). *De Cal* et al. (1990) tested the antagonist *P. frequentans* alone or in alternation with captan in the field in order to control peach twig blight. Preparation of the antagonist with nutrients gave significant reductions in the severity of disease. Combination of the antagonist with captan resulted in similar control as that provided by the antagonist or captan alone. *Madrigal* et al. (1991) made a similar study with *E. nigrum* on peach tree and they found that the control effect of the antagonist on the disease was variable. The most successful treatment was when *E. nigrum* was used in combination with captan. Further examination of *E. nigrum* showed that the fungus produced an antifungal compound, flavipin, which was toxic to *M. laxa*. *Madrigal & Melgarejo* (1994) applied this compound to spores of *M. laxa* and the level of ATP in the brown rot fungus cells dropped suddenly, which indicated that there was a strong inhibition in the respiration process. Flavipin seemed to affect also the protein synthesis but the mode of action of the compound has not been known yet. The lytic enzyme producing fungus, *P. purpurogenum* was also tested against *M. laxa*. Crude filtrates and crude enzyme preparations of the antagonist cultures produced lysis of the hyphae and spores of *M. laxa* (*Larena & Melgarejo*, 1996). *Pascual* et al., (1999, 2000) investigated the production of *E. nigrum* by substrate fermentation and the accumulation of compatible solutions in *P. frequentans*. *De Cal* et al. (2002) achieved mass conidial production of *P. frequentans* and *Larena* et al., (2003) dried *E. nigrum* conidia for obtaining self-stable biological products against *M. laxa*.

In the case of the bacterial antagonist, *Pusey & Wilson* (1984) reported that the *Bacillus subtilis* B-3 strain successfully controlled post-harvest brown rot caused by *M. fructicola*. The mechanism of the bacterium appeared to involve production of antifungal substances. Under laboratory circumstances, the antifungal substances showed almost complete suppression of the brown rot at 1 mg/ml concentration (*McKeen* et al., 1986). In 1988, the antifungal substances (iturines, antifungal peptides) were isolated by *Gueldner* et al. (1988). In the same year, pilot tests were made for commercial production and application of the *B. subtilis* B-3 strain for post-harvest control of peach brown rot (*Pusey* et al., 1988). A few years later, *Fan* et al. (2000) found a new strain of *B. subtilis* (B-192) against post-harvest brown rot in peach and nectarine. They found that this bacterium strain reduced brown rot infection with infection rates of 20% on peach and 40% on nectarine when fruits were inoculated with *M. fructicola* following the application of the biocontrol agent. Two other antibiotic-producing bacteria (*Pseudomonas corrugate* and *P. capacia*) are also known as biocontrol agents against post-harvest brown rot caused by *M. fructicola*. *Smilanick* et al. (1993) demonstrated in laboratory that both bacteria significantly reduced post-harvest brown rot decay when applied up to 12 hours after inoculation with *M. fructicola*.

Epiphytic fungi were also reported as antagonists of brown rot fungi. *De Curtis* et al., (1996) demonstrated that the epiphytic yeast, *Metschnikowia pulcherrima* was effective in reducing the incidence of *M. laxa* in peaches from 49 to 89% depending on the strain and fruit. *Wittig* et al. (1997) examined the antagonistic effects of three other epiphytic fungi (*Aureobasidium pulans*, *E. nigrum* and *Gliocladium roseum*). All the three fungi showed successful control against *M. fructicola* on blossom of cherry cv. 'Royal Anne' under field conditions.

Chemical control

Pre-harvest chemical control

Sulphur was the first pesticide used against brown rot diseases. Sulphur was applied in some regions every 7 or 14 days from blossom until fruit maturity. These control measures were responsible for a substantial reduction in fruit losses, although the results were not satisfactory.

During the 1950s, protective fungicides were introduced, such as captan and dichloran, with better efficacy than sulphur. Captan was superior to dichloran for brown rot control. Captan, for control of blossom diseases of stone fruit crops, required a minimum of two applications (one at pink bud and the second at full bloom) to provide protection for susceptible blossom tissues. These treatments did not control blossom blight effectively, but even so, farmers used them often during the blossom period. A mixture of the above two active ingredients was also used to control brown rot during post-harvest.

Later, in the 1970s, another group of fungicides appeared with very good efficacy against brown rot – the benzimidazole fungicides. Two applications (one at pink bud stage and at full bloom) provided excellent control of brown rot in almost all regions, due to their curative and protective mode of action. Moreover, *Osirio* et al., (1994) noted that even one application of benomyl at pink bud reduced blossom blight by 92% and the fungicide translocated systemically into the non-exposed internal blossom tissues (pistils and stamens). However, extensive application of benzimidazoles caused fungicide resistance. Benzimidazole resistance was first registered in orchards where benzimidazoles, mainly benomyl, were used against brown rot. *Shabi & Ogawa* (1981) isolated monoascosporic isolates resistant to benomyl from a peach orchard in California. These isolates were obtained from apothecia of *M. fructicola*, in which benomyl-sensitive ascospores were also present. To minimise the problems of resistance, mixtures of benomyl or thiophanate-methyl with captan, sulphur and maneb were sprayed where resistance was not established (*Zehr*, 1982).

In the 1980s, another group of fungicides was registered with the potential to control brown rot – the dicarboximides, in which vinclozolin, iprodione and prochloraz are included. However, after a few years of application, resistance against this fungicide group was also registered (*Zehr*, 1982).

