AN ABSTRACT OF THE THESIS OF

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Brown rot materially reduces the yield by causing blossom blight and fruit rot of stone fruit trees in western Oregon. Severe outbreaks of the disease occur during bloom and harvest whenever high levels of inoculum within the orchard corresponded with elevated temperature (51-17°C) and prolonged rainy periods. Two species of brown rot fungi, Monilinia laxa and M. fructicola were found. Early studies, by Evans and Owens, 1941, in western Oregon found M. fructicola was the dominant species. In our studies, 89-96% of the infected blossoms, and 83-95% of the infected fruits yielded M. laxa. Only 4-11% of the infected blossoms, and 5-17% of the infected fruit samples yielded M. fructicola. Cultures of M. laxa were easily distinguished from M. fructicola, by their lobing of the colony margins, and branched germ tubes on 2% PDA. Hyphal anastomosis of germinating conidia were commonly present with M. fructicola but were absent with M. laxa. Resistance to .5 ppm benomyl was not detected in the isolates of Monilinia spp. obtained during our studies. M. laxa and M. fructicola were equally effective in producing blossom blight. Conidial

inoculation of ripe sweet cherry fruit with M. fructicola produced a higher incidence of infection (65-83%) than M. laxa (58-75%). The major differences in pathogenicity of M. laxa and M. fructicola was the production of larger cankers and more sporodochia on peach cultivars infected by M. laxa. Neither of the species produced sporodochia on sweet cherry twigs.

No significant difference at $P \leq 0.05$ was observed in pathogenicity among isolates of M. laxa obtained from sweet cherries and other stone fruit cultivars.

Sequential samples of the infected fruit spurs collected during the 1980-1981 seasons indicated that the primary source of inoculum was conidia from sporodochia produced on previously infected fruiting spurs. Secondary inoculum from earlier infected blossoms infects additional blossoms, green fruit and eventually ripe fruit. A small percentage (2-3%) of green sweet cherry fruit was infected by M. laxa causing green fruit rot under very wet conditions. Latent mycelium in green fruit later developed into typical fruit rot at harvest (5-10%). Such infection prior to full ripeness of fruit can increase the amount of available inoculum for rot of ripe fruit in the orchard and storage. The increased level of soluble solids, brought about by cellular and biochemical processes during fruit maturity, was used as a measure of fruit ripeness and was positively correlated with the amount of fruit rot. The amount of soluble solids utilized by M. laxa also increased with fruit maturity.

# Brown Rot of Sweet Cherry in Western Oregon

by

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I DEDICATE THIS THESIS

TO MY PARENTS.

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## INTRODUCTION

Cherry production is Oregon's second largest fruit industry. In 1979 and 1980 the state produced 38,000 and 33,000 tons of sweet cherries worth $20.7 and 15.9 million, respectively, and 2,000 and 2,500 tons of sour cherries worth $1.8 and 1.01 million (OSU Extension 1981 County and State Agriculture estimates). In the Willamette Valley, the frequent combination of high rainfall and mild temperatures during the bloom period favors the development of brown rot blossom blight. Later fruit rot develops from previously infected blossoms. In the drier cherry producing areas east of the Cascades, blossom blight is insignificant, but when unseasonal rain occurs fruit rot can be severe. Barss (5,6), in his studies of the brown rot, confirmed the widespread occurrence of the disease in western Oregon and reported crop losses of up to fifty percent. There is probably an even greater loss from Monilinia blossom blight and fruit rot than is reported (personal observation). In order to control brown rot disease effectively, the causal organism and the disease cycle must be understood; this included a knowledge of the climatic factors that interact with disease development. Previous workers (7,9,76) have reported species of M. laxa and M. fructicola were present in the same orchard in western Oregon. In preliminary work, we found M. laxa more frequently than M. fructicola in isolations from sweet and sour cherries, peaches and prune. The present population study was initiated to determine which

Monilinia spp. were responsible for blossom blight and fruit rot in western Oregon. Orchards surveys were conducted between 1979-81 in an attempt to identify the relative importance of Monilinia species and disease incidence during both the blossom and fruit ripening periods.

In order to determine if there were significant differences between species, a comparative study between western Oregon isolates of M. laxa and M. fructicola was done to investigate radial growth rate, conidia germination, sporulation and development of tolerance to benomyl. Susceptibility to brown rot varies widely among related host species and even within varieties of particular stone fruit species (10,11,40,72,104). In cherries a notable difference in susceptibility exist between sour and sweet cherries and from one variety to another (104). In order to determine pathogenicity of Monilinia isolates from Oregon an experiment was designed to test their pathogenicity on various stone fruit cultivars.

Considerable variation has been observed between isolates within each of the brown rot species (38,39,91,103,104). Ezekiel (31) noted wide variation in the cultural characteristics, effects on inoculated fruit and the production of oxidase within M. fructicola. Killian (62) studied culture of M. laxa isolated from cherries in Europe, and he distinguished two physiological forms. A study was done to determine the variability of M. laxa isolates obtained from sweet cherry and other stone fruit cultivars in western Oregon.

Considerable literature has accumulated on aspects of over-wintering of brown rot fungi (11,53,54,62,101). It is generally

accepted that mummified fruit provided the major source of primary inoculum (11,81,82,87). Cankers and blighted twigs of stone fruit cultivars may provide additional inoculum (7,9,12,104). In the present study, an attempt was made to determine the role of 1) mummified fruit, 2) blighted blossoms, and 3) fruit peduncles remaining on the tree as inoculum in overwintering of Monilinia spp.

Under certain conditions the brown rot disease may become epidemic during both blossoming and harvest (54,55,63). Short and long term infections have been recognized on many crops (32,54,78,84). These infections may cause disease symptoms well after infection and subsequent development of conidia to provide inoculum for further spread of the fungus. Latent infection of green apricot and peach has been confirmed (54,57,93). An experiment was conducted to detect if latent infections are part of the brown rot disease cycle in sweet cherry.

Change in host susceptibility as tissue mature has been reported in related crops (20,25). Wade and Jerome (57,92) recorded loss of disease resistance in fruits of Prunus spp. to Monilinia fructicola as they develop from the pre-pit hardened to post-pit hardened stages. Experiments were designed to detect the level of soluble solid content (essentially sugar) at which sweet cherries become susceptible.

Thus the experiments presented in this thesis have been designed to: I) Ascertain the importance of different Monilinia spp. in Oregon cherry orchards by isolating from the existing populations, and then comparing the colonies characteristics, germination, sporulation, resistance to benomyl and pathogenicity of the resulting isolates.

II) To determine the intraspecies variability of M. laxa. III) To investigate the source of primary inoculum, the extent of latent infection and the correlation between soluble solid content and fruit rot development.

# LITERATURE REVIEW

## I. A. Disease Development Caused By *Monilinia* spp.

The brown rot disease of stone fruits can be of considerable economic importance and may cause major losses. The blossom phase of the disease known as "brown rot blossom blight" and "Monilinia blossom blight", attacks apricots, almonds, cherries, plums, prunes, flowering quince, pears, apples and certain wild species of prunus (11,104). The disease has been reported from Australia, Europe, Eastern Asia, New Zealand, North America, Manchuria, South America, Japan (27,102), North Africa and recently has been reported in Northern Iraq (1). In North America the disease has been reported in the states and provinces along the Pacific coast (5,7,9,76,81), in some fruit growing districts in Michigan, Wisconsin and New York (16,102), as well as the states along the Atlantic coast (102).

Brown rot is the name given to the disease of deciduous tree fruit caused by species of *Monilinia*. Originally two brown rot diseases were reported from Europe; one caused by *M. laxa* on stone fruit and one caused by *M. fructigena* on pome fruit. However, later the common brown rot disease of stone fruit in North America was recognized as a third distinct type and the causal organism named *M. fructicola* (45,64). Hewitt and Leach reported both *M. fructicola* and *M. laxa* on stone fruit in California (4,45). In western Oregon and Washington *M. laxa* and *M. fructicola* were also reported in the stone fruit orchards (5,76). In California the two species exhibited a degree of host specificity (69,70). While *M. fructicola* occurs

frequently on peaches causing blossom blight and fruit rot, M. laxa was seldom found in this host (4); in contrast on apricot and almond M. laxa occurs frequently and M. fructicola is rare.

Jackson (1917) recorded the imperfect stage on pears in Oregon, and Barss assigned the name M. oregonensis to this fungus. M. oregonensis was later shown to be M. laxa (49,76).

The time and manner of introduction of M. laxa into North America is unknown (12). In the Pacific coast states, M. laxa has become the major brown rot organism in some localities (5,6,7,9). Variation within the American species of brown rot fungi was reported by Read (82), but he stated that all species were the same after culturing under uniform conditions. Ezekiel (31) showed conclusively that strain of M. fructicola collected from different parts of the United States differ widely in their characteristic in culture, in rotting fruit, in oxidase production, and size of conidia, and that these differences were due to genetic variation rather than modification of the environment. Evans and Owens (29) studied two Monilinia species causing blighted blossoms and rotting fruit of sweet cherry trees from orchards located in the central counties of the Willamette Valley of Oregon. They reported the percent of M. laxa and M. fructicola isolated from blighted blossom and rotted fruits.

## B. Comparative Studies of M. laxa and M. fructicola

Differences in the habit of growth and in the mode of conidia germination of M. laxa and M. fructicola have been recorded (11,30 38,80,104). The growth characteristics on freshly prepared potato

dextrose agar have afforded a reliable procedure for distinguishing M. laxa from M. fructicola. On this medium, the M. laxa grows more slowly than M. fructicola; colonies are lobed instead of having smooth entire margins. Hyphal anastomoses seldom occur in M. laxa but frequently occur in M. fructicola (70,99,104). Hoffman (46,47) carried out cytological studies on a single conidial culture from wild strains of M. fructigena, he obtained a large number of conidia isolates of the parent type, and detected different morphotypes. Similar results were obtained for M. laxa (48). Hoffman therefore concluded that mycelia of both species were di- or hetero-karyotic. Nuclear migration between mycelia take place following fusions, and when different mycelia are involved, heterokaryosis may result (46, 48). The hyphal growth of M. laxa is in a different manner from those of the M. fructicola and M. fructigena (11,70,99), and the type of growth is described as scorpioid and geniculate. One of the most characteristic cultural features of Monilinia species is the alternation of bands of relatively sterile mycelium with bands of sporogenous hyphae that bear chain of conidia to give zonation across the petri dish or over the surface of infected fruit surface.

The most characteristic cultural feature of M. laxa is its lobed mycelial growth, M. fructicola usually forms colonies with entire smooth margins when both are grown on PDA at 24°C. The aerial vegetative mycelium of M. laxa are usually sparse, while denser bands of sporogenous tissue often develops concentrically in M. fructicola around the inoculum (104). The bands are usually light buff to white color in M. laxa but tan in M. fructicola. Black stromatal

crusts were developed by M. fructicola in the medium after the mycelium has filled the plates (99), but not in M. laxa.

The conidia of the brown rot fungi germinate on moist substrate. A very detailed study was carried by Lin (64) who found that under the condition of his experiments, M. fructicola conidia seldom germinated in pure water; occasionally a small percentage of conidia formed minute germ tubes. Under suitable conditions, a germ tube is produced after three to four hours of incubation; sometimes germination takes place while the spores are still attached in the chain (97). Hall (36) described nuclear changes during conidia germination of M. fructicola. When the germ tube has grown about one millimicron in length one of nuclei moves into the germ tube and then is followed by the other as the germ tube elongates. Germ tubes of germinating conidia of M. laxa are strongly curved (geniculate) and branched, while those of M. fructicola are straighter and less branched (70,99,103). The type of growth exhibited by the germ tube has been used to distinguish between brown rot fungi species (70, 104).

Developmental studies on conidia of M. fructicola have been carried out by Hall (37), on all three Monilinia species by Willette and Calonge (100), and on M. laxa by Hashmi, et al. (41). There are no apparent differences in conidial antogeny among species. In pure culture, M. laxa and M. fructicola produce regular conidia and may develop microconidia; these are spherical bodies about 3 μ in diameter (39). Most workers have been unable to germinate microconidia, and it was considered that they were not viable reproductive bodies,

although Humphrey (50) claimed that he saw them germinate and give rise to the Monilinia form in culture. Regular conidia are the main propagules of the brown rot fungi (100).

Since Sckolnic (88) demonstrated that the failure of dodine to control apple scab disease was caused by development of tolerance in the apple scab fungus, Venturia inaequalis (ckc.) Wint (58), a great number of reports of tolerance to other fungicides have appeared (33, 59,65,89). Many of these dealt with tolerance of such species as Cercospora, Botrytis, Venturia and Monilinia to benomyl and related benzimidazoles (32,89,95,105). These fungicides frequently gave superior control of the diseases, but after continuous use their effectiveness often diminished (59). These failures have been attributed to tolerance of the pathogen through selection and proliferation from initial components of tolerant strains of the pathogen which existed within the natural population (26). Since the selection of fungi tolerant to chemical often occurs, monitoring for tolerance is desirable (26,65,89,95).

The main commercial crops that are host to the three species of Monilinia include apples (Malus domestica L.), pear (Pyrus communis L.), quince (Cydonia oblongata Mill), the prunus species of peach (P. persica (L.) Batch), apricot (P. armeniaca L.), plum (P. domestica L.), sweet cherries (P. avium L.), sour cherries (P. cerasus L.) and almond (P. amygdalus Batsch). The host range of M. laxa f. mali is narrow, and in commercial orchards damage is confined to the apple blossoms and sprus (11).

The degree of susceptibility to brown rot varies widely among

related species and among varieties of particular species (68,72,104, 106). The susceptibility of Morello cherry has been noted more than sweet cherry in Britain and other parts of Europe (104). Kock (1910) recorded observation of 27 varieties of cherries; it was suggested, however, that the differing degrees of infection might have been due to differences in the time of flowering as related to the weather condition. Among sweet cherries it was noted by Hart (40) that the variety Merton Heart is markedly resistant to blossom blight (M. laxa). Elssman (28) tested sour cherry varieties in Bavaria and found that one variety proved to be immune to M. laxa, that others developed gummosis, but not wilting, and that severe wilting was never accompanied by gummosis.

All parts of the flower are subject to attack. The petals, calyx lobes and the stigmas and stamen can be a site of infection (12). The first sign is a small brown spot on the petal, calyx lobe or the tip of the pistil. In warm, moist weather the discoloration spreads rapidly. The infected tissue turns dark brown and the discoloration may extend through all the flower parts, down the pedicel and into the young fruit. The discolored flower parts wither to give a typical blighted appearance.

The first infections of the spring develop when the conidia land and penetrate the flower. Blighted flowers result in reduced fruit set and may infect the fruitlet. The latter may not become apparent until later in the season (56,92). The disease eventually kills the flower parts infected and then begins to invade the base of the flower. After the flower has been killed, the fungus often grows down the flower stalk

to the spur on which the flower is borne (12,104). In sweet cherries (where the flowers are borne on spurs on two-year-old twigs or older branches), when a single flower of cluster becomes infected, the fungus may extend into the spur, killing all of the flowers and young leaves on that spur (7,11,12).

There is evidence that the flowers are most susceptible to infection when fully open (23). Chester (1893) was able to produce blossom blight of peach by artificial inoculation of fully open flower. Detached blossom of stone fruit species have since been artifically inoculated with M. laxa (12,104) and M. fructicola (81,94). Blossoms of peach in the orchard have been successfully inoculated with M. laxa (3) and M. fructicola (12).

Twig blight, leaf infection and wither tip are not common on cherries, However, when blossom blight is particularly severe, emerging leaves may become infected. The fungus then extends into the shoot and produces a shoot wilt (104). Subsequently area beyond the point of infection becomes dark brown and withered (104). If the infection starts on a leaf originating from a spur then all the leaves of that spur wither and the fungus extends into the twig or branch producing a canker (104). Such cankers may girdle the twig and cause the death of the terminal portion. The fungus grows from the floral parts through the peduncle into the twig where the infected tissues are seen as brown collapsed areas. Gum accumulates on the surface of the infected tissues (9,11). Usually the fungus is not visible on either twigs or canker during the summer. The fungus passes the winter in the infected tissue and appears at the surface during early spring as

grey tufts (sporodochia) (16,79,101).

Twigs have been successfully inoculated with M. laxa (7) and M. fructicola (32). Grover (35) suggested a method for resistance assessment in vitro, but Cross-Raymond (23,24) favored a twig inoculation method for M. laxa.

Fruit rot is very important phase in the life cycle of the brown rot fungi. Either of the two brown rot fungi may cause symptoms in cherry but sometimes one fungus predominates over the other (4,19, 102). The disease begins as a dark brown circular spot which spreads rapidly over the fruit. As the disease progresses from small lesions, the fungus ruptures the epidermis of the fruit to form small tufts over the surface. Often the tufts are arranged in concentric bands around the infection site to give a characteristic bull's eye appearance (98,100). The affected tissues remain relatively firm and dry in contrast to rots caused by other fungi (102). The diseased fruits frequently fall to the ground, but sometimes when ripe fruit is infected, it remains hanging on the tree and gradually shrivel into mummies (92,104).

Fruit infection in the orchard is quite common but occasionally is insignificant (10). Infection begins at a wound, frequently due to rain cracking or insect damage (11,12). Infection may take place at any time during the growing season, but symptoms develops more rapidly as the fruit approach maturity (93,104). Losses from the rain cracking of cherries with subseqeutn fruit rot are very serious in some parts of the state, particularly on Bing cherries (5,76). Jerome (56) suggested that the resistance of fruit to penetration is reduced

as it ripens, and spores on the surface of the fruit are then able to become established within the tissue. Morphological resistance has been observed in some varieties on the basis of texture of skin and pentosan of cherry fruit (18,25). Inoculation of peach, apricot, almond, cherry fruit has been made successfully by several workers under laboratory and field conditions with M. laxa and M. fructicola (12,19,103).

