Identification and Etiology of Visible Quiescent Infections of *Monilinia fructicola* and *Botrytis cinerea* in Sweet Cherry Fruit

J. E. Adaskaveg, H. Förster, and D. F. Thompson, Department of Plant Pathology, University of California, Riverside 92521

ABSTRACT

Adaskaveg, J. E., Förster, H., and Thompson, D. F. 2000. Identification and etiology of visible quiescent infections of *Monilinia fructicola* and *Botrytis cinerea* in sweet cherry fruit. Plant Dis. 84:328-333.

Visible quiescent infections were detected as small (<1 mm) necrotic flecks on green cv. Bing cherry fruit and as reddish halos surrounding tannish spots (1 to 2 mm) on immature, yellow-pink cv. Rainier cherry fruit in commercial orchards in California. *Monilinia fructicola* or *Botrytis cinerea*, the fungal pathogens causing brown rot and gray mold of cherry fruit, respectively, were isolated from most of the viable infections. *M. fructicola* was isolated more frequently from quiescent infections than *B. cinerea* in two years of the study, whereas similar isolation frequencies for both fungi were obtained in the other two years of sampling from one commercial Rainier cherry orchard. Using immature-pink Bing fruit that were inoculated in the laboratory, significantly more visible quiescent infections than active decay were reproduced in 6-, 9-, or 12-h wetness-period treatments after inoculation as compared to 18- or 24-h wetness periods where more active decay developed. Non-visible quiescent infections of *M. fructicola* or *B. cinerea* of immature Bing and Rainier fruit collected 2 weeks before harvest were identified on surface-sterilized, paraquat-treated fruit. In both years of the study, significantly more brown rot and gray mold occurred on the surface-sterilized, paraquat-treated fruit than on the non-treated or surface-sterilized fruit, indicating the presence of non-visible quiescent infections by these fungi in cherry fruit. Thus, for the first time, we demonstrated the presence of visible quiescent infections caused by *M. fructicola* and *B. cinerea* and we confirmed the occurrence of non-visible quiescent infections in sweet cherry fruit in California.

Additional keywords: epidemiology, latent or dormant infections

Monilinia fructicola (G. Wint.) Honey (brown rot) and *Botrytis cinerea* Pers.:Fr. (gray mold) are the primary causes of pre- and postharvest fruit decay of sweet cherry, *Prunus avium* (L.) L. in California and other stone-fruit-growing regions of the world (15). Brown rot of fruit may occur as green fruit rot with active decay of immature, green fruit or as active decay of mature, ripe fruit in the field and in storage. The common occurrence of necrotic flecks on green fruit or small reddish lesions on yellow-red fruit of sweet cherry cultivars (e.g., Rainier, Royal Ann, or Corum) have suggested the presence of quiescent or dormant infections by decay fungi on cherry and other stone fruit. Furthermore, the often rapid increase in fruit-decay incidence, regardless of contact-fungicide applications used as protectant treatments, have also suggested established internal brown-rot infections in stone-fruit crops. Postharvest losses also can be se-

vere, even when environmental conditions at harvest time are unfavorable for the infection of ripe fruit. Although some authors have described latent, quiescent, and dormant infections as synonymous (19,21), others have separated the terms into non-visible (latent) and visible (quiescent) infections (7,16). In this study, we define quiescent infections, similar to Jarvis (10), as visible and non-visible infections that are established when environmental conditions or host physiology are conducive for penetration, but not for active growth of the pathogen. Thus, the parasitic relationship is quiescent or dormant until a later time. If still viable, under conducive conditions (e.g., with fruit ripening or with favorable temperatures) these infections may start actively growing and may cause rapid fruit decay. Quiescent infections have been described or were suggested from other stone-fruit crops, such as apricot and peach (11,21), plum (13,16), and cherry (12). On cherry (5,22), plum (13), and apple (1), as well as on other crops (3,8), non-visible quiescent fungal infections have been demonstrated in surface-sterilized, paraquat-dipped green fruit. The method involves treating collected fruit with paraquat, a herbicide that does not affect fungal infections, thus allowing these fungi to grow saprophytically

on senescent plant tissue. In these studies involving the paraquat method, the detection of non-visible quiescent infections was emphasized. The occurrence of visible quiescent infections on cherry fruit has not been studied.

