Incidence of Latent Infection of Immature Peach Fruit by *Monilinia fructicola* and Relationship to Brown Rot in Georgia

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ABSTRACT

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Peach fruit are most susceptible to infection by *Monilinia fructicola* during the preharvest ripening stage. Although various sources of inoculum for preharvest infection have been characterized, the role of latent infection of immature fruit in the carryover of *M. fructicola* from the spring (blossom blight phase) to the preharvest period (fruit rot phase) is unknown for the southeastern United States. From 1997 to 1999, immature peach fruit were collected at 14-day intervals from orchards in middle and northern Georgia. Fruit were surface disinfested and treated with paraquat (1997) or frozen overnight (1998 and 1999) to induce tissue senescence and activate latent infections. Across sites and years, the incidence of latent infection remained low until the final sampling date 7 to 12 days before harvest. The incidence of latent infection on the final sampling date ranged from 0 to 22.0% and correlated significantly with both the incidence of blossom blight earlier in the season (r = 0.9077, P = 0.0332) and the incidence of fruit rot at harvest (r = 0.9966, P = 0.0034). There also was a significant association between the incidence of latent infection at the onset of pit hardening (between 7 and 10 weeks before harvest) and subsequent fruit rot incidence (r = 0.9763, P = 0.0237). Weather variables (cumulative rainfall or rainfall frequency) alone did not correlate with fruit rot incidence (P > 0.05), whereas combined latent infection–rainfall variables did. The results suggest that latent infections can serve as a source of inoculum for subsequent fruit rot in peach orchards in Georgia. Despite its significant association with fruit rot incidence, the potential for using latent infection incidence as a biological indicator of disease risk at harvest may be limited; the assessment of latent infection during the fruit ripening stage (similar to the timing of the final sampling date in this study) would not provide sufficient lead time for preharvest disease management decisions, whereas an earlier assessment (e.g., at the onset of pit hardening) would require large sample sizes due to the low incidence of latent infection present during that period.

Additional keywords: epidemiology, *Prunus persica*

The brown rot fungus *Monilinia fructicola* (G. Wint.) Honey is economically the most important pathogen of peach (*Prunus persica* (L.) Batsch) in Georgia (30). Direct yield losses result from infection of flowers (blossom and twig blight) and from fruit rot at harvest and postharvest; indirect losses are due to the cost of fungicide application during bloom and during the pre- and postharvest periods. In most years, direct losses from *M. fructicola*-incited blossom blight are minor (23,32,33); however, cankers, formed on twigs as a result of blossom blight, may serve as a source of inoculum for infections that occur during the preharvest fruit ripening stage, particularly in early maturing cultivars (23,32,33). Other within-orchard inoculum sources for preharvest fruit infections include conidia produced on thinned fruit on the ground (1,9,17) and on aborted, nonabscised fruit in the tree (1,17). By contrast, conidia produced on overwintered fruit mummies in early spring do not survive long enough to cause fruit rot (24).

Immature stone fruits generally do not exhibit symptoms or signs of infection by *M. fructicola* unless ingress and colonization are favored by prolonged rain or high humidity following injury. Even without wounding, however, immature fruit may harbor symptomless (latent) infections. Latent infections may become active as the fruit ripen, thus becoming a possible means of carryover of *M. fructicola* from the spring to the preharvest period. Latent infections by *M. fructicola* or the closely related *M. laxa* have been documented in apricot (25,28,29), peach (15,20,25), plum (21,25), prune (20), and cherry (8,31). Michailides et al. (19,20) reported positive relationships between the incidence of latent infection in immature French prune, nectarine, and plum and fruit rot severity at harvest and postharvest in California. Similarly, Northover and Cerkauskas (21) determined that latent infections in European plum in Ontario, Canada, occurred throughout the growing season and correlated positively with fruit rot incidence at harvest. These authors concluded that latent infections were most important in humid temperate regions, where they may readily progress to fruit rot.

