

Susceptibility of Immature and Mature Sweet and Sour Cherries to *Monilinia fructicola*

J. NORTHOVER and A. R. BIGGS, Agriculture Canada, Research Station, Vineland Station, Ontario L0R 2E0

ABSTRACT

Northover, J., and Biggs, A. R. 1990. Susceptibility of immature and mature sweet and sour cherries to *Monilinia fructicola*. Plant Dis. 74:280-284.

Fruits of Vista and Bing sweet cherries and Montmorency sour cherry were harvested at weekly intervals between shuck fall and full maturity in 1986 and 1987. Unwounded fruits were inoculated individually with a 30- μ l drop containing 10^6 , 10^5 , 10^4 , or 10^3 conidia of *Monilinia fructicola* per milliliter. Fruits were evaluated for lesion development after incubation for 6 days at 20 C and relative humidity above 95%. Sweet cherries were more susceptible to infection than sour cherries at intermediate and low inoculum concentrations. Initially the immature fruits of both species were as highly susceptible to infection as mature fruits at inoculum concentrations of 10^6 conidia/ml. Host resistance rose with the onset of pit hardening but decreased 3 wk before maturity, coinciding with yellowing and reddening of the epidermal tissue. Fungicide protection against brown rot appears warranted at shuck fall and before harvest for both sweet and sour cherries. Midseason protection appears necessary for sweet cherries but not for Montmorency sour cherry.

Brown rot of stone fruits, caused by *Monilinia fructicola* (Wint.) Honey, is an annual concern under the temperate conditions of southern Ontario. Crop losses at harvest of unprotected sweet cherries (*Prunus avium* (L.) L.) usually are very high, and weekly fungicide protection during fruit development is a commercial necessity. On Montmorency sour cherry (*P. cerasus* L.), by contrast, one fungicide application during bloom and one preharvest application generally suffice for adequate protection against brown rot.

In general, immature stone fruits are less susceptible than mature fruits to infection by *M. fructicola* (2,3,5,10), although in a companion study (1), peach fruits before pit hardening were as susceptible as mature fruits. No comparative susceptibility data were available for commercial sweet or sour cherry cultivars.

Our objective in this study was to determine whether different commercial fungicide programs for sweet and sour cherry cultivars are justified by a difference in susceptibility to *M. fructicola* during fruit maturation.

MATERIALS AND METHODS

Fruits were collected from fungicide-free mature trees of sweet cherry cultivars Vista and Bing and sour cherry cultivar Montmorency. For each cultivar, 500–600 fruits with attached stems were collected at weekly intervals commencing

at shuck fall (26 May 1986, 21 May to 2 June 1987) and continuing until fruit maturity (1–21 July 1986, 24 June to 9 July 1987), with the dates varying for the different cultivars.

In the laboratory, the fruits remained unwashed, but the stems were trimmed to less than 6 mm. Fifty fruits were arranged noncontiguously and with suture side uppermost on sterile 13 \times 13-mm galvanized steel mesh screens, supported within 42 \times 29 \times 6-cm aluminum baking trays. In the early-season samples, fruits too small to be supported by the screen were attached to double-sided adhesive tape attached to the screen.

On each sample date, 100 fruits of each cultivar were inoculated with each inoculum concentration. Each fruit was inoculated on the suture with a 30- μ l drop of a conidial suspension of *M. fructicola* isolate S.4 (benomyl-sensitive) grown on potato-dextrose agar (PDA) and prepared as described previously (1). The conidial suspension was adjusted to twice the required concentration and mixed with an equal volume of double-strength Miller's germination solution (6) to give concentrations of 10^6 , 10^5 , 10^4 , and 10^3 conidia/ml. The lowest concentration was used only in 1987. Fruits in the control treatment received drops of single-strength Miller's solution. Germination of conidia on PDA was examined for each weekly inoculation to verify high (>95%) viability.

