

The brown rot fungi of fruit crops (*Monilinia* spp.): II. Important features of their epidemiology

(Review paper)

Holb I. J.

Department of Plant Protection, Centre for Agricultural Sciences, University of Debrecen, 138 Böszörményi St.,
4032 Debrecen, Hungary

Summary: Plant disease epidemiology provides the key to both a better understanding of the nature of a disease and the most effective approach to disease control. Brown rot fungi (*Monilinia* spp.) cause mainly fruit rot, blossom blight and stem canker which results in considerable yield losses both in the field and in the storage place. In order to provide a better disease control strategy, all aspects of brown rot fungi epidemiology are described and discussed in the second part of this review. The general disease cycle of *Monilinia fructigena*, *M. laxa*, *M. fructicola* and *Monilia polystroma* is described. After such environmental and biological factors are presented which influence the development of hyphae, mycelium, conidia, stroma and apothecial formation. Factors affecting the ability of brown rot fungi to survive are also demonstrated. Then spatio-temporal dynamics of brown rot fungi are discussed. In the last two parts, the epidemiology of brown rot fungi was related to disease warning models and some aspects of disease management.

Key words: epidemiology, brown rot fungi, *Monilinia* spp., *Monilia polystroma*, disease cycle, environmental and biological factors, spatio-temporal dynamics, disease warning, disease management

Introduction

Today, there is little doubt that plant disease epidemiology provides the key to both a better understanding of the nature of a disease and the most effective approach to disease control. Plant disease epidemiology is the overall study of disease epidemics, the comprehensive analysis of the interaction among three constituents: the host, the pathogen and the environment (Van der Plank, 1963; Zadoks & Schein, 1979). Since the early nineteenth century, the epidemiology of fruit pathogens including *Monilinia* spp. has been studied thoroughly. Rapid development of our knowledge on the epidemiology of brown rot fungi started at the end of World War II. For fifty years, research into several parts of the brown rot disease cycles has been conducted and the results have been involved in disease management practice. For the last two decades, the complexity of brown rot epidemics, including molecular examinations, the spatio-temporal modelling approach, disease threshold and disease warning, has been involved in most epidemiological studies.

The aim of the first part of this review paper was to summarise the important features of the biology of four brown rot fungi, including *Monilinia fructigena* (Aderh. & Ruhl.) Honey, *Monilinia laxa* (Aderh. & Ruhl.) Honey, *Monilinia fructicola* (Wint.) Honey and *Monilia polystroma* van Leeuwen (Holb, 2003b). Now, in this second part, an attempt is made to describe and discuss all aspects of the epidemiology of the four brown rot fungi. A detailed overview of the disease cycle of each species and the environmental and biological factors affecting disease epidemiology is given. Then, the spatio-temporal dynamics of

the disease epidemics and the relationship between brown rot epidemiology and disease warning models are shown and, finally, the relationship between brown rot epidemiology and disease management is discussed. In the third part of this review, the possibilities for disease control against brown rot fungi will be demonstrated.

Disease cycle in general

Disease cycles of brown rot fungi are very similar except for the greater importance of the sexual stage in *M. fructicola* than in the other species. *M. fructicola* is the only species in the life cycle of which apothecia play a role in nature, although the occurrence of apothecia varies by region (Kable, 1965b; Landgraf & Zehr, 1982; Sanoamuang et al., 1995). Apothecia of *M. fructigena* rarely form and, if they do, then only in the field during the second spring after infection (Harada, 1977; Willetts & Harada, 1984). Apothecia of *M. fructigena* were successfully produced in the field and also in the laboratory (Harada, 1977; Barta & Harada, 1986). However, *M. laxa* failed to produce apothecia in several attempts and the perfect stage of this fungus has also been recorded only in a few cases in the field (Willetts & Harada, 1984). The infrequent production of apothecia by *M. fructigena* and *M. laxa* suggests that ascospores are not important as a source of inoculum in the disease cycles of these species (Byrde & Willetts, 1977; Willetts & Harada, 1984). The perfect stage of *M. polystroma* is not known yet. Its features are very similar to those of *M. fructigena*.

The possible sources of brown rot fungi for primary infection are:

- overwintering of conidia on mummified fruits on the tree or on the ground;
- conidia, produced on fruit mummies in the spring;
- ascospores, produced in apothecia on mummified fruits on the ground;
- and conidia, produced on infected blossom, twigs, peduncles and cankers on branches (Anderson, 1956).

Normally, there are several successive cycles of secondary infection on young or maturing fruits or blossoms, twigs and other parts of the host. Any infected tissue in which the moisture content is sufficient for sporulation may serve as a source of inoculum for secondary infection. Also, infected crab apples and ornamental trees growing near the cultivated crops may serve as a source of inoculum. These conidia provide the inoculum for other parts of the same or the neighbouring trees (Byrde & Willetts, 1977; Batra, 1991). The spores are dispersed by wind, water splash or vectors such as insects and birds and when they reach susceptible tissues, infection may take place under favourable environmental conditions (Pauvert et al., 1969; Lack, 1989). Mycelium of the fungus spreads via blossoms and fruits to woody tissues. After penetration of fruits, there is active mycelial growth, and the hyphae in the outer tissues of the fruit become closely interwoven to form a stroma (Byrde & Willetts, 1977). The extent of stromatal formation depends on relative humidity, air temperature after penetration and the degree of fruit maturity at the time of infection (Willetts, 1968a). The fungal development stops when the nutrients in the fruit become depleted and temperature decrease. The fruit becomes a wrinkled mummy and, in this form, as well as in cankers in the branches, the fungus overwinters (Willetts, 1968a).

Fruits may become infected at harvest time and then fruit rot develops during the post-harvest period (Byrde & Willetts, 1977). Infection may also spread quickly during storage, by mycelial contact between infected and healthy fruits (Willetts & Bullock, 1993).

Disease cycle of *Monilinia fructigena*

The main primary inoculum of *M. fructigena* consists of conidia that are produced on mummified fruits which have remained in the orchard from the previous season to spring (Figure 1). Mummified fruits mainly overwinter on the tree, but sometimes they can survive on the orchard floor. Knoche et al. (2000) showed that mummified fruits on apple cv. 'Elstar' could remain firmly attached to spurs far into the next growing season. A combination of humid weather followed by a temperature of 15–20 °C in springtime induces the formation of fresh conidia on the surface of mummified fruits (Byrde, 1954). Xu et al. (2001b) demonstrated that conidia produced on overwintered mummified fruits in spring may survive for long periods and therefore become an

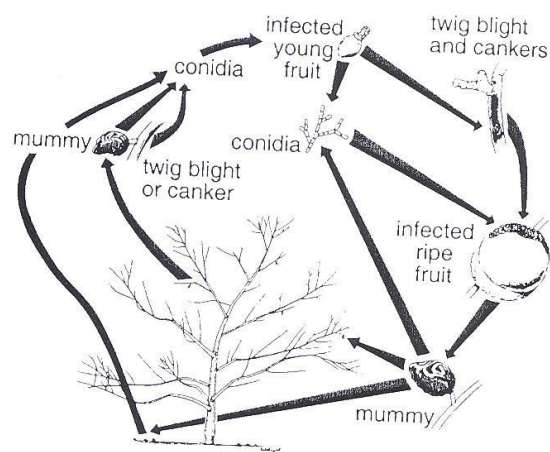


Figure 1 Disease cycle of *Monilinia fructigena* (Aderhold & Ruhland) Honey (adapted from Byrde & Willetts, 1977)

inoculum source for a long time during the growing season. However, Biggs & Northover (1985) have observed that not all mummified fruits sporulate in the spring. Furthermore, Van Leeuwen et al. (2002b) demonstrated that fruits of apple cv. 'Golden Delicious' infected at the beginning of October had not mummified, but after overwintering sporulated profusely under optimal conditions. Consequently, all (mummified and not mummified) infected fruits can be a source inoculum in the next spring if they overwinter. *M. fructigena* is mainly a wound pathogen. Xu & Robinson (2000) showed that no brown rot developed after inoculation of non-wounded apple fruits with a concentration of 7.5×10^5 conidia/ml. Therefore, successful infection requires fruit skin injuries, which can be caused by abiotic (spring frosts, hail) as well as biotic factors (insects, birds, man). Infection mainly takes place via cracks and wounds in the fruit skin (Xu & Robinson, 2000). However, a few studies emphasised that infection can also take place via fruit-to-fruit contact (Van Leeuwen et al., 2000) or via lenticels (Horne, 1933). After infection, a brown lesion develops on the fruit, which is later ruptured by numerous sporodichia (conidial pustules) bursting through the cuticle of the fruit. In the first crop of conidia, millions of spores are produced per fruit, which are subsequently dispersed by air, water or vectors such as insects and birds (Moore, 1950; Croxall et al., 1951; Pauvert et al., 1969; Lack, 1989; Tobin et al., 1989; Van't Westeinde, 1999; Holb, 2003a). A part of these conidia initiates new infections, although healthy fruits are also infected by fruit-to-fruit contact. With *M. fructigena*, the first infected fruitlets in pome fruit orchards usually appear 5–7 weeks after the bloom, and subsequently infection of healthy fruits occurs continuously up to harvest time (Van Leeuwen et al., 2000). No exact data are available for pre-harvest losses caused by *M. fructigena*, but low or moderate losses (1–15%) (Van Leeuwen et al., 2000; Xu et al., 2001b) to extremely high losses (40–60%) are reported (Wormald, 1954; Holb, 2003a). Diseased fruits either stay on the tree or fall to the