The goals of our study were to determine the etiology of small necrotic flecks on green fruit and small reddish lesions on yellow-red fruit as possible quiescent fungal infections in sweet cherry and to reproduce symptoms of visible quiescent brown-rot infections in the laboratory. Another focus of our research was to substantiate the presence of non-visible quiescent infections in cherry fruit in California. The results of our research will help to further elucidate the disease cycle of brown rot and other decay fungi of sweet cherry fruit in California.

MATERIALS AND METHODS

Isolations from symptomatic epicarp tissue of cherry fruit. A total of 25 immature, yellow-pink cv. Rainier fruit showing reddish circular halos with tan centers (approximately 1 to 2 mm in diameter) were collected 2 to 3 weeks before harvest from each of four to six single-tree replications. Samples were collected in each of four years (1993 to 1996) in one commercial orchard (Bellota, CA) where no fungicides were applied for disease management. Similarly, 20 fruit samples of green cv. Bing fruit showing small necrotic flecks (<1 mm) were collected 3 to 4 weeks after full bloom in two commercial orchards (Lodi, CA) in one year (1993). Fungal isolations were made from epicarp tissue of Rainier fruit showing reddish symptoms or Bing fruit showing small necrotic flecks. For this, approximately 25 mm² of epicarp tissue was surface sterilized with hypochlorous acid (400 mg HOCl/liter) for 1 min. The tissue was then rinsed in autoclaved distilled water, placed onto potato dextrose agar (PDA), and incubated in the dark at 25°C. Fungal isolates were identified based on cultural and microscopic morphology. Data for percent isolation of fungal decays (brown rot or gray mold) were transformed using arcsine-square root percentage transformation and analyzed using general linear model procedures (PROC GLM) of SAS (version 6.12, SAS Institute, Cary, NC). Data obtained for each of the four years for Rainier fruit and one year for Bing fruit were analyzed separately. Means of incidence of *M.*

Corresponding author: J. E. Adaskaveg
E-mail: jim.adaskaveg@ucr.edu

Accepted for publication 29 November 1999.

Publication no. D-2000-0120-01R
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fruticola and *B. cinerea* isolated from fruit were compared using least significant difference procedures of SAS (version 6.12).

Inoculation of cv. Bing cherry fruit in the laboratory. Detached, immature cv. Bing cherry fruit with no visible symptoms of quiescent infections were collected 2 to 3 weeks before harvest in an experimental orchard located on the campus of the University of California, Davis. Maturity of fruit was determined by surface color and concentration of soluble solids in degrees Brix (18) for three 10-fruit replicates. The latter was measured using a handheld refractometer (Fisher Scientific, Pittsburgh, PA). Each cherry fruit was then inoculated with a 20- μ l droplet of water or a conidial suspension containing 3×10^4 spores/ml of *M. fruticola* (isolate 94-6, obtained from sweet cherry in California). For production of conidia, *M. fruticola* was grown on clarified V8 agar in the dark at 25°C for 8 to 10 days. Inoculated fruit were incubated in the dark at 20°C, >95% relative humidity (RH). The droplets were carefully dried using tissue paper after 1, 6, 9 (year 2 only), 12, 18, and 24 h and were air-dried for 30 min. Fruit were then incubated at 20°C, >95% RH, for up to 10 days. Fruit were evaluated for the presence of visible active decay and visible quiescent infections. The latter were defined as reddish circular halos with tan centers. After evaluation, a sample of 4 or 5 symptomatic fruit were surface sterilized and fungal isolations were made using the methods described above. This experiment was done once in each of two years (1996 and 1999) using eight fruit in each of the four to five replications for each wetness period. Data for incidence of infection (quiescent infections or active decay) were transformed using the arcsine-square root percentage transformation and analyzed for each wetness period using PROC GLM. Each year was analyzed separately. Means of quiescent infections and active decay were compared using least significant difference mean separation procedures of SAS (version 6.12). Additionally, data for average severity of active decay (lesion diameter in mm) was regressed on wetness-period duration for each experiment using PROC GLM of SAS to compare slopes, midpoints, and y-intercepts.

