Significance of Thinned Fruit as a Source of the Secondary Inoculum of *Monilinia fructicola* in California Nectarine Orchards

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ABSTRACT

Hong, C. X., Holtz, B. A., Morgan, D. P., and Michailides, T. J. 1997. Significance of thinned fruit as a source of the secondary inoculum of *Monilinia fructicola* in California nectarine orchards. Plant Dis. 81:519-524.

The significance of thinned fruit as a source of secondary inoculum in the spread of brown rot, caused by *Monilinia fructicola*, under semi-arid weather conditions of the San Joaquin Valley in California, was investigated in seven nectarine orchards in 1995 and 1996. Between 6 and 60% (depending on the orchard) of thinned fruit showed sporulation by *M. fructicola*. Brown rot was significantly less severe at preharvest (five orchards) and postharvest (one orchard) on fruit harvested from trees in plots from which thinned fruit were completely removed than on those in plots from which thinned fruit were not removed. *M. fructicola* sporulated more frequently on thinned fruit placed into irrigation trenches than on those left on the dry berms in tree rows. The incidence of preharvest fruit brown rot increased exponentially as the density of thinned fruit increased on the orchard floor. These results suggest that thinned fruit left on the floor of nectarine orchards can be a significant inoculum source of secondary infections. Removal or destruction of thinned fruit should reduce brown rot in nectarine and possibly other stone fruit orchards under semi-arid California conditions.

Additional keywords: disease control, epidemiology, *Prunus*, stone fruits

Brown rot, caused by *Monilinia fructicola* (G. Wint.) Honey, is one of the most destructive diseases of stone fruits (*Prunus* L.) in the United States (22,28,40), Canada (1-3), Australia (5-7), and New Zealand (4,8). *M. fructicola* usually causes blossom blight in the spring, which develops into a twig blight and canker and provides inoculum for latent infection of green fruit (15,26,35,37, and T. J. Michailides, unpublished data). Subsequently, brown rot develops on fruit at the end of the season (harvest) and after harvest (during storage and transport to markets) (5). Blossom blight and fruit brown rot are the most important phases epidemiologically and economically.

Inoculum sources for primary infection of blossoms by *M. fructicola* have been extensively investigated under various weather conditions (1,5,11-13,18,22) since the late nineteenth century (30). Comparatively little has been done on the relationship between sources of inoculum and infection of immature and mature fruit.

Pioneer work on the sources of secondary inoculum of brown rot was done by Landgraf and Zehr (22) in South Carolina and Biggs and Northover (1) in Canada. They considered non-abscised, aborted fruit in trees and thinned fruit on the orchard floor as important sources of the secondary inoculum for fruit brown rot under their humid climates (1,22). However, there are no reports on the contribution and importance of thinned fruit as a source of the secondary inoculum under the semi-arid weather conditions of California.

This paper reports the investigation of the importance of thinned fruit as a source of secondary inoculum for fruit brown rot in seven nectarine (*Prunus persica* var. *nectarina* (Ait.) Maxim.) orchards in the semi-arid San Joaquin Valley of California in 1995 and 1996. The specific objectives of this study were to determine the incidence of *M. fructicola* conidial sporulation on thinned fruit, study the significance of thinned fruit as a source of the secondary inoculum of *M. fructicola* in irrigated nectarine orchards, and determine the relationship between the density of thinned fruit on the orchard floor and the incidence of fruit brown rot.

MATERIALS AND METHODS

Orchards and weather data. Field experiments were conducted in five nectarine orchards in 1995 (coded as 951 to 955) and two in 1996 (coded as 961 and 962) (Table

1), located near Parlier, Reedley, or Easton, all in Fresno County. Nectarine cultivars included in the experiments were Fantasia (orchards 951, 953, and 962), which is one of the latest maturing freestone nectarine cultivars, Summer Grand (orchards 954 and 961), which is the most common cultivar in production in California, August Red (orchard 952), and May Diamond (orchard 955) (Table 1). Trees were planted 5 to 6 by 5 to 6 m and ranged in age from 9 to 22 years during the seasons when experiments were conducted. Strips of 1-m-wide reflective plastic film were placed between tree rows in orchards 961 and 962 in early June to enhance the fruit color.