In the 1990s, the use of sterol biosynthesis inhibitor (SBI) fungicides was common in apple orchards due to their activity against apple scab (*Venturia inaequalis*). SBI fungicides are also an important group of antifungal agents used against fruit rot diseases. For triazole, imidazole, pirimidine, and piperazine derivates, the primary mode of action is the inhibition of C14 demethylation (DMI) in sterol biosynthesis (*Siegel*, 1981; *Van den Bossche* et al., 1984). It is presumed that the depletion of functional sterols and the accumulation of sterol intermediates lead to a disruption of membrane functions and to growth inhibition (*Nes*, 1973, *Siegel*, 1981; *Van den Bossche* et al., 1984). *Wilcox* (1990) studied the post-infection and anti-sporulant activities of some SBI fungicides in the control of *Monilinia fructicola* on sour cherry, such as tebuconazole, propiconazole, myclobutanil, fluzilazole, triforine and fenarimol. All of them gave 97 to 100% control when applied 24 hours after inoculation. However, when the period following inoculation was longer, the degree of control was strongly influenced by the concentration of the inoculum and by the fungicide applied. When applications were made 72 hours after inoculation, almost no control was achieved. Of SBI fungicides, tebuconazole and propiconazole showed the best post-infection and anti-sporulant activities.

Careful monitoring of orchards with reduced spray schedules is essential. In this case, when the first symptoms are observed, very effective fungicides should be sprayed against brown rot fungi in the first two applications. If other inoculum sources appear during the season, another application with an effective fungicide should be performed (*Zehr*, 1982). Normally, 2 to 4 applications are performed during blossom. Later during fruit ripening, two or three other applications may be executed depending on the inoculum pressure.

Below, we discuss the specific elements of pre-harvest control of *M. laxa* and *M. fructigena* separately.

Pre-harvest chemical control of M. laxa

Rudolph (1925) developed a protective spray schedule that has proved relatively effective on apricots in California. The trees were sprayed with Bordeaux mixtures when the blossoms were at pink bud stage. Where the disease has been severe, two sprays were advised, one at the tight cluster stage and one at full bloom. These sprays were phytotoxic to the floral parts of the trees. Adequate control of blossom and twig blight of almond and apricot, caused by *M. laxa*, using eradicant fungicides was first achieved by *Wilson* (1942). Wilson successfully directed control efforts toward the reduction of the primary inoculum source by spraying in the dormant season with arsenite compounds. Because of the phytotoxicity of arsenites to almond trees, *Wilson* (1950) and *Ogawa* et al. (1967) tried sodium pentachlorophenate (SPCP) and found that it destroyed sporodochia of *M. laxa*. By the end of the 1970s, monocalcium arsenate was banned and replacement of SPCP was needed because it was hazardous to the applicator. Therefore, new products began to be tested. *Ramsdell & Manji* (1969), *Ramsdell* et al. (1970) and *Ramsdell & Ogawa* (1973a,b) demonstrated that early dormant benomyl sprays markedly reduced the development of *M. laxa* on almond and apricot. *Ramsdell & Ogawa* (1973a) reported that a dormant benomyl spray reduced the number and size of sporodochia of *M. laxa* arisen from blighted almond twigs. Addition of oil to the dormant benomyl spray enhanced sporodochial inhibition by providing longer residual action, although the initial deposit was less. Oil increased the penetration of benomyl into the bark and provided additional activity against the fungus. *Ramsdell & Ogawa* (1973b) evaluated the systemic activity of methyl 2-benzimidazolcarbamate (MBC) and benomyl when they were sprayed before bloom. Pre-bloom sprays of benomyl gave excellent control of *M. laxa* blossom and twig blight of almond. Sprays of 1.4 kg/ha and 2.8 kg/ha bemonyl with or without oil were equally effective. Benomyl + MBC applied with or without oil to branches of covered trees at green tip or pink-bud stages protected all blossom parts at full bloom. The authors also demonstrated that benomyl and MBC had a similar degree of fungitoxicity to *M. laxa* conidia. Benomyl resistant *M. laxa* was detected by *Ogawa* et al. (1984) in apricot orchards. However, its population has not increased (*Michailides* et al., 1986), and the isolates appeared to be less pathogenic than the benomyl sensitive ones collected from severely diseased almond orchards (*Cañez & Ogawa*, 1985). Other materials, such as the SBI and the dicarboximide fungicides, are effective against *M. laxa*. *Latorre & Lolas* (1986) reported on the good effectiveness of several sterol-inhibiting fungicides against *M. laxa* in sweet cherry. Derivatives of SBI and dicarboximide fungicides (e.g. triazole, piperazine, pyrimidine and imidazole) also showed good activity against *M. laxa* in field tests on several stone fruit crops in California (*Ogawa* et al., 1988). *Zhang* et al. (1991) evaluated the sensitivity of several sterol biosynthesis inhibitors to isolates of *M. laxa* and *M. fructigena*. They found that cyproconazole and difenoconazole strongly inhibited the mycelial growth of isolates of both fungi. The high efficacy of cyproconazole and difenoconazole is further underlined by the minimum inhibitory concentration which was more than thirty times lower for these two SBI-fungicides than for myclobutanil and triadimenol. In a similar study, *Osirio* et al. (1994) compared iprodione fungicide and benomyl for *in vitro* inhibition of mycelial growth of *M. laxa* and *M. fructicola*, for suppression of anther infection of almond blossom in the laboratory, and for control of brown rot of blossom and twigs of almond in the field. The fungicide was active against both the benomyl-sensitive and benomyl-resistant isolates of *M. laxa* and *M. fructicola*. In the laboratory studies, they showed that anther infection was suppressed when open blossoms were sprayed with iprodione within 24 hours after

inoculation with a benomyl-sensitive isolate of *M. laxa*. In the field study, applications of iprodione at pink bud (closed blossom) and full bloom (opened blossoms) effectively reduced brown rot twig blight of almond.