ground. In the tree canopy, diseased fruits gradually dry out and shrivel, and turn into firm structures (mummified fruits). These mummified fruits are the main survival structures of the pathogen during wintertime, although in a few cases it was also observed to survive as mycelium in infected fruits, twigs and branches (Byrde & Willetts, 1977). In springtime, periods of rain stimulate the uptake of moisture by the dried-out, mummified fruits. Subsequently, these mummies produce a new crop of conidia (Byrde, 1954); this forms the primary inoculum to start an epidemic the following year.

Disease cycle of Monilinia laxa

The life cycle of *M. laxa* is quite similar to that of *M. fructigena* (Figure 1). One of the basic differences is that *M. laxa* is more virulent than *M. fructigena* or *M. fructicola* at flowering. *M. fructigena* and *M. fructicola* usually cause more damage to ripening fruits than *M. laxa*. *M. laxa* survives from one season to the next in twig cankers, blighted blossoms, peduncles, and in the rotted, mummified fruits hanging in the tree. Conidia begin to develop on these parts in spring. The production of *M. laxa* conidia from mummified fruits, blighted spurs and flowers was thoroughly studied on sweet cherry (Stensvand et al., 2001). Large numbers of conidia are produced on mummified cherry fruits left hanging on the trees for 2–3 years after infection. The production of conidia from overwintered fruit mummies was more than 10 times higher compared to the conidial production from an overwintered fruit spur or newly infected flower. The highest sporulation on mummified fruits and fruit spurs occurs prior to flowering, and very few conidia are produced at harvest. Flowers infected in the spring produce most conidia during the first two months after infection. The newly infected flowers produce more than 10 times more conidia than infected, overwintered flowers in the following spring. Consequently, an abundant and timely supply of inoculum is strategically located in the tree when blossoms emerge in the spring (Stensvand et al., 2001).

The conidia are blown about by wind (Wilson & Baker, 1946; Corbin et al., 1968; Corbin & Ogawa, 1974; and washed about by rain (Corbin et al., 1968). When they lodge on susceptible tissue, they germinate in two to four hours if moisture is present and temperature is favourable. A high percentage of newly formed conidia are germinable, and if not exposed to direct sunlight and high temperature, they retain their viability for months (Corbin et al., 1968; Corbin & Ogawa, 1974). *M. laxa* attacks the blossoms of cherry, plum, almond and apricot, producing extensive flower and twig blighting. The critical period for flower infection extends from the time the unopened flowers emerge from the winter buds until the petals are shed. There is evidence that the flowers are most susceptible to infection when fully open, although some infection through the side of the floral tube may also occur. Calavan & Keitt (1948) reported that the most frequent sites of infection in cherry blossoms are the anthers, stigmas, and petals. In apricot and prune, the sepals are susceptible, as are the other floral parts. In almond,

stigma infection is most common, with anthers and petals being the next most frequently infected (Ogawa & McCain, 1960).

At ordinary springtime temperatures, three to six days elapse between blossom infection and the first evidence of necrosis. This is followed by rapid necrosis of the entire blossom. Infection and development of disease symptoms occur over a relatively wide temperature range between 4 and 30 °C (Calavan & Keitt, 1948). Low-moisture conditions limit infection; little or no infection occurs in rainless weather, even if humidity is high (Weaver, 1943). Most stone fruits, such as nectarine and plum, are resistant to penetration and disease expression at the pit hardening stage (Fourie & Holz, 2003a, b). This indicates that infection at this stage should not contribute to a gradual build-up of secondary inoculum in the orchards. However, resistance to penetration and disease expression decreases with fruit maturity and by harvest a large amount of secondary inoculum is produced (Fourie & Holz, 2003a, b). During the season, several successive cycles of secondary infection can occur on fruits particularly near fruit maturing and mummified fruits are formed.

Finally, blighted blossoms, peduncles, twig cankers, and mummified fruits can serve as a survival structure for the fungus.

Disease cycle of Monilinia fructicola

The fungus survives the winter in several ways (Figure 2):

- As mummified fruits hanging in the tree; here conidia are produced on the surface of the fruit in spring.
- As mummified fruits on the ground; on such fruit the fungus produces the typical pseudosclerotial mat (stroma) from which the apothecia arise. Apothecia are never produced from nonstromatized or recently-infected (fleshy) fruit (Holtz et al., 1998). The apothecia appear and mature at the time the host blossoms in the spring. They discharge their spores into the air for a few weeks and then disintegrate.
- As mycelium in blossom parts, peduncles, and twigs killed by the pathogen the previous year (Sutton & Clayton, 1972). Sporulation on peduncles, twigs and branch cankers occurs frequently in the eastern United States and Australia (Kable, 1965b) but is less common in California. Sources of inoculum for South Carolina peach orchards were found to be nonabscised aborted fruit, infected thinned fruit on the ground, and infected fruit on wild plum trees (Landgraf & Zehr, 1982).

In spring, apothecia may develop from the overwintered pseudosclerotial mat (stroma) when the asci are mature and conditions are favourable, the ascospores are discharged causing the primary infection. Apothecia develop in areas where the soil is moist in the spring. Ascospores are forcibly ejected into the air and are carried by air currents about the

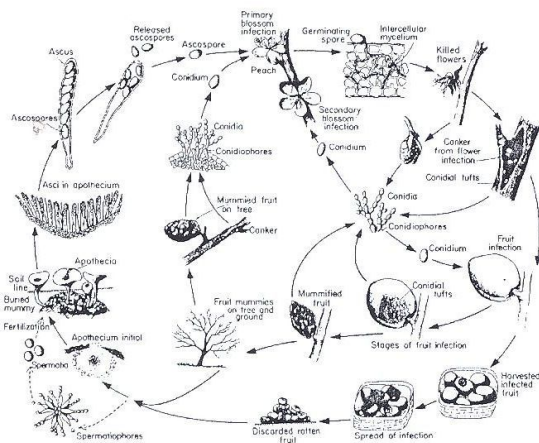


Figure 2 Disease cycle of *Monilia fructicola* (Aderhold & Ruhland) Honey (adapted from Agrios, 1997)

orchard. Slight disturbances in the air (which change the humidity) initiate ascospore discharge. The liberation of ascospores normally coincides with the emergence of young shoots and blossoms of plants. The primary cycle can also begin with the conidia formed on mummified fruits on the tree and on other infected host tissues (twig cankers and blighted flowers that remain in the tree). Conidia are freely disseminated by moving air, rainwater (Jenkins, 1965; Kable, 1965a) and insects (Tate & Ogawa, 1975). Conidia produced on mummified fruits may also survive the winter and cause infection in spring (Bertram, 1916). These are formed at the end of the season, possess a thicker wall, and are more resistant than those produced during the early summer. However, it is generally accepted that peduncles and fruit mummies producing new pustules provide the major inoculum sources. Mycelia in buds and leaves do not seem to contribute to a great extent to primary infections (Byrde & Willetts, 1977).

Ascospores or conidia produced from mummies cause blossom blight in the spring under favourable conditions (Byrde & Willetts, 1977; Kable 1965b; Sholberg et al., 1981; Landgraf & Zehr, 1982). After infection, initial hyphae colonise infected floral parts, then the mycelium pushes outward through the epidermis and forms numerous conidial tufts on the infected tissues. In the meantime, the mycelium rapidly grows on the tissues of blossom petioles and from there into the fruit spurs and the twigs. In the twig, a reddish-brown shield-shaped canker forms. Infected twigs often become girdled and die. Primary infections of blossom can also function as a source of latent infection of fruit (Jerome, 1958; Jenkins & Reinganum, 1965; Tate & Corbin, 1978; Gubler et al., 1987; Cruickshank & Wade, 1992; Wade & Cruickshank, 1992). When microclimatic conditions are unfavourable, these primary infections can remain latent until conditions are favourable for disease development that leads to fruit rot. The level of latent infection in fruits is influenced by both primary and secondary infections (Wade,

1956; Wade & Cruickshank, 1992; Luo et al. 2001a). The latent infection could occur over the whole season under favourable conditions.