## II. Intraspecies Variability of M. laxa Isolates in Oregon

Ezekiel (30) reported the behavior of different strains when seeded in combination of 2 per plate at different points in potato dextrose agar plates. He did not observe any macroscopic reaction where the two colonies grew together. On microscopic observation it was found that the fungus produced a dark line type reaction. Ezekiel grouped more than thirty strains of M. fructicola (syn. S. americana) into six strains differentiated by their habits of growth on PDA at 25°C in tube cultures at 15°C in plate cultures. The strains retained their differences under widely different environments and after successive passages through artificial media. Seal (83) distinguished at least two biological forms of American Monilinia which showed constant cultural difference when grown on various substrate and under different environmental conditions. Robert and Dunegan (81) also found variation within strains on different media. Jehl (52) and Matheny (64) made comparative studies of M. laxa found in Europe and America. Both concluded

that there are two species and that the one common to the United States was M. laxa (syn. S. cinerea). From culture studies they decided that there were two forms on cherries which they designated M. laxa (syn. S. cinerea f. avium) on sweet cherries and M. laxa (syn. S. cinerea f. cereasi) on sour cherries.

## III. Aspects of the Disease Cycle of Brown Rot on Sweet Cherry

The brown rot fungi survive the adverse conditions of winter as mycelium in fruit mummies, fruit peduncles, cankers on twig and branches. Considerable literature has accumulated on the survival aspects, and some reviews have been published (42,52,55,70,101,104). Inoculum from the twig and canker phases of the disease was discussed by Robert and Dunegan (81). The wood of infected twigs turned brown as they wilted this discoloration may extend up to 60 cm. back from the mummy and blighted blossom. The fungus mycelium was recovered from the tissue beyond the discolored margin (14,15,68). Smith (1891) found extensive gum pockets full of mycelia of Monilinia in the region of the cambium and primary phloem of freshly blighted peach twigs (104). Jehle (52), Robert and Dunegan (81) reported that M. fructicola mycelium lived from year to year in cankers and that these cankers could increase in size. Mycelium in peach trees survived for five to six months when it was not subject to desiccation during summer in New South Wales, but the fungus failed to survive in apricot for more than eight months when part of this period was during the summer (60,62).

Viability of Monilinia conidia on fruit mummies and peduncles under field conditions in Australia has been reported by Shepherd (85). Abundant sporulation was observed on infected fruits, and 90% of the conidia were viable; after overwintering on mummies hanging in the trees. Similar results were obtained from fruit peduncles. The loss of viability of conidia from mummies of the brown rot fungi is difficult to determine, and there is great variability according to the condition under which the mummies are kept (11,101). It has been suggested that the breakdown of stroma may in part be due to the activities of other micro-organisms in the soil or on the mummies (102). However, Pollock (1918) reported that stromatized plum produced apothecia of M. fructicola after ten years in soil (104). Jenkins (55) studied the conidia longevity of M. fructicola under field conditions and found that less than one percent of conidia remained viable after exposure for eight days in the tree canopy. Conidia in contact with unsterilized soil have lost their viability within 24 hours (55,86). However, Kable (60) found that conidia of M. fructicola survived for up to four months on peach leaf surfaces in the orchard. Jenkins (55) considered biotic rather than physical degradation to be the major cause of viability loss. Germinating spores of M. fructicola can retain their viability after at least one period of drying (86), but the percentage of conidia that germinated was low (86). Jerome (56) demonstrated that conidia of M. fructicola retained their viability when kept in the laboratory at temperatures from 2 to 24°C for eight weeks with 40-98% relative humidity.

Infected twigs, fruit peduncles and mummies remained on the tree

for several months and are subjected to extreme drying from sun, wind and possibly frost. Yet, since new crops of conidia are produced on these tissues after rains commence, the fungus is able to survive long periods of dehydration (60).

It is generally accepted that fruit mummies, plus fruit peduncles, provides the most important source of the primary inoculum. Other sources may be cankers, twigs, and the remains of blighted blossoms from previous years. In many orchards, mummies and cankers are removed at pruning time, but fruit peduncles and blighted blossoms usually remain on the tree and have tended to be overlooked as source of inoculum (60,87). Kable (60) concluded that abundant sporulation takes place on the infected peduncles in the early growing season and the spores provide inoculum for blossom infection. Byrde (10) estimated that the plum and apple fruits infected with M. laxa and M. fructigena produced approximately 41-64 pustules per mummy respectively and each mummy could produce up to $4 \times 10^6$ conidia. Shepherd (85) estimated that an average peach fruit mummy has the potential to produce $10^6$ viable conidia of M. fructicola during early spring. Infected peduncles when incubated in moist chambers, produced $1.9 \times 10^4$ to $9.8 \times 10^4$ conidia, with 92% viability (85).

Apothecia of M. laxa are extremely rare. Aderhold and Ruhland (3) described the spbcies on the basis of a few apothecia. Wormald and Harrison (38,39,103,104) described apothecial development in England, but no apothecia of this fungus have been reported in the United States (102). Apothecia of M. fructicola develop in areas where the soil are protected from drying by weeds or debris. Ezekiel

(30) reported that a soil with a pH below 7.0 favors apothecial development, whereas an alkaline soil does not. Norm Dobie found apothecia in a peach orchard near Albany (unpublished data). Only one report of the production of apothecia by M. fructicola in culture has been published by Japanese workers (11). The most detailed morphological study of apothecial development by the brown rot fungi is that of Norton et al. (67). Huber and Baur (7) concluded from their investigation in western Washington that apothecia of M. fructicola were found in prune orchard, but apothecia have not been found originating from cherry mummies.

The brown rot disease of stone fruit caused by Monilinia species normally reach epidemic proportions during bloom and when the fruit is fully mature (92). A film of moisture is required for spore germination. This moisture may be provided by injured host tissue or by rain, dew or fog (22). Fruit may become infected due to skin splitting and insect damage. Conidia may enter through an injured fruit tissue, or they may germinate and penetrate the uninjured epidermis (56). Short and long term latent infection and quiescent infection have been recognized by many workers (2,49,53,56,78). Despite the use of protectant spray schedules, incomplete control of the disease on peaches, following a season of severe blossom infection (61). To account for these facts, a latent infection of green fruits has been postulated. Evidence of latent infection on apricots has been recorded (53,56,61), but no data on cherries. Jenkins and Reinganum (53) proposed a quiescent infection as distinct from latent infection because the pathogen induces a reaction large enough to be seen. Gauman (32) specifies that a latent infection is

one which produces no symptom. Simmond (84) described Gleosporium musarum, the anthracnose fungus of bananas, as causing latent infection of green fruit. Powelson (78) showed that Botrytis cinerea can cause incipient infection on strawberry. Adams, et al. (2) described a similar feature of the infection of oranges by Colletotrichum gleosporioides. Establishment of latent infection by stomatal penetration was described for Botrosphaeria ribis on avocado by Horne and Palmer (49). Several workers deduced from laboratory studies that direct infection could occur in noninjured peach fruit (61). However, Corbin (21) suggested that fruit injury explains the establishment of most brown rot infection in the field.

The young green fruit ordinarily is fairly resistant to attack, but some infection may follow in the wake of driving rain and hailstorms causing wounds (22). When the fruit is fully grown and begins to ripen, the susceptibility increases markedly. At this time the fruit is undergoing pronounced physiological and anatomical changes which may account for its decrease in resistance. Young rapidly growing fruit may heal potential infection sites more rapidly than mature fruit (56). Wade and Jerome (56,92) recorded loss of disease resistance to M. fructicola (Wint) Honey in fruit of prunus spp. as they develop from the green pre-pit hardened stage to the post-pit hardened stage. Jerome (56) showed the relation between fruit skin injuries and infection on mature and near-mature peaches. As fruit mature, however, the lag phase shortens and there is an increasing possibility of epiphytotics occurring (20,56). A linear relation was found between the growth rate and utilization of soluble

solids by brown rot fungi (35). Making use of this index, cherry varieties were tested for determining resistance against the brown rot organism. Using the low soluble solid utilization index of the two brown rot fungi, Grover (35) reported that the cultivars Karassa severa and Griotte du pay possessed natural resistance to brown rot.

# MATERIALS AND METHODS

## I. A. Disease Development Caused by *Monilinia* spp.

A survey of the *Monilinia* species that attacks sweet cherries in Oregon was carried out in orchards located (Linn and Yamhill counties) in the Willamette Valley. During these surveys, collections were made from blossoms, rotten fruit and from mummified fruits that remained on the trees. Cultures of *Monilinia* were obtained from blighted blossoms collected from randomly selected trees in each orchard from 'Corum', 'Napoleon' and 'Black Republican' cultivars. One thousand blossoms were collected from each tree. Ten different trees were used each time when an orchard was sampled. *Monilinia* cultures were also obtained from fruits collected randomly prior to harvest according to the same sampling procedures. Samples were brought to the laboratory, surface sterilized in .05% sodium hypochlorite (10% chlorax) for 5-10 minutes, then in 70% alcohol for two minutes. The tissues were rinsed with distilled water for less than one minute and plated on a selective media developed by Phillip (75), and on freshly prepared 2% potato dextrose agar. Plates were incubated at 24°C temperature.

The cultures of *Monilinia* spp. obtained in this study, were identified by their cultural characteristics on 2% PDA. The characters used such as lobing of the colony margins, branched germ tubes of *M. laxa*, and smooth entire margin, straight germ tubes of *M. fructicola*. The common occurrance of anastomosis when isolates of *Monilinia* spp. were mixed as reported by earlier workers (38,70,99,103).

Field assessment of brown rot blossom blight of sweet cherry cultivars was started in 1979, at the plant pathology field lab, then was expanded to three locations in 1980, and four locations in 1982. Brown rot blossom blight development during 1981 was assessed at 3 stages of bloom development in four locations in western Oregon under orchard conditions. This evaluation was based on the number of blighted blossom from 10,000 flowers collected at random from ten trees in the orchard for each cultivar. At harvest, fruit was collected with the same sampling procedures described earlier during the blossom period. The fruits were not collected from the same tree twice. The apparent infection rate (r) was calculated from disease incidence during bloom period by using $(\log_e \frac{1}{1-X})$ due to slow multiplication and secondary spread of inoculum to cause new blossom infection. During fruit ripening (r) was calculated from the incidence of fruit rot use $(\log_e \frac{X}{1-X})$ because of fast sporulation and subsequent secondary spread to cause further fruit infection.

## B. Comparative Studies of *M. laxa* and *M. fructicola*

Cultures of both *Monilinia* spp. were obtained from infected parts of stone fruit trees. Mycelial agar discs, 5 mm in diameter of both *Monilinia* species were inverted and transferred to the center of five plates of 2% PDA replicated twice. Colonies diameter to measure the growth rate were started after 48 hours and then on a daily basis for five days. The effect of temperature on growth rate was determined at 15, 20 and 24°C using 10 plates per experiment for each species.

Conidia for germination tests were obtained from 7 days' old cultures of M. laxa and M. fructicola growing on oatmeal agar. 200 conidia adjusted by means of hemacytometer were seeded on 2% water agar. Five plates were used per each test and each test was replicated three times. The plates were incubated at 24°C temperature during germination. Germ tube counts were started at four hours and continued for 24 hours.

A sporulation study of the two species was made by placing mycelial agar discs from a 2-5 days old culture on oatmeal agar, V-8 media, prune, peach and cherry agar. Conidia were counted by placing a block of the sporulating fungus one centimeter square in 10 ml of distilled water. A ml of the spore suspension was counted by means of hemacytometer. Five plates, replicated twice, were used for each media mentioned above.

Resistance to Benomyl

Commercially prepared benomyl (Benlate 50 WP, E.I. DuPont De Nemours and Company, Wilmington, DE 19898) was suspended in water and appropriated quantities of the suspension were added to 2% (PDA) at 45°C to give concentrations of 0.5, 1, and 5 ppm. After the addition of benomyl, 20 ml of the media was poured into each petri dish. Isolates were obtained from sporulating lesions on the blighted blossoms and fruits of sweet and sour cherry, prune, 'Elberta' and 'Red Haven' peach cultivar. Growth on benomyl-amended PDA was determined by inoculating petri plates with 5 mm diameter agar discs' taken from four days old cultures of the

indicated isolates of M. laxa and M. fructicola growing on freshly prepared PDA. The discs were inverted and transferred to the center of five plates at each benomyl concentrations, and to five plates of PDA used as a control. The test was repeated twice. In all tests, measurements of growth were made at 48 hours and then on a daily basis until the fungus growth in the PDA media reached the edge of the petri dish.

Germination on benomyl amended PDA was determined by seeding a 200 conidia per ml mixture from each of the isolates mentioned above. Five plates were used in each test. Each test was repeated twice. The percentage of conidia that germinated was counted after six and 12 hours of incubation at 24°C. Benomyl concentrations used in this test were .5, 1, and 5 ppm.

Supplementary test of benomyl tolerance were done to investigate the effect of this fungicide used as a blossom spray against both of the Monilinia that cause blossom blight. A total of 100 blossoms that were between the popcorn and full bloom stage were selected from three sweet cherry cultivars (Corum, Napoleon, Black Republican) at the Botany and Plant Pathology Field Laboratory. The blossoms were covered with plastic sheets and attached into a metal rack in crisper boxes. The boxes had been filled with one cm of water at the bottom, and were kept at 24°C. The blossoms were sprayed with benlate at 4 oz/100 gallons, the recommended rate for field application. One hundred blossoms were sprayed with distilled water as check. Forty-eight hours after the benlate spray, a conidial suspension of 50,000/ml of both M. laxa and M. fructicola, obtained

from cultures grown on oatmeal agar, were sprayed on the blossoms until run off. The blossoms were incubated at 24°C for five days. Blighted blossoms were determined by visual observation of necrosis on the petals and caylax lobes.

Conidia obtained from these infected blossoms were transferred into benlate amended media at concentration of .5, 1, and 5 ppm to measure the radial growth to determine if any resistance had developed to benomyl during the experiment.

Comparative Pathogenicity of M. laxa and M. fructicola Using Prunus Cultivars

Blossom Inoculation: Conidia for inoculation were obtained from a seven-day-old culture grown on oatmeal agar. The conidia of M. laxa and M. fructicola were inoculated into both unopen and open blossom clusters. Conidia suspension of 50,000/ml of water were counted by the hemacytometer. The twigs bearing blossom clusters were selected and dipped in 70% alcohol for two minutes then rinsed with distilled water. The spore suspension was applied with an atomizer until run off and the blossoms were placed on metal racks in a food crisper box with one cm of water at the bottom to prevent drying. The boxes then were incubated at 24°C. In all tests, each of the cultures producing conidia originated from a single conidia transfer. Blossom inoculation experiments were repeated during two successive bloom periods.

Blossom inoculations of three sweet cherries cultivars were conducted under field conditions in 1981. Each inflorescence that was selected contained recently opened flowers. Five hundred flowers

from each cultivar were inoculated by atomizing a 50,000 conidia/ml suspension of both M. laxa and M. fructicola. Blighted blossoms were counted seven days after inoculation on the basis of the symptoms on the petals and calyx lobes. Symptoms consisted of necrosis in the pedicles and the formation of the conidia on the flower cups.

Twig Inoculation: One-year-old twigs of commercial sweet cherry cultivars, sour cherry, 'Elberta' and 'Red Haven' peaches, were inoculated with single spore isolates of M. laxa and M. fructicola growing on 2% PDA. Inoculations were made also on seven stone fruits with isolates that originated on its respective host. Current year twigs from sweet cherry seedlings were inoculated with eight single spore isolates of M. laxa and two of M. fructicola. Inoculation of the sweet cherry seedlings were made in the greenhouse twice over a period of two years. In all twig inoculations, five-millimeter mycelium plugs, from four-day-old cultures, were inserted into a 2 cm slit made by a sterile scaple. The slit was wrapped with parafilm in order to maintain humidity. The parafilm was removed seven days after inoculation. The data was taken by measuring the canker length after 15 days. Six months later, these inoculated twigs were brought into the laboratory to assess the sporodochia development.

Fruit Inoculation: One hundred healthy fruit each from all three cultivars of sweet cherry, 'Corum', 'Napoleon' and 'Black Republican' were collected two weeks before harvest in 1980 and 1981, from non-sprayed trees at the Botany and Plant Pathology Field Laboratory. The fruits were surface sterilized in 70% alcohol for five minutes and then were rinsed with distilled water. Fruits were inoculated by atomizing

conidial suspensions of 50,000 conidia/ml of M. laxa and M. fructicola until run off. The fruits were arranged in crisper boxes with wet paper towels at the bottom and incubated at 24°C.

Fruit inoculation was also made on Elberta peach fruit. The Elberta peach fruits were inoculated by transferring 5 mm diameter mycelial plugs from the edge of eight active growing single spore isolates of M. laxa and two of M. fructicola. Fruit of Elberta peach were collected at random from non-sprayed trees starting three weeks before harvest and continued for two weeks. Holes were made by a 5 mm in diameter cork borer on ten fruits. Mycelial plugs were inserted and then the holes were sealed with adhesive tape. The fruits were incubated in crisper boxes with a wet paper towel at the bottom, at 24°C. Results in inoculated cherries were recorded as the number of rotten fruit with sporulation after four days. In 'Elberta' peach, lesion diameter and sporulation intensity were recorded.

## II. Intraspecies Variability of M. laxa Isolates in Oregon

M. laxa isolates were obtained from infected parts of sweet cherry cultivars ('Corum', 'Napoleon', 'Black Republican', and 'Bing'), sour cherry 'Montmorency', 'Red Haven' and 'Elberta' peaches and 'Early Italian' prune. Freshly prepared potato dextrose agar (PDA) was used routinely as the culture media. Prune agar was prepared from pitted 'Early Italian' prune. Each 1 lb. of pulp was diluted with

500 ml water and boiled gently for two hours. The resulting extract was strained through 2 layers of cheesecloth. Peach agar was prepared from pitted 'Elberta' peach fruit by the same procedures. Cherry agar was prepared from 500 g of cherries pulps added to 1000 ml water. 300 ml of cherry extract was added to 700 ml water and 20 g agar and sterilized at 110°C for one hour.