Detection of non-visible quiescent infections. A total of 250 immature cherry fruit were collected 2 weeks before harvest from five cv. Rainier commercial orchards in each of two years (1993 and 1994) and from three cv. Bing commercial orchards in one year (1994). All orchards were located in Lodi, California. Fruit were collected from five trees in each orchard, combined, and then divided into three equal groups (replications). Each group was then sub-divided into another three groups with equal numbers of fruit. Treatments were (i) untreated, (ii) surface sterilized using a hypochlorous acid (HOCl,

400 mg/liter) plus Triton X-100 (0.01%) solution for 2 min similar to the methods used by Rosenberger (16), and (iii) surface sterilized as in treatment (ii) and then dipped for 2 min in 6 g/liter of paraquat (Gramoxone, Zeneca Ag, Wilmington, DE) plus 0.01% Triton X-100. Fruit were then air dried and placed onto wire racks in clear plastic containers (32 by 24 by 11 cm) containing 100 ml of water (the fruit were not in contact with the water). Fruit were incubated under fluorescent light (200 lux; Sylvania Cool White, FC8T9-CW-RS, Danvers, MA) for 5 to 7 days at 25°C, >95% RH.

Fungi associated with the decay of each fruit were identified either by making fungal isolations or by microscopic examination of sporulating fungi on the fruit

surface. Three replicated samples of 25 fruit were used for each treatment and for each orchard sampled. Data for incidence of fungal decays (brown rot and gray mold) were transformed (arcsine-square root percentage transformation) and analyzed separately for each cherry cultivar using PROC GLM of SAS (version 6.12). For the two experiments (one experiment per year) using cv. Rainier fruit, variances of treatments were evaluated using Bartlett's test of homogeneity of variance. Data with homogenous variances were combined and treatments (non-treated, surface-sterilized, and surface-sterilized and paraquat-treated) were compared. For cv. Bing and cv. Rainier fruit, means for the incidence of decay caused by *M. fruticola* or *B. cinerea* for each treatment were

Fig. 1. Quiescent infections caused by *Monilinia fruticola* on sweet-cherry fruit. (A) Diseased floral tissues (left), green fruit rot (center), and visible quiescent infections (right; necrotic flecks, arrowheads) on the surface of green cv. Bing cherry fruit (2 $\times$). (B) Visible quiescent infections (arrowheads) on immature cv. Rainier cherry fruit. Each quiescent infection had a tan center with a reddish halo (2.5 $\times$). (C) Selected stages of brown rot infections on laboratory-inoculated, immature Bing cherry fruit collected 2 to 3 weeks before harvest (0.75 $\times$). Fruit were inoculated with conidia of *M. fruticola*, provided wetness periods of 6, 9, 12, 18, or 24 h (from left to right), and incubated for 4 to 6 days at 20°C and > 95% relative humidity. Quiescent infections (arrowheads) were observed in the 6-, 9-, and 12-h wetness periods and active decay (double arrowheads) was observed in the 12-, 18-, and 24-h wetness periods.

separated using least significant difference procedures of SAS (version 6.12).

RESULTS

Fungal isolations from symptomatic epicarp tissue of cherry fruit. In field observations on green cv. Bing fruit, lesions developed as small necrotic flecks (<1 mm) that were occasionally associated with an adjacent fruit developing green fruit rot (Fig. 1A). Significantly ($P < 0.05$) more *M. fructicola* (40 and 28% for orchard 1 and 2, respectively) was isolated from the necrotic flecks than *B. cinerea* (20 and 0% for orchard 1 and 2, respectively) from Bing fruit for the two orchards sampled. On immature cv. Rainier fruit, reddish spots with tan centers (1 to 2 mm in diameter) developed 2 to 3 weeks before harvest (Fig. 1B). The presence of *M. fructicola* and *B. cinerea* in these reddish spots was demonstrated by fungal isolations from the fruit epicarp after surface sterilization. Quantitative differences in isolation frequencies of fungi isolated from reddish lesions of Rainier fruit were obtained in the four years of this study (Fig. 2A to D). In two years, significantly ($P < 0.01$) more *M. fructicola* was isolated from these lesions compared to *B. cinerea* (Fig. 2A and D), whereas, in the two other years, there was no significant difference ($P > 0.10$) between isolation frequency of the two fungi (Fig. 2B and C). Other fungi that were incidentally isolated from both the Bing and Rainier fruit were *Alternaria alternata*, a *Cladosporium* sp., *Sclerotinia sclerotiorum*, and *Penicillium* spp.