In Hungary, in the early 1920s, *Béla Husz* proved the fungicide activity of Bordeaux mixture against *M. laxa* during bloom. Later in the 1950s, good control was achieved with a trichotecin antibiotic suspension sprayed during bloom in sour cherry orchards (*Berend*, 1957). *Paszternák* et al. (1982) suggested fenarimol (Rubigan 12 EC, 0.04%) or mankoceb (Dithane M 45, 0.3%) against *M. laxa* in sour cherry orchards during bloom when 30–40% of the blossoms are open. Moreover, they recommended an additional ftalanil-acid (Nevirol 20 WP, 0.05%) spray to increase the vitality of the stigmata. Another fungicide experiment against *M. laxa* conducted in Hungarian cherry orchards concluded that, depending on weather conditions, cherry trees should be sprayed 2 or 3 times during bloom in order to protect the flowers on the trees (*Schweigert*, 1996). Tebuconazole (Folicur 250 EW, 1 L ha^{-1}), proclorase (Sporgon 50 WP, 0.6 kg ha^{-1}), hexaconazole (Anvil SC, 0.3 L ha^{-1}) and penconazole (Topas 100 EC, 0.5 Lha^{-1}) provided excellent control against brown rot during bloom. Moreover, good fungicide activity was found in commercial cherry orchards by using procimidon (Sumilex 50 WP, 1 kg ha^{-1}), ciproconazole+captan (Atemi C, 1.5 kg ha^{-1}) and iprodione (Rovral 25 FW, 2 kg ha^{-1}) (*Schweigert*, 1996). *Véghelyi* (1996) and *Glits* (2001) suggested a spray with elementary sulphur (3%) or copper sulphate or copper hydroxide (1–1.5%) in early spring during the dormant bud stage. This should be followed with another copper spray at green tip stage. It is important to spray during bloom with captan, benomyl and penconazole fungicides which are not harmful to bees. *Glits* (2001) suggested other systemic fungicides, such as triforine, cyprodinil, miclobutanile and vinclozolin, against brown rot during bloom. According to EU regulations, the use of triforine is banned after 1 May 2004 in Hungary.

Pre-harvest chemical control of M. fructicola

Pre-harvest control of *M. fructicola* focuses on both blossom blight and fruit rot. Fungicides for blossom protection should be applied before rains, when about 5 percent of the blossoms are open, and again at 70 percent bloom (*Ogawa & English*, 1991). In the 1950s, liquid lime sulphur applications were suggested against blossom blight (*Ogawa* et al., 1954). However, authors noted that lime sulphur applications on blossoms may result in severe damage resembling that caused by *M. fructicola*. Eradication of incipient fruit infection on cling peaches following rains during the last three weeks before harvest was shown to be possible worth ground application of liquid lime-sulfur within 37 hours from the beginning of rain (*Ogawa* et al., 1954). In later studies, it was proved that benomyl and thiophanate-methyl gave more effective control than earlier fungicides (*Gilpatrick*, 1973; *Ogawa* et al., 1968; *Ogawa* et al., 1967; *Tate* et al., 1974) and can be applied as early as the pink-bud stage of bloom. Their application at this time protects the anthers from infection.

Szkolnik (1981) tested the protective and after-infection activity of sterol inhibitors and dicarboximides against *M. fructicola*. He concluded that protection against brown rot blossom blight was excellent with a bloom spray to sour cherry in the greenhouse with sterol inhibitors and prochloraz and with benomyl and vinclozolin. A post-infection spray with sterol inhibitors, prochloraz, triadimefon, triforine and iprodione 18 or 24 hours after inoculation gave excellent blight control. *Lade & Christensen* (1971) noted that 30–40 ppm of triarimol provided 90–100% brown rot (*M. fructicola*) control in Michigan and in New York, in the USA. Aircraft applications of the systemic fungicides have provided excellent coverage as well as disease control (*Ogawa* et al., 1972; *Ogawa* et al., 1985). *Van Geluwe* et al. (1981) evaluated the length of protective activity of sterol inhibitors for control of brown rot blossom blight in peach cultivars. They found that mean percentages of infected blossom among cultivars in the 5, 6 and 7 day treatments were 17.5, 33.3 and 51.7, respectively, compared to 69.2% of the untreated control. They concluded that sterol inhibitors provided effective disease control under heavy disease pressure when the fungicide was applied up to 5 days prior to bloom. *Dahmen* et al. (1988) found that ultrastructural damage to cell membranes become apparent in *M. fructicola* when they used sterol inhibitors. Many of the germ tube tips of the fungus ruptured 2–4 hours after initiation of the fungicides. In this study, the effects of sterol inhibitors fenpropimorph, imazalil, flutriafol, triadimenol, propiconazole and penconazole on growth and on cell electrolyte leakage were also compared. Among fungicides, propiconazole and penconazole caused the greatest electrolyte leakage in *M. fructicola*. The authors concluded that direct action on fungal cell membranes of *M. fructicola* may be a second mechanism of action for propiconazole and penconazole. *Osirio* et al. (1994) compared iprodione with benomyl for *in vitro* inhibition of mycelial growth of *M. laxa* and *M. fructicola*, which was demonstrated in the section of 'Pre-harvest chemical control of *M. laxa*'. A few years later, *Northover & Cerkauskas* (1998) examined the effect of several fungicides on brown rot incidences of European plums (*Prunus domestica* 'Stanley'). They found that when five sterol-inhibiting fungicides were applied twice at mid-season to Stanley trees having fruits with a high incidence of latent infection, then only tebuconazole gave temporary suppression of *M. fructicola* in excised immature fruits. In a recent study, the interactions between components of fungicide mixtures were evaluated against *M. fructicola* (*Emery* et al., 2002). Two-way mixtures of commercial formulations of propiconazole with either benomyl, captan, chlorothalonil, cyprodinil or vinclozolin were evaluated *in vitro* for potential synergism in inhibiting *M. fructicola*. Experiments included each active ingredient at low, medium and high concentrations in all possible pair-