Two to three fungicide sprays were applied to control blossom blight from February to March (except for orchards 952 and 953) in 1995 and 1996, and one to two sprays were applied to control fruit brown rot before harvest in 1995 (Table 1). Fungicides applied included benomyl, captan, chlorothalonil, iprodione, myclobutanil, and triforine (4,22,29).

Orchards were irrigated from late March until the end of October (data after harvest not shown); irrigation schedules varied from orchard to orchard (Table 1). Usually, orchards were irrigated every 5 to 20 days, resulting in four to seven irrigations between fruit thinning and harvest. Local weather data were obtained from the California Irrigation Management Information System (CIMIS).

Effect of completely removing thinned fruit on brown rot. Experiment A was conducted in three orchards (951, 952 and 953) in 1995 and two orchards (961 and 962) in 1996. Treatments consisted of A1, in which thinned fruit were completely removed from the plots, and A2, in which thinned fruit were not removed and were supplemented by an equal amount of thinned fruit obtained from A1 plots. In orchard 962, a third treatment, A3, was included, in which fruit thinned by the grower were left undisturbed. There were three replicated plots per treatment in all orchards, except for 952 and 953, which had four replications per treatment. Each plot consisted of 8 × 8 trees. Treatments in each orchard were arranged in a randomized block design.

Before fruit thinning, the four center trees and four corner trees in each plot were flagged. Green fruit were thinned

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Accepted for publication 10 February 1997.

manually by growers and counted under the four center trees in each plot. Stage of fruit development at thinning was determined by cutting fruit, and no fruit were thinned before pit-hardening in any of our experimental orchards. Eleven to fifteen days after thinning, 200 thinned fruit were selected randomly in the irrigation trenches and on the dry berms under each of 4 center trees in orchards 951 and 953. The fruit were examined individually for visible conidial sporulation of *M. fructicola*, and the number of thinned fruit exhibiting sporulation recorded. Thinned fruit were examined only in the trenches around the 4 center trees in A2 plots in orchards 961 and 962. Thinned fruit with sporulation of *M. fructicola* were recorded weekly in orchard 961, but only twice in orchard 962. The incidence of thinned fruit exhibiting the sporulation in each plot was calculated and the data were analyzed using Proc analysis of variance (ANOVA) of SAS (Version 8.6e, SAS Institute Inc., Cary, NC). Means were separated by Fisher's least significant difference at $P = 0.05$.

Fruit brown rot was assessed by observing sporulation of *M. fructicola* on 200 fruit selected arbitrarily in each of the 4 center trees in all plots, either once or twice before the first fruit harvest. Because fruit mature at different times, several harvests occur per season. The incidence of preharvest fruit brown rot was calculated for each replicated plot and data were analyzed using Proc ANOVA and means were also separated by Fisher's least significant difference at $P = 0.05$.

Fifty fruit with no obvious symptoms of brown rot were harvested from each of the 4 center trees in each replicate, placed into a paper box (2 layers with 25 fruit per layer), transported to the Kearney Agricultural Center postharvest laboratory, and stored at 4°C for 7 days to restrict

Rhizopus rot (10) and simulate commercial storage. The fruit were then incubated at 20°C and >95% relative humidity for 3 days before examining for postharvest diseases. Brown rot and other diseases were recorded every other day for 3 to 5 days. The incidence of postharvest fruit brown rot was calculated for each plot and the incidence data of each orchard and assessment were also analyzed using Proc ANOVA. Treatment mean incidence data of 5 orchards and 2 to 3 assessments were pooled and further analyzed using the sign test (20).