Blossom blight may cause severe yield losses on stone fruits by reducing the number of flowers and twigs. Moreover, infected flowers and twigs with sporulation serve as a source of secondary inoculum for infection of fruit (Sholberg et al., 1981; Landgraf & Zehr, 1982). However, e.g. in California, the main inoculum sources for secondary infection are conidia produced on the thinned infected fruits on the orchard floor (Hong et al., 1997).

A few green fruits may become rotted in early summer. This is thought to result mainly from quiescent (incipient) infections (Tilford, 1936) or insect wounds (Tate & Ogawa, 1975), because direct infections require over 30 hours of continuous moisture. However, Biggs & Northover (1988a) have shown that young peach fruits are highly susceptible to infection, then, the fruits become resistant at pit hardening, and later they become increasingly susceptible at two to three weeks before full ripeness. Although the injury of the fruit may lead to an increase in infection, the fungus readily infects when no wound or fruit-to-fruit contact is present (Michailides & Morgan, 1997). It commonly enters the fruit by the way of trichome (hair) sockets. Rains and accompanying high humidity favour infection. Thus, the disease occurs most frequently and causes the greatest destruction in the more humid fruit-growing areas (Sonoda et al., 1967); nevertheless, the disease may also appear during a rainless period. Dew at night probably provides the moisture for spore germination and infection. Fruit infection is most common during the last few weeks before harvest. Infected fruits sporulate and increase the secondary inoculum. There are several successive cycles of secondary infection on different plant parts. Finally, fungus can survive on twig cankers, blighted blossoms, peduncles, and mummified fruits, as we described earlier.

When the fruits are not picked until fully ripe, heavy losses in the field are common. Fresh-market fruit for shipping are picked before they are fully ripe; consequently, loss in the field is relatively low. However, loss during shipment and at market may be heavy, as the fungus develops rapidly when packaged fruits are removed from cold storage. Fruit infection also takes place after harvest in storage. Mycelium can directly attack healthy fruits in contact with infected ones. Healthy fruits may also be attacked by conidia at any time between harvest and use by the consumer (Agrios, 1997).

Disease cycle of *Monilia polystroma*

The anamorph of the fungus was described by Van Leeuwen et al. (2002a), however, no record is available about the perfect stage of the fungus. It is supposed that the apothecia of Japanese *M. fructigena* observed by Batra & Harada (1986) in the field is probably the perfect stage of the recently described *M. polystroma*, but this has not been proven yet. The life cycle of *M. polystroma* is not clearly

known either; however, it is supposed that the fungus has a very similar life cycle as that described for *M. fructigena* (Van Leeuwen et al., 2002a).

Environmental and biological factors affecting disease epidemiology

Several abiotic and biotic factors affect the development of the brown rot fungi, changing the disease development and dynamics of brown rot epidemic during the season. In this section, the factors affecting the development of hyphae, mycelium, conidia, stroma and apothecial formation of the brown rot fungi are discussed. Information on the factors influencing survival is also given.

Factors affecting the development of hyphae, mycelium, conidia and stroma

Temperature

Byrde & Willetts (1977) stated that the optimum temperature for mycelial development and sporulation is about 25 °C for all brown rot fungi. However, Willetts & Harada (1984) stated that for most *Monilinia* spp., the optimum temperature for mycelial growth ranges from 15 to 20 °C, only *M. laxa* requires 25 °C for optimal growth. Usually, stroma formation of *Monilinia* spp. in culture or on fruit tissues takes place at 15–20 °C after a 4 to 8 weeks' incubation. However, Terui & Harada (1966) and Harada (1977) concluded that stroma required a further incubation of 4–8 weeks at 20–30 °C before they were fully mature.

The optimum temperature range for the germination of spores of *M. fructicola* has been recorded as 20–25 °C (Weaver, 1950) and 21–27 °C (McCallan, 1930; Wellman & McCallan, 1942). Biggs & Northover (1988b) observed that although the fungus can grow slowly at 1.7 °C to 4.4 °C, its optimum temperature is 22.2 °C to 23.9 °C. At 23 °C or above, it produces visible symptoms on infected fruit within two days and can completely rot the fruit in four or five days. The optimum temperatures for infection of peach and sweet cherry fruits are 22.5 °C to 25 °C and 20 °C to 22.5 °C, respectively (Biggs & Northover, 1988b). In general, optimal temperature range for infection of stone fruits by *M. fructicola* is between 20 and 25 °C (Corbin, 1963; Phillips, 1984; Biggs & Northover, 1988b) and the optimum temperature for penetration of blossoms is 25 °C (Weaver, 1950; Luo et al. 2001b). Wilcox (1989) demonstrated that incidence of *M. fructicola* on blossom blight of sour cherry increased when inoculated plants were incubated at 8, 12, 16, 20 °C for 5 to 10 hours of wetness, and reached the level of 85 to 90% after 24 hours of wetness. Hong & Michailides (1999) found that optimal mycelial growth of the fungus occurred at –1 MPa osmotic potential and 25 °C and sporulation at –3 MPa and 20 °C on potato dextrose agar (PDA). In a field study, Watson et al. (2002) showed that the frequency of *M. fructicola* sporulation on overwintered infected tissues was greater at 15 and 23 °C than at 4 or 12 °C.

In *M. laxa* studies, infection and development of disease symptoms occurred over a relatively wide temperature range (4 °C to 30 °C), the optimum being about 24 °C (Calavan & Keitt, 1948). Mycelial growth and germination of conidia on peach-agar at temperatures ranging from 25 to –4 °C decrease with temperature and cease at –4 °C (Tian & Bertolini, 1999). At germination temperatures of 0 and 5 °C, larger conidia are produced and they germinate earlier with longer germ tubes than those produced at 25 °C (Tian & Bertolini, 1999). Conidia produced at the lower temperatures (0 and 5 °C) and inoculated onto wounded nectarines stored at 0 °C were highly infective and infectivity decreased when the conidia were produced at 25 °C (Tian & Bertolini, 1999). Tamm et al. (1995) established a non-linear model to describe the incidence of infection as a function of temperature and wetness duration for blossom blight of sweet cherry.

In a *M. fructigena* study in the UK (Xu et al., 2001a), it was shown that the rate of germination initially increases with temperature to a maximum at approximately 23–25 °C and then decreases (Figure 3). 70% of viable conidia had germinated within 2 hours at 20 and 25 °C. The rate of colonization on detached fruits increases log-linearly with increasing temperature (Table 1). No sporulation on detached fruits occurs at 5 or 25 °C and sporulation appears to be unaffected by either temperature (10–20 °C) or RH (45–98%) once infection is established (Figure 4). In a

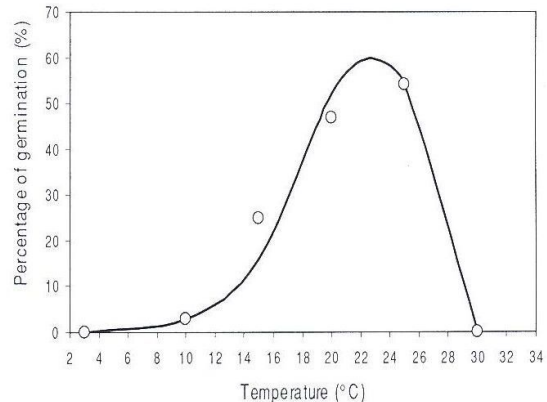


Figure 3 Relationship between the average percentage germination of *Monilinia fructigena* and temperature assessed on PDA media after two-hour germination (adopted from Xu et al., 2001a)

Table 1 The colonisation rate ($\text{cm}^2 \text{day}^{-1}$) of *Monilinia fructigena* on apple fruit in relation to temperature and relative humidity (adopted from Xu et al., 2001a)

Relative humidity (%)	Temperature (°C)				
	5	10	15	20	25
45	–0.2	0.6	1.5	1.8	2.2
75	–0.5	0.2	0.9	1.7	1.8
85	–0.7	0.5	0.7	1.6	2.1
98	–0.4	0.3	–0.9	1.4	1.5

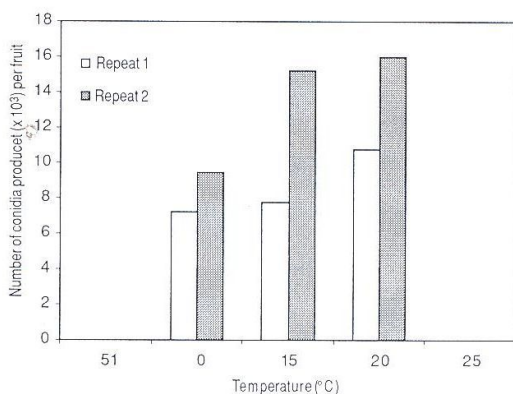


Figure 4 Histogram of numbers of *Monilinia fructigena* conidia produced on detached apple fruits over five temperatures at 75% RH (adopted from Xu et al., 2001a)

Dutch study, Van Leeuwen et al. (2002b) examined the effect of four temperature regimes (9–10, 14–15, 19–20 and 24–25 °C) on sporulation of *M. fructigena*-diseased fruit mummies after 4, 8 and 12 weeks of incubation. They found that the numbers of fruits sporulating decreased markedly from 9–10 °C to 24–25 °C regimes after a 12-week incubation. In a post-infection regime of 24–25 °C and 65–75% relative humidity, the number of sporulating pear cv. 'Conference' fruits decreased rapidly after 4 and 8 weeks of incubation, and sporulation had completely ceased after a 12-week incubation (Van Leeuwen et al., 2002b). Temperature requirements of *M. polystoma* are similar to those of *M. fructigena* (Van Leeuwen et al., 2002a).

