PREDICTION OF BROWN ROT OF DRIED PLUM: SPORE INOCULUM POTENTIAL IN ORCHARDS AND BEST TIME PERIOD FOR DETERMINING FRUIT LATENT INFECTION CAUSED BY MONILINIA FRUCTICOLA

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ABSTRACT

Two studies were conducted this year towards the general goal to predict brown rot of dried plum (*Prunus domestica* L. cv. 'French'). We investigated periodically daily spore density of *Monilinia fructicola* in the air in two dried plum orchards located in Glenn (Orchard 1) and Tehama (Orchard 2) Counties. A spore trap was installed in each orchard, and daily spore density was determined microscopically from sticky tape samples obtained from the spore traps. Relatively high spore densities of *M. fructicola* in the air were observed from late March to early April in these two orchards. Later on, the spore densities in the air generally decreased to a low level. After June in Orchard 1, the spore density in the air remained at a very low level, but in Orchard 2, the density fluctuated to some degree on some dates of sampling. Very low level of blossom infection was observed in either of these orchards and no fruit infection detected in midseason.

In the second study, inoculation experiments were performed in ten dried plum orchards located in eight counties of California. Inoculations were conducted five times from full bloom to the first harvest stages in most of these orchards. For each inoculation, 20 branches bearing 30 - 40 fruit were selected, and each fruit was injected with about 0.1 ml of a 10,000 spores/ml suspension of M. fructicola. The inoculated fruit were kept in the trees until harvest, and percentage of fruit rot per branch was determined at different times during the season. Development of fruit rot on inoculated fruit was recorded periodically. The earlier the fruit inoculation, the slower development of fruit rot recorded. In other words, fruit infections (inoculations) that occurred in late season developed fruit rot faster than infections occurred in early season. A linear regression between rate of development of fruit rot (% fruit rot per day) and the days after full bloom was obtained. Based on information relevant to possible risk index of fruit rot for different inoculations (infections) that occurred over the season, a diagram was produced, which shows different recommendations on how to determine latent infection. Three options are given in this diagram relevant to determining latent infection: not recommended, recommended, and strongly recommended. These recommendations also take into account low, moderate, and high levels of spore inoculum potential in dried plum orchards. This diagram can serve as a reference for growers for timing the determination of latent infection in dried plums.

INTRODUCTION

Brown rot of stone fruit is caused mainly by the fungal pathogen *Monilinia fructicola*. Blossom blight and fruit rot are two major phases of this disease (1). Under favorable conditions, ascospores and/or conidia produced from mummies infected by *M. fructicola* on the orchard floor or conidia from mummies on the trees could serve as inoculum sources that cause blossom blight in the spring. These primary infections could be latent until favorable conditions are encountered and become fruit rot (2,15). Infections of fruit could continue during mid- and late-season. The main inoculum sources during this period in California in nectarine and dried plum orchards were determined to be sporulation of *M. fructicola* on the thinned fruit on the orchard floor (3,9).

Previous findings on the brown rot disease include: 1) a relationship among blossom infection and environments (8); 2) a seasonal pattern of blossom and fruit susceptibility over the growing season (5); 3) seasonal patterns of risky factors affecting fruit infections (6); 4) a relationship between inoculum potential on fruit and environments (4); 5) effects of orchard cultural practices on dynamics of inoculum potential in orchards (9); and 6) threshold conditions that lead latent infection to fruit rot (7). Based on these findings, we have established a preliminary Decision Support System (DSS) for IPM of Prune Brown Rot (10) located at http://tjm.uckac.edu. The existing DSS thus far includes a basic system for IPM of blossom blight and a preliminary model for decisions on fungicide application during mid-season.

Information that is needed for improving the existing DSS includes: a) the development of a fast and accurate method for estimating spore inoculum potential in orchards over the season; b) the determination of possible risk of fruit rot development from latent infection; and c) the best time period to efficiently determine latent infection levels in dried plum orchards. In previous research, we determined the dynamics of spore density in the air in orchards by using spore traps and the effect of orchard cultural practices on the spore dynamics of the pathogen. Generally, the highest spore inoculum potential was observed at bloom and then inoculum density decreased, unless there were improper fruit thinning and timing of irrigation in the orchard. Continuation of this study is needed to confirm the above conclusions.