wise combinations. The inhibition of the radial growth of two isolates of *M. fructicola* was not significantly different ($P > 0.01$) from that predicted by a simple model of independent action for any of the fungicide-concentration combinations, indicating the absence of synergism between active ingredients. Results were similar when mixtures of propiconazole with benomyl, chlorthalonil or cyprodinil were evaluated on peach fruits treated with fungicide. While fungicide mixtures are useful in delaying the development of fungicide resistance, they are unlikely to be used in practice unless synergistic interactions allow for applications at reduced concentrations. The absence of synergism suggests that little incentive exists for favouring propiconazole-based fungicide mixtures over a rotating schedule of fungicides for control of and resistance management in *M. fructicola*.

Protection of fruits from infection of *M. fructicola* can be achieved only if fungicides are applied before free moisture occurs on the fruit. Aircraft or ground sprays are effective if performed before rains. Repeated ground spray applications are beneficial in sprinkler-irrigated peach orchards but not in prune orchards (*Ogawa & English*, 1991).

Fruits with quiescent infections usually develop rot during the last month before harvest, regardless of the application of protective fungicides. *Manji & Ogawa* (1987) demonstrated that if green cherry fruits showed quiescent infection of *M. fructicola*, spray applications of iprodione, triforine and benomyl reduced the incidence of decayed fruits at harvest.

Post-harvest chemical control

To prevent infections at harvest time, during storage and transport, fruits should be picked and handled with care in order to avoid injuries that favour disease development. Damaged fruit should not be stored. Surveys in the USA indicated that fruits are often free of decay producing organisms when they enter packing houses, but can be contaminated by *M. fructicola* spores when exposed to unsanitary conditions in the packing house (*Smith* et al., 1971). Therefore, some authors suggested several methods to reduce the contamination of fruits before they are transported to the place of storage: first, more frequent cleaning of the hydro-cooler and dump tanks, second, chlorination of the cooling or damp tanks, third, hot water treatment of fruits (52 °C for 1–2 minutes) and fourth, cooling of fruits right before storage in air rather than in water. *Sommer* (1982) gave some additional measures to minimise post-harvest diseases in the storage places. First, fruits should be harvested at optimum maturity. Fruits should be cooled to the lowest temperature that will not damage them. If possible, controlled or modified atmosphere should be applied during storage and transport.

Before storage or during storage, several methods or chemicals can be applied to reduce post-harvest decay of fruits caused by brown rot. The most important ones are listed below.

Fungicides and inorganic salts

Post-harvest application of benomyl effectively controlled brown rot of peaches, nectarines and plums infected by *M. fructicola* (*Ogawa* et al., 1968; *Wells & Gerdts*, 1971; *Wells*, 1972). A study of *Szkolnik* (1981) evaluated the effectiveness of certain fungicides against post-harvest rot caused by *M. fructicola*. Protection of sweet cherry fruits against brown rot with a 30-second post-harvest dip was excellent with sterol inhibitors, prochloraz, fenarimol, triforine and vinclozolin, and fair with captan, triadimefon and iprodione. Fruit dip for 30 seconds 24 hours after inoculation gave excellent after-infection control with sterol inhibitors, prochloraz, fenarimol and triforine, and fair with triadimefon, benomyl and iprodione. However, *Spotts* et al. (1998) examined the effect of the single pre-harvest application of iprodione on brown rot in stored sweet cherry fruits and they found that iprodione at 1.13 kg a.i. ha^{-1} reduced brown rot in stored sweet cherry fruits. However, significantly better control of brown rot was obtained when cherry fruits that received a pre-harvest iprodione application were also treated with a post-harvest dip in a suspension of a yeast species (*Cryptococcus infirma-miniatus*) containing 0.5 to 1.5×10^8 CFU ml^{-1}. In another study, *Northover & Cerkauskas* (1998) examined the effect of several fungicides on brown rot incidences of European plums (*Prunus domestica* cv. 'Stanley') with a high incidence of symptomless latent infections of *M. fructicola*. Fruits were harvested soft-ripe or firm-ripe, surface disinfested in NaOCl, soaked for 4 minutes in fungicide suspensions and incubated for 7–11 days at >95% RH at 20 °C. Using soft-ripe fruits, most fungicides reduced brown rot relative to the water check after 7 days of incubation, with tebuconazole and flusilazole being numerically superior. Using firm-ripe fruits, five sterol-inhibiting fungicides and iprodione reduced brown rot infections after 7 days of incubation, with tebuconazole, flusilazole and myclobutanil being numerically superior.