Inoculation of cv. Bing cherry fruit in the laboratory. Immature cv. Bing cherry fruit were inoculated with water or conidial suspensions of *M. fructicola* and were kept wet for selected time periods. In both years, light-pink to pink cherry fruit were selected. Average concentration of soluble solids was $14.8 \pm 0.2^\circ\text{Brix}$ for the first experiment and $7.6 \pm 0.7^\circ\text{Brix}$ for the second experiment. Circular reddish lesions developed 4 to 6 days after inoculations with conidia of *M. fructicola* in both experiments (Fig. 3A and B). These lesions were similar in appearance to those observed on fruit in the field; however, they were larger, with diameters between 4 and 6 mm (Fig. 1C, left fruit, and Fig. 3). Most of these lesions developed no active decay even after incubation periods of up to 11 days. In re-isolations from sub-samples of inoculated fruit showing reddish halos with tan centers, only *M. fructicola* was recovered. No symptoms developed on fruit inoculated with only water.

In these inoculations, significantly ($P < 0.05$) more of the circular reddish spots were present as compared to active decay at wetness periods of 1 h in the first experiment (Fig. 3A), 6 and 12 h in both experiments (Fig. 3A and B), and 9 h in the second experiment (Fig. 3B). At wetness periods of 18 to 24 h, incidence of active decay was significantly higher than the incidence of reddish circular lesions on fruit in both experiments (Fig. 3A and B). Incidence (Fig. 3A and B) and severity (Fig. 4) of active fruit decay increased with longer duration of wetness periods after

inoculation. The regressions of severity of active decay on wetness period duration for each year were highly significant ($P < 0.01$) and increased linearly with a positive slope ($R^2 > 0.90$). The regression lines for each year that the experiment was done were not significantly different ($P > 0.05$) in slope, midpoint, or y-intercept (Fig. 4).

Detection of non-visible quiescent infections. When fruit were collected 2 weeks before harvest and treated with paraquat, decay fungi grew saprophytically on the dead fruit tissue. Variances of the treatments were homogeneous for the repeated treatments. Thus, the databases for the two years were combined for each decay fungus (Fig. 5A and B). For both decays, incidence was significantly ($P < 0.05$) higher for the surface-sterilized, paraquat-treated fruit compared to fruit that was only surface sterilized. Incidence of brown-rot decay of the non-treated fruit was not significantly ($P > 0.10$) different from the other two treatments (Fig. 5A), whereas incidence of gray-mold decay of the non-treated fruit was similar to the surface-sterilized fruit (Fig. 5B). Similar results were obtained with cv. Bing cherry fruit. Here, incidences of brown rot and gray mold were highest for the surface-sterilized, paraquat-treated fruit (5.6 and 29.5%, respectively). Significantly less decay ($P < 0.05$) was found in the surface-sterilized and the non-treated fruit. The incidence of brown rot was 0 and 1.9% and of gray mold was 8.2 and 8.7% for surface-sterilized and non-treated fruit, respectively.

In both years, decay fungi colonized 90 or 98% of the surface-sterilized, paraquat-treated cvs. Rainier or Bing fruit. In addition to *M. fructicola* or *B. cinerea*, other fungi that caused decay included *A. alternata*, *Alternaria* spp., *Aureobasidium pullulans*, *Cladosporium* spp., *Epicoccum nigrum*, *Mucor racemosus*, *Penicillium* spp., *Rhizopus stolonifer*, *S. sclerotiorum*, an unidentified Coelomycete, and unidentified yeasts.