Estimating latent infection during the growing season is meaningful for both pre- and post-harvest (the latter only for fresh-market fruit) disease management, and reducing latent infection can become important in disease management. Our existing DSS can be used to help growers make a decision on fungicide application in the mid-season by using results from overnight freezing/incubation technique (ONFIT; the protocol of this technique has been posted at http://tjm.uckac.edu/TJM-Site/onfit/ONFIT.htm). For the system to estimate what the risk could be that latent infections become fruit rot, it is required that the level of latent infection is determined first and provided. A previous study determined the risk of latent infections becoming fruit rot before harvest (7). Obviously, periodic determinations of latent infection over the season could be desirable,

but they cannot be practical. The critical period (best time period) for an efficient determination of latent infection needs to be defined and could be of major importance for the user of the DSS. If determination of latent infection was done only once or twice per season, its application in and use of DSS would be easy and practical.

Field experiments were performed in 2002 to help identify the critical period for determining the level of latent infection in dried plum orchards. Development of fruit rot originated from latent infections was studied from results of our field inoculation to determine the critical infection period that may relate to a high risk or fast fruit rot development. These results are helpful to determine when a fungicide application, which is done to reduce the risk of latent infection, could be most effective during the season in reducing fruit rot before harvest.

OBJECTIVES IN 2002:

- 1. To continue to determine the spore densities of *M. fructicola* in the air in two dried plum orchards.
- 2. To determine the development of fruit rot from latent infections over the growing season
- 3. To determine the best time period for quantifying latent infections in dried plum orchards.

PROCEDURES

1. Continue to determine the spore densities of *M. fructicola* in the air of dried plum orchards.

Spore trap samples. One Burkard spore trap was installed on 20 March 2002 in each of the two dried plum orchards selected in Glenn and Tehama Counties, respectively. The orchard in Glenn County was just next to the orchard where we determined spore density in 2001. The orchard in Tehama County was about 2 miles south from the orchard where we determined spore density in Tehama County in 2001. Slides with spore trap sticky tape samples were collected weekly and the daily spore density was determined with a compound microscope at the Kearney Agricultural Center in Parlier. A curve showing densities of spores (spores/liter/minute) in each orchard's air over the season was produced.

To determine infection levels, blossom and fruit samples were collected in early- and mid-season from each orchard. At full bloom, about 500 blossoms were randomly collected in each orchard, surface sterilized with 300 ml of a 10% bleach solution for 3 minutes, and washed with sterile distilled water five times. The blossoms were separately placed on top of two layers of sterile wet paper towels in a sterile plastic container. The containers were incubated at room temperature, $23 \pm 2^{\circ}$ C (73 to 77° F) for 7 days, and the blossoms showing sporulation of *M. fructicola* on the stem were recorded. Thus, the incidence of blossom infection as a percentage of the total number of the blossoms was calculated for each sample.

Symptomless fruit samples were taken on May 15 and June 4 at pit hardening and embryo growth stages (13), respectively. In each sampling, 300 fruit were randomly collected from 10 trees in each orchard. The Overnight Freezing Incubation Technique (ONFIT; 11,12) was used to determine the incidence of brown rot latent infection for each sampling.

Molecular procedure. We also developed a fast and reliable method to detect spore density in the air by using molecular techniques. In our previous studies on genetic and population structures of M. fructicola, we observed that the microsatellite primer M13 amplified a DNA fragment of approximately 740 bp in size from each of more than 500 isolates of M. fructicola collected worldwide (Luo, et al., unpublished data). In this study, we cloned and sequenced the characterized amplification region to develop nested PCR assays for detection of M. fructicola. Nested primer pairs (external and the internal), specific to M. fructicola were designed using a computer program (Xprimer). The specificities of primers were tested using 30 isolates of M. fructicola collected from different hosts throughout California in different years, as well as eight isolates of M. fructicola from other states in the US and other countries, and 25 isolates of other fungal species associated with stone fruit. To determine how sensitive (i.e., how many spores of M. fructicola) these primers can detect, we preformed sensitivity tests by diluting the known amounts of DNA (from 0.1 ng to 1 fg, i.e., from 10⁻¹² to 10⁻¹⁵ grams) and DNA extracted from known numbers of spores of M. fructicola. These DNAs were used as templates for PCR amplifications of unknown spore samples.