The inoculated fruits were incubated at 20 C for 22 hr at relative humidity (RH) above 95% in a stainless steel wet room. They were then examined for the presence of the inoculum drops and were then moved to a well-ventilated room at 20 C and 60% RH, where the inoculum

drops dried over a 2-hr period. Fruits were then returned to the wet room and incubated at 20 C and RH above 95%. They were examined daily and after 6 days were evaluated for percentage fruit infection. The criterion used for assessing infection was the same as that used in our companion study of peach fruits (1), except that only fruits with inoculum drops still present 22 hr after inoculation were evaluated. Fruits were considered infected when lesions were greater than and were centered on the inoculation site.

Percentage infection data were transformed to the arcsine-square root percentage (9) and were analyzed for each cultivar separately using the general linear model procedure (SAS Institute, Cary, NC) with least squares analysis of variance and type III sums of squares for unbalanced linear models with randomized design. Data for the 2 yr (1986 and 1987) were treated as blocks, and differences between the averaged inoculum concentration effect at different sampling dates were examined with Duncan's multiple range test (9).

The stage of fruit development was defined at each collection date. Ten fruits of each cultivar were examined, and their length, greatest width, weight, surface color, pulp color, and concentration of soluble solids (degrees Brix) (measured with a hand refractometer [T/C, American Optical, Buffalo, NY]) were recorded. Ten fruits were cut with a knife to determine the incidence of hardened pits. Mean maximum and mean minimum temperatures and rainfall for the 6–8 days preceding each fruit collection were obtained from the meteorological records for Vineland Station kept by the Horticultural Research Institute of Ontario, Ontario Ministry of Agriculture and Food.

RESULTS

The incidence of brown rot-infected fruits of the three cherry cultivars was affected to the greatest extent by differences in inoculum concentration (Table 1). The effect of sample date was also highly significant, and interactions between inoculum concentration and sample date were significant for each cultivar, although of lesser magnitude.

Infection of fruits of both sweet cherry cultivars inoculated with 10^6 conidia/ml exceeded 93% throughout both growing seasons, except on the first collection date in 1987 (Figs. 1 and 2), when a slightly lower conidial concentration

Accepted for publication 19 October 1989 (submitted for electronic processing).

than intended was used. Similarly high incidences of infection were obtained with 10^5 conidia/ml in 1987. However, with a concentration of 10^4 conidia/ml in 1987 (Fig. 2B), Bing fruits were highly infected on 27 May (shuck fall, or shedding of the corolla tube) and again on and after 17 June but showed low infection on 10 June. A similar response pattern was noted during the corresponding period in 1986 with inoculum of 10^5 conidia/ml (Fig. 1B). However, Vista fruits did not show a reduction in infection in early June in the corresponding treatments. With the lowest inoculum concentrations in both years (Figs. 1A and 2A), Bing fruits were infected only on 26–27 May (shuck fall) and in early to mid-July (fruit maturity). In contrast, Vista fruits showed low to moderate infection throughout both seasons in response to these inoculum levels.

Montmorency sour cherry fruits were almost completely resistant to infection after inoculation with an inoculum concentration of 10^4 conidia/ml throughout both seasons (Figs. 1A and 2B). Fruits inoculated with 10^5 conidia/ml demonstrated an early and midseason resistance followed by a progressive increase in susceptibility beginning 2–4 wk before fruit maturity (Figs. 1B and 2C). At the highest inoculum concentration (10^6 conidia/ml), a very different pattern emerged. In both years, Montmorency fruits were susceptible at the shuck fall stage, with infection incidences of 87 and 88%, but within 2 wk infection declined to 3–7%, then rose quite rapidly and exceeded 90% at fruit maturity (Figs. 1C and 2D).

The proportion of fruits with hardened pits reached 100% between 2 and 10 June in both years (Tables 2 and 3). This period coincided with declining incidences of infection with intermediate and high inoculum concentrations for Bing sweet and Montmorency sour cherry, respectively. The subsequent increases in infection were associated with rapidly rising concentrations of soluble solids to 13.0–14.2° Brix for Bing and 8.4–10.8° Brix for Montmorency, which occurred 1–3 wk after 100% pit hardening.

Fruit surface tissues began to yellow at or 1 wk after 100% pit hardening. The appearance of red pigment coincided with average soluble solids greater than 11° Brix for Vista and Bing, but the relationship was less consistent for Montmorency. Changes in the color of fruit pulp followed color changes of the surface tissues. Fruit reddening was associated with increased susceptibility to infection of Bing fruits and especially Montmorency fruits.