Relative humidity and moisture

For normal development, vegetative mycelium must have contact with water, and even a slight desiccation stops mycelial growth either permanently or until water is again available in a liquid or vapour state (Byrde & Willetts, 1977). Growth *in vitro* of *M. fructicola* was found to be uninterrupted at relative humidities above 96%; however, growth was retarded below 93% (Weaver, 1950). In nature, moisture is available from infected host tissue and the type and condition of the infected tissue greatly affect the amount of mycelium produced. At very high relative humidities, or when the tissue is water-soaked, infection of woody tissues such as twigs may become severe, but under less humid conditions the activities of the fungus are seriously curtailed or stopped (Weaver, 1950; Corbin & Cruickshank, 1963). Weaver (1950) found that penetration of peach blossom by *M. fructicola* was greatly influenced by atmospheric relative humidity. In a saturated atmosphere, entry was through any of the floral parts, except the sepals, but at relative humidities of 80% or lower, infection was only through stigmas. A dense hyphal mat develops over the surfaces of infected fruits or cultural medium that have been incubated under warm, humid conditions or on the mummified fruits on the

tree or on the ground when the weather was warm and wet (Woronin, 1900; Willetts, 1968b). Apart from high moisture levels, an abundance of soluble nutrients associated with softening of fruits is needed for mat-growth. In a part of the study of Northover & Biggs (1995), detached cherries were inoculated with *M. fructicola* and incubated for 22, 36 and 48 hours wetting at 20 °C. They found that longer wetting durations of 36 and 48 hours, increased mycelial, lesion and sporodochial development on cv. 'Bing' sweet cherry fruits. In a sour cherry study, Koball et al. (1997) found that when detached sour cherry blossoms were inoculated with conidia of *M. fructicola* and subjected to a standard 8-hour wetting treatment at 20 °C, blossom blight incidence was proportional to relative humidity. In the same study, the authors also demonstrated that relative humidity during incubation had an important influence on blossom blight development. In the case of *M. fructigena*, Xu & Robinson (2000) determined the effect of the duration of wetness periods on conidial infection of fruits in relation to wound age. They demonstrated that increasing the duration of wetness periods reduced the incidence of *M. fructigena* on older wounds. Recently, Fourie & Holz (2003a) showed that increased wetness and wetness duration markedly increased the penetration of hyphae and the disease expression of *M. laxa* on ripening nectarine fruits.

Conidiophores normally develop under drier conditions than vegetative mycelium and hence an increase in relative humidity often suppresses conidiophore and conidial production and stimulates vegetative growth. The septa that separate conidia of the spore chains do not develop under moist conditions (Willetts & Calonge, 1969) and relatively low relative humidities are needed for fragmentation of conidial chains (Byrde, 1954). A detailed study of the effect of moisture on the sporulation of *M. fructicola* on apricots was made by Corbin & Cruickshank (1963) who found that the water content of tissues greatly influenced the initiation and intensity of sporulation. They found that water-soaked plant tissues produced conidia more quickly than did tissues that contained less moisture. Conidia were most readily produced on fruitlets and fruits, while sporulation on peduncles and mummified fruits depended upon the moisture levels of these tissues. *M. fructicola* produced conidia continuously if there was only minimal moisture loss from the sporulating tissue, and if the atmospheric relative humidity was between 94 and 100%. When there was a significant water loss, which was found at relative humidities of less than 94%, sporulation intensity was reduced. Corbin & Cruickshank (1963) also observed that alternation of high and low relative humidities produced greater intensity of sporulation than that produced when the atmospheric humidity was maintained at a continuously high level. Alternating conditions can occur frequently in nature such as in showery weather, which results in repeated drying and remoistening of fruit mummies. Luo et al. (2001a) showed that both sporulation intensity and duration of sporulation increased as water content of thinned fruits increased from 13.4 to 67.2% (Figure 5). Increased inoculum concentration

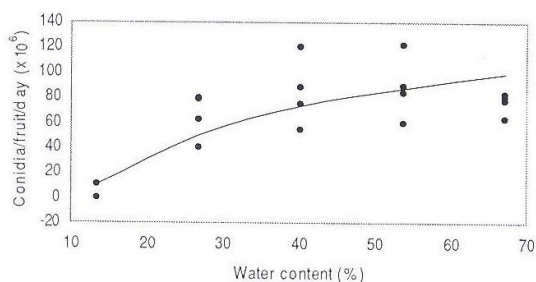


Figure 5 Relationship between water content (%) of thinned prune fruit and sporulation intensity (conidia/fruit/day) of *Monilinia fructicola* (adopted from Luo et al., 2001a)

(from 8,000 to 24,000 conidia per millilitre) and wetness duration (from 4 to 16 hours) increased the percentage of fruits with latent infections. In a field study, Watson et al. (2002) demonstrated that 12 hours of wetting at ranges of 5 to 23 °C was sufficient for sporulation of overwintered cankers in peach twigs caused by *M. fructicola* (Table 2). The number of twig cankers supporting sporulation increased with the time of wetting up to 72 hours. Given the additional moisture requirements for spore germination, ingress and infection, 17 to 30 hours of wetting or high humidity during bloom may be needed for blossom blight to occur (Watson et al., 2002). Xu et al. (2001a) studied the effects of relative humidity on conidial germination and sporulation of *M. fructigena*. They showed that conidia only germinated under near-saturation humidity when it was greater than or equal to 97% RH. In the same study, sporulation of *M. fructigena* at 10–20 °C appeared to be unaffected by the relative humidity once infection was established.

High relative humidities are required for good stroma formation of brown rot fungi (Willets & Harada, 1984). Fruits become dry during the mummification process and survive unfavourable conditions in winter. After overwintering, when the temperature rises and the mummies are wetted, either by the uptake of free water or water vapour, conidia develop. The minimum moisture content of mummies for sporulation to take place at 26 °C was found to be 21% (Byrde & Willets, 1977).

Light

Hall (1933) found that mycelial growth of *M. fructigena* was greater in intermittent light and darkness than in continuous darkness; if acidic conditions were maintained. Carlile (1965) observed that any departure from optimal nutritional conditions caused staling of cultures of *M. fructigena* and that this was more marked in dark-grown cultures than in illuminated ones. Hall (1933) observed that in darkness the rate of linear growth was slower but the total dry weight of the colonies was greater than under illumination. Under alternating light and darkness, the weight of mycelium was less than in darkness, the plectenchyma were relatively thin and aerial tuft-like masses of mycelium bearing conidia were abundantly formed in zones. In continuous light, there was very little plectenchyma developed with only diffuse aerial mycelium and spores. A characteristic of the growth of many fungi, including the brown rot species, is the development of zonation in culture or on host tissues. Usually the growth of colonies is unzoned for the first few days, even under conditions that are optimal for the development of concentric ridges. Hall (1933) observed that growth of ridges was mainly during the dark period in *M. fructigena* cultures, while the hollows developed in the light.

The response of *M. laxa* to light is similar to that of *M. fructigena*. Under conditions of alternating light and darkness, ridging is produced by *M. laxa* but aerial mycelium is less floccose than in cultures of *M. fructigena* and there are no obvious differences between the densities of vegetative mycelium in the light and darkness zones. In continuous darkness, a dense plectenchyma develops, with occasional tuft-like hyphal masses; marginal lobing and staling take place during the growth of the colony (Byrde & Willets, 1977).