- 2. Determine the development of fruit rot from latent infections over the growing season.

 and.
- 3. Determine the best time period for quantifying latent infections in different dried plum orchards.

Experimental design:

In 2002, in cooperation of farm advisors, we continued to conduct multi-location experiments. Ten dried plum orchards located in Tulare, Fresno, Madera, Yuba, Butte, Sutter, Glenn, and Tehama Counties, were selected. Two random trees in each orchard were periodically (at different fruit developmental stages) inoculated. In each tree, 10 branches each bearing 30 – 40 fruit were identified, and each fruit was inoculated by injecting about 0.1 ml of a 10,000 spores/ml suspension of *M. fructicola*. The inoculated fruit were kept on the trees until harvest. On each inoculation date, the number of fruit with brown rot for each branch that had been inoculated the previous inoculation date was recorded, and the percentage of fruit rot for each inoculated branch was calculated. Table 1 lists the inoculation dates and the corresponding fruit rot recording dates for each inoculation.

Data analysis:

Determination of fruit rot development for each inoculation experiment. On each disease recording date and for each orchard and inoculation date, the average percentage of fruit rot from the 20 inoculated branches was calculated. Also, for each disease recording date and each inoculation, the mean percentages of fruit rot (PFR) for all 10 orchards were also determined. The PFRs versus days after full bloom were used to draw the disease developmental curve for each inoculation. For all curves, the percentage of fruit rot on the inoculation date was assigned as 0, and the days after full bloom were used.

Calculation of rate of development of fruit rot and regression of rate over days after full bloom. For each inoculation, the rate of development of fruit rot (r) was calculated by using the following formula: $r = (PFR_t - PFR_{t-1}) / D$, where PFR_t is the percentage of fruit rot at time t and PFR_{t-1} is the percentage of fruit rot at time t-1 (meaning at the previous recording time of fruit rot). For the 4th and 5th inoculations that the fruit rot was only recorded once after inoculation, the fruit rot on the inoculation date was assigned as 0% to calculate r values. For the 1st, 2^{nd} , and 3rd inoculations, the 0% fruit rot on inoculation date was also used to calculate r values for the time period from the inoculation date to the first disease recording date. The average fruit rot developmental rate was calculated from the rates of two time periods of disease recordings for the 1st, 2nd, and 3rd inoculations.

Table 1. Locations, corresponding dates of fruit inoculation with *Monilinia fructicola*, and dates of fruit rot recording in 2002.