To measure the radial growth of M. laxa isolates 5 mm mycelial discs were transferred into the center of five plates from each media replicated twice. Colony diameter was measured seven days from the transfer time. The plates were incubated at 24°C. In germination test, 200 conidia from each isolate were seeded on 2% water agar. The percent of germinated conidia were counted after six hours on the basis of the presence of branched germ tubes. Sporulation was determined by transferring mycelial discs on freshly prepared 2% PDA twice. Conidia formation were measured by the number released by atomizing each plate with 10 ml of distilled water. One ml of the resulting conidial suspension was counted by means of a hemacytometer. Conidial production of these isolates was also tested by inoculating ripe 'Elberta' peach fruit. Five mm mycelium plugs were inserted into surface sterilized peach fruits. Conidial production on the lesion induced by the fungus was evaluated 72 hours after inoculation, half of each rotted fruit was washed in 50 ml water, and a one ml sample was counted by using a hemacytometer.

M. laxa isolates were mixed in different combination to determine if anastomoses occurred. The mycelium was investigated microscopically where two isolates comes in contact. They were also observed to deter-

mine if sclerotia like bodies developed when these culture aged. In addition to the isolates obtained from Oregon's orchards a California isolate was obtained from Ogawa and another isolate was obtained from the U.S.D.A. Mycology Lab., Beltsville, MD.

Pathogenicity tests to compare M. laxa isolates were carried out by means of single conidia cultures. The isolates were grown on 2% PDA for four days. Inoculations were made into one-year-old twigs of sweet cherry seedlings in the greenhouse, and commercial cherry cultivars under field conditions. Five mm mycelium discs were transferred into a 2 cm slit made on each of ten twigs. The twigs were wrapped with parafilm to keep the inoculated area moist. The data were taken after 15 days by measuring the canker length as criteria for pathogenicity. Sporodochia production on the inoculated twigs were assessed six months later. The pathogenicity test also involved the inoculation of 'Elberta' peach fruits at different stages of maturity. In this test, lesion diameter (size) developed by each isolate plus sporulations intensity were assessed 72 hours after inoculation.

## III. Aspects of the Disease Cycle of Brown Rot on Sweet Cherry

### A. Source of Primary Inoculum

Mummies and tagged blighted spurs were collected from the trees and the ground at the Botany and Plant Pathology Field Laboratory during the winter of 1980 and 1981. The mummies and the blighted

spurs were brought to the laboratory at monthly intervals. The sporulation on these mummies or twigs and the germinability of conidia was evaluated from the middle of December to the middle of March. The germination of conidia was measured by seeding approximately 200 conidia on each of five plates of 2% water agar. The percentage of germination was determined by the presence of germ tubes after six hours incubated at 24°C. Sporulation was determined by the tufts of conidia on the mummies or the spurs.

In 1981, 300 spurs were counted on each of ten trees selected at random from three sweet cherry cultivars ('Napoloen', 'Corum', and 'Black Republican') at the Botany and Plant Pathology Field Laboratory. Another 2,000 spurs were collected at random from 'Napoleon' and 'Corum' trees at two other locations (Lewis and Brown Horticulture Farm and Conklin Orchard). The blighted spurs were brought to the laboratory and divided into two parts. One part was thoroughly surface sterilized in .05% sodium hypochlorite (10% chlorax) for 10 minutes without rinsing, to determine whether the fungus on the surface or within the spur tissues. These blighted twigs were incubated at 24°C inside crisper boxes with a wet paper towel at the bottom. The second part was not surface sterilized and was incubated as mentioned above. Counts were made 36-40 hours later to assess the sporodochia development on the blighted blossoms and the fruit peduncles attached to those spurs.

B. Latent Infection

A study was carried out during springs of 1980 and 1981 to

investigate the phenomona of latent infections of sweet cherry fruit under Oregon conditions. In earlier works (53,56,61) the suggestion was made that latent infection induced by M. fructicola on apricots and peaches occurred early during fruit development, but such infection did not develop and cause rotting until the fruit matured. Samples of 100 green fruit was collected at random from 'Corum', 'Napoleon' and 'Black Republican' sweet cherry cultivars at the Botany and Plant Pathology Field Laboratory. The fruit was surface sterilized in .05% sodium hypochlorite (10% chlorax) for five minutes and then in 70% alcohol for two minutes. A corresponding sample of 100 fruits was surface sterilized and sprayed with 50,000 conidia/ml until run off. A third sample of 100 fruits was kept without surface sterilization. Checks were sprayed with equal volumes of distilled water. The samples were placed inside sterile crisper boxes with a wet paper towel at the bottom. The boxes were kept at 24°C. Fruits which showed small brown lesion were excised for histological studies. The tissue was fixed in FAA (50% (or 70%) ethyl alcohol, 90 cc, glacial acitic acid, 5 cc and Formalin, 5 cc) (57). The tissue was then dehydrated and embedded in paraffin. The sections, 12 μ in thickness, were immersed in parasol (Stockwell's solution) to remove heavy deposities in the necrotic areas. The sections were put in a warm oven at 32°C overnight and then stained in safranin and fast green (57). Samples of tissue were also taken from fruit showing sunken light brown spots and plated on freshly prepared 2% PDA.

Another experiment during study was made to determine if Monilinia

propagules could remain on the fruit surface, especially at the concaved stem end. 10 ml of sterile water was used to wash each stem ends from 25 green fruits of three sweet cherry cultivars. One ml of the suspension was plated on both fresh 2% PDA and on a selective media for Monilinia species (75).

Another sample of 100 fruits was taken from the treated trees with fungicide (benlate) during bloom period. This was done to determine if benlate treatments had an effect on the incidence of green fruit rot (to support the hypothesis of latent infection). The fruit samples were treated the same way as mentioned earlier. Samples were taken from ('Corum', 'Napoleon', and 'Black Republican') sweet cherries.

Fruit samples were also collected three weeks before harvest from four orchards to assess the latent infection during fruit ripening. One hundred fruit samples were used from both 'Corum' and 'Napoleon'. One hundred fruits from each cultivar were surface sterilized in 70% alcohol for five minutes rinsed with distilled water, then incubated inside crisper boxes at 24°C. Another sample the same size was incubated without surface sterilization. Counts made on the number of fruits showing brown rot symptoms after four days of incubation.

## C. Relationship of Fruit Soluble Solid Content With Fruit Rot Development

A survey of the cherry orchards located in Linn-Benton, Yamhill and Marion counites were carried out to investigate the stage of fruit development in relation to brown rot symptom. Sweet cherry fruits from

both 'Corum' and 'Napoloen' were used. Field assessment of brown rot was determined by sampling 1,000 fruits from each tree, from a total of ten trees for each cultivar. Any given tree was sampled only once. Sampling started four weeks before harvest and continued through harvest on a weekly basis. The samples were brought into the laboratory and separated on the basis of brown rot symptoms to give the approximate percentage of the disease incidence under orchard conditions.

Samples of 100 sound healthy fruits were used for inoculation with M. laxa under the laboratory conditions. The fruits were surface sterilized in .05% sodium hypochlorite for ten minutes, dipped in 70% alcohol for two minutes then rinsed with distilled water. The fruits were inoculated by means of spraying 50,000 conidia/ml suspension applied until run off. Checks were sprayed only with water following surface sterilization. The fruits were incubated in crisper boxes at 24°C. Moisture was provided by means of a wet paper towel at the bottom of the boxes. Fruit inoculation was made every week starting approximately four weeks before harvest. To measure the soluble solid content (%) of the fruit, 1/2 ml of fruit juice was measured by Zeiss hand refractometer. Twenty fruits at random from each sample were measured before inoculation, then another 20 fruits were measured after fruit rot developed to determine how much soluble solid had been utilized by M. laxa.

# RESULTS

## I. A. Disease Development Caused by Monilinia spp.

A survey of 4 orchards in western Oregon (Willamette Valley) were made in order to determine the Monilinia species causing cherry blossom blight and fruit rot. Cultures of Monilinia species were obtained by planting tissue from infected blossoms, spurs, fruit peduncles and mummies. Although a number of different isolates were found, most of the isolates were easily identified as either M. laxa or M. fructicola. Using such characters as lobing of colony margins, branching of germ tubes and presence of anastomosis that were widely accepted to differentiate between the brown rot fungi (69,103). As many as 100 different isolates were obtained from commercial orchards during the bloom and at fruit ripening stage. The relative occurrance of M. laxa and M. fructicola during a three year period is reported in this study (Tables 1, 2). M. laxa was isolated more frequently in all of the sampling sites. The percentage of M. fructicola was always low (Tables 1, 2).

The development of brown rot blossom blight on sweet cherry cultivars was evaluated under field conditions. Data were based on the examination of 10,000 blossoms each sampling period. The result of blossom infection in 1979 (Table 3) showed a high disease incidence relative to 1980 and 1981 seasons. Rainfall was 3.29 during the blossom period which interferred with bloom spray schedules between the popcorn stage and petal fall of both 'Black Republican' and 'Corum' cultivars. During 1981 blossom infection was relatively

Table 1. Percentage of *Monilinia* spp. isolated from infected blossoms of sweet cherry cultivars in the Willamette Valley of Oregon during 1979-1981 bloom periods.

| Location (counties) | Source of Isolation (host) | % Colonies (of *M. laxa* and *M. fructicola*) Isolated from Infected Blossoms 1979 *M. laxa* (%) | 1979 *M. fructicola* (%) | 1980 *M. laxa* (%) | 1980 *M. fructicola* (%) | 1981 *M. laxa* (%) | 1981 *M. fructicola* (%) |
|---|---|---|---|---|---|---|---|
| Plant Path. | Corum | 94 (1) | 6 | 89 | 11 | 90 | 10 |
| Field Lab. | Napoleon | 95 | 5 | 93 | 7 | 93 | 7 |
| (Linn) | Black Republican | 92 | 8 | 94 | 6 | 96 | 4 |
| Lewis Brown | Corum | - (2) | - | 91 | 9 | 92 | 8 |
| (Linn) | Napoleon | - | - | 93 | 7 | 94 | 6 |
| Conklin | Corum | - | - | 91 | 9 | 90 | 10 |
| Orchard | Napoleon | - | - | 94 | 6 | 92 | 8 |
| (Yamhill) | | | | | | | |
| Schindler | Corum | - | - | - | - | 94 | 6 |
| Orchard | Napoleon | - | - | - | - | 93 | 7 |
| (Marion) | Black Republican | - | - | - | - | 95 | 5 |

(1) Percentage of cultures isolated from blighted blossoms.
(2) Sampling not done.

Table 2. Percentage of Monilinia spp. isolated from rotten fruit of sweet cherry cultivars in the Willamette Valley of Oregon during 1979-1981 fruit harvest period.

| Location (counties) | Cultivars | % Colonies Isolated From Rotten Fruit 1979 M. laxa | 1979 M. fructicola | 1980 M. laxa | 1980 M. fructicola | 1981 M. laxa | 1981 M. fructicola |
|---|---|---|---|---|---|---|---|
| Plant Path. | Corum | 83 (1) | 17 | 89 | 11 | 91 | 9 |
| Field Lab. | Napoleon | 89 | 11 | 83 | 17 | 95 | 5 |
| (Linn) | Black Republican | 85 | 15 | 88 | 12 | 96 | 4 |
| Lewis Brown | Corum | - (2) | - | 89 | 11 | 90 | 10 |
| (Linn) | Napoleon | - | - | 85 | 15 | 95 | 5 |
| Conklin | Corum | - | - | 89 | 11 | 94 | 6 |
| Orchard | Napoleon | - | - | 88 | 12 | 95 | 5 |
| (Yamhill) | | | | | | | |
| Schindler | Corum | - | - | - | - | 93 | 7 |
| Orchard | Napoleon | - | - | - | - | 95 | 5 |
| (Marion) | Black Republican | - | - | - | - | 92 | 8 |

(1) Percentage of cultures of both M. laxa and M. fructicola isolated from rotten fruit.
(2) Sampling not done.

Table 3. Field assessment of brown rot blossom blight of cherry cultivars under orchard conditions in the Willamette Valley.

| Location (counties) | Host | % of Infected Blossoms (1979) | (1980) | (1981) |
|---|---|---|---|---|
| Plant Pathology | Corum | 12.8 (1) | 4.43 | 5.20 |
| Field Lab. | Napoleon | 24.0 | 3.91 | 5.25 |
| (Linn) | Black Republican | 19.5 | 17.35 | 6.30 |
| | Montmorency Sour Cherry | 34.0 | 29.00 | 14.00 |
| Lewis Brown Farm | Corum | - (2) | 6.90 | 3.60 |
| (Linn) | Napoleon | - | 14.50 | 2.20 |
| Conklin Orchard | Corum | - | 5.60 | 4.10 |
| (Yamhill) | Napoleon | - | 9.60 | 3.10 |
| Schindler Orchard | Corum | - | - | 17.20 |
| (Marion) | Napoleon | - | - | 8.00 |
| | Black Republican | - | - | 54.40 |

(1) Based on number of infected blossoms out of 10,000 collected at random from ten trees per orchard.
(2) Sampling not done.

low in the orchards at Linn and Yamhill counties. However, blossom infection was higher at the Schindler orchard located in Marion county. This particular orchard was about 300 yards from the river, and surrounded by hills. (Note: Total amount of rain was 2.40 inches between the first and the third week of April with an average temperature of 17°C). This weather was favorable for blossom infection by the fungus in cherry bloom. This orchard was 10 days earlier than the other orchards used in these experiments and the bloom spray was delayed due to the rain during the bloom period.

Brown rot blossom blight development during 1981 was assessed at popcorn, full bloom and petal fall stages under field conditions. The percentage of blossom blight traces the progression of disease symptoms during bloom (Table 4). The blossom blight was more on 'Corum' than 'Napoleon' in the Linn county orchards. At the Schindler orchard, blossom blight symptoms were more severe on 'Napoleon' than 'Corum'. There was a linear relation between the percentage of blossom blight ($\log_e \frac{1}{1-X}$) and the stage of the bloom developments. The calculated apparent infection rate of the disease progress during the bloom period was (r. 005/day) for 'Corum' in the Linn and Yamhill county orchards. However, it was (r. 056/day) at the Schindler orchard. In 'Napoleon' the apparent infection rate was (r .003/day) in the Linn and Yamhill county orchards, but it was (r .042/day) at the Schindler orchard.

The fruit rot phase of the disease was assessed three weeks before harvest in 1981. The fruit samples were evaluated on the

Table 4. Brown rot blossom blight development during 1981 in the Willamette Valley.

| Location (counties) | Sweet Cherry Cultivars | % of Infected Blossoms Stage Development | | |
|---|---|---|---|---|
| | | Popcorn | Full Bloom | Petal Fall |
| Plant Pathology Field Lab. (Linn) | Corum | 2.9* | 7.0 | 13.0 |
| | Napoleon | 0.3 | 0.7 | 1.5 |
| | Black Republican | 1.4 | 3.1 | 5.8 |
| Lewis Brown Farm (Linn) | Corum | 0.3 | 2.1 | 3.6 |
| | Napoleon | 0.6 | 0.8 | 2.2 |
| Conklin Orchard (Yamhill) | Corum | 1.1 | 2.6 | 4.1 |
| | Napoleon | 0.3 | 1.4 | 3.1 |
| Schindler Orchard (Marion) | Corum | 5.6 | 10.6 | 17.2 |
| | Napoleon | 12.0 | 43.0 | 54.4 |

(*) Based on examination of 10,000 blossoms collected at random from 10 trees in each orchard.

basis of brown rot symptoms followed by isolation from the infected areas. The percentage of fruit rot was the highest at the Schindler orchard (Table 5). The fruit rot incidence was higher on 'Corum' than 'Napoleon' in all locations except the Linn county site (Lewis Brown Farm) (Table 5). Showery weather with 2.40 inches rainfall during the third week of June and the 4th of July, caused fruit cracking on 'Corum' more than 'Napoleon' due to the earlier maturity of 'Corum'. The split fruit provided a good infection court for the brown rot conidia.

The apparent infection rate for infected fruit was calculated based on the percentage of fruit rot during the 3 week sampling period, prior to harvest (Table 5 and Figures 1, 2). The percentage of the infected fruit was low during the first sample, but increased rapidly as harvest approached (Table 5). Showery weather starting in the third week of June caused cracking and fruit split, and also provided enough moisture for germination of conidia on the remains of blighted blossoms remained on the trees. There was some differences between the orchards in the amount of the disease during sampling period (Table 5).

## B. Comparative Studies Between *M. laxa* and *M. fructicola*

### 1. Colonies Characteristics

Differences were observed in the growth habit of *M. laxa* and *M. fructicola* on fresh prepared 2% potato dextrose agar, prune and peach agar. The most characteristic feature of *M. laxa* was the

Table 5. Brown rot development on sweet cherry cultivars during the 1981 fruit ripening in the Willamette Valley.

| Location (counties) | Sweet Cherry Cultivars | % of Brown Rot on Fruit Before Harvest 3 weeks | 2 weeks | 1 week |
|---|---|---|---|---|
| Plant Pathology Field Lab. (Linn) | Corum | 2.40 (1) | 4.15 | 15.00 |
| | Napoleon | 1.97 | 4.41 | 9.33 |
| Lewis Brown Farm (Linn) | Corum | 7.20 | 15.71 | 22.20 |
| | Napoleon | 1.60 | 6.14 | 28.72 |
| Conklin Orchard (Yamhill) | Corum | 5.95 | 16.25 | 21.50 |
| | Napoleon | 1.60 | 11.50 | 12.50 |
| Schindler Orchard Grand Island (Marion) | Corum | 28.75 | 57.00 | 63.00 |
| | Napoleon | 32.00 | 44.00 | 49.00 |
| Ron Schindler Orchard (Yamhill) | Corum | 29.80 | 36.50 | 40.00 |
| | Napoleon | 5.00 | 8.00 | 12.00 |

(1) Based on examination of 10,000 fruit collected at random from ten trees in each orchard.