Effect of moving thinned fruit from berms to irrigation trenches on fruit brown rot. Experiment B was carried out in orchards 954 and 955 in 1995. Treatments included in this trial were B1, in which thinned fruit were moved from berms into irrigation trenches, and B2, in which thinned fruit were not disturbed. There were three 8 × 8 tree replicates of each treatment and the two treatments were arranged in a randomized block design. Sporulation of *M. fructicola* on thinned fruit was assessed by examining 200 fruit in the trenches, and 11 to 26 naturally

Fig. 1. Daily rainfall and maximum relative humidity at a representative weather station (Parlier, California), during May to August of 1995 (top) and 1996 (bottom).

Table 1. Fungicide applications and irrigation schedule applied of orchards used in the study

Orchard code	951	952 ^x	953	954	955	961	962
Location	KAC	Reedley	Reedley	Parlier	Parlier	Parlier	Easton
Nectarine cultivar	Fantasia	August Red	Fantasia	Summer Grand	May Diamond	Summer Grand	Fantasia
Tree age (years)	21	9	17	17	10	18	22
Thinning date	4/27 ^y	6/6	5/17	5/3	5/9	5/13	5/18
No. of thinned fruit/tree	960	162	314	1547	351	2114	500
1st blossom spray	2/13 benomyl			2/12	2/12	2/24	2/22
(date and fungicides)	/iprodione	NA ^z	NA	iprodione	iprodione	iprodione	iprodione
2nd blossom spray	2/17			2/21 benomyl/	2/21 benomyl/	3/4 myclobutanil/	3/1
(date and fungicides)	iprodione	NA	NA	chlorothalonil	chlorothalonil	captan	iprodione
3rd blossom spray				3/8	3/8	3/9	3/8
(date and fungicides)	NA	NA	NA	captan	captan	chlorothalonil	benomyl
1st preharvest spray	6/29	7/18	6/23	6/19	6/16		
(date and fungicides)	iprodione	triforine	triforine	triforine	triforine	NA	NA
2nd preharvest spray	7/7		7/4				
(date and fungicides)	triforine	NA	triforine	NA	NA	NA	NA
Irrigation dates	5/1,10,22, 6/1,8,19, 7/3,10	4/25, 5/1,11,25, 6/9,16,22, 7/2,7,11,15, 7/28, 8/15	3/22, 4/25, 5/22, 6/5,20,25, 7/9,15,20	5/5,27, 6/14,26, 7/7,18	5/4,26, 6/10,28, 7/16,29	4/18, 5/3,17, 6/2,13,30, 7/2,21	4/7,23, 5/4,17,29, 6/8,17,26, 7/4,10

^x In orchard 952, every other tree row was flooded with each irrigation in July.

^y Month/date(s).

^z Fungicides were not applied.

fallen fruit found on the berm under each of 4 center trees in either orchard, as described previously. Pre- and postharvest fruit brown rot in these orchards were assessed and the data were analyzed as in experiment A.

Relationship between fruit brown rot and thinned fruit in orchards with nectarine cultivars of the same maturity.

Since there is no apparent difference in susceptibility among nectarine cultivars to brown rot (28), disease incidences of different cultivars of the same maturity were compared. Nectarine fruit in orchards 951, 953, 954, and 955 matured and were harvested in early- to mid-July, so data of preharvest fruit brown rot in A2 plots in orchards 951 and 953, and B2 plots in orchards 954 and 955, were pooled. The density of thinned fruit in A2 plots was calculated by doubling the orchard average number of thinned fruit per tree, since these plots received about equal amounts of thinned fruit moved from A1 plots in each orchard. The relationship between incidence of brown rot and the number of thinned fruit per tree was examined with Proc REG of SAS, after transforming the disease incidence data with the natural log. Similarly, the density of thinned fruit was calculated for A2 plots of orchards 961 and 962.