Cultures of *M. fructicola* sporulate profusely to produce a continuous carpet of spores under continuous darkness and uniform temperature. Under alternating light and darkness, concentric ridges and hollows are formed; ridges coincide with growth during the dark periods and hollows with growth in the light (Byrde & Willets, 1977). *M. fructigena* requires light for the production of conidia, while *M. fructicola* needs darkness for sporulation. Van Ende & Cornelis (1970)

Table 2 Sporulation of *Monilinia fructicola* on peach twig cankers in response to temperature and wetting period^a (adopted from Watson et al., 2002)

Temperature (°C)	Hours of wetting						Average ^y
	12	24	36	48	60	72	
5	0.8	2.5	6.8	6.8	7.5	10.0	5.7 a
11	3.3	10.8	21.7	24.2	26.7	26.7	18.9 a
15	15.8	25.8	32.5	37.5	40.8	40.8	32.2 ab
23	19.0	31.7	37.5	44.2	45.8	46.7	37.5 b
Mean ^z	9.8 a	17.7 b	24.6 c	28.1 cd	30.2 d	31.0 d	

^a Data show percentage of cankers with sporulation at each wetting period and temperature. Each number is an average of three replicate sampling times.

^y Means followed by the same letter in the average temperature column are not significantly different (least significant difference=17.4, P=0.05).

^z Means followed by the same letter across wetting periods are not significantly different (least significant difference=3.7, P=0.05).

suggested that light stimulates the development of conidiophores by *M. fructicola* but inhibits conidial formation. They referred to the production of a photoproduct (P310) which accumulated at higher levels in mycelium growth in light of short wavelength and of high intensity. Zonation can be induced in cultures of *M. fructicola* by the introduction of amino acids into the medium (Jerebzoﬀ, 1956).

On potato sucrose agar, Harada (1977) found that light inhibited stroma formation by most Japanese *Monilinia* spp. However, alternating light and dark periods were not necessary for stroma development by *M. fructicola* when the fungus was grown in fruit tissues. Therefore, Willetts & Harada (1984) concluded that during stroma development there is a complex interaction of light with other environmental factors.

Wavelength irradiation and zonation

The brown rot fungi have been used on several occasions for the study of the spectral regions that are active in the induction of zonation in fungi. Jerebzoﬀ (1956) suggested that only visible radiations with wavelengths shorter than 500 nm are active in the production of zonation in cultures of *M. fructicola*. Blue light (440–490 nm) is the most effective for sporulation and vegetative growth by *M. fructigena*, and there is a decreasing stimulatory effect in the red, orange, yellow and green regions. Five-day-old *M. fructicola* cultures incubated in darkness absorbed light from the shorter wavelengths, with the strongest peak at 400 nm, a sharp one at 365 nm and the smallest peak at 455 nm. The most effective wavelengths for the stimulation of growth of fertile conidiophores are in the near ultraviolet at 390 and 370 nm and the blue spectral region at about 420 and 470 nm (Byrde & Willetts, 1977).

Marcaki et al. (1998) studied the effects of 0, 1.5 and 3 KGy gamma-irradiation on fungal growth of *M. fructigena*. Results showed clearly that all radiation doses induced a delay in fungus development, even when it was not directly affected.

Factors affecting apothecial formation

Climatic conditions are obviously of great importance and are probably responsible for the vernal development of apothecia. Norton et al. (1923) noted that when peaches bloomed early then apothecia were produced correspondingly early. This suggests that the environmental conditions that bring about the breaking of dormancy of fruit trees also influence apothecial formation. Some obvious factors are temperature, moisture content of the soil and of the mummies, humidity of the atmosphere, type and intensity of light (Byrde & Willetts, 1977).

Temperature

Temperatures best suited for initiation and differentiation of apothecia of the brown rot fungi are lower than those for mycelial growth and sporulation. Ezekiel (1924) concluded that a period of incubation at cold temperatures is required

for the initiation at apothecia by *M. fructicola*. However, Harrison (1933) obtained the perfect stage of this fungus after a winter when there were only a few frosts, suggesting that an extended period of incubation at low temperatures is not essential for successful apothecial production. Optimum temperature for apothecial initiation and then development is about 15 °C (Elliott, 1965). Ogawa & English (1991) demonstrated that moderate spring temperatures (10 °C to 15.5 °C) favour development of apothecia while colder weather deters it. Apothecia of *M. fructicola* were obtained on well-developed stromata grown on potato sucrose agar and on apple tissues at 24 °C for 3 months; the stromata were kept for a further period of 3 months at 8 °C and then within several days of transference to 12 °C apothecial initials were observed (Terui & Harada, 1966; Harada, 1977). With respect to a period of incubation at low temperatures as a prerequisite for apothecial initiation, it is probably significant that most members of the *Sclerotiniaceae* are low-temperature organisms. Their survival depends to a large extent on the resistance of the stroma to severe winter conditions. Presumably, during the resting period there is greatly reduced metabolic activity and possibly metabolism along different pathways that, when growth conditions improve, could lead to the initiation of apothecia rather than conidial production or external vegetative growth. It seems likely that any factor that inhibits the two latter processes could, when other conditions are suitable, promote the formation of the perfect stage (Byrde & Willetts, 1977). In a field study, Holtz et al. (1998) observed that leaving peach and nectarine mummies on the soil surface versus burying them 2 to 3 cm depth did not affect the development of apothecia. However, apothecia were only produced from mummies that were subject to an 8-week or greater cold-temperature incubation while in contact with soil. In the laboratory, apothecia were only produced from mummies that were partially buried in moist sand and stored without light at 2 °C and >97% relative humidity (RH) for more than 8 weeks prior to incubation for 2 weeks (12, 15, or 20 °C) with a 12-h photoperiod (Holtz et al., 1998). In another field study, Hong & Michailides (1998) determined the effect of temperature on the discharge and germination of ascospores by apothecia of *M. fructicola*. They found that the period of discharge decreased as temperature increased from 10 to 25 °C. The greatest discharge occurred with apothecia at 15 °C, followed by those incubated at 20, 10, and 25 °C (Table 3). The germination of ascospores of *M. fructicola* and the length of germ tubes increased as temperature increased from 7 to 15 °C; however, increasing temperatures above 15 °C did not increase either ascospore germination or length of germ tubes.

Moisture

Apothecia develop only on fruit mummies on the ground and they have never been reported on mummified fruits still hanging from the trees. This situation is probably associated with the greater desiccation of the fruit in the tree compared with that in the soil. Mummies have a reduced ability to absorb moisture during periods of rain or from atmospheres

Table 3 Effect of temperature on the total discharge of ascospores by apothecia of *Monilinia fructicola* (adopted from Hong & Michailides, 1998)

Location, group	Number of apothecia examined	Total discharge (x 10 ⁶ ascospores/apothecium) at temperature (°C)					p ^w	Group mean
		10	15	20	25			
Reedley								
96-1	24	2.14 b	5.49 a	1.58 b	1.21 b	0.0165	2.6 c	
96-2	24	1.41 b	4.16 a	1.62 b	1.41 b	0.0184	2.2 c	
96-3	24	0.90 a	7.01 a	3.65 a	1.76 a	0.4142	3.3 bc	
97-2	18	1.83 a	... ^x	2.05 a	0.33 a	0.3650	1.4 c	
Sanger								
97-1	24	8.02 a	6.63 a	12.60 a	4.03 a	0.3076	7.8 a	
97-3	24	2.90 b	12.30 a	11.20 ab	5.24 ab	0.0992	9.7 a	
97-4	20	3.22 a	9.42 a	8.36 a	1.92 a	0.2456	6.5 ab	
Grand mean		2.92 c	7.50 a	5.87 b	2.27 c	0.0004 ^y	0.0097 ^z	

^w Level of significance of the difference of the total ascospore discharge among incubation temperatures using Proc GLM of SAS. Each value is the mean of six replicates of apothecia and the numbers followed by the same letter within a row (group) did not differ significantly according to Duncan's multiple range test at P=0.05.

^x No data were collected because of incubator failure.

^y Level of significance of the difference among grand means by analysis of variance on combined data after they were normalized by dividing treatment means by their group means. Grand means followed by the same letter did not differ significantly according to Duncan's multiple range test at P=0.05.