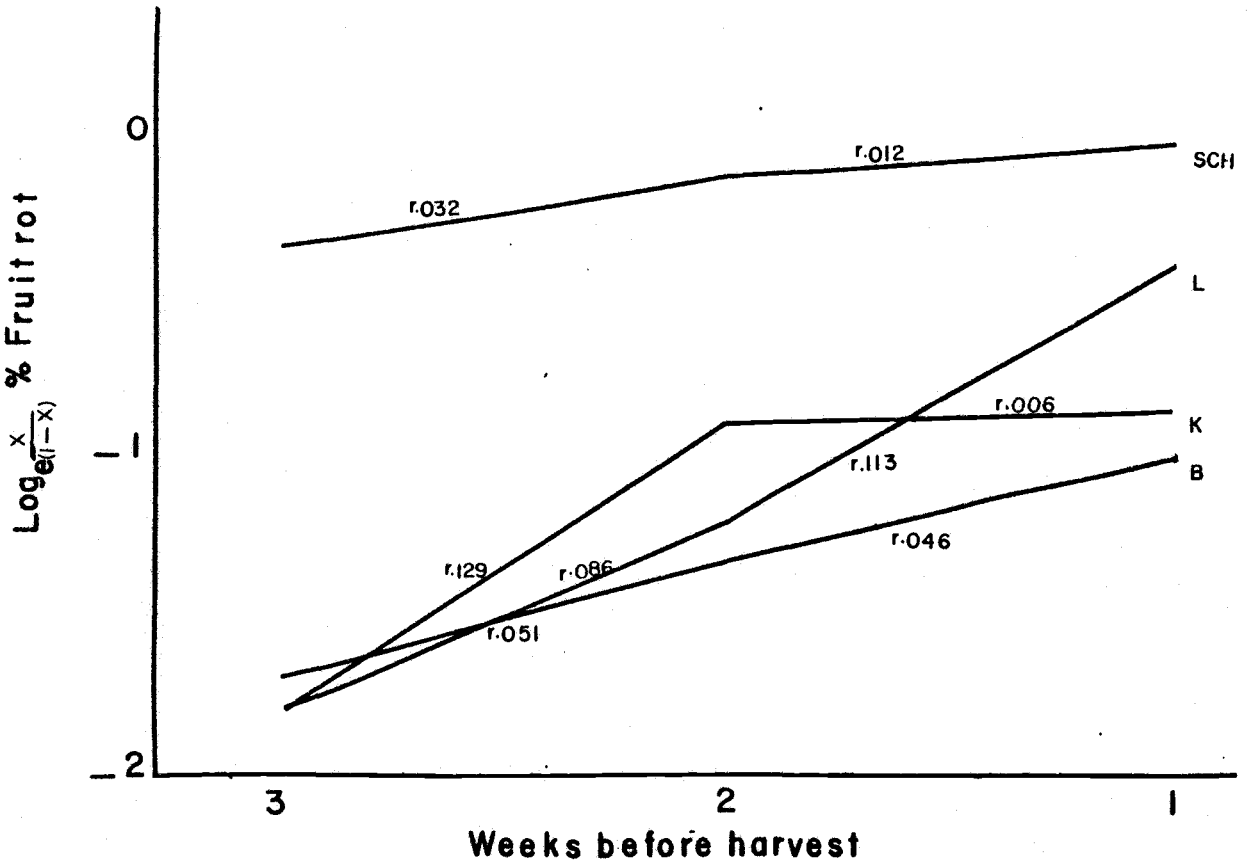


Figure 1. Apparent infection rate (r) for brown rot during ripening of sweet cherry 'Napoleon' cultivar at four locations in western Oregon measured as percentage of infected fruit. Sch = Schindler, L = Lewis Brown Farm, K = Conklin, B = Botany Farm

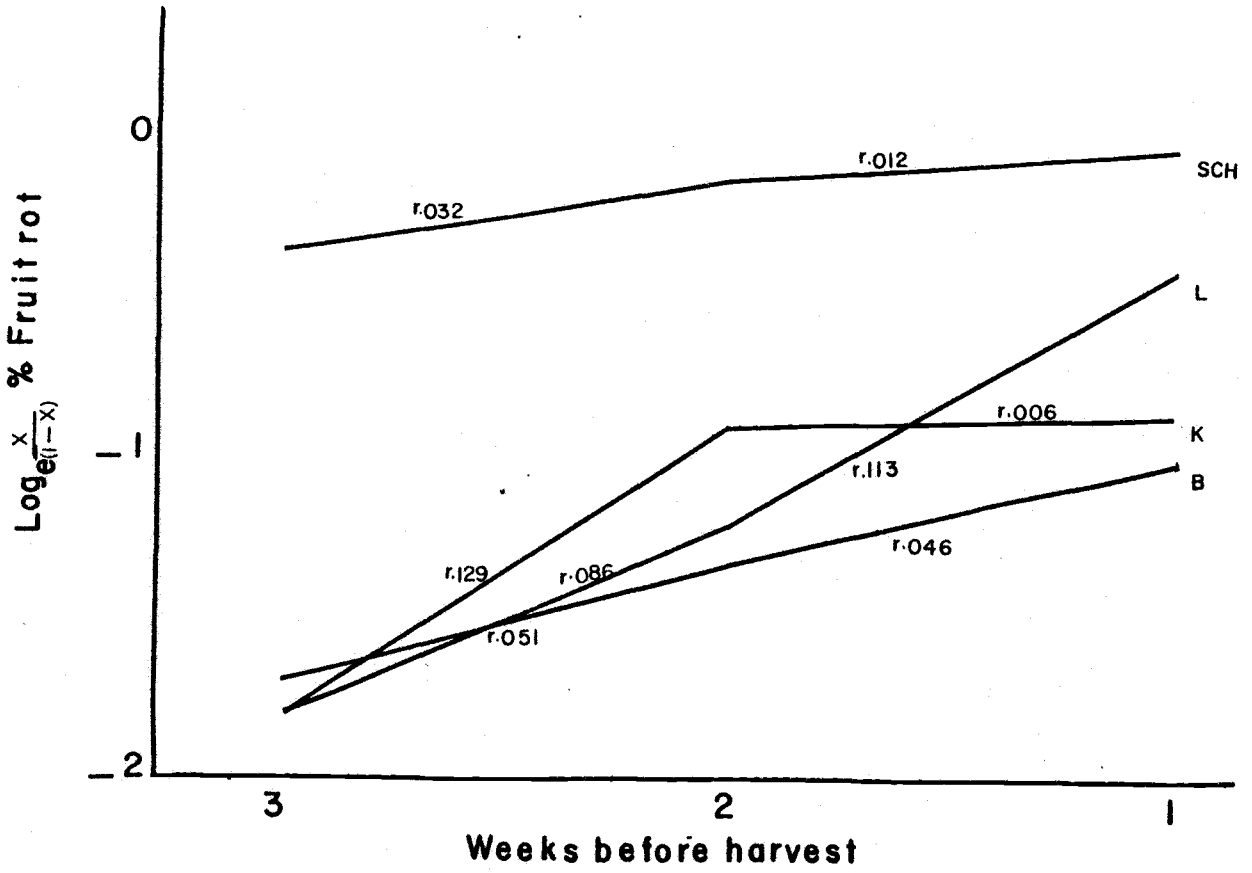


Figure 2. Apparent infection rate (r) for brown rot during ripening of sweet cherry 'Corum' cultivar at four locations in western Oregon measured as percentage of infected fruit. Sch = Schindler, L = Lewis Brown Farm, K = Conklin, B = Botany Farm

lobes or zonation type of radial growth. M. fructicola had a smooth entire margin type of radial growth (Figure 3). M. fructicola grew faster in 2% PDA than M. laxa. Anastomoses were lacking in M. laxa, while they were very common in M. fructicola. M. fructicola produced dark pigmented sclerotia like bodies when the culture aged. Production of such structure was lacking in M. laxa. Radial growth of M. laxa and M. fructicola showed significant differences in their colony diameter when both were incubated for seven days at the same temperature (Figure 4).

Temperature Relationship. The effect of temperature on the radial growth of M. laxa was studied and compared with M. fructicola. Each species was grown on freshly prepared 2% PDA, at temperatures of 15°, 20° and 24°C. Measurement of the radial growth was made every day starting two days after the initial transfer. Radial growth of both organisms was greatest at 24°C. In a seven day period, M. fructicola grew faster than M. laxa at all temperatures tested (Figure 4). Growth rate (colony diameter) at each temperature was significantly different between the two species at 5% level.

2. Germination

Conidia from M. laxa isolates frequently germinated with branched germ tubes. Straight germ tubes were more common in M. fructicola and branching usually started as the germ tube reaches 100 μ or more. Percentage of conidia germination of M. laxa were lower than M. fructicola (Table 6 and Figure 5). Conidia of M. fructicola germinated more readily in water agar than M. laxa. Conidia of both species, regardless

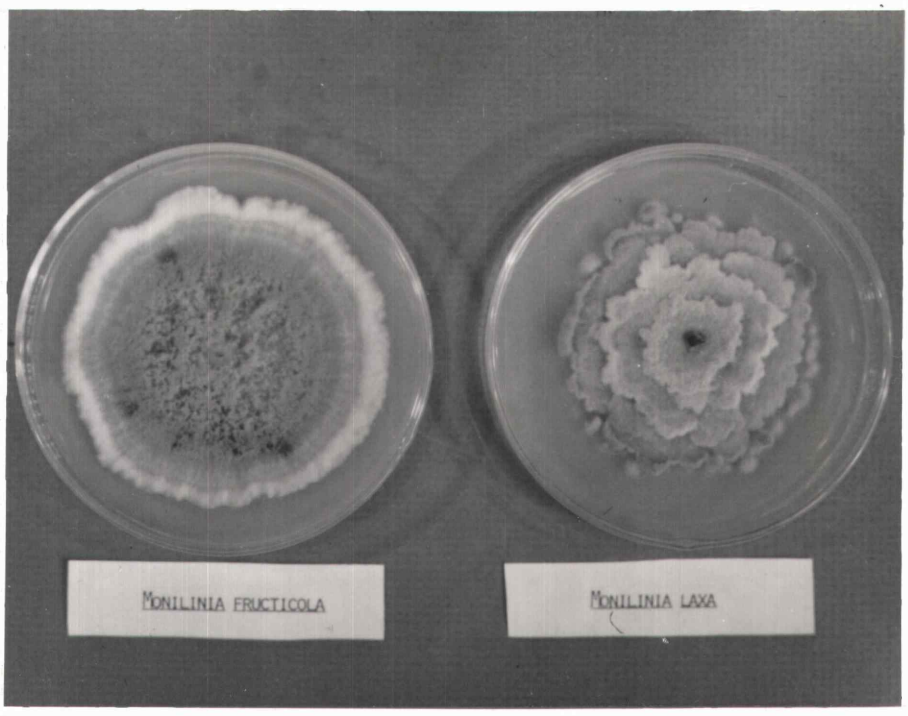


Figure 3. Colonies appearance of M. laxa and M. fructicola on 2% PDA.

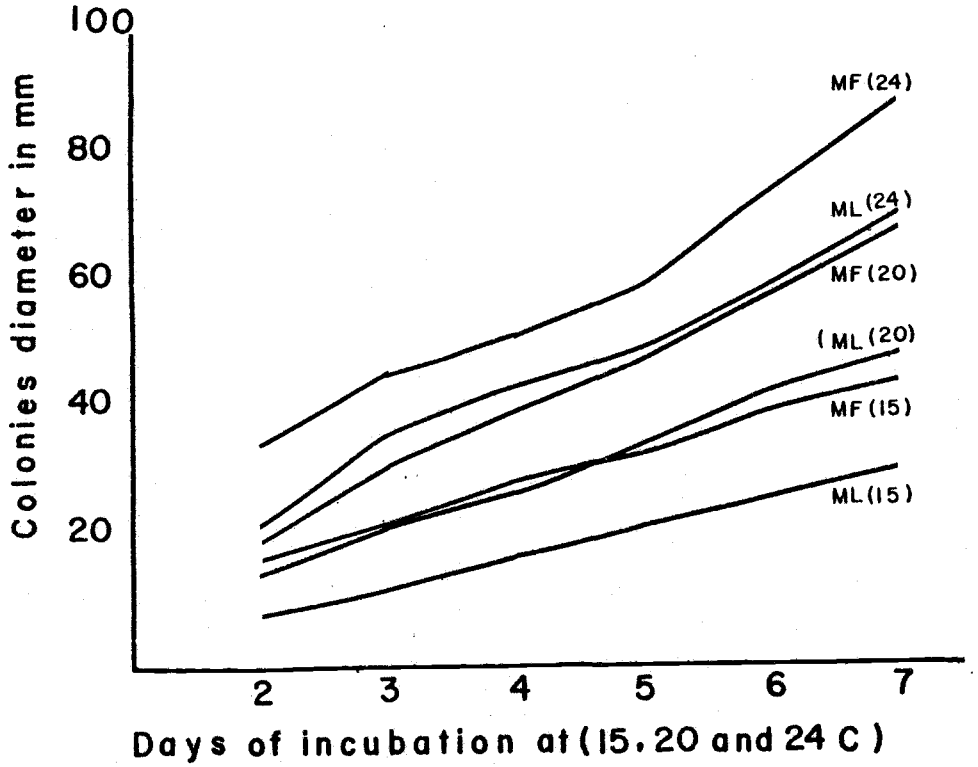


Figure 4. Effect of different temperature on the mycelial growth of M. laxa and M. fructicola.

Table 6. Germination percentage of *M. laxa* and *M. fructicola* incubated at 24°C temperature.

| Organism | % of Germination in Hours on Water Agar | | | | | |
|---|---|---|---|---|---|---|
| | 4 | 8 | 12 | 16 | 20 | 24 |
| *M. laxa* | 17a | 40a | 67a | 79a | 82a | 85a |
| *M. fructicola* | 20a | 50b | 75b | 80a | 90b | 95b |

Numbers followed by the same letter in each column do not differ significantly at P = .05, L.S.D. test.

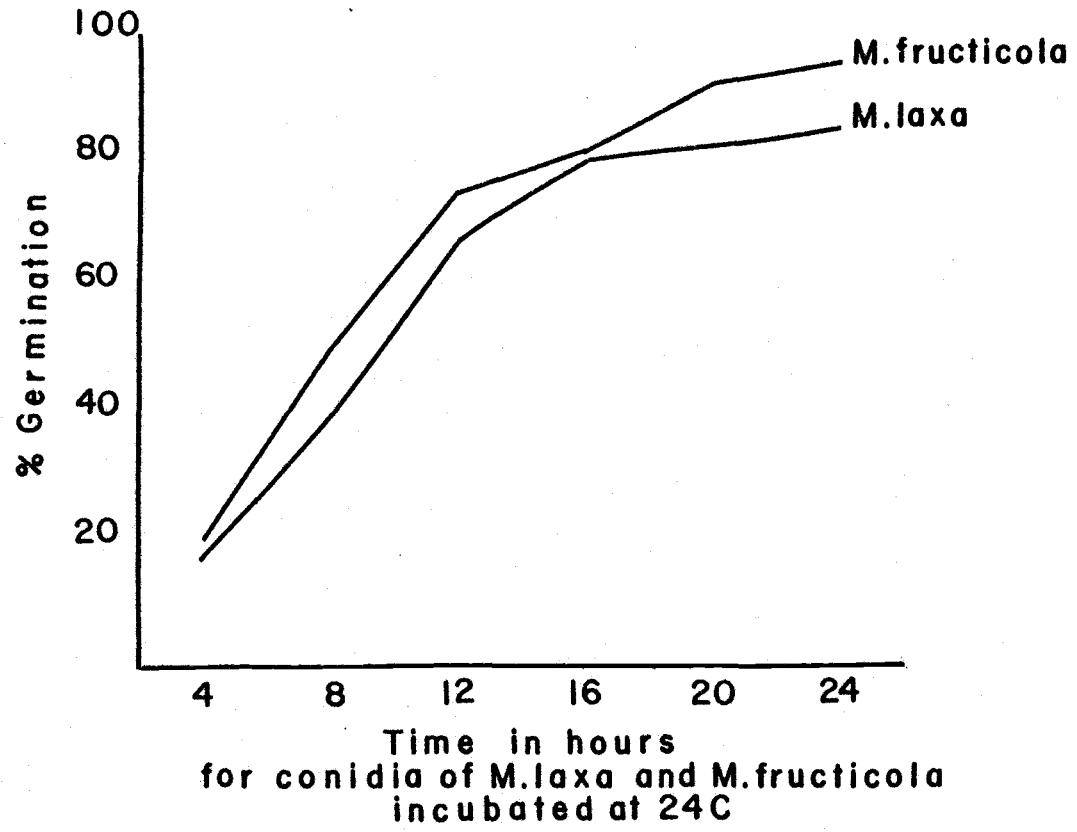


Figure 5. Conidial germination of both M. laxa and M. fructicola at the same temperature.

of their age, required at least four hours of incubation in water agar before germination. Germination of conidia were optimum at 24°C for both species. There was a significant difference in the percentage of conidial germination except after 4 and 16 hours of incubation (Table 6).

3. Sporulation

When the brown rot fungi was grown on oatmeal agar and incubated at 24°C in continuous light, aerial hyphae of M. fructicola began to form conidial chain after 48 hours, while M. laxa did not form conidia until 70 hours. However, when both species were grown on prune, peach and cherry agar, they sporulated earlier, M. fructicola relatively sporulated faster than M. laxa on these media. Production of conidia per square centrimeter was approximately $9 \times 10^4$ and $7.5 \times 10^4$ for M. fructicola and M. laxa respectively.

4. Resistance to Benomyl

In the presence of benomyl, at a concentration of .5 ppm neither the M. laxa nor M. fructicola isolates produced significant mycelial growth. Conidia of all isolates were able to germinate on benomyl amended PDA after 12 hours (Table 7), but germination was reduced from non-amended media. None of the isolate were able to germinate or produce significant mycelial growth at 5 ppm. After 12 hours of incubation at 24°C, conidia germination was reduced at 1 ppm more than .5 ppm.