In peach, latent infections can occur during all stages of fruit development but their role in the epidemiology of brown rot is uncertain. Kable (13,15) concluded that only latent infections near harvest are important in the development of fruit rot in the semiarid climate of southeastern Australia. Landgraf and Zehr (17) surveyed potential inoculum sources for fruit infection by *M. fructicola* in South Carolina peach orchards but did not include latent infection. They did acknowledge, however, the need to evaluate the role of latent infection in the humid southeastern United States. A better understanding of the importance of latent infection in the epidemiology of brown rot could facilitate the early detection of increased fruit rot risk before harvest, thereby increasing lead time for disease management decisions (8,18–20).

The objectives of this exploratory study were to monitor the seasonal dynamics of latent fruit infection by *M. fructicola* in peach orchards in Georgia and to determine the relationship between latent infection at select stages of fruit development and fruit rot at harvest and postharvest. The incidence of blossom blight and surface contamination of fruit by *M. fructicola* also were measured to obtain a comprehensive assessment of inoculum dynamics during the entire period of fruit development.

MATERIALS AND METHODS

Orchard sites. From 1997 to 1999, mature trees in experimental and commercial peach orchards were assessed for several *M. fructicola*-related disease variables as described below. Cultivars included Blake at the University of Georgia Horticulture Farm in Oconee County in northern

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Georgia (1997 to 1999), Sunprince in a commercial orchard in Crawford County in middle Georgia (1998), and an unknown cultivar in an abandoned commercial orchard in Peach County in middle Georgia (1999). Except for the Peach County orchard, which had not been managed for 2 years, general orchard management followed standard commercial practices (10). Half the trees sampled in the Oconee and Crawford County orchards did not receive any fungicide applications, whereas sprays in the other half were limited to use of wettable sulfur (9.1 to 12.1 kg a.i./ha) for control of peach scab (*Cladosporium carpophilum*) applied at 10- to 14-day intervals starting at shuck split until approximately 4 weeks before harvest. Data from the two groups of trees were combined.

Assessment of blossom blight. In the Oconee County orchard in 1997, all twigs with cankers or gumming at the base of flowers, presumed to be a result of blossom blight, were collected after petal fall from 12 trees. Twigs were surface disinfested for 1 min each in 70% EtOH and 0.5% NaOCl (10% household bleach), and symptomatic tissue was placed on Monilinia-selective medium (MSM; 22) to determine the incidence of infection by *M. fructicola*. After 1 week at room temperature (23 to 25°C), dishes were examined for *M. fructicola* based on characteristic colony morphology.

In 1998 and 1999, blossom blight incidence was assessed visually in each orchard at full bloom. Twenty blossoms were examined on each of 10 trees to determine the presence of symptoms or signs of infection by *M. fructicola*.

Sampling of immature fruit and assessment of surface contamination and latent infection. Immature fruit were collected at 14-day intervals, beginning at shuck fall. In each orchard on each sampling date, 120 to 170 nonwounded fruit were selected arbitrarily from the same group of trees used previously for assessment of blossom blight. Fruit were washed, in batches of 10 fruit, in sterile distilled water containing 0.01% Tween 80 for 15 (1997) or 30 (1998 and 1999) min on an orbital shaker. To evaluate the incidence of fruit surface contamination by conidia of *M. fructicola*, aliquots of the wash water were spread onto MSM (five dishes per group of 10 fruit). After 1 week at room temperature, dishes were examined under a dissecting microscope to determine the presence of *M. fructicola* colonies.

Immediately following the washing step described above, fruit were surface disinfested for 10 s in 70% EtOH, followed by 4 min in a fresh solution of 0.5% NaOCl. In 1997, two thirds of the fruit were submerged in a filter-sterilized solution of paraquat at 6,000 µg/ml (Gramoxone Herbicide; Zeneca Ag Products, Wilmington, DE) for 1 min to induce tissue senescence and activate latent infections (21). The remaining fruit (untreated check) were placed in sterile distilled water for 1 min. In 1998 and 1999, after surface disinfestation, two thirds of the fruit were frozen overnight at –12°C to induce tissue senescence (18,20). The remaining fruit were not frozen and served as a check. After treatment, all fruit were incubated individually in autoclaved tissue culture vessels at room temperature in the dark. After 1 week, fruit were assessed for signs of *M. fructicola* and fruit diameters were measured with a digital caliper.