DISCUSSION

Our study is the first report on the natural occurrence of visible quiescent infections on maturing cv. Rainier and green cv. Bing cherry fruit caused by either *M. fructicola* or *B. cinerea*. The presence of visible quiescent infections was confirmed by direct isolation of these pathogens from symptomatic fruit. Based on these isolations, symptoms caused by *M. fructicola* or *B. cinerea* were indistinguishable. Visible quiescent infections from peach (11), apricot (11), and plum (9,16,17,) have been described as small lesions (<2 mm in diameter) or halo spots. In our study, visible quiescent infections on sweet cherry ranged from small necrotic flecks that occurred on green fruit of Bing cherry to reddish halos that surrounded tannish spots or lesions on Rainier fruit collected 2 to 3

Fig. 2. Average percent incidence of *Monilinia fructicola* and *Botrytis cinerea* isolated from visible quiescent infections from cv. Rainier cherry fruit collected in a commercial orchard in California. Fruit were obtained from non-fungicide-treated trees in each of four growing seasons from 1993 to 1996. For each year, bars followed by the same letter were not significantly different.

weeks before harvest. Visible reddish lesions on nearly mature Rainier or other yellow-red cherry cultivars, such as Corum and Royal Ann, are very likely to occur also on maturing Bing fruit. In our laboratory inoculation studies, quiescent infections were reproduced on detached immature Bing fruit. Longer incubation periods (11 days), however, resulted in a decrease in visible symptoms with the development of fruit pigmentation during incubation (data not shown). This may explain why visible quiescent infections have not been observed on maturing Bing fruit under field conditions. Visible symptoms may occur for only a short period of time before fruit ripening and development of fruit pigmentation.

The isolation frequencies of *M. fructicola* and *B. cinerea* from cvs. Rainier and Bing fruit indicated that not all of the visi-

ble quiescent infections were viable or resulted in active fruit decay. Isolation frequencies of these fungi from Rainier fruit were also different for each of the four years of the study. Different environmental conditions during each growing season could possibly be more or less conducive for the establishment and viability of quiescent infections. Furthermore, the exact age of the naturally occurring visible quiescent infections was not known at the time of sampling. In old infections, the pathogen may have died before establishing an active decay. Other researchers have indicated that many of the quiescent infections on stone fruit (up to 98%) may become non-viable over time (9,11,21). The exact period of susceptibility for establishment of quiescent infections is unknown. As indicated in other studies (11), however, eradicant sprays during blos-

soming of peach and apricot were effective in reducing a proportion of quiescent fruit infections. Furthermore, Northover and Biggs (12) indicated that immature and nearly mature (3 weeks before harvest) fruit were most susceptible, whereas fruit at pit hardening were least susceptible to active-decay brown rot. Thus, quiescent infections possibly can be established during several periods during fruit development.

For the first time, visible quiescent infections were reproduced experimentally on cherry fruit. Inoculation studies to reproduce visible quiescent infections have been done on apricot (11,21) and peach (11). In our study, however, we defined conducive wetness periods at constant temperature, relative humidity, and stage of fruit development for the establishment of quiescent infections. A maximum of 50 to 60% of the fruit developed visible quiescent infections using between 9 and 12 h of wetness followed by incubation at 95% RH at 20°C. Similar data were obtained for immature (3 to 4 weeks before harvest) cv. Rainier fruit (J. E. Adaskaveg, unpublished). Any visible infection that did not develop into active decay after 10 days at 20°C was considered quiescent. Incipient infections that have been defined as symptomless, actively growing infections (14) would have developed into active decay during the 10-day incubation period at 20°C and >95% RH. This environment is highly conducive for active decay and the temperature is within the optimal temperature range for spore germination and growth of the pathogen (2). Longer wetness periods (e.g., 18 to 24 h) after inoculation at this temperature did result mostly in active decay. The development of visible quiescent infections as reddish halos is most likely an active host response to fungal penetration. Histological and biochemical information, however, has not

Fig. 3. Percent incidence of visible quiescent infections (mean $\pm$ standard deviation) and active decay (mean $\pm$ standard deviation) caused by *Monilinia fructicola* on laboratory-inoculated immature cv. Bing cherry fruit collected 2 to 3 weeks before harvest. Fruit were inoculated with conidia of *M. fructicola*, provided wetness periods of 1, 6, 9, 12, 18, or 24 h, and incubated for 4 to 6 days at 20°C and >95% relative humidity. (A, B) correspond to the years 1996 and 1999, respectively, when the experiments were conducted.