Fruit of each cultivar underwent a period of rapid growth during which individual fruit weight doubled within a week to more than 3 g. This growth occurred generally 2–3 wk after 100% pit

hardening, and in 1987 (Table 3), it coincided with increased susceptibility to infection following the temporary decline at pit hardening in both Bing and Montmorency. Fruit width increased proportionally more than fruit length during both years for all three cultivars.

The proportion of all the inoculum drops (0 – 10^6 conidia/ml) that remained on the sweet cherry fruits for 22 hr (i.e.,

drop stability) was as low as 20% at shuck fall (21 May 1987) but increased abruptly to 87–98% coincident with hardening of the pits (Tables 2 and 3). On sour cherry fruits, drop stability was 68–74% before pit hardening, but it increased erratically to 98–99% on mature fruits.

In early June 1986 and 1987, when resistance in Bing and Montmorency fruits increased temporarily, no extreme

Table 1. Least squares analysis of variance for percentage fruit infection of cherry cultivars Vista, Bing, and Montmorency sampled approximately weekly and inoculated with conidia of *Monilinia fructicola*^a

Source	Vista		Bing		Montmorency	
	df	Mean squares ^b	df	Mean squares ^b	df	Mean squares ^b
Inoculum						
concentration	3	24,700**	3	46,946**	3	27,855**
Sample date	5	2,518**	7	2,873**	8	3,295**
Replication	3	115	3	72	3	40
Block (year)	1	7,394**	1	8,903**	1	124
Inoculum × sample date	15	476*	20	683**	21	968**
Residual	140	281	173	291	167	169

^aEach fruit was inoculated with a drop containing 10^3 , 10^4 , 10^5 , or 10^6 conidia/ml and incubated for 24 hr at 20 C. The percentage of fruit infected at 6 days was determined. Mean squares were derived from type III sums of squares for unbalanced linear models and a randomized block design over time using transformed data.

^bAsterisks indicate significance at $P \leq 0.01$ (**) and $P \leq 0.05$ (*).