Assessment of fruit rot. When fruit reached a maturity level between commercially ripe and tree ripe (ground color between 4 and 5 on the Clemson color chart; 6), fruit rot incidence was determined in each orchard except the Oconee County orchard in 1997. At least 12 arbitrarily selected fruit per tree were assessed for symptoms and signs of infection by *M. fructicola*; the assessment was made in the same group of trees used previously for assessment of blossom blight and sampling of immature fruit. At the same time, 12 nonwounded fruit per tree without symptoms or signs of infection by *M. fructicola* were collected for postharvest disease assessment from the same group of trees. These fruit were misted with tap water, placed on plywood racks surface disinfested with 0.5% NaOCl, covered with plastic film, and incubated at room temperature in the dark. After 5 days, the incidence of postharvest fruit rot was assessed based on the presence of symptoms or signs of infection by *M. fructicola*.

Data analysis. Based on the incidence of latent infection, the area under the latent infection curve (AULIC), in units of percent-days, was calculated by trapezoidal integration (3). Using combined data from all sites and years, correlation coefficients were computed to explore linear relationships among disease variables (incidence of blossom blight, AULIC, latent infection on select sampling dates, and fruit rot at harvest and postharvest). Correlations between disease variables and rainfall (as measured by Georgia Automated Environmental Monitoring Network weather

Table 1. Disease variables associated with infection by *Monilinia fructicola* in Georgia peach orchards

Site and year	Blossom blight incidence (%)	AULIC (percent-days)[a]	Final incidence of latent infection (%)[b]	Fruit rot incidence at harvest (%)	Postharvest fruit rot incidence (%)[c]
Oconee County 1997	0	0	0	…[d]	37.0
Oconee County 1998	0.1	10.4	0.3	7.0	18.0
Oconee County 1999	0.5	84.6	9.0	32.0	90.0
Crawford County 1998	1.0	8.4	0.4	3.3	9.0
Peach County 1999	4.0	234.6	22.0	64.0	100

[a] Area under the latent infection curve (from shuck fall until 7 to 12 days before harvest).
[b] Determined on fruit sampled 7 to 12 days before harvest.
[c] Determined after storage for 5 days at room temperature in a high-humidity environment.
[d] Not determined.

Table 2. Correlation matrix of disease variables associated with infection by *Monilinia fructicola* in Georgia peach orchards[a]

	Blossom blight incidence	AULIC[b]	Final incidence of latent infection[c]	Fruit rot incidence at harvest	Postharvest fruit rot incidence[d]
Blossom blight incidence	1.0000	0.9280 (0.0229)	0.9077 (0.0332)	0.8541 (0.1459)	0.6190 (0.2656)
AULIC	…	1.0000	0.9978 (0.0001)	0.9907 (0.0093)	0.8535 (0.0658)
Final incidence of latent infection	…	…	1.0000	0.9966 (0.0034)	0.8840 (0.0466)
Fruit rot incidence at harvest	…	…	…	1.0000	0.9213 (0.0787)
Postharvest fruit rot incidence	…	…	…	…	1.0000

[a] Total of five trials carried out between 1997 and 1999; data on fruit rot incidence at harvest were missing for one of the trials. Values are correlation coefficients and corresponding *P* values (in parentheses).
[b] Area under the latent infection curve (from shuck fall until 7 to 12 days before harvest).
[c] Determined on fruit sampled 7 to 12 days before harvest.
[d] Determined after storage for 5 days at room temperature in a high-humidity environment.

stations near the orchards) also were examined. Rainfall was summarized as cumulative precipitation and the number of rainy days from 15 March to harvest; this interval encompassed the entire fruit development period. Rainfall also was summarized for the 15- and 30-day periods before harvest, assuming that rain during these periods may have been associated most closely with fruit rot at harvest. All correlation analyses were done with the PROC CORR procedure of the Statistical Analysis System (SAS Institute, Cary, NC). Correlation coefficients were considered statistically significant at $\alpha = 0.05$.