Fig. 4. Regression of average lesion diameter of active brown-rot decay caused by *Monilinia fructicola* of immature cv. Bing cherry fruit collected 2 to 3 weeks before harvest on wetness-period duration in laboratory inoculation studies. Fruit were inoculated with conidia of *M. fructicola*, provided wetness periods of 1, 6, 12, or 18 h, and incubated for 4 to 6 days at 20°C and >95% relative humidity. Data are shown for each year the study was done.

been obtained for sweet cherry. In apricot, a hydrophobic suberin layer separated quiescent infections of *M. fructicola* from healthy host tissue (21). Furthermore, the volatile compounds acetaldehyde and ethanol that were produced in ripening fruit were correlated with the activation of fungal growth from quiescent infections (4). Other possible mechanisms involved in the establishment of quiescent infections have been reviewed (2,10).

The results of this study also substantiate the existence of non-visible quiescent infections caused by *M. fructicola* and *B. cinerea* on cvs. Rainier and Bing sweet cherry fruit in California similar to other stone fruit crops including peach (11), apricot (4,11,20,21), and plum (9,13,16,) grown elsewhere. Northover and Biggs (12) suggested that quiescent (latent) infections also occur on sweet cherry. Subsequently, Wittig, Johnson, and Pscheidt (22) showed that non-visible quiescent infections occur on green sweet cherry fruit. The paraquat detection system was useful for demonstrating the presence of the brown rot and gray mold pathogens in cherry fruit in California collected 2 weeks before harvest when no visible symptoms were evident. The higher incidence of brown rot and gray mold in the surface-sterilized, paraquat-treated fruit compared to the surface-sterilized fruit cannot be attributed to surface contamination by conidia of these fungi during short incubation times (4 to 6 days). As in similar studies that were done previously for brown rot of plum (13) and for fruit pathogens of apple (1), fungal growth resumes on the senescent or dead tissue of fruit treated with paraquat.

In our study, *M. fructicola* and *B. cinerea* were the only or most common fungi

isolated from naturally occurring visible quiescent infections. Species of *Alternaria*, *Cladosporium*, *Penicillium*, and *Sclerotinia* were isolated only infrequently. In four years of sampling in one Rainier orchard, visible quiescent infections were either mainly caused by *M. fructicola* or by *M. fructicola* and *B. cinerea* in approximately equal proportions. In the studies to detect non-visible quiescent infections using the paraquat technique, greater fungal diversity was observed on the paraquat-treated fruit. In this treatment, fungi other than *M. fructicola* or *B. cinerea* mostly caused fruit decay. Some of these fungi (e.g., *Cladosporium* spp., *Alternaria* spp., *A. pullulans*, *E. nigrum*, *Penicillium* spp., and *S. sclerotiorum*) have also been found by others on decaying cherry fruit (5,6). In our study, sterilization times longer than 2 min possibly might have resulted in a lower incidence of other fungi as indicated in other studies (14).

Our studies indicate that visible and non-visible quiescent infections are common on maturing sweet cherry fruit in California. We show that many of the necrotic flecks and reddish halos are visible quiescent infections of *M. fructicola* or *B. cinerea*. Therefore, quiescent infections may be critical components of the disease cycle of these decay fungi in California, depending on the climatic conditions during a given season. Thus, the quantitative detection of quiescent infections would be helpful in the prediction of pre- and post-harvest brown rot and gray mold of sweet cherry. One method for detecting quiescent infections is the paraquat technique. The practical use of this method in a management program for determining the amount of fungal infections in fruit, however, will require specialized training in fungal iden-

tification. Ideally, a method is needed that does not require long incubation times (i.e., days) and expertise in identifying fungi and that will facilitate risk assessments of naturally infected fruit. Additionally, the method should help in decision-making processes for pre- and postharvest disease management practices (e.g., fungicide applications) and for postharvest shipping strategies for sweet cherry.

ACKNOWLEDGMENTS

We thank the California Cherry Board for financial support, P. R. Renner and L. Wade for technical assistance, and the late J. M. Ogawa for his encouragement and enthusiasm for this research.

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Fig. 5. Average percent incidence of decay caused by (A) *Monilinia fructicola* and (B) *Botrytis cinerea* of non-treated (control), surface-sterilized, or surface-sterilized and paraquat-treated cv. Rainier cherry fruit collected 2 weeks before harvest. Data were combined for two experiments (one experiment each in 1993 and 1994) and the average values are presented. Bars followed by the same letter were not significantly different.

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