Conway (1981) used alternative treatments with calcium chloride against post-harvest brown rot caused by *M. fructicola*. He treated harvested cv. 'Redhaven' peaches with a 0, 2, 4, or 6% calcium chloride solution either by dipping, vacuum infiltration or pressure infiltration. Twenty-four hours later, the treated peaches were wounded on two sides and inoculated with a conidial suspension of *M. fructicola*. After storage at 20 °C for 5 days, the rate of decay was assessed. The best treatment was the 4% $CaCl_2$ solution pressure infiltrated resulting in 50% less decay than observed on the non-treated fruits. Moreover, *Biggs* et al. (1997) examined the effects of calcium salts on growth, polygalacturonase (PG) activity, and infection of peach fruits by *M. fructicola*. They found that calcium hydroxide, calcium oxide, calcium silicate, and calcium pyrophosphate reduced growth by approximately 65% on amended potato-dextrose agar (PDA) after 7 days compared to the control. Fungal PG activity was also inhibited by calcium salts. Greatest inhibition of PG was achieved by using calcium

propionate followed by calcium sulfate, tribasic calcium phosphate, calcium gluconate, and calcium succinate. When the inoculum was sprayed on detached fruits, the incidence and severity of brown rot were the lowest on fruits that had been dipped in solutions of calcium propionate or calcium silicate. When the inoculum was applied as a localized drop to wounded fruits that had been dipped in a solution containing 1,200 mg of calcium per litre, brown rot severity was the lowest for fruits treated with calcium oxide and calcium hydroxide. For non-wounded fruits and drop inoculations, calcium hydroxide was the most effective in reducing brown rot incidence, and all salts reduced rot severity similarly (*Biggs* et al., 1997). In the same year, *Margosan* et al. (1997) examined the combination of ethanol and hot water to control post-harvest decay of peaches and nectarines. Spores of *M. fructicola* were immersed in water or 10% ethanol for 1, 2, 4, or 8 min at temperatures of 46 or 50 °C to determine exposure times that would produce 95% lethality. Fruits infected with *M. fructicola* were immersed in hot water alone or hot water with ethanol to control decay. Immersion of fruits in water at 46 or 50 °C for 2.5 minutes reduced the incidence of decayed fruits from 82.8% to 59.3 and 38.8%, respectively Immersion of fruit in 10% ethanol at 46 or 50 °C for 2.5 minutes further reduced decay to 33.8 and 24.5%, respectively. Decay after triforine (1,000 mu g ml^{-1}) treatment was 32.8%. The flesh of ethanol-treated fruits was significantly firmer, with approximately 4.4 N force, than that of control fruits among seven of nine cultivars evaluated (*Margosan* et al., 1997). In a different study, *Mari* et al. (1999) examined the effects of different concentrations of peracetic acid (PAA) and chlorine dioxide (ClO_2) for post-harvest control of *M. laxa* in stone fruits. Complete inhibition of conidia germination was observed with PAA at 500 mu g ml^{-1} after 5 minutes of contact with conidia and with ClO_2 at 50 mu g ml^{-1} after 1 minute of contact with conidia. The PAA treatment was also effective 1 hour after pathogen inoculation but only on plums, for which a 1,000 mu g ml^{-1} treatment significantly reduced decay incidence by 50%. In a semi-commercial test, pathogen conidia dipped for 20 minutes in PAA at 250 mu g ml^{-1} or in ClO_2 at 10 mu g ml^{-1} or for 5 minutes in PAA at 250 mu g ml^{-1} were completely inhibited, and no brown rot was observed in inoculated wounded nectarines and plums.

Fumigation

Eckert & Kolbezen (1966, 1970) showed that fumigation of fruits with gaseous 2-aminobutanate greatly reduced peaches rot by *M. fructicola*. Exposure of the fruits for 4 hours containing 100–200 ppm (v/v) gaseous 2-aminobutanate reduced decay by 90% or more. The fruits tolerated dosages 5–10 times greater than those required for effective control and the fruits contained less than 10 mg kg^{-1} amine after 4 hours of fumigation. *Abeles & Pusey* (1982) successfully used carbon disulfide (CS_2) and carbon monoxide (CO) as fumigants to control *M. fructicola* in stored fruits. In a study made in the 1990's, acetic acid was an effective post-harvest fumigant to destroy fungal spores on peaches, nectarines, apricots, and cherries (*Sholberg & Gaunce*, 1996). *Sholberg & Gaunce* (1996) showed that decay by *M. fructicola* on peaches was prevented by as little as 1.4 or 2.7 mg l^{-1} acetic acid, respectively. Cultivar 'Harbrite' peaches fumigated with 2.7 mg l^{-1} acetic acid were slightly injured; the phytotoxicity was indicated by light brown streaks. Cultivar 'Glohaven' peaches treated in the orchard with captan at 5% bloom, full bloom, ripening fruit, and 2 days before harvest, then fumigated with 2.7 mg l^{-1} acetic acid after harvest, had significantly less post-harvest brown rot (12.5%) than fruits treated with captan alone (25%). Brown rot of cv. 'Tilton' apricots was reduced from 100 to 25% by fumigation with 2 mg l^{-1} acetic acid without signs of severe phytotoxicity. A few years later, *Sholberg* et al. (2000) used vinegar vapour to reduce post-harvest decay of harvested fruits. They demonstrated that the effect of vapours of several common vinegars containing 4.2% to 6% acetic acid effectively prevented conidia of brown rot (*M. fructicola*) from germinating and causing decay of stone fruits. Fruits were fumigated in sealed containers in which vinegar was dripped onto filter paper wicks or vapourized by heating from an aluminum receptacle. Vapour from 1 ml of red wine vinegar (6% acetic acid) reduced decay by *M. fructicola* on cv. 'Sundrop' apricots from 100% to 0%. According to the above results, *Sholberg* et al. (2000) suggested that vinegar vapour could be an effective alternative to liquid biocides such as sodium hypochlorite for sterilization of surfaces contaminated by conidia of fungal pathogens. In a recent study, *Liu* et al. (2002) demonstrated that thymol vapour reduced post-harvest brown rot of apricots and plums. Fumigation with 1 mg l^{-1} of thymol vapour reduced mean colony diameter of *M. fructicola* from 49 mm in the control to 13 mm when the conidia were cultured on potato dextrose agar. Fumigation of apricots with 2 mg l^{-1} of thymol vapour reduced the germination of *M. fructicola* conidia to 2% compared to 98% on untreated fruits. The incidence of brown rot was reduced to 3% and 32% when cv. 'Manch' apricots were fumigated with thymol or acetic acid at 5 mg l^{-1}, respectively, compared to the 64% incidence in untreated fruits. *Liu* et al. (2002) also demonstrated that fumigation of cv. 'Violette' plums with thymol or acetic acid at 8 mg l^{-1} reduced brown rot from 88% in the control to 24% and 25%, respectively. Fumigation of cv. 'Veeblue' plums with thymol at 4 mg l^{-1} reduced brown rot from 56% in the control to 14%. Moreover, *Liu* et al. (2002) noted that fumigation of apricots with thymol resulted in firmer fruits and higher surface browning, but total soluble solids and titratable acidity were not affected. Fumigation of plum fruits with thymol resulted in higher total soluble solids, but firmness and titratable acidity were not affected. *Liu* et al. (2002) also noted that thymol fumigation caused phytotoxicity on apricots but not on plums.

