Influence of Environment and Inoculum Density on the Incidence of Brown Rot Blossom Blight of Sour Cherry

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I gratefully acknowledge J. R. Nevill, E. M. Bourett, and L. M. Pscheidt for technical assistance; J. Barnard for statistical analysis; and R. McMillen-Sticht for assistance in preparation of the illustrations.

Research supported in part by a grant from the New York State Integrated Pest Management program.

Accepted for publication 25 October 1988 (submitted for electronic processing).

ABSTRACT

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.

When potted Montmorency sour cherry trees were inoculated in full bloom with 5,000 conidia/ml of *Monilinia fructicola*, placed into mist chambers at 8, 12, 16, or 20 C for 0–24 hr, then incubated for 7 additional days at 20 C and 60–70% relative humidity (RH), blossom blight incidence was proportional to the temperature and duration of the wetting period. Disease incidence was nil without wetting and minimal with 3 hr of wetting, regardless of temperature, but was 5, 7, 28, and 72% with 5 hr of wetting at 8, 12, 16, and 20 C, respectively. Disease incidence increased to 21, 78, 90, and 98% after 10 hr of wetting at the same respective temperatures, and reached a level of 85–97% after 24-hr wetting periods, regardless of temperature. The influence of time and temperature on the rate of conidium germination in vitro closely paralleled the influence of wetting duration and temperature on the rate of increase of disease incidence. When potted Montmorency trees were inoculated with either 50, 500, or 5,000 conidia/ml of *M. fructicola*, placed into mist chambers for 8–24 hr at the abovementioned temperatures, and subsequently incubated at 20 C and 85–90% RH, blossom blight incidence was influenced by inoculum concentration in addition to temperature and wetting duration. Production of conidia of *M. fructicola* was assessed for 3 yr on mummified Montmorency fruits collected regularly from orchard trees between bud break and harvest, yielding only 2–18% as many conidia during the sample periods before petal fall as during the peak sampling period before harvest. These data suggest that under New York conditions, mummified fruits are often a more important inoculum source for initiating fruit rot than blossom blight.

Additional keywords: Prunus cerasus.

Brown rot is a ubiquitous disease of stone fruits in New York State. Although both the "European" (causal agent = *Monilinia laxa* (Aderh. & Ruhl) Honey) and "American" (causal agent = *M. fructicola* (Wint.) Honey) forms have been reported in the region, *M. fructicola* is considered to be the far more common pathogen (6).

M. fructicola can cause both blossom blight and fruit rot on susceptible hosts (4), yet the relative importance and relationship of these two phases of brown rot have not been documented on sour cherry (*Prunus cerasus* L.), the most widely planted stone fruit species in the Great Lakes region. Blossom blight may cause severe losses on peach by reducing the number of flowers capable of developing into fruits (11) or by reducing the amount of bearing surface as a result of subtending twig cankers (19); however, in the eastern United States blossom blight is considered to more frequently cause economic damage on peach by providing a source of inoculum for subsequent rot of the ripening fruit (11). Wilson and Ogawa (18) also have noted the general primacy of blighted blossoms as a source of inoculum for rot of stone fruits in California, as has Kable (7) for apricots in New South Wales, Australia.

Nevertheless, the perennial importance of blossom blight is subject to question. In reviewing peach brown rot, Roberts and Dunnegan (12) concluded that control measures for the blossom blight phase are needed only in certain seasons, even on relatively susceptible varieties. Anderson (2) noted that blossom blight is relatively rare in the midwestern United States, although he recognized that sporulating blossoms can represent a significant threat to ripening fruit in regions where blossom blight is common. Recent epidemiological studies of peach brown rot in South Carolina (8) and the Niagara Peninsula of Ontario, Canada (3), suggest that blighted blossoms are a relatively minor source of inoculum during the preharvest period, although their importance as a bridge (7) between overwintering inoculum sources and the major sources of infection before harvest (i.e., aborted or thinned fruitlets) was not investigated in these studies.