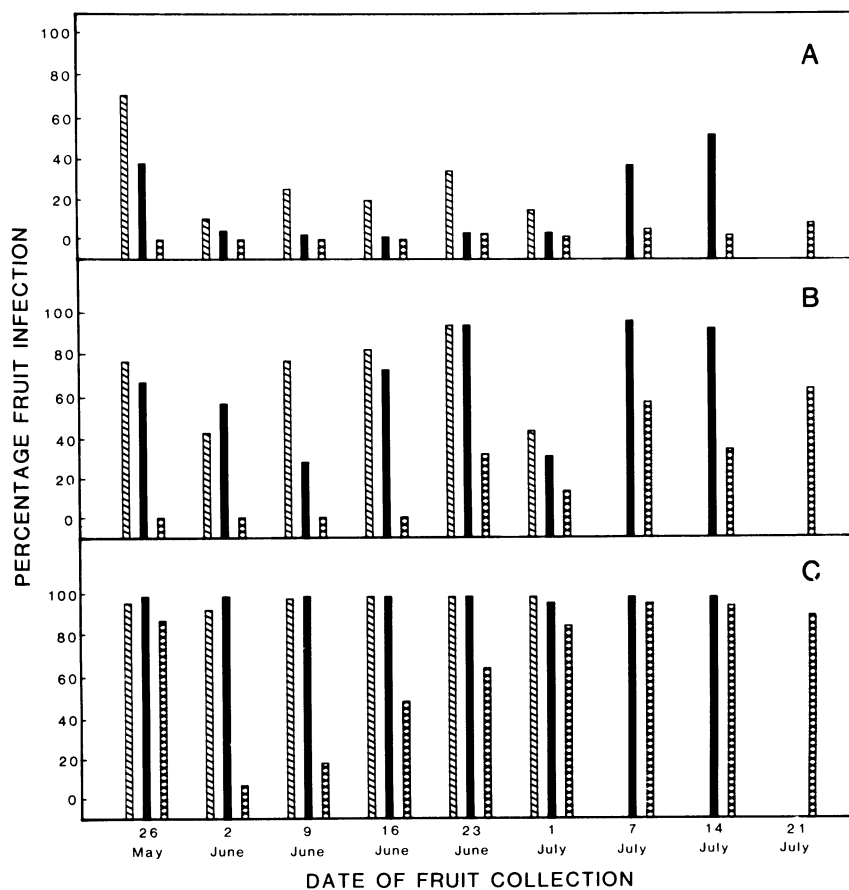


Fig. 1. Percentage infection of cherry fruits sampled between shuck fall and fruit maturity in 1986 and inoculated with a drop of inoculum containing (A) 10^4 , (B) 10^5 , or (C) 10^6 conidia of *Monilinia fructicola* per milliliter. For each fruit collection date, the infection data are shown as bars for (left to right) Vista (hatched), Bing (solid), and Montmorency (diamond pattern). Vista and Bing fruits were unsuitable for use in experiments on 7–21 July and 21 July, respectively, because of overmaturity.

climatic conditions preceded fruit sample collection. The maximum temperature was only 25.5 C and 30.4 C during the weeks before 2 June in 1986 and 1987, respectively (Table 4). No rain fell during the week before 2 June 1987. Comparable or higher temperatures were associated with the period of increased susceptibility of the more mature fruits sampled in early July.

DISCUSSION

Unwounded fruits of sweet cherry cultivars Vista and Bing were more susceptible to infection with low and intermediate concentrations of *M. fructicola* conidia than those of Montmorency sour cherry. However, at the

high concentration of 10^6 conidia/ml, the mature fruits of both sweet and sour cherries were equally infected. The degree of susceptibility varied with the maturity of the fruit (sampling date), and this factor interacted with inoculum concentration to affect the incidence of infection differently for the two cherry species. Host resistance in sour cherry was overcome by the highest inoculum concentration of 10^6 conidia/ml early and late in the season, but the fruits remained relatively resistant to infection in midseason. A similar pattern was evident in the response of Bing sweet cherries to 10^5 and 10^4 conidia/ml in 1986 and 1987, respectively, but moderate midseason resistance was overcome by

a 10-fold increase of inoculum concentration in both years.

The susceptibility of small, green fruits at shuck fall has not been described previously for sweet or sour cherry. This phenomenon is more transient than that described for Redhaven and Loring peaches (1), possibly because the earlier-maturing cherry fruits develop more rapidly. In each of the three species, the midseason elevated resistance corresponded to the onset of pit hardening. In peach, the increased resistance was a broad response to 10^4 , 10^5 , and 10^6 conidia/ml. However, for Bing sweet cherry it was evident only with 10^5 and 10^4 conidia/ml in 1986 and 1987, respectively, and for the more resistant sour cherry, the phenomenon was evident only with concentrations of 10^6 conidia/ml in 1986 and 10^5 and 10^6 conidia/ml in 1987. An inoculum concentration of 10^6 conidia/ml is probably as high as might occur under field conditions, since a comparable value was found in water drops running over heavily sporulating sweet cherry fruit (J. Northover and A. R. Biggs, unpublished). Thus, under orchard conditions, sour cherry and peach fruits would be expected to be resistant to infection by *M. fructicola* immediately after pit hardening.

These data relate to senescing detached fruits, which could be more susceptible to *M. fructicola* than inoculated immature fruits attached to trees. Additional research is needed to determine the susceptibility of maturing cherries on trees. The single-drop inoculation of fruits in the laboratory also differs from natural inoculation in the orchard, where inoculum may be deposited simultaneously at several sites on a fruit surface. The latter process could alter the host response to infection and hence the estimate of susceptibility.

The increase in fruit susceptibility to *M. fructicola* infection after pit hardening was associated with elevated Brix readings, increased fruit size, and fruit reddening. Fruit reddening is easily observed and is the most convenient indicator of increased susceptibility to infection of Bing and Montmorency fruits. Commercial growers use fruit reddening as the stage at which to enhance their protection programs.