Fungicide resistance

In the past decades, fungal resistance to fungicides has become an increasingly important problem. Brown rot resistance to benzimidazole, dicarboximide, EBI and DMI fungicides were reported and strategies against resistance were developed. These are listed and discussed below.

After only a few years of application of **benzimidazoles**, benomyl-tolerant isolates of the brown rot fungi, *M. fructicola* and *M. laxa* were found in 1974 in stone fruit orchards of California (*Tate*, 1974; *Tate* et al., 1974). Repeated applications of benomyl during bloom and pre-harvest have resulted in the selection of benomyl-resistant *M. fructicola* in Australia (*Whan*, 1976), in the states of Michigan (*Jones & Ehret*, 1976) and California (*Ogawa* et al., 1988) and *M. laxa* in California (*Cañez & Ogawa*, 1982; *Ogawa* et al., 1984). *Jones & Ehret* (1976) characterised the virulence, sporulation and growth of benomyl-tolerant isolates. They concluded that the tolerant isolates grew more slowly, produced less conidia and were less virulent than the sensitive ones. However, benomyl at concentrations of 150 to 300 μg ml^{-1} protected fruits inoculated with a sensitive isolate but these concentrations were ineffective against the tolerant isolates. Moreover, benomyl-tolerant isolates were also tolerant to other benzimidazole fungicides such as thiophanate-methyl. In an attempt to delay the development of benomyl-resistant isolates as well as to ensure disease control, monitoring of benomyl-resistant *M. fructicola* strains was suggested in California (*Ogawa* et al., 1981). *Ogawa* et al. (1981) showed that benomyl or benomyl-captan combination applications effectively controlled the disease in orchards with low populations of low-level resistant *M. fructicola*. However, the benomyl-captan combination failed in orchards with high populations of low-level resistant *M. fructicola*. *Ogawa* et al. (1981) suggested that non-benzimidazole fungicides should be used in these orchards according to orchard monitoring for resistant *M. fructicola*. Manufacturers often provided that benomyl should be used only in a mixture with other fungicides. However, field tests indicated that benomyl combined with less effective compounds does not delay the selection of resistant populations (*Szkolnik* et al., 1978). *Sonoda* et al. (1983) showed that with low populations of benomyl-resistant *M. fructicola*, effective disease control can be obtained with benomyl sprays. There is laboratory evidence that benomyl-sensitive isolates tend to predominate over resistant isolates when inoculated onto injured peach fruits (*Sonoda* et al., 1982a,b). A few years later, the parasitic fitness of benomyl-resistant and benomyl-sensitive *M. laxa* was also examined in the field in California (*Cañez & Ogawa*, 1985). In this study, almond blossoms were inoculated with benomyl-resistant and benomyl-sensitive *M. laxa* isolates. Inoculation with sensitive isolates resulted in greater number of blighted blossoms, shorter latent period, greater spore production and larger cankers on the twigs than inoculation with resistant isolates (*Cañez & Ogawa*, 1985). *Michalaides* et al. (1986) examined several prune and apricot orchards for detecting benomyl-resistant isolates of *M. laxa* and *M. fructicola*. They found that *M. laxa* isolates were sensitive to benomyl, however, *M. fructicola* isolates were resistant to benomyl at a level of 1 μg ml^{-1} in all sampled orchards. In addition, they detected *M. fructicola* isolates resistant to benomyl at levels of 4 μg ml^{-1} in several prune and apricot orchards. *Ogawa* et al. (1988) reported that the frequency of benomyl-resistant isolates increased from 20% to almost 90% after a single benomyl application. This suggests that reintroduction of the benimidazole fungicides in areas with resistant strains is, at best, likely to provide only short-term disease control. By the early 1990's, benzimidazol-resistant isolates of *M. fructicola* persisted in field populations in Australian (*Penrose*, 1990) and New Zealand orchards (*Braithwaite* et al., 1991) and were competitive in laboratory experiments (*Sanoamuang & Gaunt*, 1991). The spread and persistence of benomyl-resistant *M. fructicola* strains was reported also in South Carolina peach orchards by the early 1990's (*Zehr* et al., 1991). Recently, *Ma* et al. (2003) identified and characterised low and high levels of resistance to the benzimidazole fungicides, benomyl and thiophanate-methyl, in field isolates of *M. fructicola*. Results from microsatellite DNA fingerprints showed that genetic identities among the sensitive populations, low-resistant, and high-resistant isolates were very high (>0.96). Analysis of DNA sequences of the beta-tubulin gene showed that the low-resistant isolates had a point mutation at codon 6, causing a replacement of the amino acid histidine by tyrosine. Codon 198, which encodes a glutamic acid in sensitive and low-resistant isolates, was converted to a codon for alanine in high-resistant isolates. Based on these point mutations in the beta-tubulin gene, *Ma* et al. (2003) developed an allele-specific PCR assays for rapid detection of benzimidazole-resistant isolates of *M. fructicola*. *Yoshimura* et al. (2004) found that the frequency of resistance to the benzimidazole thiophanate-methyl was 75% in isolates collected from 1992 to 1998 and 22% in isolates collected in 2002. Three groups having distinct ranges of values for 50% effective concentration were identified: benzimidazole-sensitive, low-resistant and high-resistant isolates. The use of thiophanate-methyl at 300 μg ml^{-1} (half dosage) and 600 μg ml^{-1} (full dosage) effectively reduced the percentage of blighted blossoms caused by the benzimidazole-sensitive isolates but not that caused by the low-resistant or high-resistant isolates. *Yoshimura* et al. (2004) also noted that the high-resistant isolates caused significantly greater blossom blight than low-resistant isolates at either dosage levels.