| | Inoculation | Date of recording | | Inoculation | Date of recording |
|----------|-------------|-------------------|----------|-------------|---------------------|
| Location | Date | fruit rot | Location | Date | fruit rot |
| | | | Tehama- | | |
| Tulare | 20-Apr | April 29, July 9 | 2 | 22-Apr | May 17, July 10 |
| | 14-May | June 20, July 9 | | 17-May | June 19, July 10 |
| | 1-Jun | June 20, July 9 | | 4-Jun | June 19, July 10 |
| | 20-Jun | 9-Jul | | 19-Jun | 10-Jul |
| | 9-Jul | 25-Jul | | 10-Jul | 24-Jul |
| Butte | 22-Apr | May 17, July 10 | Madera | 19-Apr | May 8, July 9 |
| | 17-May | May 17, July 10 | | 8-May | June 21. July 9 |
| | 4-Jun | 19-Jun | | 31-May | June 21, July 9 |
| | 19-Jun | 10-Jul | | 21-Jun | 9-Jul |
| | 10-Jul | 24-Jul | | 9-Jul | 25-Jul |
| Sutter | 17-May | June 4, July 24 | Glenn-1 | 22-Apr | May 17, July 10 |
| | 4-Jun | 19-Jun | | 17-May | May 17, July 10 |
| | 19-Jul | 24-Jul | | 4-Jun | June 19, July 10 |
| | | | | 19-Jun | 10-Jul |
| KAC | 11-Apr | April 19, July 11 | | 9-Jul | 24-Jul |
| | 8-May | June 20, July 11 | | | |
| | 31-May | June 20, July 11 | Glenn-2 | 17-May | May 17, July 10 |
| | 20-Jun | 11-Jul | | 4-Jun | June 19, July 10 |
| | 11-Jul | 11-Jul | | 19-Jun | 10-Jul |
| Tehama- | | | | 9-Jul | 24-Jul |
| 1 | 22-Apr | 10-Jul | | | |
| | 17-May | June 19, July 10 | Yuba | 22-Apr | May 17, July 10 |
| | 4-Jun | June 19, July 10 | | 17-May | May 17, July 10 |
| | 19-Jun | 10-Jul | | 4-Jun | 19-Jun |
| | 10-Jul | 24-Jul | | 19-Jun | 19-Jul |
| | | _ · · · · · · · · | | 10-Jul | 24-Jul |
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The average fruit rot developmental rate from the 10 orchards was calculated for each inoculation date, and a regression between the rates of the five inoculations and days after full bloom was conducted. A linear regression equation was used to calculate the possible fruit rot developmental rate on a certain day t (r_t) after full bloom, and the r values were produced for every 5 days from March 20 (full bloom) to July 31. These r values could represent the potential fruit rot developmental rates when inoculations (=infections) occur on the corresponding dates. Therefore, these rates were also used to calculate how many days (D_t) are needed from the day t that the fruit rot developmental rate could reach a 100%. The $D_t = 100\%$ / r_t , where r_t is the fruit rot developmental rate at the day t after full bloom.

We used the following ratio to calculate a Risk Index at the day t (RI_t), where RI_t = C_t/D_t , where C_t is number of days from the current date to August 15, and D_t is the number of days required so that the fruit rot developmental rate reaches 100%, which was calculated with the above equation. The values of RI_t greater than 1.0 were used in further analysis, and the RI_ts with values less than 1.0 were not taken into account in the analysis, since on these dates it would not be necessary to determine latent infection. We arbitrarily assigned 1.0, 1.25 and 1.5 of RI values to represent low, moderate, and high possibilities that the infection of fruit on a certain date could reach 100% fruit rot before or on August 15.

Development of a diagram for helping in deciding on determining latent infection during the mid-season. Based on the Risk Index (RI) curve, we developed a diagram to help growers decide when it is necessary to determine latent infection during the growing season. In this diagram, we also considered the low, moderate, and high inoculum potential levels in the orchards. Recommendations for determining latent infection are given at a 5-day interval from May 1 to July 31. Three recommendations for determining latent infection were used as follows: not recommended, recommended, and strongly recommended.

RESULTS

1. Determination of spore density in the air of dried plum orchards.

Spore trap samples. Figure 1 shows the ranges of spore density of M. fructicola in the air over the season in 2002 in the two commercial orchards. Generally, spore densities were lower compared with those observed in 2001 (9), and relatively higher spore densities (20–45 spores/liter/min) were recorded during the end March to beginning of April (Figure 1). In Orchard 1, the spore density decreased continuously after the highest peak observed in late March through the beginning of June and remained at very low levels through the end of the season. In Orchard 2, some small peaks of spore density in the air were observed early in the season (late March) and in the mid-season (early in June). Although some relatively high spore densities were observed in some days, the spore density was generally low in the two orchards included in this study (Figure 1). Blossom infection was about 3% in Orchard 1 and only 0.24% in Orchard 2. No fruit infection was observed in spring and mid-season in either of these orchards. The results implied very low infection levels over the season in these two orchards in 2002.

Molecular procedure. The nested PCR primers specific to M. fructicola were developed based upon the sequence of a species-specific DNA fragment amplified by microsatellite primer M13. The external primer pairs EMfF + EMfR and the internal primer pair IMfF + IMfR amplified a 571 bp and a 468 bp fragment, respectively, from M. fructicola, but not from any other fungal species present in stone fruit orchards. The nested PCR assay was sensitive enough to detect the specific fragments in 1 fg (10⁻¹⁵ of a gram) of M. fructicola or in the DNA from only two spores of M. fructicola. This technique could detect as few as 200 spores of M. fructicola caught on spore-trap tapes. The results of this study indicate that microsatellite regions could be useful in developing highly sensitive PCR detection systems for phytopathogenic fungi, including M. fructicola. Although

more research is needed in this area, nested PCR assays have a potential to detect spores of *M. fructicola* in the air of stone fruit orchards. Such molecular detections of *M. fructicola* spores in orchards could be used in more refined epidemiological studies of the brown rot disease in stone fruits.