When immature cherry fruits are very small, the sharply curved surface limits adhesion of inoculum drops to the fruit surface and results in low drop stability. The inoculum drop moved slowly over the surface of many of the fruits, possibly by a slow wetting action, and ran off the fruit, in sharp contrast to the high degree of stability of drops deposited on mature fruits with obviously waxy cuticles. The frequent runoff of inoculum-containing raindrops under orchard conditions may have considerable epidemiological significance. It would promote faster drying of fruits and a

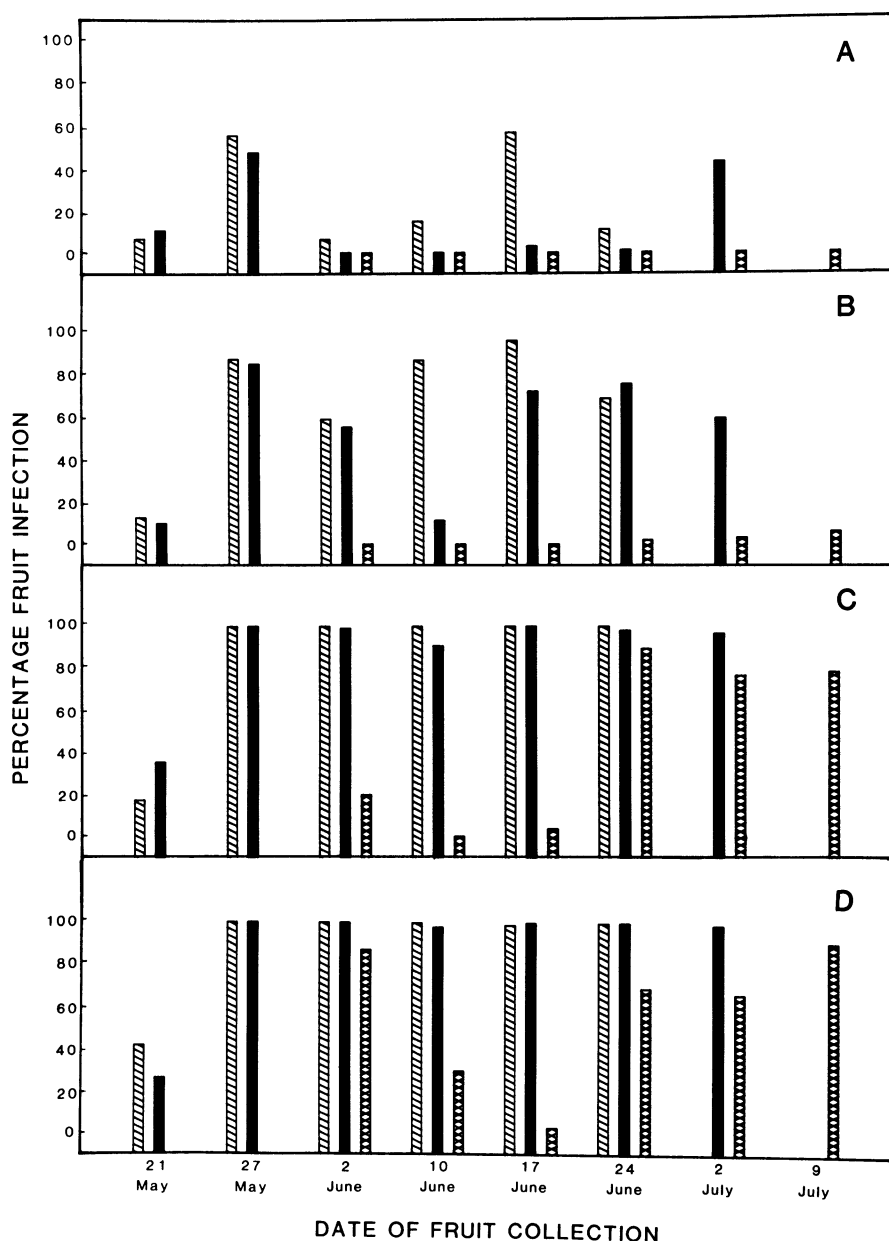


Fig. 2. Percentage infection of cherry fruits sampled between shuck fall and fruit maturity in 1987 and inoculated with a drop of inoculum containing (A) 10^3 , (B) 10^4 , (C) 10^5 , or (D) 10^6 conidia of *Monilinia fructicola* per milliliter. For each fruit collection date, the infection data are shown as bars for (left to right) Vista (hatched), Bing (solid), and Montmorency (diamond pattern). On some dates fruits were unsuitable for use in experiments because they were either too small or overmature.