Benomyl was sprayed on the blossom of sweet cherry at 300 ppm

Table 7. Effect of benomyl in PDA medium on the germination of conidia of M. laxa and M. fructicola isolates in Oregon.

| Isolates | % Germination of Conidia After 6 Hours on Media Containing Benomyl | | | | % Germination of Conidia After 12 Hours on Media Amended With Benomyl | | | |
|---|---|---|---|---|---|---|---|---|
| | 0.0 | .5 ppm | 1 ppm | 5 ppm | 0.0 | .5 ppm | 1 ppm | 5 ppm |
| M. laxa | | | | | | | | |
| C | 70(1) | 45 | 12 | 0 | 100a(2) | 85a | 42a | 0 |
| N | 72 | 42 | 15 | 0 | 100a | 82a | 40a | 0 |
| BR | 65 | 36 | 10 | 0 | 98a | 84a | 39a | 0 |
| S | 64 | 40 | 20 | 0 | 96a | 87a | 35a | 0 |
| B | 68 | 38 | 18 | 0 | 100a | 80a | 45a | 0 |
| P | 66 | 40 | 14 | 0 | 100a | 80a | 43a | 0 |
| E | 61 | 36 | 12 | 0 | 92a | 81a | 39a | 0 |
| RH | 59 | 35 | 18 | 0 | 96a | 79a | 38a | 0 |
| M. fructicola | | | | | | | | |
| E | 57 | 35 | 19 | 0 | 100a | 84a | 42a | 0 |
| RH | 62 | 40 | 22 | 0 | 100a | 85a | 46a | 0 |

(1) Average percent of conidia germinated on PDA out of 200 conidia/ml used per test, using 5 plates for each isolate.

C = Corum; N = Napoleon; BR = Black Republican; S = Sour Cherry; B = Bing; P = Prune; E = Elberta peach; RH = Red Haven peach.

(2) Figures followed by the same letter in any column do not differ significantly (P = .05) by Duncan's Multiple Range Test.

and the blossoms were inoculated with conidia suspension of both M. laxa and M. fructicola. Benomyl at this rate, provided a good control against both Monilinia spp. (Table 8). Isolations from infected blossoms in this experiment produced no significant mycelial growth when transferred to benlate amended PDA at .5 ppm.

5. Comparative Pathogenicity of M. laxa and M. fructicola Using Prunus Cultivars

Blossom Inoculation. Both M. laxa and M. fructicola were able to cause a high percentage of blossom blight. The number of blossoms showing (necrosis of the peduncles and other floral parts) were recorded (Table 9). Open blossoms were more susceptible to infection than the closed blossoms. There was no significant differences in the number of infected blossoms between the two species of brown rot fungi when they were inoculated on these sweet cherry cultivars.

Flower of 'Napoleon', 'Corum' and 'Black Republican' sweet cherries were inoculated at the Botany and Plant Pathology Field Laboratory. The percentage of blighted blossom infected with M. laxa were 80%, 89%, and 93% on 'Napoleon', 'Corum', and 'Black Republican' respectively. The inoculation with M. fructicola gave 82%, 87%, and 89% respectively on these same cultivars. Some of the inoculated flowers fell off before infection extended into the axis, in the rest of the infected flower, the fungus grew into the peduncle and killed all the flowers on the spur. Isolation from these spurs confirmed that the causal agents were the original organisms. The wilting of the emerging leaves adjacent to the

Table 8. Effect of 300 ppm benomyl spray on blossoms of sweet cherry cultivars inoculated with both M. laxa and M. fructicola isolates.

| Host | Time of Application | % of Infected Blossoms M. laxa | M. fructicola | Check A | Check B |
|---|---|---|---|---|---|
| Corum | popcorn to full bloom | 12 (1) | 8 | 0 | 100 |
| Napoleon | - | 14 | 7 | 0 | 96 |
| Black Republican | - | 10 | 11 | 0 | 95 |

(1) Total of 100 blossoms were used in each test.

A = Sprayed with distilled water only.
B = Blossoms were inoculated with Benlate sprays.

Table 9. Comparison of percentage blossom blight in three sweet cherries cultivars inoculated in the laboratory.

| Sweet Cherry Cultivars | Percentage Blossom Blight | | | | Check Distilled $H_2O$ |
|---|---|---|---|---|---|
| | *M. laxa* | | *M. fructicola* | | |
| | (Closed) | (Open) | (Closed) | (Open) | |
| Corum | 34 | 86 | 41 | 89 | 0 |
| Napoleon | 29 | 84 | 35 | 92 | 0 |
| Black Republican | 32 | 92 | 39 | 85 | 0 |

inoculated flower cluster were first noticed 15 days after the flower inoculation. The inoculated flowers showed an early browning of the stylet and a premature withering of the stamen and calyx lobes. Tufts of grey conidia sporulation was observed on the flower cup.

One-year-old twigs were inoculated on sweet cherry, sour cherry, and peach cultivars under field condition and sweet cherry seedling in the greenhouse. There was no difference in the canker size produced by either species on sweet cherry cultivars (Table 10). However, M. laxa produced larger cankers than M. fructicola on sour cherry and the two peach cultivars (Table 10). M. fructicola produced only significantly larger cankers on 'Red Haven' peach. Sporodochia were only produced on twig of 'Montmorancy' sour cherry, 'Elberta' and 'Red Haven' peach cultivars inoculated with both species (Table 10). Although the number of sporodochia was higher on these twigs inoculated with M. laxa (Table 10). Both species failed to produce any sporodochia on the inoculated twigs of any sweet cherries cultivars (Table 10). There was no difference in the number of conidia produced per sporodochia (Table 10). The inoculated checks failed to produce measureable cankers or sporodochia.

Monilinia species isolates from sweet cherry produced smaller cankers than isolates from peaches when they were reinoculated onto their original host under field conditions (Table 11). Sporodochia were produced only on sour cherry and two peach cultivars but none were produced on sweet cherry twigs (Table 11). There was a poor correlation between the rate of canker increase per month and the number of sporodochia produced on the inoculated tiwgs of these

Table 10. Canker size and sporodochia production in seven stone fruit cultivars inoculated with M. laxa and M. fructicola at the Botany and Plant Pathology Field Laboratory in Corvallis, OR.

| Host | Mean Canker Size in Cm ML(1) | MF(1) | Average No. of Sporodochia ML | MF | Average No. of Conidia From Sporodochia ML | MF |
|---|---|---|---|---|---|---|
| Corum | 3.8 | 3.4 | 0 | 0 | 0 | 0 |
| Napoleon | 3.9 | 3.6 | 0 | 0 | 0 | 0 |
| Bing | 4.0 | 5.2 | 0 | 0 | 0 | 0 |
| Black Republican | 4.8 | 3.6 | 0 | 0 | 0 | 0 |
| Montmorency Sour Cherry | 7.0 | 4.5 | 5 | 3 | $0.50 \times 10^4$(2) | $0.27 \times 10^4$ |
| Elberta peach | 9.5 | 3.4 | 4 | 2 | $0.21 \times 10^4$ | $0.38 \times 10^4$ |
| Red Haven peach | 31.0 | 14.0 | 38 | 8 | $0.42 \times 10^4$ | $0.45 \times 10^4$ |

(1) ML = Monilinia laxa; MF = Monilinia fructicola
(2) Indicates number conidia per one milliliter of water

Table 11. Rate of canker increase in Cm. on seven stone fruit cultivars reinoculated with their original isolate of Monilinia spp. under field conditions.

| Stone Fruit Cultivars | Size of Canker in Cm 15 days Later | 6 months Later | Rate of Increase of Canker/Month | Average No. of Sporodochia Produced After 6 Months | % Germination of Conidia From Sporodochia |
|---|---|---|---|---|---|
| Napoleon | 3.86 | 5.92 | .34 | 0.0 | - |
| Corum | 3.79 | 6.30 | .42 | 0.0 | - |
| Black Republican | 4.96 | 5.76 | .13 | 0.0 | - |
| Bing | 4.30 | 6.78 | 1.13 | 0.0 | - |
| Montmorency Sour Cherry | 4.22 | 6.53 | 1.04 | 2.8 | 85 |
| Elberta peach | 6.74 | 12.92 | 2.15 | 5.0 | 79 |
| Red Haven peach | 8.56 | 16.60 | 2.77 | 2.0 | 82 |

seven stone fruit cultivars, with correlation coefficient r = .28.

Inoculation of sweet cherry seedling with both M. laxa and M. fructicola isolates showed no significant differences between the two species with respect to the size of canker produced on the inoculated twigs (Table 12). Under natural conditions sporodochia did not produce on sweet cherry due to rare canker development. However, when twigs of sweet cherry seedling inoculated with M. laxa and M. fructicola isolates sporodochia were produced. M. laxa isolates produced more sporodochia than M. fructicola (Table 12).

Fruit Inoculation. One hundred healthy fruit from three sweet cherry cultivars were collected and inoculated with M. laxa and M. fructicola. Both Monilinia spp. caused a high percentage of fruit rot (Table 13). The three sweet cherry cultivars were susceptible to both organisms. M. fructicola caused more severe fruit rot symptoms than M. laxa in all cultivars tested (Table 13). There was no difference in the length of time before sporulation.

Radial growth on PDA, sporulation intensity and virulence (measured by the size of lesion on the inoculated 'Elberta' peach fruit) were determined for isolates of M. laxa and M. fructicola (Table 14). Growth of M. laxa isolates were slower than M. fructicola after 96 hours. M. laxa isolates from sweet cherries produced only mycelial growth, but Monilinia isolates from peaches form conidia on the inoculated fruit 3 weeks before harvest (Table 14). M. fructicola isolates sporulated heavily in comparison to M. laxa isolates on inoculated fruit 2 weeks before harvest. All isolates of both species sporulated about the same when inoculated onto fruits one week prior

Table 12. Susceptibility of sweet cherry seedlings inoculated with different isolates of M. laxa and M. fructicola under greenhouse conditions using the canker length and sporodochia production.

| Criteria Used | Sweet Cherry Cultivars Original Isolates Sources CML | NML | BML | BRML | Peach Cultivars EML | EMF | RHML | RHMF | Sour Cherry SML | Prune PML | Check |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Mean Canker Length in cm. 15 days after inoculation | 5.5 | 4.0 | 4.32 | 5.18 | 5.20 | 4.63 | 5.12 | 4.42 | 5.18 | 4.82 | 0 |
| Total No. of Sporodochia produced on 5 inoculated twigs | 20 | 20 | 29 | 10 | 13 | 3 | 8 | 4 | 12 | 10 | 0 |

ML = M. laxa; MF = M. fructicola

C = Corum; RA = Royal Ann; B = Bing; BR = Black Republican; E = Elberta peach; RH = Red Haven; S = Montmorency Sour Cherry; P = Early Italian prune

Check. .5 inoculated twig with clear PDA only.

Table 13. Comparison of fruit rot percentage on three sweet cherry cultivars resulting from infection from M. laxa and M. fructicola in the laboratory.

| Sweet Cherry Cultivars | Fruit Rot Percentage Resulted From Artificial Inoculation With | | Check Distilled $H_2O$ Only |
|---|---|---|---|
| | M. laxa | M. fructicola | |
| Corum | 64(1) | 79 | 0 |
| Napoleon | 58 | 65 | 0 |
| Black Republican | 80 | 82 | 0 |

(1) Based on 100 fruits used per test, repeated twice over two years.

Table 14. Linear mycelial growth, sporulation intensity and virulence on harvested Elberta peach fruits against *M. laxa* and *M. fructicola* isolates incubated at 24°C.

| Isolates | Diameter of Colonies (mm) 96 hours | Sporulation Intensity 72 Hours After Inoculation — Weeks Before Harvest 3 | 2 | 1 | Lesion Diameter in (mm) on Fruits 72 Hours After Inoculation — Weeks Before Harvest 3 | 2 | 1 | Check |
|---|---|---|---|---|---|---|---|---|
| *M. laxa* | | | | | | | | |
| C | 40(1) | MS(2) | SS | SSS | 35(3) | 72 | 80 | 0 |
| N | 48 | MS | SS | SSS | 41 | 78 | 96 | 0 |
| BR | 55 | MS | SS | SSS | 51 | 74 | 94 | 0 |
| S | 54 | MS | SS | SSS | 40 | 83 | 92 | 0 |
| B | 59 | SS | SS | SSS | 53 | 86 | 90 | 0 |
| P | 50 | MS | SS | SSS | 48 | 82 | 88 | 0 |
| E | 55 | SS | SS | SSS | 51 | 83 | 92 | 0 |
| RH | 48 | SS | SS | SSS | 61 | 72 | 94 | 0 |
| *M. fructicola* | | | | | | | | |
| E | 62 | SS | SSS | SSS | 65 | 78 | 85 | 0 |
| RH | 65 | SS | SSS | SSS | 69 | 80 | 84a | 0 |

(1) Average of 5 plates repeated twice.
(2) MS = mycelial growth; SS = sporulation moderate, SSS = sporulation heavy.
(3) Average lesion diameter of 10 inoculated fruits read 72 hours after inoculation.
Check fruit were inoculated with 5 mm plain PDA plugs.
C = Corum; N = Napoleon; BR = Black Republican; S = Montmorency Sour Cherry; B = Bing; P = Prune;
E = Eliberta peach; RH = Red Haven peach

to harvest. Virulence, as determined by the lesion size on the fruit, showed M. laxa isolates developed smaller lesion than M. fructicola on fruits inoculated 3 weeks before harvest (Tbale 14). However, all isolates of both species developed large lesion on fruit two and one week before harvest (Table 14).

## II. Intraspecies Variability of M. laxa Isolates in Oregon

Colonies Characteristics. M. laxa isolates were obtained from infected blossoms, spurs, rotted fruits and mummies. There was no significant differences ($P \leq 0.05$) in radial growth when measured after 7 days at different media (Table 15). Mixing these isolates in different combinations produced a dark line reaction when two isolates come in contact. No anastomoses were observed.

The occurrence of branched germ tubes were consistent in all M. laxa isolates tested. There was no difference in the percentage of the conidia germination among isolates. Conidia were germinated readily on water agar after 6 hours of incubation. All isolates when put on PDA for the first time tended to be slow in conidia formation. However, when these isolates were transferred for the second time, they produced more conidia within 70 hours. The average conidia size produced from individual isolates were not significantly different (Table 15). Growth rate and sporulation were favored when these isolates grew on prune and peach agar.

When a mycelium plug of 5 mm in diameter was inserted into ripe 'Elberta' peach fruits, sporulation was faster. Heavy sporulation

Table 15. Radial growth of M. laxa isolates in mm on different media seven days after incubation at 24°C.

| Isolates | Media | | | | Ave. Conidia Size in μ |
|---|---|---|---|---|---|
| | PDA | Prune Agar | Peach Agar | Cherry Agar | |
| C | 63(1) | 72 | 71 | 67 | (9-15 x 6-10)(2) |
| N | 64 | 70 | 69 | 73 | (9-17 x 6-10) |
| BR | 62 | 69 | 73 | 68 | (10-16 x 7-9) |
| B | 63 | 68 | 69 | 72 | (10-17 x 7-9) |
| S | 61 | 67 | 65 | 70 | (10-15 x 6-9) |
| P | 65 | 69 | 71 | 71 | (10-17 x 6-10) |
| E | 64 | 69 | 70 | 73 | (9-17 x 6-9) |
| RH | 63 | 70 | 71 | 71 | (11-17 x 6-8) |

(1) Average of 5 plates replicated twice.
(2) Average size of 50 conidia.

C = Corum; N = Napoleon; BR = Black Republican; B = Bing; S = Montmorency Sour Cherry; P = Italian prune, E = Elberta peach; RH = Red Haven peach

was obtained within 96 hours after inoculation of peach fruit (Table 14). Isolates differed in the number of conidia that were formed on the inoculated peach fruits, when they were measured by washing half the fruit in 50 ml of distilled water, then counted by hemacytometer. The average number of conidia were between $2.5 \times 10^5$ to $8 \times 10^6$. The highest conidia production was from a peach isolate.

Pathogenicity. Pathogenicity was tested on one-year-old inoculated twigs of two sweet cherry and two peach cultivars under field conditions and on sweet cherry seedlings in the greenhouse. The canker size and the number of sporodochia were used to determine variation and pathogenicity (Table 12,16,17). There was no difference between the isolates with respect to the cankers size produced on the inoculated twig of sweet cherry cultivars (Table 16). There was a difference in canker size between sweet cherry and peach cultivars. Isolates from peach produced larger cankers on peach twigs. Canker size was smaller on sweet cherry and sweet cherry seedlings than on peach for all isolates (Table 12). The number of sporodochia was varied from isolate to isolate, especially when inoculated into peach cultivars (Table 17). All isolates failed to produce sporodochia on 'Napoleon', however, some isolates produced a few sporodochia on 'Black Republican' (Table 17). There was also some variation in sporodochia production between the isolates on sweet cherry seedlings (Table 12). There was no strong correlation between the canker size and number of sporodochia produced on 'Elberta' and 'Red Haven' peach cultivars ($r = .40$ and $r = .09$ respectively).

Table 16. Pathogenicity of M. laxa isolates inoculated on sweet cherry and peach cultivars under field conditions.

| Host Inoculated | Source of Isolates: Sweet Cherries C[3] | N | B | BR | Sour Cherry S | Peach RH | E | Prune P | Check[2] |
|---|---|---|---|---|---|---|---|---|---|
| Sweet Cherry | | | | | | | | | |
| Black Republican | 5.22[1] | 4.68 | 4.68 | 4.96 | 4.16 | 3.43 | 4.42 | 4.12 | 0 |
| Napoleon | 3.92 | 3.86 | 5.71 | 5.68 | 4.62 | 4.61 | 4.53 | 3.77 | 0 |
| Peach | | | | | | | | | |
| Elberta | 6.72 | 6.38 | 5.80 | 6.41 | 7.13 | 8.20 | 6.74 | 4.82 | 0 |
| Red Haven | 7.67 | 7.82 | 7.25 | 8.94 | 7.04 | 8.50 | 7.84 | 5.94 | 0 |

(1) Average canker size (cm) 15 days after inoculation.
(2) Five twigs inoculated with PDA 5 mm plugs.
(3) C = Corum, N = Napoleon, B = Bing, BR = Black Republican, S = Montmorency Sour Cherry, RH = Red Haven, E = Elberta and P = Italian prune.