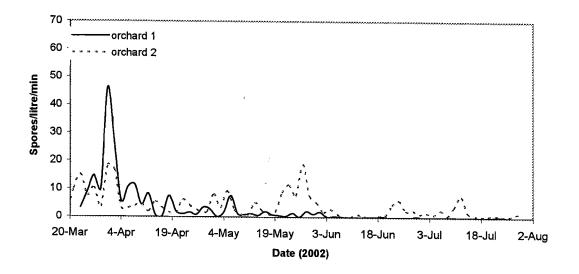


Figure 1. Dynamics of daily spore density of *Monilinia fructicola* in the air as determined from samples taken from Burkard spore traps located in two dried plum orchards. Orchard 1 was located in Glenn County and Orchard 2 in Tehama County.

2 and 3. Determine the development of fruit rot from latent infections and the best time period for quantifying latent infections in dried plum orchards.

Different developmental curves of fruit rot were observed for different inoculation dates over the season (Figure 2). In general, the earlier the inoculation date, the slower the development of fruit rot observed. For the first inoculation date (on 22 April), the fruit rot reached about 15.8% on May 17 and about 26.6% on July 11. For the second inoculation date (17 May), the fruit rot reached 52% on July 10. For the third inoculation date (4 June), the fruit rot reached 48.9% on June 20 and 58.7% on July 10. The incidence of fruit rot reached 70%, 20 days after the fourth inoculation date (20 June); and 51.8%, 14 days after the fifth inoculation date (10 July) (Figure 2). It is obvious from these results that fruit rot developed faster for the later than the earlier inoculation (infection) dates (Figure 2).

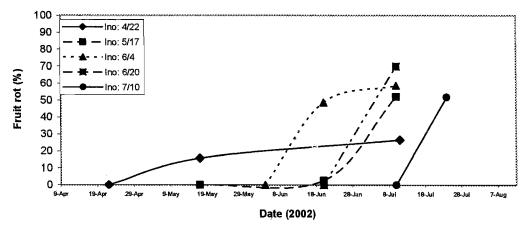


Figure 2. Development of fruit rot recorded on branches of dried plum trees inoculated with spores of *Monilinia fructicola* at different dates over the season. Inoculation experiments were conducted in ten different orchards in several Counties in California. Each dot represents the average of fruit rot from 20 branches inoculated each time for these ten locations.

A linear regression between fruit rot developmental rate (% fruit rot /day) and the days after full bloom with a high R² of 0.96 was obtained (Figure 3). The results demonstrated that the fruit rot developmental rate increased along with the season, namely, fruit rot developed faster in later than in earlier dates in season, suggesting that the risk of fruit rot development from latent infection could be higher for infections of fruit that occur later than those that occur earlier in season.

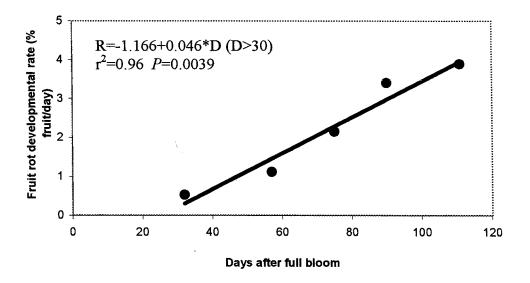


Figure 3. Linear relationship between days (D) after full bloom and fruit rot (caused by *Monilinia fructicola*) developmental rate (R). Each dot represents the average of fruit rot developmental rate from ten dried plum orchards with five inoculations during the season per orchard.