^z Level of significance of the difference among group means by Proc GLM. Group means followed by the same letter within the last column did not differ significantly according to Duncan's multiple range test at P=0.05.

with very high relative humidity and to retain it long enough for apothecial initiation and differentiation (Byrde & Willetts, 1977; Willetts & Harada, 1984). Cunningham (1922) noted that apothecia were more readily produced from mummies buried in hard and compacted soil than in loose soil. Apothecia are found in areas of orchards where the soil remains damp due to the shading by trees or buildings; also litter often covers mummies on which ascocarps develop. All of these are factors related to the maintenance of high moisture levels in the soil, especially in the cool conditions of early spring when evaporation will be at a relatively low level. Ogawa & English (1991) also demonstrated that apothecia develop in areas where the soil is moist in the spring, but they seldom occur, e.g. in California, where the weather becomes dry just before and during bloom. Under such conditions, these structures are found only where the soil is protected from drying by weeds or debris. Also, showery weather often precedes the discovery of the perfect stage in the field. In the laboratory, moist sand and a complete cover of damp moss have been used to maintain high moisture content of mummies on which apothecia of *M. fructicola* were produced (Terui & Harada, 1966). In New Zealand peach orchards, Tate & Wood (2000) observed that first apothecia appeared at the beginning of bloom after 15 mm rain but shrivelled after a few days of drying weather. Main apothecial emergence peaked and declined at full bloom and petal fall, respectively. Apothecia on bare ground were short-lived compared with those protected by overhanging grass. Holtz et al. (1998) showed that nectarine and peach mummies that were incubated at >97% RH for less than 8 weeks or incubated at <90% RH never produced apothecia when stored at 2 °C and then transferred to warmer temperatures with light. In

orchard experiments, apothecia were only observed in plots with non-disturbed and wet orchard floor vegetation (Holtz et al., 1998).

Light

Diffused sunlight or fluorescent illumination above 1500 lux intensity with a 12-hour photoperiod is needed for complete differentiation of the *Monilinia* spp. apothecium (Harada, 1977).

Other physical conditions

Other physical conditions must also be involved in the initiation, development and maturation of apothecia. Low pH of soils, the depth at which mummies are buried in the soil, and the position in which the mummy falls in the orchard are only a few of the other factors that may affect initiation and differentiation of ascocarps (Byrde & Willetts, 1977). Ezekiel (1923) reported that soil with a pH below 7.0 favours apothecial development, whereas an alkaline soil does not. The infrequency of formation of apothecia by *M. laxa* and *M. fructigena*, the sometimes erratic development of ascocarps by *M. fructicola* and the difficulties of producing the perfect stage in the laboratory suggest that a number of exacting and interacting conditions, both of the environment and of the fungus, must be fulfilled before ascocarps are produced (Willetts & Harada, 1984).

Factors affecting the survival of brown rot fungi

Extremes of temperature

Jerome (1958) demonstrated that conidia of *M. fructicola* retained their viability when kept in the laboratory at

temperatures from 2 to 42 °C for 8 weeks with relative humidities in the range of 40–98%. *Ezekiel* (1924), *Wormald* (1954), and *Willets* (1969) showed that conidia germinate and mycelia start to develop at 0 °C but the rates are very slow; mycelial growth stops at 30–35 °C, and the death point of mycelium is about 50 °C. Spores probably survive extremes of temperatures better than mycelia. Dormant and germinating spores of *M. fructicola* survive sub-zero temperatures for at least several days (*Smith et al.*, 1965). *Bertram* (1916) reported that in Vermont, conidia of *M. fructicola* produced in the autumn remained viable throughout winter. When temperatures of –12 °C to –22 °C prevailed, however, few conidia survived. *Jenkins* (1968) showed that in either sun or shade, the reduction in viability of *M. fructicola* conidia was less than 1 percent. Temperatures of about 50 °C for intervals as short as 30 sec significantly reduced percentage germination and hot broth killed the spores more quickly than hot air; hot air was less lethal at a relative humidity of 50% than at RH 80 or 90% (*Smith et al.*, 1965; *Smith & Blomquist*, 1970). Ultra-structural changes were observed in spores of *M. fructicola* after heat treatment (*Baker & Smith*, 1970). Anoxia appears to increase the resistance of spores to subsequent heating (*Bussel et al.*, 1971). Stroma would be expected to have lower thermal death points than vegetative mycelium as they have become adapted to survive at sub-zero temperatures; probably their highest thermal death points are not significantly different from those of mycelia (*Byrde & Willets*, 1977).

Maximum temperatures in regions where fruit trees are grown rarely exceed 40–50 °C, and these high temperatures would prevail for only a few hours during the growing season. Minimum temperatures may be at sub-zero levels for a few days to a few months each winter. Usually the host and its pathogens will be dormant during these cold periods. To some extent, mycelia formed within host tissue will be protected against low and high temperatures by the plant. If the host and the fungus survive, further mycelial development and sporulation will take place when the environment becomes more favourable (*Byrde & Willets*, 1977).

Cold resistance is probably dependent on some basic mechanisms which are similar to adaptations that are found in a range of plants. One of the mechanisms is 'slow air drying', which results in the removal of free water of the fungus tissues and most of the water that remains will be bound water that will not be available for the formation of intracellular ice (*Byrde & Willets*, 1977). In this way, fungi prevent the formation of intracellular ice. In nature, mummies remaining for some time on trees and on the surface of soils become air-dried and should be in suitable state to survive extremes of temperature. Mummies that become buried in soil may be subjected to some air-drying before they are covered with soil or litter; also the active extrusion of water will reduce the moisture content of mummies. Other mechanisms apart from slow air-drying must operate in resistance to sub-zero temperatures as, on

occasions, mummies with high moisture contents survive very low temperatures (*Willets*, 1971). High concentrations of solutes within the cell depress the freezing point and reduce the formation of intracellular ice crystals. Medullary hyphae are generally rich in nutrient reserves, some of which increase the osmotic concentration of the cells. In general, the principles involved in resistance to high temperatures appear to be similar to those which apply to survival against cold (*Byrde & Willets*, 1977). Heat resistance of microbial cells increases with decreasing humidity. Even small changes in the water content may have considerable influence in survival owing to the greater stability of proteins in a dry state. As in resistance to low temperatures, some chemicals will protect against high temperatures. These substances replace bound water by forming hydrogen bonds with proteins and thereby retain the integrity of macromolecules (*Byrde & Willets*, 1977).

Radiations

Conidia and mycelia of brown rot fungi formed on the surface of plant parts or fruit mummies that remain hanging on the tree are exposed to high levels of solar radiation. However, fruit mummies that fall to the ground are protected from the harmful effects of such radiation. Most of the data available on the effect of radiation on fungi have been obtained from laboratory studies and little is known of the chronic effects of solar radiations that reach the surface of the earth. *Thanos* (1951) showed that there was a significant reduction in the viability of spores of *M. fructicola* after short exposures to UV radiations in the laboratory. In a recently made study, *Marquenie et al.* (2002) examined the effect of different ranges of UV-C ($\lambda = 254 \text{ nm}$) light (from 0.01 to 1.50 J cm^{-2}), on the development of the *Monilinia fructigena* on sweet cherry. After an UV-C treatment of 0.50 J cm^{-2} , complete spore inactivation of the fungus was found. *Shepherd* (1968) demonstrated that in exposed situations, conidia of *M. fructicola* were killed relatively quickly but, when they were protected from short wavelength solar radiation, survival was for longer periods. In the same study, considerably higher dosages of radiation were necessary to kill spores at low relative humidity than at saturation. Melanized structures are more resistant to UV light than are hyaline ones (*Sussman*, 1968); the structure and composition of the rind provide a protective barrier for the more loosely interwoven medullary hyphae; possibly mucilage on hyphae and over the exposed surfaces of mummies gives some protection, and the low moisture contents of air-dried tissue may prevent damage to essential proteins and other macromolecules by solar radiations (*Byrde & Willets*, 1977).

Competition with micro-organisms

Apart from the effects of temperature, humidity, moisture and solar radiations on survival of the brown rot fungi, biological factors must also be considered. This was well demonstrated by *Lockwood* (1960) who obtained varying

degrees of lysis of living and dead mycelia of *M. fructicola* by microbial extracts. In early studies, workers demonstrated that the melanin or related substances may have great ecological significance in reducing damage by the lytic action of micro-organisms (Potgieter & Alexander, 1966; Bloomfield & Alexander, 1967; Chet et al., 1967). They supposed that melanin gives protection against biological degradation. Two mechanisms have been suggested: the hyphal wall is protected by a deposit on the outer surface of the hyphae and/or by the formation of a complex with the components of the wall, particularly with chitin; melanin inhibits the enzymes, mainly chitinases and glucanases, associated with biological degradation.

Potgieter & Alexander (1966) demonstrated that chlamydospores and sclerotia were particularly resistant to biological degradation in soils. However, the degree of resistance is dependent on soil type and other environmental factors. This is well illustrated by the rapid and complete degradation of mummified fruits of brown rot fungi in some soils but their excellent preservation in others. Obviously, the types of micro-organisms present in a particular soil are of considerable importance together with physical factors such as temperature and moisture of the soil. The claim that a period of chilling is necessary for apothecial formation may be related to the lack of activity of antagonistic micro-organisms at low temperatures and consequently better preservation of stromatic tissue in which ascocarps can be more readily initiated and then develop. Dry and acid soils also restrict microbial degradation of mummies, and this could explain why stromatic hyphae retain their viability for long periods under these conditions.