Table 2. Phenological characteristics^v of sweet and sour cherries and percentage infection of fruits collected during 1986 and inoculated with *Monilinia fructicola*

Cultivar Collection date	Fruit infection ^{w,x} (%)	Fruit length/ width (mm)	Fruit weight (g)	Surface color ^y	Pulp color ^y	Pits hardened (%)	Soluble solids (° Brix)	Drop stability ^z (%)
Vista								
26 May	82 a	13.0/10.9	0.9	G	G	0	6.5	40
2 June	49 d	15.2/11.6	1.1	G/Y	G/Y	100	10.5	87
9 June	67 b	17.0/13.8	1.6	G/R	G/P	100	11.0	94
16 June	67 b	19.4/18.1	3.3	Y/R	Y/R	100	9.8	99
23 June	72 a	23.3/22.2	6.3	O/R	O/P	100	16.2	100
1 July	52 c	23.9/23.1	6.9	R	R	100	18.2	100
Bing								
26 May	65 b	15.0/11.9	1.1	G	G	10	5.8	66
2 June	54 c	15.9/12.6	1.4	G	G	100	9.2	98
9 June	43 d	15.7/13.3	1.6	G/Y	G	100	9.4	98
16 June	58 c	17.6/16.0	2.4	G/Y	G/Y	100	7.8	100
23 June	66 b	20.3/21.3	4.8	Y/R	Y/O	100	14.2	99
1 July	44 d	22.2/24.2	6.8	O/R	Y/O	100	17.8	99
7 July	78 a	23.7/25.0	7.9	R	O/P	100	21.6	99
14 July	82 a	24.2/25.6	8.9	R	P/R	100	21.6	99
Montmorency								
26 May	29 d	11.4/ 9.4	0.6	G	G	0	5.8	68
2 June	2 f	13.0/11.8	0.9	G	G	70	9.1	87
9 June	6 f	13.1/12.2	1.0	G	G	100	7.8	96
16 June	16 e	14.0/13.2	1.3	G/P	G/Y	100	8.4	94
23 June	33 c	15.7/16.2	2.6	Y/O	Y	100	9.8	79
1 July	33 c	18.5/21.0	5.0	O	Y/O	100	10.8	93
7 July	53 a	18.0/20.7	4.5	O/R	O/P	100	13.6	98
14 July	44 b	18.5/21.6	5.4	R	O/P	100	14.2	98
21 July	54 a	18.2/21.2	5.0	R	O/R	100	12.6	99

^v Phenological data were obtained by examining 10 fruits of each cultivar on each sampling date.

^w The percentage of 300 fruits, 100 of which were drop-inoculated with 10⁴, 10⁵, or 10⁶ conidia/ml, with lesions larger than the inoculation site after incubation for 6 days at 20 C.

^x Within each cultivar, means followed by dissimilar letters are significantly different ($P \leq 0.05$) by Duncan's multiple range test, using transformed data.

^y G = green, Y = yellow, O = orange, P = pink, R = red.

^z The percentage of 400 inoculated fruits on which the inoculum drop remained at the inoculation site until the end of the inoculation period.

Table 3. Phenological characteristics^v of sweet and sour cherries and percentage infection of fruits collected during 1987 and inoculated with *Monilinia fructicola*