Blossom blight occurs only infrequently on sour cherries in New York State, yet many growers have been hesitant to alter their traditional protective fungicide programs in the absence of supportive epidemiological or biological data. Zehr (19) has recently outlined a disease management program for peaches in South Carolina in which blossom sprays are reduced or eliminated depending primarily on the perceived level of overwintering inoculum sources within or immediately external to the orchard. Climatological factors are deemphasized under this program, due to the generally warm, humid environment favoring epidemic development of brown rot in the southeastern United States. However, the cool climate that often prevails during bloom in the sour cherry growing districts adjacent to the Great Lakes, and evidence that this species is relatively less susceptible to blossom blight than peach (14), collectively suggest that an independent set of parameters might be developed to assess the need for blossom blight sprays on sour cherry under New York conditions. Accordingly, this study was initiated to determine the influence of temperature and wetness duration on disease incidence; the interaction of these environmental variables with inoculum density; and the seasonal production of conidia of *M. fructicola* within selected orchards. A brief portion of this work has been published previously (17).

MATERIALS AND METHODS

Controlled environment studies. Plant material used in all tests consisted of 3–10-yr-old sour cherry trees (Montmorency cultivar) on Mahaleb rootstock grown in 30-cm-diameter clay pots. Trees were maintained outdoors during the growing season, stored indoors at 1 C during the winter, and induced to bloom in the spring by repotting and transfer to a greenhouse. Immediately before inoculation, unopened blossom buds were removed from the trees, leaving about 100–200 fully opened blossoms per tree.

Inoculum was prepared by growing a benomyl-sensitive isolate

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of *M. fructicola* (reisolated from inoculated Montmorency blossoms before the start of each experimental season) on canned peach halves for 1 wk at 22–25 C and rinsing the conidia that developed on the fruit surface into a blender with a stream of distilled water. Spore chains were broken up by operating the blender for 60 sec at alternating high and low speeds, the concentration of conidia was determined with a hemacytometer, and the final suspension was adjusted to the desired concentration with distilled water. Blossoms were then uniformly inoculated with a fine mist of the conidial suspension, delivered by a spray paint apparatus connected to a source of compressed air at a pressure of 1.75 kg/cm^2, until numerous droplets beaded on the petals.

Trees were moved into one of four mist chambers maintained at 8, 12, 16, or 20 C immediately after inoculation. Individual trees were then removed from each chamber after various wetting periods had elapsed, maintained under ambient laboratory conditions for 30–60 min until the blossoms had dried, and transferred to a controlled environment for the duration of the incubation period. Blossoms were examined daily from 3–7 days after inoculation and rated as diseased if a necrotic lesion had expanded into the base of the calyx. Diseased blossoms were recorded and excised after each examination, and a final count of uninfected blossoms was made to calculate disease incidence. In the first set of experiments, all blossoms were inoculated with a suspension containing 5,000 conidia/ml, subjected to a wetting period of 0, 3, 4, 5, 6, 8, 10, 12, or 24 hr at one of the four abovementioned temperatures, and subsequently incubated in a growth chamber maintained at 20 C with a 14-hr photoperiod. Approximately 1 cm of water was maintained on the floor of the chamber to provide a high atmospheric relative humidity (RH) (about 60–70% as measured by a hygrothermograph located 60 cm above the floor). The experiment was repeated six times to provide six replicate data points per treatment, and for each temperature the data were described by the Gompertz model. The Gompertz equation is of the form $Y = A + C*[-\mathrm{EXP}(-B[X - M])$, where Y represents disease incidence; A and C represent the lower and upper asymptotes, respectively; M is a location parameter; X represents the hours of blossom wetting after inoculation; and B represents the rate of increase of disease incidence (13).

In a second experiment, blossoms were inoculated with either 50, 500, or 5,000 conidia/ml, and subjected to wetting periods of either 8, 12, or 24 hr at the same four temperatures. Subsequent incubation was provided in a sealed chamber with continuous fluorescent lighting; a temperature of 20 C; and the RH maintained at 85–90% (as measured with a sling psychrometer) by wetting the floor and walls and minimizing air circulation. This experiment was repeated four times to provide four replicate data points, and the data were subjected to an analysis of variance for a 4 (temperatures) × 3 (inoculum concentrations) × 3 (wetting durations) factorial design.