Resistance in *M. fructicola* to the **dicarboximide fungicides**, iprodione, procymidone and vinclozolin, has been reported (*Sztejnberg & Jones*, 1978; *Ritchie*, 1981, 1983; *Penrose* et al., 1985). Firstly, *Sztejnberg & Jones* (1978) reported *M. fructicola* resistance to dicarboximide fungicides. They isolated the fungus from the field and it showed resistance to dicarboximides in laboratory. However, they did not report whether the resistance was field resistance associated with poor disease control. *Ritchie* (1983) showed that strains of *M. fructicola* resistant to dichloran, iprodione,

procymidone and vinclozolin produced smaller lesions or sporulated less, or both, on untreated fruits than did sensitive parental strains. *Ritchie* (1983) demonstrated that dicarboximide-resistant isolates of *M. fructicola* were less parasitically fit and would not be apt to rapidly increase to a dominant population level. However, iprodione-resistant *Botrytis cinerea* has become dominant in grape vineyards in France and strawberry fields in California. Therefore, it can be concluded that laboratory tests may not reliably indicate the parasitic fitness of isolates that develop under field conditions. *Benes & Ritchie* (1984) provided evidence of increased melanin content in the resistant isolates of *M. fructicola*. They demonstrated that dicarboximide-resistant strains of *M. fructicola* could be distinguished from sensitive strains by their darker mycelial pigmentation. *Penrose* et al. (1985) reported field occurrence of vinclozolin resistance in *M. fructicola* in New South Wales fruit orchards. The resistance occurred in those orchards where dicarboximides had been used for over four seasons. *Sanoamuang & Gaunt* (1991) indicated that dicarboximide-resistant *M. fructicola* may not overwinter as effectively in mummified fruits and twig cancers as the dicarboximide-sensitive ones. *Elmer & Gaunt* (1994) demonstrated that the competitive ability of resistant isolates was less compared to sensitive ones. This explains the fact that resistant strains declined in the field population not treated with dicarboximide fungicides (*Braithwaite* et al., 1991; *Elmer & Gaunt*, 1993). *Elmer & Gaunt* (1993) demonstrated that dicarboximide-resistant strains of *M. fructicola*, in contrast with benzimidazole-resistant strains, did not persist in field populations unless dicarboximide fungicides were used regularly during the season. *Sanoamuang & Gaunt* (1995) showed that resistant isolates were as virulent and pathogenic as sensitive isolates on flowers and fruits. However, in the same study, it was also shown that dicarboximide-resistant isolates poorly sporulated after survival on twig cancers. This phenomenon may explain their decline in the absence of fungicide selection pressure. *Beever* et al. (1989), *Rewal* et al. (1991) and *Staub* (1991) demonstrated that dicarboximide-resistance in pathogens is often associated with reduced fitness. However, causation is still required because of the potential for recombination of the genes coding dicarboximide resistance with those conferring enhanced survival and other aspects of fitness (*Sanomuang* et al., 1995). *Yoshimura* et al. (2004) noted that iprodione has been used in the United States for about two decades, but resistance to iprodione in *M. fructicola* has not yet been reported in the field.

Reese & Moore (1982) induced resistance in *M. fructicola* to an **SBI compound**, Nustar through ultraviolet radiation of spores, but these isolates lost their resistance after three to nine transfers on a fungicide-free medium. The potential of *M. fructicola* to build up resistance against SBI fungicides *in vitro* was also shown by *Nunninger-Ney* (1988). To minimise the risk of such a resistance build-up, *Zhang* et al. (1991) recommended using the SBI-fungicides only in combination with a protective fungicide such as captan or dithianon. Recently, *Zehr* et al. (1999) determined the baseline sensitivity of *M. fructicola* to propiconazole in a peach orchard not previously exposed to demethylation-inhibiting (DMI) fungicides, using the concentration in an agar medium required to suppress radial growth of mycelium by 50%. The baseline sensitivity was found to be approximately 0.03 mu g ml^{-1}. Prolonged, regular exposure of the natural population of *M. fructicola* to propiconazole in the test orchard over a 3-year period (29 total applications) resulted in a wider range of sensitivity among isolates than was observed in the initial population. Comparisons with isolates from commercial orchards where DMI fungicides were used regularly showed that sensitivities were similar to that of the test orchard that had been exposed to propiconazole for the 3-year period. *Yoshimura* et al. (2004) noted that the demethylation-inhibiting fungicides have been used widely for the last years, and no resistance against them has been found in *M. fructicola* in California However, Schnabel et al. (2003, 2004) reported propiconazole resistance of *M. fructicola* in Georgia, and Huy suggested that the MFABC1 gene might be a DMI fungicide resistance determinant in *M. fructicola*.