Cultivar Collection date	Fruit infection ^{w,x} (%)	Fruit length/ width (mm)	Fruit weight (g)	Surface color ^y	Pulp color ^y	Pits hardened (%)	Soluble solids (° Brix)	Drop stability ^z (%)
Vista								
21 May	20 d	13.5/10.5	0.8	G	G	0	4.9	38
27 May	86 a	14.5/11.0	1.1	G	G	0	5.2	26
2 June	66 c	15.0/12.8	1.3	G	G	70	5.5	97
10 June	76 b	20.5/16.0	3.0	G/Y	G/Y	100	9.6	96
17 June	88 a	20.3/18.3	3.6	R	Y/P	100	12.0	99
24 June	70 bc	21.9/20.6	4.9	R	R	100	13.0	98
Bing								
21 May	20 e	13.5/11.0	0.8	G	G	0	5.6	20
27 May	83 a	14.7/12.0	1.2	G	G	0	5.9	49
2 June	64 c	14.8/13.0	1.4	G	G	100	7.2	95
10 June	50 d	17.5/15.6	2.3	G/Y	G	100	7.8	95
17 June	69 bc	19.8/19.9	4.2	P/R	G/Y	100	13.0	98
24 June	69 bc	20.9/23.2	6.2	R	P	100	12.9	97
2 July	75 b	22.9/24.1	7.3	R	R	100	15.0	95
Montmorency								
2 June	27 c	12.2/10.7	0.8	G	G	80	6.2	74
10 June	7 cd	11.6/13.1	1.0	G/Y	G	100	7.0	64
17 June	2 d	14.0/12.7	1.4	G/R	G	100	5.2	86
24 June	40 ab	15.9/16.6	3.0	O/R	Y	100	10.8	95
2 July	37 b	17.2/20.0	4.4	O/R	Y	100	11.0	98
9 July	44 a	17.1/20.1	4.3	R	Y	100	12.2	98

^v Phenological data were obtained by examining 10 fruits of each cultivar on each sampling date.

^w The percentage of 400 fruits, 100 of which were drop-inoculated with 10³, 10⁴, 10⁵, or 10⁶ conidia/ml, with lesions larger than the inoculation site after incubation for 6 days at 20 C.

^x Within each cultivar, means followed by dissimilar letters are significantly different ($P \leq 0.05$) by Duncan's multiple range test, using transformed data.

^y G = green, Y = yellow, O = orange, P = pink, R = red.

^z The percentage of 500 inoculated fruits on which the inoculum drop remained at the inoculation site until the end of the inoculation period.

Table 4. Mean maximum and mean minimum temperatures and precipitation for the 6–8 days preceding cherry fruit sampling dates in 1986 and 1987

Sample date	Temperature (C)		Precipitation (mm)
	Maximum	Minimum	
26 May 1986	17.4	9.9	35.4
2 June 1986	25.5	13.2	22.0
9 June 1986	19.9	10.1	16.4
16 June 1986	22.7	12.8	22.6
23 June 1986	23.3	12.6	7.6
1 July 1986	23.2	13.7	13.2
7 July 1986	26.4	16.9	37.0
14 July 1986	23.0	15.4	26.8
21 July 1986	26.5	18.2	47.4
21 May 1987	17.5	8.6	4.8
27 May 1987	20.5	9.7	13.6
2 June 1987	30.4	18.8	0.0
10 June 1987	26.0	17.6	8.2
17 June 1987	26.9	14.6	11.0
24 June 1987	24.8	14.6	30.2
2 July 1987	24.3	16.6	30.2
9 July 1987	27.4	18.4	10.6

lower incidence of infection than might be expected from the high degree of susceptibility demonstrated by the laboratory inoculations.

Several authors have examined the susceptibility of immature stone fruits to *M. fructicola*, but only Corbin (2) included a species of cherry (ornamental). Corbin spray-inoculated wounded fruits and showed that immature fruits produced sporodochia more slowly than ripe fruits. Similar responses of unwounded fruits have been shown for peach (5) and plum (10), but we did not make comparable observations in the present study with cherries. The high degree of susceptibility of detached immature cherries to infection under laboratory conditions and of peaches in a companion study (1) contrasted with the low incidence of infection of field-incubated immature peaches and plums inoculated with *M. laxa* (3). However, incidents of severe brown rot and sporulation on immature sweet cherries were observed in commercial orchards in 1986 and 1989 and resembled the disease symptoms observed on the laboratory-inoculated cherries (A. R. Biggs and J. Northover, unpublished). Some infections of field-incubated cherries may remain latent until the fruit ripens, as has been found with peach (3,4) and plum (3,8).