The sample size, n, required to reliably estimate the incidence of latent infection, X, was calculated with the equation $n = [s_x/(m_x \times CV)]^2$, where m_x and s_x are, respectively, the mean and standard deviation of X (16) determined from multiple 80-fruit samples; CV, the maximum allowable coefficient of variation around X, was set at 0.25.

RESULTS AND DISCUSSION

Blossom blight occurred only sporadically, consistent with previous reports on this phase of the disease cycle of *M. fructicola* in the eastern United States (1,5,17,24,32,33). The incidence of blossom blight ranged from 0% in Oconee County in 1997 to 4.0% in Peach County in 1999 (Table 1). The lack of disease management for 2 years in the abandoned commercial orchard in Peach County may explain the greater disease incidence there.

Fruit rot incidence at harvest was variable across sites and years, ranging from 3.3% in Crawford County in 1998 to 64.0% in Peach County in 1999 (Table 1). Disease incidence was similarly variable in postharvest assessments, varying from 9.0% in Crawford County in 1998 to 100% in Peach County in 1999 (Table 1). Postharvest disease incidence tended to correlate positively with disease incidence at harvest ($r = 0.9213$, $P = 0.0787$; Table 2).

No surface contamination of immature fruit by *M. fructicola* was detected in the 1997 trial. The incidence of fruit surface contamination was sporadic in most other trials (Fig. 1A), indicating that inoculum often may have been limiting. Conidial contamination was, however, detected frequently on fruit collected in the unmanaged Peach County orchard (Fig. 1B). In this orchard, sporulation of *M. fructicola* on infected fruit was observed approximately 5 weeks before harvest. Fruit with early symptoms of brown rot generally also showed symptoms of insect injury, primarily oviposition scars caused by the plum curculio (*Conotrachelus nenuphar*), which may have provided entry points for infection and a nutrient source for sporulation by the pathogen. The association between insect wounding and fruit rot is well documented (13,24,27). In most commercial orchards, however, insect injury of fruit is rare due to rigorous insecticide programs (10).

The incidence of latent infection of immature fruit was low across sampling dates and sites (Fig. 1A). In the 1997 trial, no latent infections were detected. At all other sites, the incidence of latent infection was low until the final sampling date, 7 to 12 days before harvest (Fig. 1B). On the final sampling date, the incidence of latent infection ranged from 0% in Oconee County in 1997 to 22.0% in Peach County in 1999 (Table 1). These values are lower than those reported by Michailides et al. (20)