We also obtained a Risk Index (RI) curve shown on Figure 4. The definition of the RI is the possibility of fruit rot reaching 100% at harvest. Three levels, 1.0, 1.25 and 1.5 of (RI) were assigned to represent the low, moderate and high risk levels, respectively. For instance, if the RI was between 1.0 and 1.25, it would relate to a low risk for fruit rot, if the RI was between 1.25 and 1.5, it would relate to a moderate risk for fruit rot, and when the RI was above 1.5, it would relate to a high risk for fruit rot development.

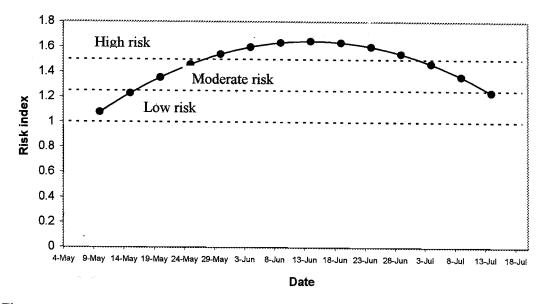


Figure 4. The risk index (RI) curve for fruit rot development caused by *Monilinia fructicola* in dried plum orchards, indicating infection dates that correspond to possible risk levels for fruit rot development to reach 100% at harvest.

Based on the information from the Risk Index curve for fruit rot development, a diagram showing different recommendations for determining levels of latent infection was produced (Figure 5). Three options are given in this diagram: a) not recommended; b) recommended; and c) strongly recommended to proceed with determination of latent infection. In generating this diagram, we also considered three situations of inoculum potential in dried plum orchards: 1) low, 2) moderate, and 3) high. This diagram can serve as a reference for the grower to decide on whether or not determination of latent infection would be required. For instance, in an orchard with a low inoculum potential, determination of latent infection is strongly recommended during 10 to 15 of June (Figure 5). In an orchard with a moderate spore inoculum potential of M. fructicola, determination of latent infection is strongly recommended during May 30 to June 30 (Figure 5). However, if an orchard has a high spore inoculum potential, determination of latent infection would be strongly recommended during May 20 to July 5 (Figure 5). Since the time required for latent infections that may occur in late-season would not be long enough for latent infections to become fruit rot at a 100% level at harvest, investigation for latent infection levels in late season in orchards with a moderate or a high inoculum potential is recommended (Figure 5).

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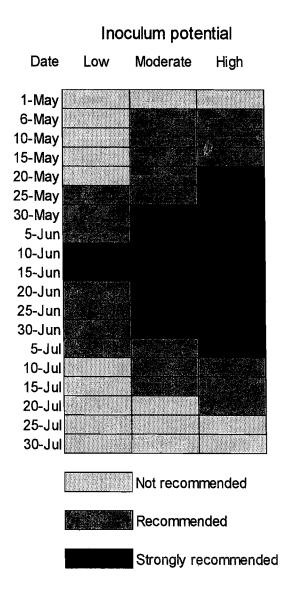


Figure 5. A chronological diagram for recommendations on making a decision for determining the level of latent infection caused by *Monilinia fructicola* in dried plum orchards. Three levels of initial inoculum potential are considered for the corresponding dates in the season.

CONCLUSIONS

1. High levels of spore densities occurred in the two dried plum orchards in the spring at full bloom. However, the pathogen's spores decreased to low levels after full bloom, and remained to very low levels after June.

- 2. Late-season infections relate to a higher risk of becoming fruit rot than early-season infections, and the development of fruit rot was faster for fruit infected late than early in season.
- 3. As shown in Figure 3, the developmental rate of fruit rot increased as the time of fruit infection after full bloom increased and approached the harvest date.
- 4. The critical period for determining latent infection of dried plums differs at different periods during the growing season. In general, the critical period for determining latent infection is in June. However, this critical period during which determination of latent infection is recommended is shorter in orchards with low spore inoculum potential than in those with moderate or high inoculum potential.
- 5. A chronological diagram was produced that can help growers decide on the time to determine latent infection in their orchard. Knowing the inoculum potential in the orchard can provide more accurate information relevant to the critical period (best time period) for determining latent infections in dried plums.
- 6. The above results will be incorporated into the existing Decision Support System for IPM of Prune Brown Rot (http://tjm.uckac.edu) to guide growers in fungicide application to manage both blossom and fruit rot in their orchards.

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