Table 17. Sporodochia production of M. laxa isolates inoculated on sweet cherry and peach cultivars.

| Host Inoculated | Source of Isolates: Sweet Cherries C | N | B | BR | Sour Cherry S | Peach RH | E | Prune P | Check |
|---|---|---|---|---|---|---|---|---|---|
| Sweet Cherry | | | | | | | | | |
| Black Republican | 0* | 0 | 2 | 2 | 3 | 0 | 2 | 0 | 0 |
| Napoleon | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Peach | | | | | | | | | |
| Elberta | 78 | 51 | 18 | 22 | 29 | 16 | 31 | 59 | 0 |
| Red Haven | 89 | 72 | 39 | 128 | 46 | 8 | 52 | 57 | 0 |

*Total number of sporodochia produced six months after inoculation on ten twigs.

C = Corum, N = Napoleon, B = Bing, BR = Black Republican, S = Montmorency Sour Cherry, RH = Red Haven peach, E = Elberta peach, P = Italian prune.

## III. Aspects of the Disease Cycle of Brown Rot on Sweet Cherry

### Source of Primary Inoculum

The isolates from infected spurs and mummified fruit samples collected in 1980 gave mainly M. laxa. The isolation from samples collected in 1981 yielded 97% M. laxa and 3% M. fructicola from mummies. However the isolation from blighted fruit spurs yielded only M. laxa (Table 18). Conidial tufts were observed on the mummified fruit samples throughout the sampling period from the middle of December to the middle of March, but sporulation on the spurs was only observed after rainfall followed by warm weather. Conidial germination was highest during the last two sample dates.

During March 1981, field counts of fruit spurs were made at three orchards in western Oregon. The percentage of blighted spurs varied from 2.8 to 5.2%. Sporodochia developed on the surface of the fruit peduncles and the dead blossoms infected from previous years, after they had been incubated at moist and warm conditions (Table 19). Sporodochia were produced mainly on the surface of these infected fruit spurs. After the fruit spurs were thoroughly surface sterilized few sporodochia were developed (Table 19). The number of sporodochia producing sites varied from orchard to orchard. At the Botany and Plant Pathology Field Laboratory double the number of sporodochia sites were produced than at either the Lewis Brown Farm or the Conklin orchard. At the Lewis Brown Farm the trees where the samples were taken had been sprayed with lime sulfur on March 12th as a late

Table 18. Mummified fruit plus tagged blighted spurs from sweet cherry cultivars as a site for overwintering of Monilinia spp. under field conditions during 1979/80 - 1980/81 seasons.

| Sampling Period | No. of Sample Evaluated Mummies | Spurs | Sporulation M | S | % Germinating | % Colonies Isolated From Mummies and Spurs Mummies M. laxa(3) | Mummies M. fructicola | Spurs M. laxa | Spurs M. fructicola |
|---|---|---|---|---|---|---|---|---|---|
| 12/15/79 | 100(1) | 50 | +(2) | - | 42 | 89 | 0 | 96 | 0 |
| 1/20/80 | 50 | 50 | + | - | 62 | 80 | 0 | 96 | 0 |
| 2/15/80 | 50 | 50 | + | - | 79 | 85 | 0 | 90 | 0 |
| 2/20/80 | 50 | 50 | + | + | 85 | 92 | 0 | 96 | 0 |
| 12/20/80 | 100 | 50 | + | - | 45 | 90 | 2 | 92 | 0 |
| 1/20/81 | 100 | 50 | + | - | 50 | 95 | 0 | 88 | 0 |
| 2/20/81 | 100 | 50 | + | - | 75 | 87 | 3 | 86 | 0 |
| 3/20/81 | 100 | 50 | + | + | 79 | 84 | 3 | 96 | 0 |

(1) Sample collected at random from sweet cherry cultivars in the Botany Farm.
(2) Sporulation indicated by the presence of ash cushion appearance on fruit.
(3) Colonies percentage based on one point of isolation from each fruit mummies or spurs.

Table 19. Blighted blossoms, fruit peduncles on spurs as an important overwintering site of Monilinia spp. in the Willamette Valley, western Oregon (incubated under laboratory conditions during early spring 1981).

| Location (counties) | Host | Total No. Of Spurs Evaluated | Total No. Of Spurs Blighted | Site of Sporodochia Development: Surface Sterile — Fruit Peduncles | Surface Sterile — Blighted Blossom On Spur From Previous Year | Non-Surface Sterile — Fruit Peduncles | Non-Surface Sterile — Blighted Blossom From Previous Year |
|---|---|---|---|---|---|---|---|
| Plant Path. | Corum | 3,000 | 32 | 1(1) | 2 | 6 | 7 |
| Field Lab. | Napoleon | 3,000 | 38 | 0 | 1 | 6 | 8 |
| (Linn) | Black Republican | 3,000 | 52 | 1 | 2 | 4 | 7 |
| Lewis Brown | Corum | 2,000 | 28 | 0 | 0 | 2 | 3 |
| Farm (Linn) | Napoleon | 2,000 | 38 | 0 | 0 | 2 | 1 |
| Conklin | Corum | 2,000 | 42 | 0 | 0 | 1 | 3 |
| Orchard | Napoleon | 2,000 | 48 | 0 | 2 | 3 | 4 |
| (Yamhill) | | | | | | | |

(1) Indicate the number of site where the sporodochia developed.

dormant spray. Sampling was done on March 17th. The Conklin orchard trees had been sprayed with benomyl plus oil between the 3rd and 5th of March. Sampling was done on March 15th from this orchard. These late dormant sprays apparently reduced the number of sites where sporodochia were produced (Table 19).

An attempt was made to locate apothecia in commercial orchards. No apothecia were found during the three years of observation. No apothecia resulted from burying mummified sweet cherry, peach and 'Early Italian' prune, fruit for six months. Mummies kept at 5°C for two years failed to form apotehcia, however, stroma did develop.

Latent Infection

When immature fruits were kept under moist and warm conditions, fruit rot symptoms developed on some fruits (Table 20). The symptoms started as small circular spots, that enlarged quickly and caused a green fruit rot. The immature cherry fruits that were thoroughly surface sterilized produced few infected fruits (Table 20). Inoculated green fruit (after surface sterilization) produced circular brown spots identical to the symptoms observed in the orchard. There was a difference in the amount of green fruit rot between treated (benlate at the rate of 4 oz/100 gallon 3 bloom sprays) and non-treated trees. The stem end wash, when plated on PDA developed typical colonies of Monilinia spp. (Table 20). This suggested that Monilinia propagules might be present on the fruit surface. Isolation from small circular brown spot from the outer skin (epidermis), but not from the actual green fruit flesh, produced colonies of Monilinia

Table 20. Incidence of infection on green fruits of sweet cherry cultivars during spring 1981.

| Host | % Latent Infection Nonsterile | % Latent Infection Surface Sterile | Surface Sterile Inoculated With 50,000/ml | Check Distilled $H_2O$ Only | No. of Colonies Isolated From Stem End Wash |
|---|---|---|---|---|---|
| Nontreated | | | | | |
| Corum | 13(1) | 2 | 10 | 2 | 3/25(3) |
| Napoleon | 12 | 2 | 16 | 1 | 6/25 |
| Black Republican | 10 | 3 | 12 | 0 | 4/25 |
| Treated(2) | | | | | |
| Corum | 2 | 0 | 4 | 0 | 1/25 |
| Napoleon | 1 | 0 | 6 | 0 | 1/25 |
| Black Republican | 2 | 0 | 5 | 0 | 0/25 |

(1) Based on 100 fruit samples.
(2) Treated with benlate at the rate of 4 oz/per 100 gallons of water.
(3) One-milliliter of alaquote sprayed evenly on PDA plate from each stem end.

when plated on PDA. Histological studies of the excised section confirmed that germinated conidia were present on the epidermis. The depth at which fungal hyphae were found in the mesophyll cells under the epidermis depended on the extent of the rotted area (Figure 7).

The incidence of latent infection was determined at four orchards in western Oregon. Even when the fruit were thoroughly surface sterilized and incubated at moist and warm conditions, brown rot symptoms developed on a number of fruits (Table 21). The amount of fruit rot varied from orchard to orchard. The incidence was highest at the Schindler orchard compared to the other locations (Table 21). There was a difference in the control measures in each orchard which may partially account for the difference in fruit rot development. The development of fruit rot in non-surface sterilized portion apparently due to the contamination on the fruit surface (Table 21).

## Relationship of the Fruit Soluble Solid Content to Fruit Rot Development

Fruits from different sweet cherry cultivars, when inoculated with the brown rot fungus in the laboratory, showed that the fruit rots symptoms by M. laxa usually depended on the cultivar and the maturity of the fruit. The soluble solids appeared to be utilized as substrate for the growth of M. laxa. To determine the relationship of soluble solids to symptom development, the initial fruit soluble solids content, the utilization of this soluble solids by the fungus after fruit rot, and the percentage of fruit rot, were determined (Table 22,

Table 21. Incidence of latent infection on sweet cherries at different locations in the Willamette Valley.

| Location (counties) | Host | % of Rotten Fruit After Surface Sterilization in 70% Alcohol — Weeks Before Harvest 3 | 2 | 1 | % of Rotten Fruit Without Surface Sterilization — Weeks Before Harvest 3 | 2 | 1 |
|---|---|---|---|---|---|---|---|
| Plant Pathology Field Lab. (Linn) | C | 5(1) | 10 | 6 | 18 | 66 | 92 |
| | N | 8 | 2 | 6 | 12 | 66 | 90 |
| Horticulture Farm (Linn) | C | 7 | 4 | 6 | 16 | 8 | 22 |
| | N | 5 | 8 | 8 | 24 | 16 | 46 |
| Schindler Orchard (Marion) | C | 10 | 12 | 12 | 92 | 96 | 100 |
| | N | 4 | 8 | 10 | 76 | 84 | 100 |
| Conklin Orchard (Yamhill) | C | 3 | 6 | 4 | 8 | 52 | 68 |
| | N | 1 | 3 | 5 | 6 | 32 | 50 |

(1) Based on 100 fruits sampled at random from the cherry orchards.

C = Corum, N = Napoleon

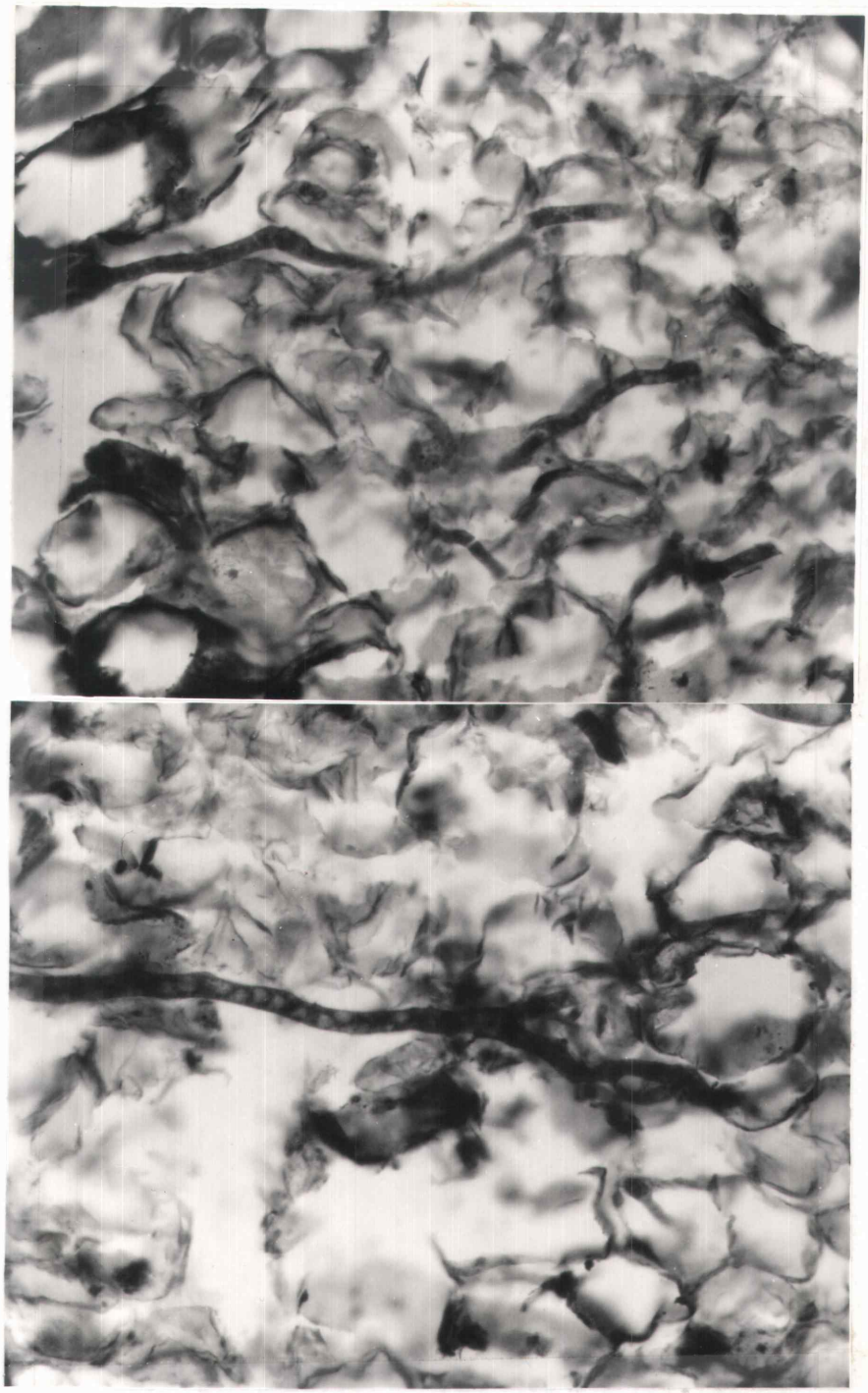

Figure 6. Cross sections of the excised green fruit of sweet cherry showing fungal hyphae of M. laxa in the epidermal layers.

Table 22. Utilization of soluble solid content by M. laxa and fruit rot development of artificially inoculated sweet cherry fruit at different stages of maturity during 1981.

| Location (counties) | Host | Initial Soluble Solid Content (%) in Fruit — Weeks Before Harvest 4 | 3 | 2 | 1 | Rotten Fruit % Developed By Artificial Inoculation — Weeks Before Harvest 4 | 3 | 2 | 1 | Correlation Coefficient Between Initial Soluble Solid Content and % of Fruit Rot r |
|---|---|---|---|---|---|---|---|---|---|---|
| Plant Pathology Field Lab. (Linn) | Corum | 8.50(1) | 10.30 | 14.75 | 17.50 | 60 | 75 | 88 | 97 | .98 |
| | Napoleon | 7.75 | 8.55 | 15.00 | 18.20 | 38 | 52 | 80 | 96 | .92 |
| Lewis Brown Farm (Linn) | Corum | 9.25 | 13.50 | 16.50 | 19.50 | 55 | 79 | 86 | 98 | .91 |
| | Napoleon | 8.65 | 10.75 | 16.25 | 19.75 | 63 | 86 | 94 | 98 | .96 |
| Schindler Orchard (Marion) | Corum | 10.65 | 11.80 | 115.20 | 18.50 | 75 | 88 | 94 | 99 | .91 |
| | Napoleon | 8.10 | 10.80 | 13.50 | 16.75 | 70 | 83 | 91 | 98 | .92 |
| Conklin Orchard (Yamhill) | Corum | 7.50 | 9.25 | 14.65 | 18.20 | 45 | 65 | 86 | 96 | .98 |
| | Napoleon | 6.70 | 9.20 | 12.20 | 16.50 | 40 | 69 | 88 | 98 | .89 |

(1) Average of 20 fruits measured by refractometer.
(2) Average of 20 fruits measured by refractometer after fruit rot had developed.

23). The results of this experiment showed that whenever the soluble solid content in the fruit was above 10% the incidence of fruit rot increased on the inoculated fruit (Table 22,23). There was a linear relation between the soluble solid content and percentage of fruit rot at different stage of maturity ($y = 43.51 + 3.04X$). There was also an increased use of the soluble solid by M. laxa. There was good correlation between the initial soluble solid content and the utilized soluble solid content and the amount of fruit rot (Table 22,23). The results of this inoculation showed that during the last 3 weeks before harvest the sugar level increased rapidly and the amount of fruit also started to increase. The amount of fruit rot assessed under orchard conditions was highly correlated with the soluble solid content in the fruit at different stage of maturity with a correlation coefficient (r between .82 to .94).

Table 23. Utilization of soluble solid content by M. laxa and fruit rot development on artificially inoculated sweet cherry fruit at different stages of maturity during 1981.

| Location (counties) | Host | Soluble Solid % Used by M. laxa After Inoculation — Weeks Before Harvest 4 | 3 | 2 | 1 | Correlation Coefficient Between Utilized Soluble Solid Content and % of Rotted Fruit r |
|---|---|---|---|---|---|---|
| Plant Pathology Field Lab. (Linn) | Corum | 1.75(2) | 2.90 | 3.20 | 3.50 | .98 |
| | Napoleon | 1.50 | 2.50 | 3.40 | 3.77 | .96 |
| Lewis Brown Farm (Linn) | Corum | 2.00 | 2.60 | 3.25 | 3.80 | .96 |
| | Napoleon | 1.75 | 2.95 | 3.20 | 4.00 | .95 |
| Schindler Orchard (Marion) | Corum | 2.80 | 3.00 | 3.50 | 3.90 | .88 |
| | Napoleon | 1.90 | 2.80 | 3.20 | 3.75 | .98 |
| Conklin Orchard (Yamhill) | Corum | 1.25 | 2.00 | 3.50 | 4.00 | .92 |
| | Napoleon | 1.30 | 2.10 | 3.25 | 3.80 | .93 |

# DISCUSSION

## I. A. Disease Development Caused by *Monilinia* spp.

Early investigation of brown rot disease on stone fruit in the Willamette Valley by Owens and Evans (29) reported that the isolation from infected blossoms yielded 73% *M. fructicola* and 27% were *M. laxa*. The isolation from rotted fruit samples gave approximately 51% of *M. fructicola* and 48% *M. laxa*. However, in our studies in the past three seasons, isolation have been made from infected blossoms, rotted fruit and fruit spurs of sweet cherry. In every orchard that was sampled *M. laxa* was more common than *M. fructicola* (Table 1 and 2). The orchard sampled included valley floor and hill sites from major producing counties.