Conidium germination studies. A suspension of conidia containing 2.4×10^5 spores/ml was prepared as described above, and 0.2 ml of this suspension was pipetted onto individual petri dishes containing 10 ml of Difco PDA (Difco Laboratories, Detroit, MI) stored in incubators at 8, 12, 16, or 20 C for 24 hr before inoculation. The conidial suspension was spread evenly over the agar surface using a sterile bent glass rod, and the dishes were immediately returned to their respective incubators. One dish was removed from each incubator at 3, 4, 5, 6, 8, 10, and 12 hr after inoculation, and a glass cover slip was placed on the agar surface in three locations of each plate, preceded by a drop of aniline blue in lactophenol to fix and stain the conidia. Dishes were then stored in a refrigerator until a determination of conidium germination could be made by examining 100 conidia within each of the three stained locations. Conidia were considered to have germinated according to one of two separate criteria: A germ tube had developed with a length $\geqslant 1\times$ the width of the conidium or a germ tube had developed with a length $\geqslant 3\times$ the width of the conidium. The experiment was repeated six times to provide six replicate data points, then the Gompertz model was used to describe the data for each temperature × germination criterion combination.

Seasonal production of inoculum. Twenty mummified fruits with characteristic symptoms of previous brown rot infection were collected at 7–14-day intervals from bud break through harvest from unsprayed Montmorency trees in each of three successive years. The phenological stage of crop development was noted before each sampling, then the mummies were brought to the laboratory, placed into a 250-ml Erlenmeyer flask with 100 ml of distilled water plus three drops of Triton X-100 surfactant, and shaken vigorously for 30 sec by hand. The concentration of conidia of *M. fructicola* within this solution was then determined with a hemacytometer. In 1985, mummified fruits were sampled from an experimental orchard in Geneva, NY; in 1986, from a commercial orchard in Williamson, NY, about 2 km south of Lake Ontario; and in 1987, from a commercial orchard in Sodus, NY, about 200 m south of Lake Ontario.

RESULTS

Controlled environment studies. When inoculum concentration was held constant at 5,000 conidia/ml, blossom blight did not occur unless trees were exposed to a wetting period in a mist chamber after inoculation; however, the rate of disease development was proportional to both temperature and wetting duration once postinoculation wetting treatments were imposed (Table 1). For instance, blossom blight incidence was 5, 7, 28, and 72% with a 5-hr wetting period at 8, 12, 16, and 20 C, respectively, and increased to 21, 78, 90, and 98% after 10 hr of wetting at the same respective temperatures. Disease incidence reached a level of 85–97% after 24 hr of wetting, regardless of temperature (Fig. 1). In experiments with variable inoculum levels, disease incidence was a function of inoculum concentration in addition to temperature and wetting duration. For instance, when blossoms were subjected to a 12-hr postinoculation wetting period at 8 C, blight incidence increased from 3 to 7 to 39% as the inoculum concentration increased from 50 to 500 to 5,000 conidia/ml, respectively. Given the same 12-hr wetting period and respective inoculum doses at 12 C, blight incidence was 10, 44, and 79%; was 11, 47, and 81% at 16 C; and rose to 26, 59, and 95% at 20 C (Fig. 2). Analysis of variance for these data (Table 2) showed a highly significant ($P < 0.001$) F-value for the main effects of temperature, concentration, and time (primarily accounted for by a linear term), and for the interactions of temperature × concentration and temperature × time ($P < 0.01$). The temperature × concentration interaction appears to result from the contrast between the relative lack of response to wetness duration with high inoculum × high temperature and low inoculum × low temperature combinations (i.e., maximum or minimum disease pressure, respectively), and the marked response with most other temperature × inoculum concentration treatments. Similarly, the temperature × time interaction appears to result from a relatively greater influence of wetting duration at 8 and 12 C than at 16 and 20 C, within the range of wetness periods examined, i.e., 8–24 hr (Fig. 2).

Conidium germination studies. The rate of germination for

TABLE 1. Rate of blossom blight development with respect to wetting duration and germination of conidia of *Monilinia fructicola* at four different temperatures, as determined by fitting the Gompertz model[a] to the data

	Disease incidence		Conidium germination (1×)[d]		Conidium germination (3×)[d]	
Temp (C)	Rate[b]	(SE)[c]	Rate[b]	(SE)[c]	Rate[b]	(SE)[c]
8	0.14	(0.02)	0.77	(0.07)	0.31	(0.03)
12	0.48	(0.07)	0.99	(0.03)	0.42	(0.02)
16	0.68	(0.09)	1.53	(0.06)	0.61	(0.08)
20	1.00	(0.12)	1.69	(0.08)	0.86	(0.03)

[a] The Gompertz model is $Y = A + C* \mathrm{EXP}[-\mathrm{EXP}(-B[X - M]]$, where Y and X represent the appropriate dependent and independent variables, respectively, and B is a rate parameter (see text).