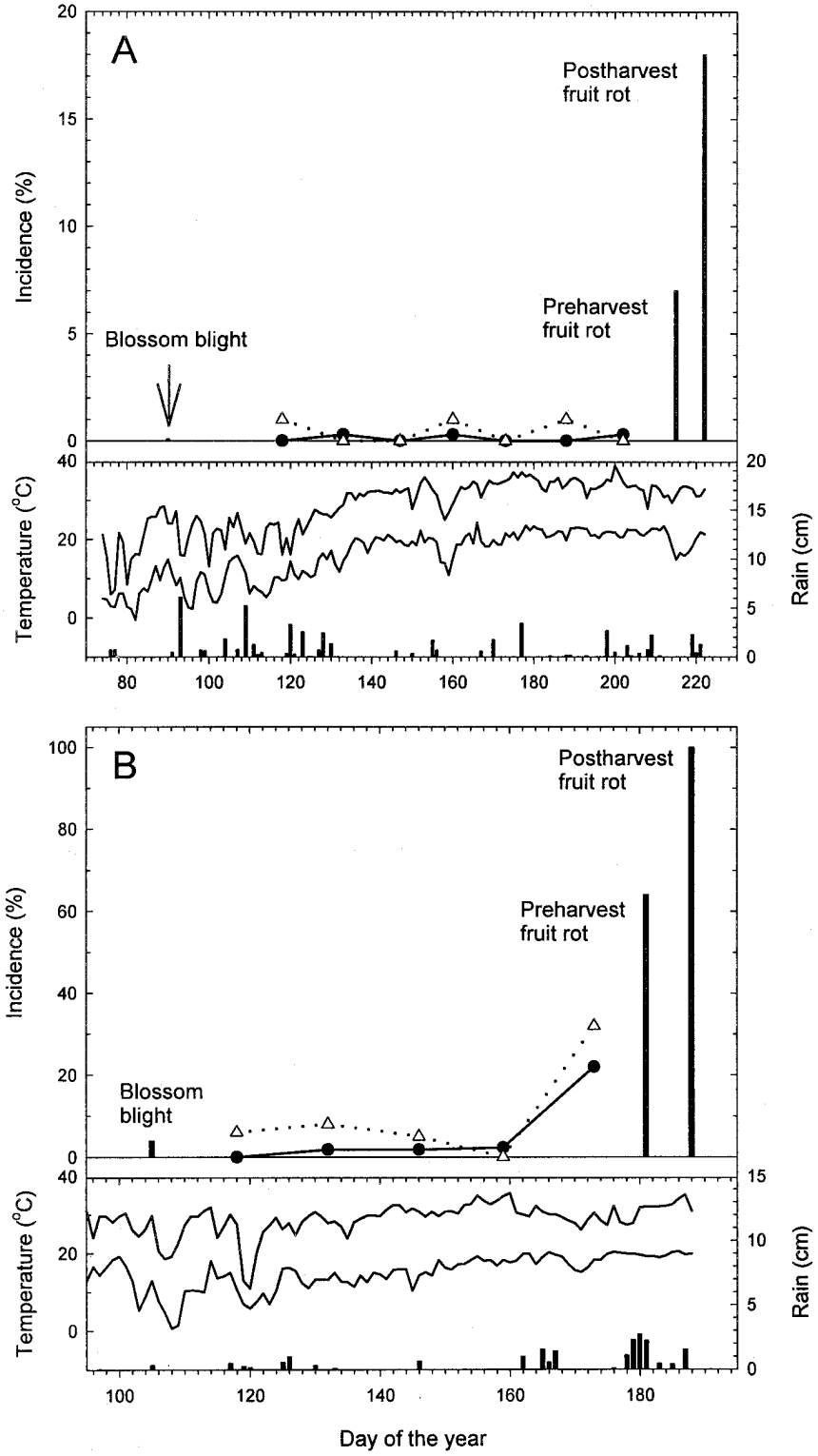

Fig. 1. Weather and disease variables associated with infection by *Monilinia fructicola* in two Georgia peach orchards, **(A)** Oconee County, 1998, and **(B)** Peach County, 1999. Upper panels show the incidence of blossom blight, surface contamination of fruit (Δ), latent infection of fruit (●), and fruit rot at harvest and postharvest. Lower panels show rainfall (vertical bars) and daily maximum and minimum temperatures. Two of five trials carried out between 1997 and 1999 are presented.

for peach and nectarine in California sampled at the onset of fruit ripening. Although these authors observed that the incidence of latent infection during fruit ripening was generally greater than the incidence of fruit rot at harvest, the opposite was true in the present study (Table 1), suggesting that the proportion of fruit rot symptoms that can be attributed to latent infections is lower in Georgia than in California. This could be due to the greater potential for secondary spread of the disease from conidia produced on symptomatic fruit in the humid weather prevailing in the peach production regions in Georgia during the preharvest period. Secondary spread, which is less likely in the semiarid production environment of California, would tend to reduce the direct contribution of latent infections to fruit rot incidence at harvest.