More than 50 years ago, Michener & Snell (1949) found that *Bacillus subtilis* secreted substances that were antibiotic to several fungi, including *M. fructicola*. Jenkins (1968) studied the longevity of conidia of *M. fructicola* in sterilized and unsterilized soils. The unsterile soil conidia failed to germinate and showed evidence of lysis. Bacteria were present in the unsterilized soils, and these were identified as a *Bacillus* species, probably *B. cereus* Frankland & Frankland. Colonies of this bacterium inhibited growth of *M. fructicola* in culture. Jenkins (1968) concluded that inactivation of conidia of *M. fructicola* in orchards is largely due to the antagonistic effect of bacteria and probably UV radiations, while other factors are of lesser importance. Other, more recent examples for antagonistic micro-organisms to brown rot fungi will be given in the third part of this review, in the chapter titled Biological control.

Spatio-temporal dynamics of brown rot epidemics

Few studies have been made to characterise temporal and spatial aspects of the increase in *Monilinia*-diseased fruits in orchards. As part of a study that dealt with fungicide-resistant strains of *M. fructicola* in New Zealand, Elmer et al.

(1998) analysed the spatial distribution of peach fruit affected brown rot at harvest time. They determined brown rot incidence during the pre-harvest period under controlled conditions. In stone fruits, disease incidence can increase very rapidly around harvest time, as mature fruits become more readily infected (Corbin, 1963; Zehr, 1982). *M. fructicola* is able to infect uninjured stone fruits even at the pre-pit-hardening stage (Corbin, 1963). Latent infections of *M. fructicola* can also play a role in the rapid disease development observed around harvest time (Jenkins & Reinganum, 1965; Northover & Cerkauskas, 1994). Latent infections occur in immature fruits particularly in a season of severe blossom infection, and after a period of quiescence that may last several weeks, rot starts to develop as the fruits ripen. In contrast, *M. fructigena* and *M. laxa* rely almost exclusively on pre-existing wounds in the fruit skin for penetration, although uninjured, ripe apples have been successfully infected via lenticels (Horne, 1933) and fruit-to-fruit contact (Michailides & Morgan, 1997). Latent infections of immature fruits as described for *M. fructicola* have never been reported for *M. fructigena*, *M. polystroma* or *M. laxa*. All these specific features greatly influence the spatio-temporal development of each brown rot fungus.

Van Leeuwen et al. (2000) and Xu et al. (2001b) studied the space-time variation of disease development of *M. fructigena* in apple. They proved the hypothesis that disease incidence increases gradually up to harvest time, unlike the rapid increase at harvest time frequently observed in stone fruits affected by *M. fructicola* or *M. laxa* (Hutton & Leigh, 1956; Zehr, 1982; Northover & Cerkauskas, 1994). Xu et al. (2001b) also clearly demonstrated that incidence of disease (percentage of fruits with brown rot) increased gradually from late July up to harvest; the final disease incidence varied with seasons and cultivars, ranging from 1 to 11%. Van Leeuwen et al. (2000) observed no marked increase around harvest time in cv. 'James Grieve'. However, in cv. 'Cox's Orange Pippin' a distinct increase in disease incidence occurred in the last 3 weeks before harvest maturity, at the same time when cv. 'James Grieve' was harvested. The increase of fruit infection of cv. 'Cox's Orange Pippin' was due to the presence of inoculum in the cv. 'James Grieve' orchard part and wounding agents (insects, birds) shifted to cv. 'Cox's Orange Pippin' after cv. 'James Grieve' was harvested. Van Leeuwen et al. (2000) demonstrated that the increase of disease incidence in cv. 'Cox's Orange Pippin' could not be explained by increased susceptibility of ripening fruits, because ripe 'Cox's Orange Pippin' apples did not become infected without injuries when sprayed with high concentrations of *M. fructigena* incubated at 16–18 °C at 90–100% RH for 7–10 days. In another study, Holb (2003a) showed that no infected fruits occurred until the end of July in organic apple orchards on cv. 'Elstar' (Figure 6). However, from late July until harvest, a rapid increase of disease incidence was observed that could be described by power functions. Holb (2003a) indicated that the increase was probably due to the presence of wounding agents and a large amount of inoculum on the orchard floor.

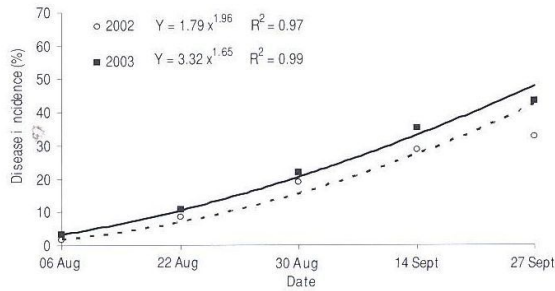


Figure 6 Disease incidence on fruits caused by *M. fructigena* on the orchard floor, in an organic apple orchard on cv. 'Elstar' in Debrecen-Pallag, Hungary, 2002 /drawn line/ and 2003 /dotted line/ (adopted from Holb, 2003a)

Elmer et al. (1998), Van Leeuwen et al., (2000) and Xu et al. (2001b) determined the spatial pattern of *Monilinia*-diseased fruits among fruit trees in time and the extent of clustering of trees with diseased fruits. Van Leeuwen et al. (2000) showed that the spatial distribution of *M. fructigena*-diseased fruits among trees was clearly clustered, indicated by highly significant Lloyd's index of patchiness (LIP) values. In time, the mean LIP values decreased as diseased fruits started to appear in trees previously devoid of diseased fruits. LIP values of the study of Van Leeuwen et al. (2000) were similar to those calculated by Elmer et al. (1998) for *M. fructicola*-diseased fruits in peach and nectarine orchards at harvest time. Van Leeuwen et al. (2000) emphasised that fruit-to-fruit contact and splash dispersal of conidia are the main mechanisms for spread of the disease. They also indicated that the spatial distribution of *M. fructigena* may depend more on behavioural characteristics of wounding agents than on inoculum concentration in the environment. Xu et al. (2001b) also demonstrated that significant aggregation of *M. fructigena*-diseased fruits among trees was detected for assessment dates when the overall incidence of disease was greater than 0–5%. On apple cv. 'Cox's Orange Pippin' and pear cv. 'Conference', significant correlation of disease incidence between adjacent trees or trees separated by one or more trees was detected, but there was no clear relationship between the correlation, the distance or time. They speculated that behavioural characteristics of wounding agents may have played an important role in influencing the spatio-temporal dynamics of brown rot on apple and pear.

Brown rot epidemiology related to disease warning models

Disease warning is partly about defining the conditions under which a pathogen, when in contact with a susceptible host, can infect and become established (Zadoks & Schein, 1979). However, disease warning schemes require, as their components, data on and an understanding of the epidemiology of the diseases and pathogens. Moreover,

understanding the interaction of host, inoculum and environment is essential for devising suitable disease warning or decision support systems. Such a system was developed for fruit diseases, such as apple scab, apple powdery mildew and fire blight. Recently, a few studies have been made to relate epidemiology and disease warning in brown rot infection caused by *M. fructicola* and *M. laxa* in order to predict infections or develop decision support models for fungicide applications during the growing season. However, no disease warning model has been constructed for *M. fructigena* and *M. polystroma*.

Luo et al. (2001b) developed a risk analysis system for blossom blight of prune caused by *M. fructicola*. A risk assessment table (Figure 7) of blossom blight was produced for different environmental conditions to guide the control of prune brown rot. The diagram is based on bloom stage, inoculum concentration and temperature. The risks were classified into four levels: no risk, low, moderate and high risk; according to values of relative risk. There was no risk of blossom blight at early bloom stage (from popcorn to full bloom) when temperature was below 10 °C and wetness duration was shorter than 4 hours (Figure 7). When wetness duration was 24 h, there was a high relative risk of blossom blight at 20 and 25 °C when inoculum potential was low or at 15 and 20 °C when inoculum potential was high. Fewer cases with a high relative risk occur at late bloom stage (from late full bloom to petal fall) than at early bloom stage. Only the 24-hour wetness duration at 20 °C with either low or high inoculum potential may be associated with severe blossom blight. Most conditions resulted in low to moderate risk of blossom blight. Luo & Michailides (2001) developed a similar risk analysis system for latent infection of prune caused by *M. fructicola*. Another study on phenological

Bloom stage	Inoculum potential	Temperature (°C)	Wetness duration (h)					
			4	8	12	16	20	24
popcorn to full bloom	low ^x	10						
		15						
		20						
		25						
	high	10						
		15						
		20						
		25						
late full bloom to petal fall	low	10						
		15						
		20						
		25						
	high	10						
		15						
		20						
		25						

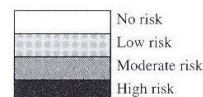


Figure 7 Relative risks of blossom blight of prunes caused by *Monilinia fructicola* in relation to different bloom stages, inoculum potentials, temperatures and wetness durations (adopted from Luo et al., 2001b)

analysis of brown rot blossom blight of sweet cherry, caused by *M. laxa*, was made by Tamm et al. (1995). They investigated the influence of temperature and wetness duration on infection incidence of sweet cherry blossoms in Switzerland. They combined Richards and Analytis' beta models into a nonlinear model to describe the infection incidence as a function of both temperature and wetness duration. The model was used to predict the possible range of incidence of blossom blight resulting from combinations of different temperatures and wetness durations.