[b] The rate parameter (B) determined from the Gompertz equation.

[c] Standard error of the rate parameter derived from the Gompertz equation.

[d] Germination determined if the germ tube length was at least equal to the width of the conidium (1×) or three times the width (3×).

conidia of *M. fructicola* was influenced by both temperature and the criterion by which conidia were deemed to have germinated. For example, when germination was assessed on the basis of a germ tube with a length $\geq 1\times$ the width of the conidium, the approximate time required to reach a 50% frequency of germination at 8, 12, 16, and 20 C was 5, 4, 3, and 2 hr, respectively; however, these respective periods increased to 11, 9, 7, and 5 hr when development of a germ tube with a length $\geq 3\times$ the conidium width was the requisite criterion for assessing germination. Similarly, the approximate durations necessary to reach a 90% frequency of germination using the $\geq 1\times$ and $\geq 3\times$ conidium width criteria were 8 and $>$12 hr at 8 C; 6 and 12 hr at 12 C; 4.5 and and 10 hr at 16 C; and 3.5 and 7 hr at 20 C, respectively (Fig. 3). The effects of time and temperature on conidium germination closely paralleled the effects of wetting duration and temperature on blossom blight incidence when inoculum dose was held constant at 5,000 conidia/ml; however, this relationship was less pronounced at 8 C than at the higher temperatures (Table 1, Fig. 3).

Seasonal production of inoculum. Relatively few conidia were produced on mummified fruits in orchard trees before and during the blossom period in each of the 3 yr of this study. However, the production of conidia began to increase sharply during the period between petal fall and the early development of red fruit color, reaching and sustaining a peak during the preharvest period (Fig. 4). The average number of conidia detected in all samplings from the white bud through petal fall periods was 18.4, 2.2, and 6.5% that of the peak value detected during the preharvest period in 1985, 1986, and 1987, respectively (Fig. 4).

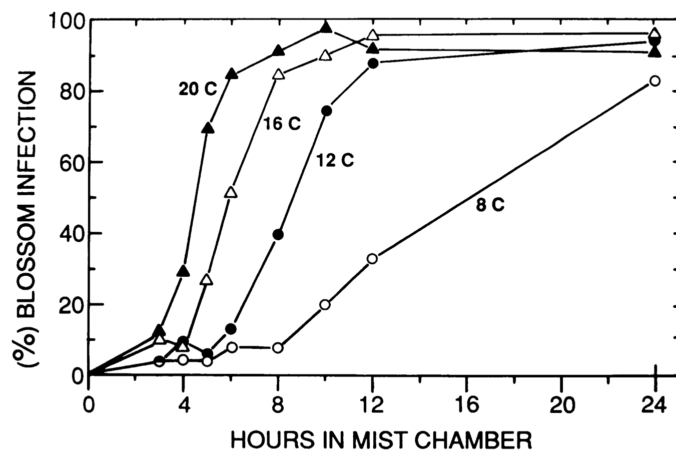

Fig. 1. Blossom blight incidence as a function of temperature and wetness duration. Potted Montmorency sour cherry trees were inoculated in full bloom with a suspension containing 5,000 conidia/ml of *Monilinia fructicola*, immediately placed into mist chambers maintained at the temperatures indicated, and removed periodically after the desired wetting period had elapsed. All trees were subsequently incubated in a common growth chamber maintained at 20 C and 60–70% RH.

TABLE 2. Analysis of variance for disease incidence of Montmorency sour cherry blossoms exposed to various levels of temperature, inoculum concentration, and wetting duration (time)

Source of variation	df	MS	*F*	*P*
Temperature	3	4,094.5	57.1	<0.001
Concentration	2	20,046.9	279.7	<0.001
Time	2	5,309.6	74.1	<0.001
Linear	1	9,807.3	136.9	<0.001
Deviations	1	811.9	11.3	0.001
Temperature × concentration	6	279.6	3.90	0.001
Temperature × time	6	243.3	3.40	0.004
Concentration × time	4	109.2	1.52	0.200
Temperature × concentration × time	12	95.8	1.34	0.208
Residual	108	71.7		