Several researchers reported that if a fungus strain is resistant to a fungicide, then other members of the fungicide group have less fungicide activity against the strain. In the case of *M. fructicola*, *Sztejnberg & Jones* (1978), *Ritchie* (1981) and *Rosenberger & Meyer* (1981) noted that fungus strains resistant to one of the dicarboximide fungicides are cross-resistant to other members of this group. *Penrose* et al. (1985) demonstrated that vinclozolin-resistant isolates were resistant *in vitro* to two other dicarboximide fungicides, iprodione and procymidone. However, the cross-resistance effect was stronger for procymidone than for iprodione.

Several **anti-resistant management strategies** were developed in order to integrate fungicide use and lower the risk of fungicide-resistance (*Staub*, 1991; *Borovinova & Sredkov*, 1996, 2003; *Schnabel* et al., 2004). A world-wide fungicide resistance committee, the Fungicide Resistance Action Committee (FRAC), has been working for several years and publishes yearly improved general recommendations of fungicide use for avoiding fungicide resistance. At the moment, six working groups exist including working groups of anilinopyrimidines, benzimidazoles, dicarboximides, phenylamides, SBI fungicides and QoI (strobilurine) fungicides. Each group gives recommendations also for other crops and plant diseases. As it was noted previously, the 2nd, 3rd, 5th and 6th groups are also important in the control of *Monilinia* species. Although, FRAC working groups do not give specific anti-resistant strategies against *Monilinia* species, the general anti-resistant strategies of the above FRAC groups are strongly advised to be used by all fruit growers. According to FRAC recommendations (*Gold*, 2004), the most essential strategies can be summarised as follows:

- Apply fungicides at effective rates and intervals according to the manufacturer's recommendations.

- The number of applications of fungicides within a total disease management program must be limited whether applied straight or in mixtures with other fungicides. Repeated application of fungicides alone should not be used on the same crop in one season against a high-risk pathogen in areas of high disease pressure for that particular pathogen.
- For crop/pathogen situations where repeated spray applications (e.g. orchard crops/powdery mildew) are made during the season, alternation (block sprays or in sequence) or mixtures with an effective non cross-resistant fungicide are recommended.
- Mixture partners for fungicides should be chosen carefully to contribute to the effective control of the targeted pathogen(s). The mixture partner must have a different mode of action; in addition, it may increase the spectrum of activity or provide needed curative activity.
- Fungicide input is only one aspect of crop management. Fungicide use does not replace the need for resistant crop varieties, good agronomic practice, plant hygiene/sanitation, etc.

Future aspects of the control measures

In the future, more attention should be paid to a forecast-based disease control and the environmentally-benign plant production systems. In order to achieve this, the grower has to know the action threshold of the brown rot and to reach information on disease warning such as weather conditions or insect population. Therefore, agrometeorological stations and PC based disease warning systems should be used in fruit orchards for precise timing of fungicide applications. In practice, growers need newly developed disease warning and expert systems, such as those developed by *Luo* et al. (2001) against *M. fructicola*.

Moreover, the grower should take it into account that different environmentally-benign production systems have different possibilities for disease control. In such systems (integrated and organic), there are less possibilities for effective disease control than in conventional production systems. Especially in organic fruit production, where only copper, sulphur, powder rock, botanical fungicides and some microbial products are approved for disease control (*Anonymous*, 1997). Under such conditions, other elements than chemical control, play a very essential role in the disease control. In integrated fruit production, most of the above mentioned fungicides (section Chemical control) are approved in Hungary, except for arsenites, SPCP, dichloran, dinitro-ortho-cresolate (DNOC) and benzimidazoles, such as benomyl and thiophanate methyl. (It should be noted that arsenites, SPCP and dichloran are banned even in conventional plant protection.) Therefore, successful control of brown rot in integrated fruit production can be achieved quite easily in the pre- and post-harvest control. However, the use of all other, non-chemical control methods has high priority especially in storage. In organic production systems, fungicides are not effective enough against brown rot causing fruit rot or bloom and branch blights. Therefore, the use of non-chemical control methods, such as legislative control measures, cultural, physical and biological control methods, as well as host resistance, are becoming more essential. However, these control measures are not effective enough for profitable organic fruit production. Therefore, the combined use of all control methods and the need for further research in this field have to be emphasised.

In the future, the role of host resistance and the use of more environmentally-safe fungicides will increase. The wide use of highly resistant cultivars and effective non-chemical methods can be predicted. Already, there are several examples of genetically modified cultivars resistant to diseases. The appearance of such a resistant or tolerant cultivar can also be predicted in the case of brown rot diseases, mainly *M. fructicola* or *M. laxa*. In most cases, environmentally-safe fungicides are more effective, but there is a higher risk of a resistance build-up than in the case of some old fungicides, such as copper or sulphur. Therefore, resistance strategies have to be followed rigorously. Probably, more practically useful biological products (antagonists) will also be produced in the future for commercial use in both the pre-harvest and the post-harvest biological control of fruits.

Acknowledgements

The study was partly supported by the Hungarian Scientific Research Fund (OTKA F043503) and a János Bolyai Research Fellowship awarded to the author.

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