The species of *Monilinia* isolated in these surveys were identified by their growth characteristics in culture such as lobing of colony margins, conidial germination and the presence of absence of anastomoses based on earlier work (38,69,99,103). In these studies 89-96% of the infected blossoms, and 83-95% of the infected fruit yielded *M. laxa*, while 4-11% of infected blossoms, and 5-17% of the infected fruit samples yielded *M. fructicola* (Table 1, 2). *M. fructicola* was never isolated from blighted sweet cherry spurs that were producing sporodochia. Isolation from the infected spurs always yielded *M. laxa*. A comparison of our data with previous reports (29) indicated that a change has taken place in the ratio of *M. laxa* to *M. fructicola* over the past few years. The destructive form of the disease characterized by blossom blight and fruit rot was caused mainly

by M. laxa. This may be explained in part if we assume large population of M. laxa conidia produced from sporodochia on overwintering sites. The rare development of such sporodochia by M. fructicola on the infected twigs of stone fruit as pointed out by Brook (9) and Robert and Dunegan (81) may account for this change of Monilinia species throughout the season. The early infection by M. laxa and subsequent preparation chiefly by means of conidia also account for the frequent isolation of M. laxa more than M. fructicola. Our knowledge of the rare development of apothecia of both may indicate its importance to M. fructicola more than M. laxa, and the abundance of sporodochia production on the overwintering sites was of considerable importance to initiate chain infection necessary for its build up.

During the bloom development the blossom blight incidence showed gradual increase towards the end of the bloom stage. The calculated apparent infection rate was different among the location where the studies were conducted. The highest rate of disease increase was observed at Schindler orchard in comparison to other locations. The differences in the apparent infection rate was attributed to the difference in the amount of initial inoculum and the effects of subsequent control program. The differences may also be due to the time of bloom or contary to 1980, the 'Corum' trees at the Schindler orchard had more blossom blight than other orchards. Such infection would supply additional inoculum to the 'Napoleon' in the same orchard to explain the high incidence of blossom blight. This heavy infection causes a dramatic reduction in the fruit set and supplies

an infection site that can spread into immature fruit. This inturn, provide a source of inoculum for mature fruit infection at harvest.

The fruit rot phase of brown rot can cause devastating loses at harvest (5,6). Over 50 percent of the fruit may be ruined (6). The fruit rot phase of brown rot was present, but not severe during the three years of this study. However, in 1981, it was estimated that about 63% of the 'Corum' and 49% of the 'Napoleon' cherries in the Schindler orchard were rotted (Table 5). Rainfall during the 3rd week of June caused more cracking of 'Corum' fruit in this particular orchard than the other three orchards. Also it caused more cracking on 'Corum' than 'Napoleon'. This is evident from the 1981 results reported in Table 5. The amount of fruit rot was markedly increased one week before harvest. This may be because the fruit is more susceptible as it approaches maturity, and/or the rapid build up of inoculum due to fast sporulation on the infected fruits. Calculating the apparent infection rate during ripening showed some variation between location (Table 5, Figure 1, 2). It seems likely that the differences were due to the amount of the infected fruit that was observed in each orchard. These differences could also be related to the heavy blossom infection at the Schindler orchard at the end of bloom period. These infections provide an early infection to the fruit and further spread within the orchard.

## B. Comparative Studies of *M. laxa* and *M. fructicola*

Colonies Characteristics. Strain intermediately between typical isolates and several cultural types of *M. laxa* and *M. fructicola* have been reported by many workers (30,31,39,105). Wormald and Ezekiel (31,104) described an isolate of *M. fructicola* that had the usual concentric rings and dense sporulation of *M. fructicola* plus the irregular, somewhat lobed margin associated with *M. laxa*. The isolates of *M. laxa* and *M. fructicola* obtained in our studies were identified by their growth characteristic in 2% PDA, by the type of conidial germination and sporulations. Since all of the isolates obtained in this research were similar to the type culture of *M. laxa* and *M. fructicola* it was not possible to explore the pathogenicity of intermediate type isolates. Colony characteristics, germination, sporulation and the presence and absence of anastomoses were similar to widely accepted as characteristic of each species. However, differences were observed in the mode of conidia formation by both species. *M. laxa* usually formed more conidia when the culture was transferred the second time which suggested that additional nutrients favored the sporulation of this fungus.

Ezekiel (30) observed interspecific and intraspecific anastromoses between *M. laxa* and *M. fructicola*. Mixing *M. laxa* and *M. fructicola* isolates may clarify the role of anastomoses between the isolates and among species. Our result showed only a dark line reaction at the point of contact between isolates of *M. laxa*. Anastomoses was observed only between different isolates of *M. fructicola*. This is

possibly due to the lack of heterokaryosis in the M. laxa and its presence in M. fructicola.

The failure of some orchardists to control brown rot blossom blight on cherries and peaches lead to the assumption that an isolate of the fungus had developed tolerance to benlate (26,33,59,64, 88). Ogawa et al. was able to detect tolerance of M. fructicola in those California orchards that had a history of repeated application of benomyl (89). Ogawa and co-workers were able to detect three distinct isolates of M. laxa tolerant to benomyl from an apricot orchard (personal communication). However, in our studies where benlate had been used, we were unable to detect the development of tolerance in either M. laxa or M. fructicola isolates. None of the isolates were able to produce significant radial growth in media amended with benlate as low as .5 ppm, however, conidial germination of both species was observed in media with low concentration of benlate (Table 7). The germ tubes were lysed quickly without radial growth development. The lack of tolerant isolates suggested that resistant isolates have not been selected out by the extensive use of the fungicide. This may be due to the Oregon recommendation that growers change to a different fungicide after the first application of benomyl. Thus even though some isolates of the pathogen may have tolerance to benomyl, they are eliminated by fungicides such as Captan or Ziram.

Inoculated sweet cherries blossom are equally susceptible to both Monilinia spp. (12,71). Our blossom inoculations in the field agreed with the results obtained from laboratory studies. This

finding agreed with the earlier work of Weaver (94) that under natural heavy blossom blight, M. laxa was found to be most important on sweet cherry blossom. Although cherry blossoms were infected with both species, M. laxa was isolated more frequently than M. fructicola. This may be because of a lower population of M. fructicola conidia.

Inoculation of one-year-old twigs of various stone fruits with M. laxa and M. fructicola, showed that a definite canker can be produced (7,24,51). In earlier work, Ogawa (71) demonstrated that differences existed between stone fruits as host for Monilinia, but there was no differences in the degree of pathogenicity between the two organisms on inoculated almond, nectarine and apricot. He concluded that cankers were the largest on almond, followed by apricot and then nectarine. In our study, M. laxa produced larger cankers than M. fructicola on 'Red Haven' and 'Elberta' peach cultivars, but M. fructicola produced larger cankers on 'Red Haven' peach. When both species were inoculated into sweet cherry they produced similar sized cankers, that were smaller than those on peach. In sweet cherry, callus periderm formation at the point of inoculation was observed to form faster than in other inoculated stone fruits. This may explain the reason for the smaller canker size in sweet cherry.

The result of reinoculating stone fruit species with isolates that originated from the same species confirmed the previous experiment, in that isolates from peach reinoculated into peach, produced larger cankers than isolates from sour into sour cherry or isolates from sweet cherry reinoculated into sweet cherry (Table 11). The

rate of canker increase per month was the smallest in sweet cherries compared to peach. M. laxa produced a few sporodochia on sweet cherry but M. fructicola never produced sporodochia on cherry cultivars (Table 10). The results of this reinoculation suggested that differences exist among stone fruit cultivars when inoculated with Monilinia spp. The result showed small cankers were produced on sweet cherry twigs as compared to sour cherry and peach twigs. Callus periderm formation was observed at the point of inoculation on sweet cherry to explain naturally the rare canker development. This also may in part explain the lack of sporodochia development on sweet cherry compared to other stone fruit.

Fruit infection may take place at any time during the season (52,55,94). Injuries produced due to fruit cracking or insect damage offer an excellent site for infection (43,72,101,104). Mature sweet cherry fruit from all tested cultivars was susceptible to attack by brown rot fungi (Table 13). Some differences between the two species were observed on the inoculated cultivars. This may be related to differences in the relative maturity of the fruit at the time of inoculation. Grover et al. (35) demonstrated that M. fructicola isolates utilized more sugar from culture media than M. laxa. Difference in the availability of fruit soluble solids may explain why M. fructicola was more aggressive than M. laxa (Table 13).

Inoculation of 'Elberta' peach fruit with isolates of M. laxa and M. fructicola showed some differences but between these organisms. The relatively slow development in unripe fruit (3 weeks before harvest) may be due to the lack of substances needed by the fungus

to colonize the tissue or simply the small volume of available soluble solids at this stage.

## II. Intraspecies Variability of *M. laxa* Isolates

Most of the *Monilinia* isolates from western Oregon orchards had characteristics widely accepted as those of *M. laxa* (11,12,38,44,48, 68,104). It was thought that the amount of variation within these isolates might be important in understanding disease development. Considerable variation had already been reported between different isolates within both species of brown rot fungi (11,12,62,104). Ezekiel (30) noted wide variation in the cultural characteristics, effects on inoculated fruit and production of oxidase. Later workers did not accept these as distinct varieties, but as variation in isolates similar to those found in most fungi (99,103). Staehelin (1946) examined the question of biologic forms of *M. laxa* on stone fruits by cross incoulation (104), and he concluded that *M. laxa* could not be subdivided into different forms. The *M. laxa* isolates obtained during our studies, showed very little variation. On culture plates there wre no significant differences in colony growth or number of conidia produced. Some differences were noted in colony color.

Oregon isolates were inoculated onto one-year-old twigs of cultivars of sweet cherry and peach in the field and seedling of sweet cherry in the greenhouse. Due to a failure of *M. laxa* to colonize sweet cherry twigs there was no difference in the size

of cankers between isolates (Table 16). The M. laxa isolates were equally pathogenic when they were inoculated into peach cultivars and sweet cherry seedlings (Table 12,16). There were no significant differences in pathogenicity between peach isolates of M. laxa inoculated into peach. The same was true for cherry isolates inoculated into cherry. However, peach isolates produced larger cankers than cherry isolates when both were inoculated into peach, and cherry isolates produced smaller sized cankers than peach isolates when both were inoculated into cherry. This suggests that host specific differences in pathogenicity do exist in isolates of M. laxa even though they were not detected in cultures.

The pathogenicity of M. laxa isolates that originated from peaches caused larger lesions than isolates from cherries when inoculated into 'Elberta' peach fruit three weeks before harvest. This may be because peach isolates were more aggressive in peach than isolates from other host. This may be related to the degree of ripeness that has some effects on readiness with which the fruit becomes infected. Cooly (18) found that ripe or nearly mature plum can be easily infected. This would indicate from the result obtained that the difference was due to the degree of fruit ripeness rather than the difference among M. laxa isolates inspite of their original source.

## III. Aspects of the Disease Cycle of Brown Rot on Sweet Cherry

Hauber and Baur (7) in western Washington, were able to observe

sporodochia on apricot and peach twigs from the latter part of December through March. They stated that under conditions of high humidity, sporodochia continued to develop into April. Since then, Jenkins (55) has shown that conidia may remain viable for 10 months in the laboratory and Kable (60) has also shown that conidia can survive up to four months in the orchard. Because there were few visible symptoms during the winter months the overwintering site of Monilinia was not clear (60,74,100,104). In these studies the Monilinia spp. were isolated from blighted blossom and fruit peduncles that remained hanging in the tree from the previous year (Table 19). The production of conidia on blighted blossoms and fruit peduncles are easliy overlooked at pruning and these provide source of primary inoculum to cause blossom infection. In Europe, Byrde and Willette (11) showed that the course of inoculum in tree was important in the blossom blight. They stated that there may be considerable variation in the level of blossom blight in adjoining trees, but there was a strong correlation between the number of blighted blossom and the number of inoculum source within the individual trees. Our results obtained from studies on the field counts of the sweet cherry spurs with blight blossom and fruit peduncles. The fungus has remained alive in the infected tissue and resume its activity, in the form of sporodochia, when conditions were favorable (Table 19). This results plus the information obtained from sampling mummies and tagged spurs (Table 18) showed that the importance of these overwintering sites of Monilinia spp. in the brown rot disease of sweet cherry.

M. laxa has been controlled by application of fungicide during dormancy to reduce the amount of primary inoculum on dead twigs of apricots (16,96). Suppressed production of M. laxa sporodochia in almond, and suppression of M. fructicola spore production on peach were achieved by a dormant benomyl spray (16,72,79). Reduction of sporodochia from the infected blossoms and fruit peduncles was achieved in the three orchards of western Oregon by a dormant spray (Table 19). The reduction of primary inoculum is probably the most practical way to reduce the total number of sprays needed for commerical orchards.

Apothecia were not produced in screen boxes buried in the orchard. Either these species do not produce apothecia under these conditions or the microbial activities in the soil destroyed the mummies as reported by earlier workers (9,11,16,86,101). Burying fruit mummies by tilling will reduce the production of conidia. The reduction of primary inoculum either by dormant sprays to destroy overwintered infections in the trees or preventing the sporulation of mummies on the ground is probably the most practical way to reduce the expense of disease control.

Wade and Morschel (92,66) demonstrated the occurrance of latent infection in green apricot fruit and Jenkin and Reinganum (53) reported on the occurrance of quiescent infection in apricot and peach. Jerome (56) concluded from her study on peach infected by M. fructicola that conidia collected among the hairs on the fruit surface but remain dormant until the fruit ripens. She suggested the low incidence of the disease in the immature fruit due to the

high mechanical resistance of the epiderm to penetration. She suggested that resistance decreased as the fruit ripens, thus she claimed latent that contamination; not latent infection was responsible for fruit rot. Powell (77), Phillips and Harvey (76) supported Jerome's conclusion, that the disease develops from conidia on the surface. However, Ogawa (71) reported that gaseous emanation from green peach fruit was toxic to conidia on the surface but those from ripe fruit of peach, apricot and cherry were stimulatory. Wade and Cruickshank (93) demonstrated the addition of extract from ripe fruit to the spore resulted in the development of infection. The results obtained in our studies from the culturing of tissue from green fruit as well as the histological evidence that the fungal hyphae was present within the tissue proved the phenomena of latent infection on sweet cherry can take place early in the season. This kind of infection may provide source of inoculum due to early sporulation on these infected fruit to the healthy fruit prior to harvest. Our data on the increase soluble solids content during fruit ripening may account for the development of the fungus that had been latent in the epidermal layer.

The application of benlate at bloom period seems to reduce the incidence of green fruit rot as indicated by the lower number of latent infections on treated trees compared to the non-treated trees. This may be because the fungicide interferred with the conidia germination in the early stages of infection. This finding agreed with earlier work of Kable (61) whow showed that the application of protective fungicide at bloom reduced the infection on peach fruit

caused by M. fructicola.

A linear relation was reported by Grover (35) between the growth rate of these fungi in culture and utilization of soluble solid. M. fructicola utilized 2.3% and M. laxa utilized 1.7% from the basal medium for their optimum growth. By determining the amount of soluble solids available to the two brown rot fungi, Grover (35) was able to determine the apparent natural resistance of sour cherry to M. laxa. The evidence obtained from our studies, showed a strong correlation between the soluble solid content and the fruit rot development at different stages of maturity (Table 22,23). Soluble solid content available to M. laxa increased as the cherry fruit approaches maturity (Table 23). The correlation of this with the growth of the fungus, can be used to determine the relative susceptibility of sweet cherry cultivars to fruit rot. Similar index could be used to determine when the grower should apply measures to protect the fruit from germinating conidia on the fruit surface prior to harvest.

# CONCLUSIONS

Evaluation of the data obtained from this research suggests the following conclusions and remarks concerning brown rot disease of sweet cherries in western Oregon.

1. Contrary to earlier surveys M. laxa was found more frequently and considered the most common species in western Oregon orchards where samples have been collected. Ninety-seven percent of the isolation from sweet cherry blossoms and more than eighty percent from the fruit were M. laxa.

2. Brown rot epidemics in western Oregon occurred when high level of inoculum corresponded with warm-moist climatic conditions. Since these climatic conditions frequently occur during bloom, it is important that the inoculum level be minimized.

3. There were no intermediate or atypical isolates of both Monilinia spp. obtained from western Oregon orchards.

4. Canker size was largest on peach followed by sour cherry and then sweet cherry when inoculated with different isolates of Monilinia spp.

5. There was no evidence of benomyl tolerance in isolates obtained from western Oregon where this fungicide had been used since the fungicide was developed in the late 1960s.

6. Host specific differences in pathogenicity exist among isolates of M. laxa obtained.

7. Histological evidence and plating tissue from symptomless green fruit contain latent Monilinia mycelium that can become active

as the fruit ripens. However, recovery of conidia from fruit wash confirms the presence of Monilinia on the fruit surface.

8. Increased susceptibility of mature fruit is probably due to the increased sugar levels available to the latent mycelium as the total soluble solids increases during the fruit ripening. Also it could be related to factors (such as biochemical and cellular processes) that modify the susceptibility of fruit to brown rot fungus.

# BIBLIOGRAPHY

1. Abbas, H. K., Damirdugh, I. S., El-Behadli and Ogasua, J. M. (1981) Monilinia laxa in stone fruit in Iraq. Plant Disease 65: 916-197.