DISCUSSION

A temperature of 20 C during wetting periods was highly favorable and clearly the most conducive for initiating blossom blight. However, Weaver (15) reported a higher disease incidence on peach with blossom wetting periods at 25 C than at 20 C. His

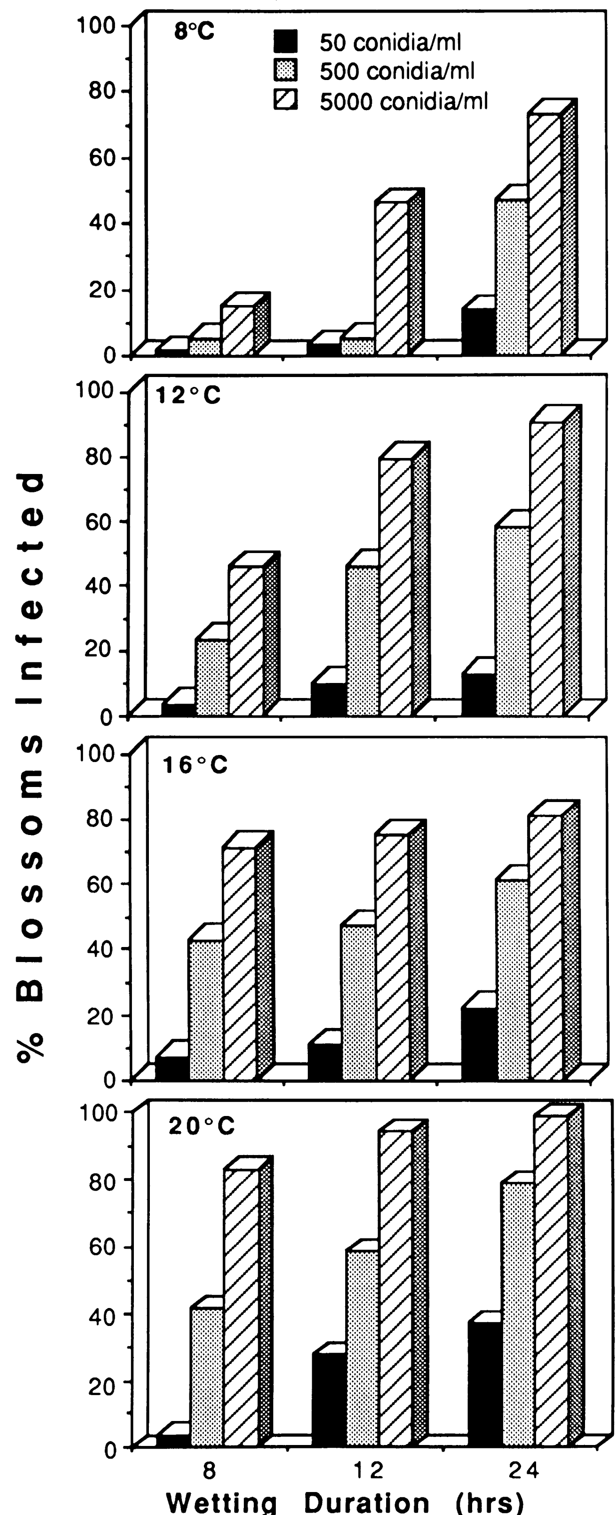

Fig. 2. Blossom blight incidence as a function of inoculum density and wetness duration at four different temperatures. Potted Montmorency sour cherry trees were inoculated in full bloom with one of three inoculum concentrations of *Monilinia fructicola*, immediately placed into mist chambers maintained at the temperatures indicated, and removed after the desired wetting period had elapsed. All trees were subsequently incubated in a common chamber maintained at 20 C and 85–90% RH.

data and others (4,16) indicate 25 C to be near optimum for vegetative growth and conidium germination by *M. fructicola* and suggest that 20 C may still be marginally below the optimum for development of blossom blight on sour cherry. In contrast, the slow rate of disease increase with respect to wetting duration at 8 C, and the anomalously low ratio of this rate to that of conidium germination (0.45 vs. 1.11–1.16 for the remaining three temperatures, using the 3× germination criterion) (Table 1, Fig. 3), suggest that 8 C is a distinctly suboptimal temperature for infection of sour cherry blossoms by *M. fructicola*. Nevertheless, disease incidence was still high at 8 C if the inoculum dose was high and the blossom wetness duration long (Figs. 1 and 2).