The differing susceptibility of sweet cherries and sour cherry to *M. fructicola*

has not been documented previously; intensive fungicide programs have been recommended in Ontario (7) for both species. Normally, to protect sweet cherries against blossom blight and brown rot, growers use two or three fungicide applications during bloom, followed by five to seven weekly applications before harvest. This contrasts with growers' programs on sour cherries, which consist usually of one application during bloom and another preharvest. Additional fungicides are applied for protection against leaf spot (caused by *Coccomyces hiemalis* Higgins) and powdery mildew (caused by *Podosphaera clandestina* (Wallr.:Fr.) Lév.), and depending on the materials, these treatments could give additional fruit protection against infection by *M. fructicola*.

The low incidence of infection of sour cherries generally observed despite their susceptibility to *M. fructicola* for several weeks before harvest could be the result of low initial inoculum levels, or it could be due to the more open branch structure of sour cherry trees compared to sweet cherry trees. This open branch structure enhances air circulation, hastens drying of fruit after rain, improves fungicide penetration, and facilitates more complete spray coverage of the smaller fruit clusters of sour cherry compared to sweet cherries.

Because sour cherry blossoms (11) and immature fruits are susceptible to

infection by *M. fructicola*, protection is advisable from bloom to shuck fall, especially if overwintering inoculum is plentiful. Additional protection appears appropriate as the fruits start to turn yellow or red, about 2 or 3 wk before harvest. The greater susceptibility of sweet cherry fruits warrants a more intensive, full-season fungicide protection program. These experimentally derived conclusions support the current management practices for sweet and sour cherries grown on the Niagara Peninsula of Ontario and provide a rationale for the empirically developed schedules for fungicide application.

ACKNOWLEDGMENTS

We are grateful to Herman Neufeld, Ann Curwin, Len Mancini, and Lawrie McEwan for their excellent technical assistance and to Wayne Wilcox and Eldon Zehr for their constructive reviews of the manuscript. This research was funded in part by grants from the Ontario Ministry of the Environment, Pesticides Advisory Committee, and the Ontario Ministry of Agriculture and Food, Pest Management Research and Services Committee.

LITERATURE CITED

- Biggs, A. R., and Northover, J. 1988. Early and late-season susceptibility of peach fruits to *Monilinia fructicola*. Plant Dis. 72:1070-1074.
- Corbin, J. B. 1962. Factors determining the length of the incubation period of *Monilinia fructicola* (Wint.) Honey in fruits of *Prunus* spp. Aust. J. Agric. Res. 14:51-60.
- Fourie, J. F., and Holz, G. 1987. Infection and decay of stone fruit by *Botrytis cinerea* and *Monilinia laxa* at different stages after anthesis. Phytophylactica 19:45-46.
- Jenkins, P. T., and Reinganum, C. 1965. The occurrence of a quiescent infection of stone fruits caused by *Sclerotinia fructicola* (Wint.) Rehm. Aust. J. Agric. Res. 16:131-140.
- 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.
- Miller, H. J. 1944. The use of *Venturia inaequalis* and *Sclerotinia fructicola* with pure chemical stimulants in slide-germination tests of fungicides. (Abstr.) Phytopathology 34:1009.
- Ontario Ministry of Agriculture and Food. 1986. Fruit Production Recommendations. Publ. 360. Queen's Printer for Ontario, Toronto, Canada. 78 pp.
- Rosenberger, D. A. 1985. Observations on quiescent brown rot infections in Grand Prize plums. Pages 19-22 in: Proceedings Brown Rot of Stone Fruit Workshop, Ames, Iowa, 1983. T. R. Burr, ed. Cornell Univ. Publ. 55. Cornell University, Ithaca, NY. 22 pp.
- Steel, R. G. D., and Torrie, J. H. 1980. Principles and Procedures of Statistics, 2nd ed. McGraw-Hill, New York.
- Valleau, W. D. 1915. Varietal resistance of plums to brown rot. J. Agric. Res. 5:365-396.
- Wilcox, W. F. 1989. Influence of environment and inoculum density on the incidence of brown rot blossom blight of sour cherry. Phytopathology 79:530-534.