The trend toward an increase in the incidence of latent infection before harvest in the present study could have been due to an increase in inoculum availability (1,9,12,17) or the increased susceptibility of the maturing fruit. Indeed, Biggs and Northover (2) reported that immature peach fruit were least susceptible during pit hardening and most susceptible during the periods preceding and following pit hardening; the latter period coincides with the phase of final fruit swell. A similar trend was observed by Ibbotson-Darhower et al. (11) in peach and nectarine.

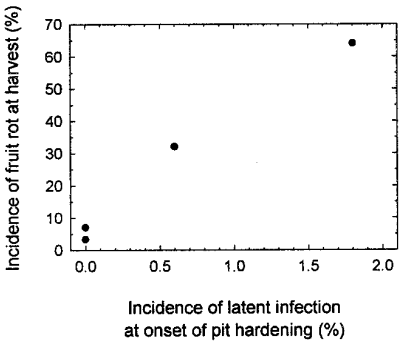

Fig. 2. Relationship between the incidence of latent infection of immature peach fruit by *Monilinia fructicola* determined at the onset of pit hardening and subsequent fruit rot incidence at harvest in Georgia peach orchards. Data are from four orchards surveyed between 1998 and 1999.

AULIC and the incidence of latent infection on the final sampling date correlated significantly with the incidence of blossom blight ($r = 0.9280$, $P = 0.0229$ and $r = 0.9077$, $P = 0.0332$, respectively; Table 2). There was, however, no significant relationship between fruit rot incidence at harvest and blossom blight incidence (Table 2). This differs from the observations of Dunegan and Goldsworthy (7), who recorded a lower incidence of fruit rot in orchards where blossom blight was controlled. Zehr (32) concluded that, although blossom blight may be an important source of inoculum for preharvest fruit infection in early and midseason cultivars, this may not be the case for late-maturing cultivars. Indeed, the presence of a strong inoculum source after flowering, such as conidia produced on thinned fruit on the ground (1,9,17) or on aborted fruit in the tree (1,17), would obscure a simple relationship between blossom blight and fruit rot at harvest.

AULIC and the incidence of latent infection on the final sampling date correlated significantly with fruit rot incidence at harvest ($r = 0.9907$, $P = 0.0093$ and $r = 0.9966$, $P = 0.0034$, respectively; Table 2). The increased incidence of latent infection on the final sampling date contributed considerably to the total variance of AULIC ($r = 0.9978$, $P = 0.0001$) and could explain the similarity in the relationships of these two variables to disease at harvest. Although the detection of latent infections during the fruit ripening period (similar to the timing of the final sampling date in the present study) is important for understanding fruit rot epidemics, it may not provide sufficient lead time for preharvest disease management decisions unless combined with a more rapid detection assay. Indeed, in four of five epidemics monitored in this study, low levels of fruit rot were detected in the orchard before the final assessment of latent infection.

To be operationally viable, a biological indicator of future fruit rot risk must allow adequate lead time before harvest and must be based on a sound biological mechanism. Immature peach fruit are most susceptible to infection by *M. fructicola* during the periods before and after pit hardening (2), which corresponds to phases of rapid expansion in fruit size (4). Thus, based on fruit diameters measured during the assessment of latent infection, the sampling dates most closely corresponding to the onset of pit hardening were identified and the relationship between latent infection on that date and fruit rot at harvest evaluated. The two variables correlated significantly ($r = 0.9902$, $P = 0.0098$; Fig. 2), suggesting that the incidence of latent infection at the onset of pit hardening might be useful as an early biological indicator of subsequent fruit rot risk with a lead time of 7 to 10 weeks before harvest. Operational use of this relationship would, however, require further optimization of detection protocols (e.g., adaptation of the polymerase chain reaction-random amplified polymorphic DNA-based assay for *M. fructicola* described recently for detection of latent infections in sweet cherry; 8). Sample size requirements for accurately estimating the incidence of latent infection would be challenging, given the low levels of latent infection throughout the season and the associated high variability. For example, based on typical means and standard deviations of latent infection incidence observed in this study, a minimum of eight 80-fruit samples would be needed if *CV* is to remain below 0.25 (data not shown).