The effects of temperature on conidium germination were similar to those reported by previous investigators (15,16). Furthermore, the coincident influence of temperature on conidium germination and disease development following controlled wetting periods (Table 1, Fig. 3) suggests that these wetting periods initiated infection by providing the requisite environment for spore germination and germ tube development. Weaver noted that free moisture was necessary for germination of conidia of *M. fructicola* on the surfaces of most peach flower parts, although he observed that germination could occur on the stigmas at relative humidities as low as 80% (15), possibly the result of stimulation by floral exudates under such conditions (10). However, conidium germination is but the first episode in a series of events culminating in blossom blight, and environmental factors are also likely to influence processes involved in the subsequent penetration and colonization of floral parts by *M. fructicola*. For instance, when peach blossoms were inoculated with conidia of *M. fructicola* and exposed to a saturated atmosphere to promote conidium germination, a much greater percentage became diseased if subsequently incubated at 15–20 C with an RH of 80 or 90% than if incubated in a greenhouse with significant fluctuations in temperature and relative humidity (15). Similarly, erratic results obtained in preliminary experiments of the present study when RH was poorly controlled following postinoculation wetting episodes (data not shown) provides additional anecdotal evidence for the influence of environment subsequent to spore gemination on ultimate disease incidence. Until more precise data on environmental influences during the incubation phase of blossom blight are available, the present data on the influence of temperature and blossom wetness duration should serve merely as an indication of relative disease potential under field conditions.

Inoculum density has heretofore been an intuitively understood yet quantitatively unexplored factor in epidemiological considerations of brown rot blossom blight. Although the data presented in Figure 2 are limited, they clearly show a pronounced effect of inoculum dose at nearly all temperature × wetness duration combinations tested, as does the highly significant *F* value for the main effect of inoculum concentration in the analysis of variance (Table 2). These data reinforce the concept that disease management programs based on environmental variables, and experimental systems designed to develop them, should account for variations in inoculum density likely to occur among different years and different orchards.

The relative importance of different potential sources of inoculum for blossom blight of sour cherry has never been documented systematically. Although no attempt was made to monitor inoculum from sources external to commercial orchards during the course of this study, apothecia and overwintering brown

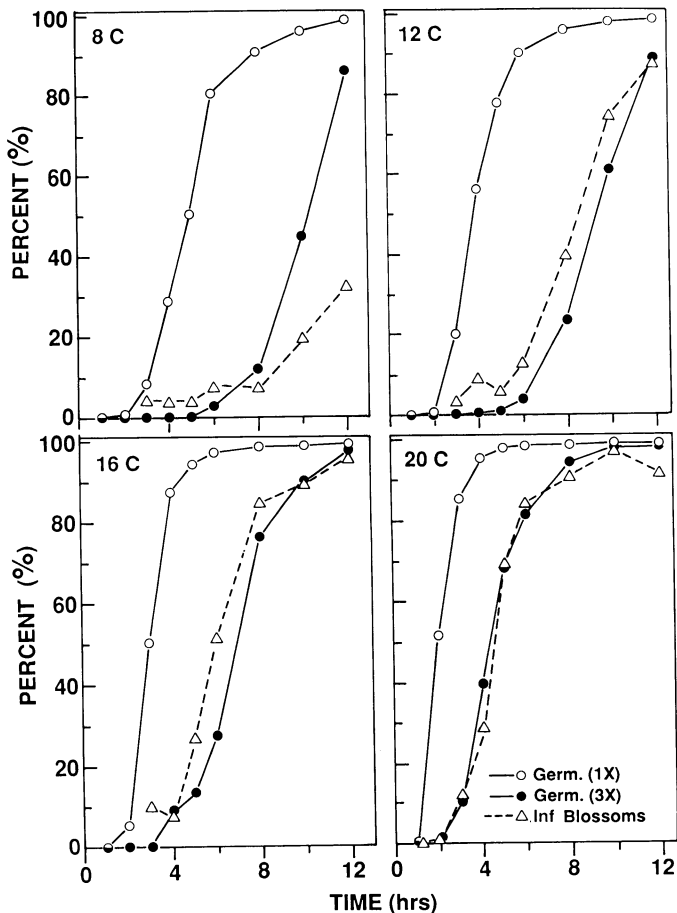

Fig. 3. Germination rate of conidia of *Monilinia fructicola* on PDA at four different temperatures. Data are presented according to two separate criteria for assessing germination: 1) presence of a germ tube ⩾ 1 × the conidium width (-o-); 2) presence of a germ tube ⩾ 3 × the conidium width (-•-). Blossom blight incidence (-Δ-) from experiments summarized in Figure 1 is included for comparative purposes.