2. Adams, D. B., McNeil, J., Hanson-Merz, B. M.; McCharthy, D. F. and Stroker, J. (1949) The estimation of latent infection in oranges. Aust. J. Sci. Res., B2, 1-18.

3. Aderhold, R. and Ruhland, W. (1905) Zurkenntnis der obotbaum-sklerotinein Arb. k. biol. abt. land-und forstw. kaiserl. Gesundheitsamte 4: 427-442.

4. Barnett, H. L. and Bodine, E. W. (1944) Results of the brown rot survey, and notes on some other diseases of stone fruits in Central California. Plant Dis. Rept. 28: 181-183.

5. Barss, H. P. (1923) Brown rot and related diseases of stone fruit in Oregon. Oregon Agr. Exp. Sta. Circ. No. 53.

6. Barss, H. P. (1925) Serious blossom blight in Pacific Northwest orchard due to species of Monilia. Phytopathology 15: 126 (Abstr).

7. Baur, K. E. and Huber, G. A. (1941) Brown rot on stone fruit in western Washington. Phytopathology 31: 718-731.

8. Brooks, G. E. and Cooley, J. S. (1921) Temperature relation of stone fruit fungi. J. Agr. Res. 22: 451-465.

9. Brooks, C. and Fisher, D. F. (1924) Prune and cherry brown rot investigation in the Pacific Northwest. U.S. Dept. of Agri-Bull. 1252: 1-21.

10. Byrde, R. J. W. (1956) The variatal resistance of fruits to brown rot. I. Infection experiments with sclerotinia fructigena Aderh and Ruhl on certain dessert culinary and cider varieties of apple. J. Hort. Sci. 31: 188-195.

11. Byrde, R. J. W. and Willetts, H. J. (1977) The brown rot fungi of fruit: their biology and control. pp. 171, Pergamon Press.

12. Calavan, E. C. and Keitt, G. W. (1948) Blossom and spur blight (Sclerotinia laxa) of sour cherry. Phytopath. 38: 857-882.

13. Carlile, M. J. and Sellin, M. A. (1963) An endogenous inhibition of spore germination in fungi. Trans. Brit. Mycol. Soc. 46: 15-18.

14. Cation, D. and Dunegan, J. C. (1949) The overwintering of Monilinia fructicola in twig cankers under Michigan conditions. Plant Disease Rept. 33: 97-98.

15. Cation, D., Dunegan, J. C. and Kephart, Joyce. (1949) The occurrence of Monilinia laxa in Michigan. Plant Dis. Rept. 33: 96.

16. Chitzanidis, A. (1971) Test of eradicant fungicides against Sclerotinia laxa on sour cherry trees. Inst. Phytopathol. Benaki Ann. (ns) 10: 119-124.

17. Chochriakova, T. M. (1971) Evaluation of cherry resistance to Moniliosis, Monilinia Cinerea. Tr. Prikladonoi Bot. Genet. Selek. 43: 231-236 (Eng. Summ).

18. Cooley, J. S. (1914) A study of the physiological relation of Sclerotinia Cinerea (Bon) Schroeter. Ann. Missouri Bot. Gard. 1: 291-326.

19. Conel, J. L. (1914) Studies of the brown rot fungus in the vicinity of Champaign and Urbana, Illinois. Phytopathology 4: 93-101.

20. Corbin, J. B. (1962) Factor determining the length of the incubation period of Monilinia fructicola (Wint.) Honey in fruits of prunus, spp. Aust. J. Agri. No. 14: 51-60.

21. Corbin, J. B., Ogawa, J. M. and Schultz, H. B. (1968) Fluctuations in numbers of Monilinia laxa conidia in an apricot orchard during the 1966 season. Phytopathology 58: 1387-1394.

22. Corbin, J. B. and Ogawa, J. M. (1974) Springtime dispersal patterns of Monilinia laxa conidia in apricot, peach, prune and almond trees. Can. J. Bot. 52: 167-176.

23. Cross-Raynaud, P. H. and Gharieni, R. (1967) Annis Inst. Natn. Pech. Ugron Tunisia 40(9), 16 pp. R. A. M. 48: 1845 (1969).

24. Cross-Raynaud, P. H. (1969) Evaluating resistance to Monilinia laxa (Aderh and Ruhl) Honey of varieties and hybrids of apricots and almond using mean growth rate of canker on young branches as a criterion of susceptibility. J. Amer. Soc. Hort. Sci. 94: 282-284.

25. Curtis, K. M. (1928) The morphological aspects of resistance to brown rot in stone fruit. Ann. Bot. 42: 39-68.

26. Dekker, J. (1976) Acquired resistance to fungicides. Ann. Rev. Phytopathol. 14: 405-428.

27. English, H. and Adriana pinto de Torres y Joyce Krikc. (1969) Reconocimiento de especies del genero Monilinia en frutales de carozo y en membrillo de flor en Chile. Agricultura Tecnica 29: 54-59.

28. Elssman, N. (1939) Prufung von Sauerkirschensorten auf ihr Verhalten gegen Sclerotinia Cinerea Schroet. Forschungsdient 7: 361-366.

29. Evans, A. W. and Owens, C_ E. (1941) Incidence of Sclerotinia fructicola and S. laxa in sweet cherries in Oregon. Phytopathology 31: 469-471.

30. Ezekiel, W. N. (1924) Fruit rotting Sclerotinias. II. The American brown-rot fungi. Bull. Md. Agric. Exp. Sta. 271: 87-142.

31. Ezekiel, W. N. (1925) Presence of European brown rot fungus in America. Phytopathology 15: 535-542.

32. Gaumann, E. (1950) Principles of plant infection. p. 442 (Croshy, Lockwood and Son, London).

33. Georgopoules, S. G. and Dovas, C. (1973) A serious outbreak of strains of Cercospora beticola resistant to benezimidazole fungicides in Northern Greece. Plant Dis. Rept. 51: 321-324.

34. Gerhardt, F., English, H. and Smith, E. (1945) Cracking and decay of Bing cherries as related to the presence of moisture on the surface of the fruit. Proc. Amer. Soc. Hort. Sci. 46: 191-198.

35. Grover, R. K. (1963) Soluble solid utilization method for determination of natural resistance in sour cherries against brown rot fungi. Indian Phytopath. 16: 205-209.

36. Hall, R. (1971) Pathogenicity of Monilinia fructicola. II. Penetration of peach leaf and fruit. Phytopath. Z, 72: 281-290.

37. Hall, R. (1972) Pathogenicity of Monilinia fructicola. III. Factors influencing lesion expansion. Phytopath. Z, 73: 27-38.

38. Harrison, T. H. (1928) Brown rot of fruits and associated diseases in Australia, Part I. History of the disease and determination of the causal organism. J. Proc. Roy. Soc. N. S., Wales, 62: 99-151.

39. Harrison, T. H. (1933) Brown rot of fruits and associated diseases of decidous fruit trees. I. Historical review and critical remarks concerning taxonomy and nomenclature of the causal organism. J. Proc. Roy. Soc. N. S., Wales 67: 132-177.

40. Hart, R. (1947) The new Merton cherries. J. Pomol. 23: 112-116.

41. Hashmi, M. N., Morgon-Jones, G. and Kendrick, B. (1972) Can. J. Bot. 50: 2419-2421.

42. Hawker, I. E. (1957) Ecological factors and the survival of fungi. Symp. Soc. Gen. Microbiol. 7: 238-258.

43. Hesse, Claron O. (1938) Variation in resistance to brown rot in apricot varieties and seedling progenies. Proc. Am. Soc. Hort. Sci. 36: 266-268.

44. Hewitt, W. B. and Leach, L. D. (1939) Brown rot Sclerotinias occurring in California and their distribution on stone fruit. Phytopathology 29: 337-351.

45. Hoffman, G. M. (1970) Nuclear condition in Monilinia fructigena and Monilinia laxa. Phytopath. Z. 68: 143-154 (English summary).

46. Hoffman, G. M. (1972) Heterokaryosis in wild strains of Monilinia laxa. Phytopath. Z. 79: 326-340 (English summary).

47. Hoffman, G. M. (1974) Occurrence of Heterokaryosis in Monilinia laxa. Phytopath. Z. 79: 193-202 (English summary).

48. Honey, E. E. (1928) The Monilioid species of Sclerotinia. Mycologia 20: 127-156.

49. Horne, W. T. and Palmer, D. F. (1935) The control of Dothiorella rot on avocado fruit. Bull. Calif. Agric. Exp. Sta. No. 594.

50. Humphrey, J. E. (1893) On Monilia fructigena. Bot. Gaz. 8: 85-93.

51. Janick, J. and Moore, J. N. (eds.) (1975) Advances in fruit breeding. Purdue, U.S.A., University Press, 640 pp.

52. Jehle, R. A. (1913) The brown rot canker of the peach. Phytopathology 3: 105-111.

53. Jenkins, P. T. and Reinganum, C. (1965) The occurrence of quiescent infection of stone fruits caused by Sclerotinia fructicola (Wint.). Rehm. Australian. J. Agric. Res. 16: 131-140.

54. Jenkins, P. T. (1965) The dispersal of Conidia of Sclerotinia fructicola (Wint.). Rehm. Aust. J. Agric. Res. 16: 627-633.

55. Jenkins, P. T. (1967) The longevity of Conidia of Sclerotinia fructicola (Wint.). Rehm. under field conditions. Aust. J. Biol. Sci. 21: 937-945.

56. Jerome, S. M. R. (1958) Brown rot of stone fruits: latent contamination in relation to spread of the disease. J. Aust. Inst. Agric. Sci. 24: 132-140.

57. Johanson, D. A. (1940) Plant Microtechnique. New York, McGraw-Hill, 523 pp.

58. Jones, A. L. and Walker, R. J. (1976) Tolerance of Venturia inaequalis to dodine and benzimidazole fungicides in Michigan. Plant Dis. Rept. 60: 40-44.

59. Jones, A. L. and Ehret, G. R. (1976) Isolation and characterization of benomyl tolerant strains of Monilinia fructicola. Plant Dis. Rept. 60: 765-769.

60. Kable, P. E. (1965) The fruit peduncle as an important overwintering site of Monilinia fructicola in the Murrumbridgee Irrigation Areas. Aust. J. Exp. Agric. Anim. Husb. 5: 172-175.

61. Kable, P. E. (1971) Significance of short-term latent infection in the control of brown rot in peach fruits. Phytopathol. Z. 70: 173-176.

62. Kilian, K. (1926) Variation des caracteres morphologiques et biologiques chez les Ascomycetes et les Deuteromyates parasites. Rev. Path. Veg. 13: 129-166.

63. Lin, C. K. (1940) Germination of the Conidia of Sclerotinia fructicola, with special reference to the toxicity of copper. Mem. Cornell Univ. Agric. Exp. Sta. 233, 33 pp.

64. Matheny, W. A. (1913) A comparison of the American brown rot fungus with Sclerotinia fructigena and S. cinera of Europe. Bot. Gaz. 56: 418-432.

65. Miller, M. W. and Fletcher, J. T. (1974) Benomyl tolerance in Botrytin Cinerea isolated from glasshouse crops. Trans. Br. Mycol. Soc. 62: 99-102.

66. Morschel, J. R. C. (1955) Brown rot of stone fruit in New South Wales. Observation and trial on the Murrumbidgee Irrigation Area. Agric. Gaz. N. S. W. 66: 146-150.

67. Norton, J. B. S. and Ezekiel, W. N. (1924) The name of the American brown rot Sclerotinia. Phytopathology 14: 31-32.

68. Ogawa, J. M. (1953) Pathogenicity comparisons between Sclerotinia laxa and S. fructicola. Phytopathology 43: 589. (Abstr.)

69. Ogawa, J. M., English, W. H. and Wilson, E. E. (1954) Survey for brown rot of stone fruits in California. Plant Dis. Rept. 38: 254-257.

70. Ogawa, J. M. and English, W. H. (1954) Means of differentiating a typical isolates of Sclerotinia laxa and S. fructicola. Phytopathology 44: 500 (Abstr.)

71. Ogawa, J. M. (1958) The influence of emanation from fruits of prunus species on spore germination of the brown rot organisms. Phytopathology 48 (Abstr.)

72. Ogawa, J. M. and English, H. (1960) Relative pathogenicity of two brown rot fungi, S. laxa and S. fructicola on twigs and blossoms. Phytopath. 50: 550-558.

73. Ogawa, J. M. and McCain, A. H. (1960) Relations of spore moisture content to spore shape and germination reaction to temperature. Phytopathology 50: 85 (Abstr.)

74. Ogawa, J. M., Mangi, B. T. and Schreader, W. R. (1975) Monilinia life cycle on sweet cherries and its control by overhead sprinkler fungicide application. Plant Dis. Rept. 59: 876-880.

75. Phillips, D. J. and Harvey, J. M. (1975) Selective medium for detection of Inoculum of Monilinia spp. on stone fruits. Phytopathology 65: 1233-1236.

76. Posey, G. B. (1915) Studies of Monilinia blight of fruit trees. Science (N.S.) 42: 583.

77. Powell, D. (1951) Phygon XL for the control of peach blossom blight. Pl. Dis. Rept. 35: 76-77.

78. Powelson, R. L. (1960) Initiation of strawberry fruit rot caused by Botrytis cinerea. Phytopath. 50: 491-494.

79. Ramsdell, D. C. and Ogawa, J. M. (1973) Reduction of Monilinia laxa inoculum potential in almond orchards resulting from dormant benomyl sprays. Phytopathology 63: 830-836.

80. Reade, J. M. (1908) Preliminary notes on some species of Sclerotinia. Ann. Mycol., Berlin, 6: 114-115.

81. Roberts, J. W. and Dunegan, J. C. (1932) Peach brown rot. U.S. Dept. Agric. Bull. No. 328, 59 pp.

82. Rudolph, B. A. (1925) Monilia blossom blight (brown rot) of apricots. Calif. Univ. Agr. Expt. Sta. Bull. No. 383.

83. Seal, J. L. (1924) Biological specialization in Sclerotinia sp., the organism causing brown rot in stone fruits. Minn. Studies Plant Sci. Studies Biol. Sci. 5: 281-283.

84. Simmonds, J. H. (1941) Latent infection in tropical fruit in relation to the part played by species of Gleosporium and Colletotrichum. Proc. Roy. Soc. Qd. 52: 92-120.

85. Shepherd, C. J. (1968) In: Report of the research work on brown rot of stone fruit (ed. Australian Brown Rot Research Committee). pp. 24-45. Government Printer, Melbourne.

86. Smith, W. L., Miller, W. H. and Bassett, R. D. (1965) Effects of temperature and relative humidity on germination of Rhizopus Stolonifer and Monilinia fructicola spores. Phytopathology 55: 604-606.

87. Sutton, T. B. and Clayton, C. W. (1972) Role and survival of Monilinia fructicola in blighted peach branches. Phytopathology 62: 1369-1373.

88. Szkolnik, M. and Gilpatrick, J. D. (1969) Apparent resistance of Venturia inaequalis to dodine in New York apple orchards. Plant Dis. Rept. 53: 861-864.

89. Szkolnik, M. and Gilpatrick, J. D. (1977) Tolerance of Monilinia fructicola to Benomyl in Western New York State Orchards. Plant Dis. Rept. 61: 654-657.

90. Tate, K. G., et al. (1974) Survey for benomyl tolerant isolates of Monilinia fructicola and Monilinia laxa in stone fruit orchards of California. Plant Dis. Rept. 58: 663-665.

91. Thind, K. S. and Keitt, G. W. (1949) Studies on variability of Sclerotinia fructicola (Wint.) Rehm. Phytopathology 39: 621-636.

92. Wade, G. C. (1956) Investigations on brown rot of apricots caused by Sclerotinia fructicola (Wint.), Rehm. I. The occurrence of latent infection in fruit. Aust. J. Agric. Res. 7: 504-515.

93. Wade, G. C. and Cruickshank, R. (1973) Abstr. papers, 2nd Int. Cong. Pl. Path., 0006.

94. Weaver, L. O. (1943) Effect of temperature and relative humidity on occurrence of blossom blight of stone fruit. Phytopathology 33: 15 (Abstr.).

95. Whan, J. H. (1976) Tolerance of Sclerotinia fructicola to benomyl. Plant Dis. Rept. 60: 200-201.

96. Wicks, T. (1981) Suppression of Monilinia laxa spore production by fungicide applied to infected apricot twig during dormancy. Pl. Dis. Rept. 65: 911-912.

97. Willetts, H. J. (1968) Factors influencing the production of Stromata and Microcanidia by Sclerotinia fructicola (Wint.), Rehm. Ann. Bot. 32: 219-232.

98. Willetts, H. J. (1968) The development of stromata of Sclerotinia fructicola and related species II in fruit. Trans. Br. Mycol. Soc. 51: 633-642.

99. Willetts, H. J. (1969) Cultural characteristics of the brown rot fungi (Sclerotinia spp.). Mycologia 61: 332-339.

100. Willetts, H. J. (1971) Survival of fungal sclerotia under adverse conditions. Biol. Rev. 46: 387-407.

101. Willetts, H. J. and Calonge, F. D. (1969) Spore development in the brown rot fungi (Sclerotinia spp.). New Phytol. 68: 123-131.

102. Wilson, E. E. and Ogawa, J. M. (1979) Fungal, bacterial and certain non-parasitic diseases of fruit and nut crops in California. Calif. Agric. Exp. Stn. Bull. 190 pp.

103. Wormald, H. (1928) Further studies of the brown rot fungi. III. Nomenclature of the American brown rot fungi. A review of literature and critical remarks. Trans. British Mycol. Soc. 13: 194-204.

104. Wormald, H. (1954) The brown rot of fruit trees. Ministry of Agric. and Fisheries (Engl.). Tech. Bull. No. 3, 113 pp.

105. Yoder, K. S. and Klos, E. J. (1976) Tolerance to dodine in Venturia Inaequalis. Phytopathology 66: 918-923.

106. Zwygart, T. (1970) Studies on host parasite interaction in Monilia diseases of fruit trees. Phytopathol. Z. 68: 97-130.