Although an association between rainfall and infection by *M. fructicola* has been noted previously (13,15,24), cumulative rainfall or rainfall frequency did not correlate with fruit rot in the present study, regardless of the period over which rainfall data were summarized (Table 3). Similar results were reported by Tamm and Flückiger (26), who studied blossom blight caused by *M. laxa* in sweet cherry. They concluded that simple weather variables alone were inadequate to predict the disease. Favorable weather and the presence of inoculum are essential for development of diseases caused by *Monilinia* spp. The combination of a rainfall variable (e.g., cumulative rainfall) with a variable reflecting inoculum dynamics before harvest

Table 3. Correlation coefficients between fruit rot incidence at harvest and postharvest, caused by *Monilinia fructicola*, and rainfall and inoculum variables in Georgia peach orchards[a]

Variables	Fruit rot incidence at harvest	Postharvest fruit rot incidence[b]
Cumulative rainfall 15 days preharvest	0.7027 (0.2973)	0.4159 (0.4861)
Cumulative rainfall 30 days preharvest	0.4110 (0.5890)	0.6377 (0.2470)
Season-long cumulative rainfall[c]	–0.3635 (0.6365)	–0.1791 (0.7732)
Blossom blight incidence × season-long cumulative rainfall	0.8922 (0.1078)	0.6753 (0.2109)
AULIC × season-long cumulative rainfall[d]	0.9843 (0.0157)	0.9409 (0.0171)
Incidence of latent infection at the onset of pit hardening × cumulative rainfall 15 days preharvest	0.9973 (0.0027)	0.8883 (0.0441)

[a] Total of five trials carried out between 1997 and 1999; data on fruit rot incidence at harvest were missing for one of the trials. Values are correlation coefficients and corresponding *P* values (in parentheses).
[b] Determined after storage for 5 days at room temperature in a high-humidity environment.
[c] Calculated from 15 March until harvest.
[d] AULIC = area under the latent infection curve (from shuck fall until 7 to 12 days before harvest).

(e.g., AULIC) can provide a derived variable that includes both provisions for disease development. This approach is suggested by observations such as those of Kable (12–14), who detected the greatest number of *M. fructicola* conidia following rain. Fruit rot incidence at harvest and postharvest was thus evaluated relative to two combined inoculum–rainfall variables (Table 3). The product of AULIC and cumulative rainfall from 15 March to harvest correlated significantly with fruit rot incidence at harvest and postharvest (r = 0.9843, P = 0.0157 and r = 0.9409, P = 0.0171, respectively), as did the product of latent infection at the onset of pit hardening and cumulative rainfall for the final 15 days before harvest (r = 0.9973, P = 0.0027 and r = 0.8883, P = 0.0441, respectively). Although the magnitude and significance levels of the correlations were not improved with combined inoculum–rainfall variables compared with inoculum variables alone (Table 2), inclusion of rainfall may render the relationships more robust over years and sites.

In conclusion, significant correlations between the incidence of latent infection of immature fruit and previous blossom blight incidence, and between fruit rot incidence at harvest and the incidence of latent infection earlier in the growing season, provide support for a role of latent infection in the carryover of *M. fructicola* from the spring to the preharvest period in the southeastern United States. Despite its significant association with fruit rot incidence, the potential for using latent infection incidence as a biological indicator of disease risk at harvest may be limited; the assessment of latent infection during the fruit ripening stage (similar to the timing of the final sampling date in this study) would not provide sufficient lead time for preharvest disease management decisions, whereas an earlier assessment (e.g., at the onset of pit hardening) would require large sample sizes due to the low incidence (<2% in all orchards) of latent infection present during that period and the associated variability.

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