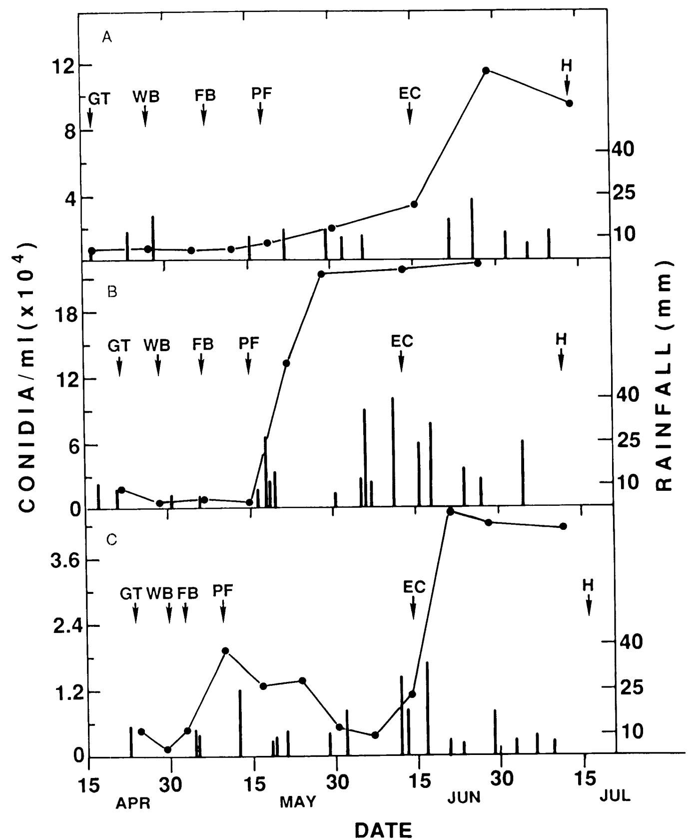

Fig. 4A–C. Seasonal production of conidia of *Monilia fructicola* from mummified Montmorency sour cherry fruits in orchard trees. Data points represent the concentration of conidia obtained from 20 mummified fruits washed in 100 ml of distilled water plus surfactant on various collection dates. Phenological stages of crop development as indicated by arrows are green tip (GT); white bud (WB); full bloom (FB); petal fall (PF); early red coloration of new fruits (EC); and harvest (H). **A,** 1987 data from orchard in Sodus, NY; **B,** 1986 data from orchard in Williamson, NY; **C,** 1985 data from orchard in Geneva, NY.

rot twig cankers were searched for within a number of orchards and never found (W. F. Wilcox, *unpublished*). However, orchard blocks or individual trees that remained incompletely harvested were very common, and infected mummies within such trees were often plentiful the following spring. Sporulation from overwintering mummies was relatively light before and during the blossom period in all 3 yr of the study, suggesting that pressure for blossom blight from this inoculum source was light. The reasons for poor sporulation during this period are uncertain, but may be related to a general lack of rainfall before bloom in the years studied (Fig. 4). Sporulation by *M. fructicola* from infected tissues is influenced dramatically by the water content therein (5), and the mummies examined may not have been hydrated sufficiently for significant sporulation until rainfall and atmospheric relative humidity increased later in the season, although this possibility was not explored. However, in contrast to limited sporulation during the early season, the intense sporulation consistently recorded during the preharvest period suggests that mummies may often constitute a significant source of inoculum for brown rot of ripening sour cherry fruits under New York conditions. These data further contrast with the general lack of recognition of mummies as a source of inoculum for preharvest infections of stone fruits (1,6,18), and with the virtual lack of sporulation reported from peach mummies during the preharvest period in South Carolina (8). Further investigation of this phenomenon among various stone fruit species would appear warranted.

With additional research into techniques for monitoring the qualitative and quantitative availability of inoculum, and a refined understanding of the role of environment during disease incubation, it may be possible to more accurately determine the need for fungicidal control of brown rot blossom blight on sour cherry, a disease of sporadic occurrence in New York and other regions (9).

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