In a recently made study, Luo & Michailides (2003) developed a preliminary decision support model to guide fungicide application to reduce risk of prune fruit rot caused by *Monilinia fructicola*. The estimated percentage of branches with fruit rot and relative probability of leading latent infection to fruit rot were used to develop the decision process (Figure 8). Four recommendations for disease management were used:

- safe, no need of fungicide application in the season;
- wait, continue to investigate latent infection;
- check reference of historical weather to decide if fungicide application is needed;
- spray fungicide immediately.

Brown rot epidemiology related to some aspects of disease management

Epidemiology as a science leads to disease management as a technology (Zadoks & Schein, 1979). Although much data on the epidemiology of brown rot fungi have been gathered during the last century (Wormald, 1954; Byrde & Willetts, 1977; Batra, 1991), the presently used disease

management strategies may still not prevent the occurrence of severe epidemics in some years. Especially in stone fruits, severe epidemics of *M. fructicola* and *M. laxa* occur under adverse weather conditions during flowering or fruit ripening (Weaver, 1950; Zehr, 1982; Wilcox, 1989). In stone fruits, fungicides are regularly applied against brown rot, primarily caused by *M. fructicola* and *M. laxa*, during blossoming and the fruit ripening period (Vályi et al., 1985; Benedek et al., 1990; Hogmire & Biggs, 1994; Szabó & Nyéki, 1995; Penrose, 1998). In pome fruits, only fruit infection is important, and no fungicidal sprays are applied specifically against brown rot in Europe (Vályi et al. 1985; 1986; Soltész & Szabó, 1997; Gonda, 1995, 2000). The flowering period constitutes a relatively short period in which susceptible tissue is available, in contrast with fruits which are susceptible to infection from 5–6 weeks after blossoming up to harvest time. Reducing the amount of primary inoculum in the direct environment to minimise flower infection was emphasised in research during the 1940s and 1950s. Several eradicant fungicides were tested for their ability to destroy conidial tufts of *M. laxa* appearing on the twigs in late winter, in order to prevent blossom infection later (Wilson & Baker, 1946; Wilson, 1950). As flowers are susceptible for only 1–2 weeks at ordinary temperatures (Calavan & Keitt, 1948), secondary inoculum hardly play a role in flower infection (Ogawa et al., 1967). In fruit infection, however, primary as well as secondary inoculum play a role, and this requires a different disease management strategy. Based on several characteristics, previously shown risk analysis and decision support have been developed on the basis of epidemiology in order to support disease control of brown rot fungi (Tamm et al., 1995; Luo & Michailides, 2001, 2003; Luo et al., 2001).

Fruit injury

In pome fruits, avoidance of fruit injury is very effective in the control of brown rot caused by *M. fructigena*. Birds (Tobin et al., 1989; Van't Westeinde, 1999) and insects (Moore, 1950; Croxall et al., 1951) are regarded the most important wounding agents. When these wounding agents at the same time act as vectors, as reported for dried fruit beetles in peach by Kable (1969), immediate infection of freshly wounded fruits occurs. As infection by aerial and water dispersed inoculum depends on the availability of (fresh) wounds and the probability of reaching them, the vector-borne mechanism is far more effective and probably the most important. The seasonal concentrations of *M. fructigena* conidia in the air in a pome fruit orchard were relatively low (Van Leeuwen et al., 2000) compared with studies done in stone fruit orchards where *M. fructicola* and *M. laxa* occurred (Kable, 1965a; Corbin et al., 1968). In order to evaluate the significance of aerial dispersed inoculum in the development of an epidemic, more knowledge about the longevity of *M. fructigena* conidia under field conditions is essential. As long as conidia deposited on non-wounded fruits remain viable, subsequent wounding may still lead to successful infection.

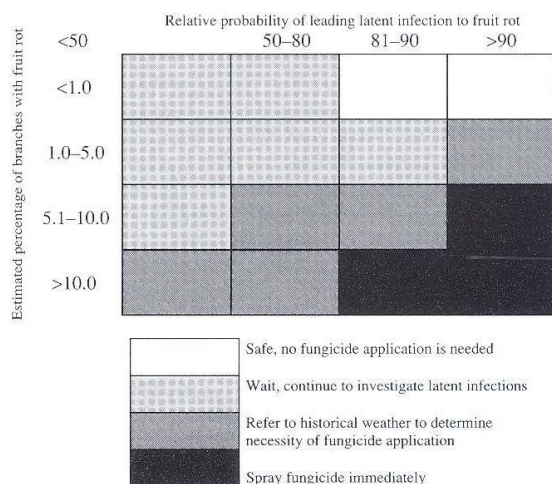


Figure 8 Diagram of decision process used in the decision support model for fungicide application to reduce risk of prune fruit rot caused by *Monilinia fructicola* (adopted from Luo & Michailides, 2003)

It has been easier to demonstrate the essential role of fruit injury in the epidemiology of *M. fructigena* in pome fruits, than to find adequate means to prevent injury. Hellmann (1998) advised immediate fungicide sprayings to diminish strong increase in brown rot after heavy hail showers, but it is doubtful whether this is economically feasible, as badly damaged fruits are of no marketable value. Bird damage is especially severe in orchards near wooded areas and brushlands, and with control methods such as shooting, gas canons and kites, growers try to combat the problem (Hasey & Salmon, 1993; Van't Westeinde, 1999). Conflicting situations sometimes occur. For example, common earwig (*Forficula auricularia*) is an important natural enemy of the woolly apple aphid *Eriosoma lanigerum* in IPM systems (Helsen et al., 1998), but earwigs have also been reported to cause small, shallow wounds in apple fruits which resulted in high brown rot incidence (Croxall et al., 1951).

Reduction of inoculum

Reduction of the amount of inoculum in the environment is another important aspect in disease management of *Monilinia* diseases. Primary inoculum in springtime originates mainly from overwintered fruits infected the previous season, although sporulation on spurs and twigs also occurs (Byrde & Willetts, 1977). Infected fruits which remain in the tree canopy, contribute to the pool of primary inoculum in the following year. It has always been recommended to remove and destroy mummified and also not mummified fruits during pruning in the dormant season (Kennel, 1968; Byrde & Willetts, 1977; Van Leeuwen et al., 2000). Fruits which drop to the ground soon after infection usually decompose quickly and thus are unlikely to contribute to primary inoculum in the next year (Willetts, 1971); however, in the case of *M. fructicola*, they can serve as an important ascospore inoculum source, producing apothecia and asci. One of the best control measures is to remove infected fruits from the tree as soon as the first brown rot symptoms appear. This (i) prevents further spread of the disease within a fruit cluster, (ii) prevents the fungus from growing into spurs/twigs, (iii) minimises the amount of secondary inoculum produced, and (iv) reduces primary inoculum for the next year. However, in practice few pome fruit growers spend time and money on this method (Van't Westeinde, 1999), and it is rarely feasible on orchards exceeding 10 ha.

Given that it is difficult to remove all infected fruit structures, an alternative approach is to minimise the amount of inoculum produced per infected fruit. After infection, the pathogen rapidly colonises the fruit and produces numerous conidia at the surface. After this first crop of conidia, the fungus can produce subsequent conidial crops from the same sporulating area (Corbin & Cruickshank, 1963). If this process of regeneration of conidia were blocked, no more conidia would be produced during the rest of the season, either after overwintering. The occurrence of many different micro-organisms on fruits previously infected by brown rot

fungi has been associated with the reduction in subsequent regeneration of conidia (Jenkins, 1968; Byrde & Willetts, 1977; Pusey & Wilson, 1984; Hong et al., 2000). Perhaps it is possible to enhance the occurrence of antagonistic fungi and bacteria on fruits previously infected by brown rot. The optimal moment of application might be just after the first *Monilinia* pustules have ruptured the fruit skin.

Acknowledgements

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

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