RESULTS

Weather data. The Parlier CIMIS station was located closest to the field experiments conducted in 1995 and in 1996. Daily rainfall and maximum relative humidity at the Parlier weather station from 1 May to 31 August, the seasonal time of nectarine fruit pit hardening and maturing in 1995 and 1996, are shown in Fig. 1. There were only 6 days of precipitation with a total of 43 mm rainfall from May to August 1995, and only 5 days of precipitation with a total of 8.9 mm rainfall during the same period in 1996. The daily maximum relative humidity ranged from 63 to 99% from May to August of 1995, and from 60 to 96% over the same 4 months in 1996. Daily maximum relative humidity was less than 90% over half of the periods in 1995 and 1996, with the weather generally being drier in 1996.

Effect of completely removing thinned fruit on brown rot. Green fruit were thinned at pit-hardening stage in all the orchards, but thinning dates differed among cultivars, orchards, and seasons (Table 1). Between 162 and 2,114 fruit per tree were thinned, depending mainly on the cultivar, season, and grower's strategy and preference.

The incidence of thinned fruit showing sporulation of *M. fructicola* was higher in the irrigation trenches than on the dry berms in orchards 951 and 953 (Table 2). The incidence of thinned fruit showing sporulation of *M. fructicola* increased in the first 3 weeks after fruit

thinning and then declined in orchard 961 (Fig. 2).

Preharvest fruit brown rot was more severe in A2 plots, where thinned fruit were added, than in A1 plots, where the fruit were completely removed in orchards 951 and 952 at the first disease assessment, orchard 953 at the second assessment (Fig. 3), and in orchards 961 and 962 (Fig. 4).

The incidence of postharvest brown rot was significantly higher on the fruit collected from A2 plots than on fruit from A1 plots or A3 plots (Fig. 4). The sign test (20) of combined data from the five orchards for the two to three assessments of postharvest brown rot further demonstrated that the probability of observing postharvest brown rot was higher on fruit from A2 plots than on fruit from A1 plots in all orchards and assessments (all postharvest assessments, $n = 14$) is 0.0002; the postharvest brown rot was significantly reduced by the practice of completely removing thinned fruit from plots. Other diseases observed included *Rhizopus* rot in 1995, and *Botrytis* and *Rhizopus* rots in 1996. *Rhizopus* rot (3.5%) was the only other disease observed on the fruit collected from orchard 952 at the last assessment in 1995. *Botrytis* rot (0.2%) was observed on fruit collected from orchards 961

and 962, whereas *Rhizopus* rot (0.2%) was observed only on the fruit collected from orchard 962 at the last assessment.

Effect of moving thinned fruit from berms to irrigation trenches on fruit brown rot. The incidence of thinned fruit showing sporulation of *M. fructicola* was higher in the irrigation trenches than on the dry berms in orchards 954 and 955 (Table 2). No differences were observed between the incidence of both pre- and postharvest brown rot on fruit from B1 and B2 plots (data not shown).

Relationship between brown rot and thinned fruit in orchards with nectarine cultivars of the same maturity. Preharvest brown rot increased as the number of thinned fruit per tree increased in orchards in 1995 (Fig. 5). The relationship between brown rot incidence (y , %) and the number of thinned fruit per tree (x) was best described by the equation $y = e^{0.0012x}$ ($P = 0.0466$, $R^2 = 0.91$). Orchard 952 was excluded from the regression analysis because green fruit were thinned and mature fruit were harvested about 1.5 months later than the fruit in the other orchards. A similar relationship was observed in 1996; in orchard 961, both thinned fruit (4,228/tree) and incidence of postharvest brown rot (13.5%) at the second disease

Table 2. Sporulation of *Monilinia fructicola* on thinned nectarine fruit in irrigation trenches and on dry berms in four irrigated orchards (1995) and one orchard (1996)

Orchard	Exp.	Thinned fruit exhibiting <i>M. fructicola</i> conidial sporulation (%)		
		Date examined	Trenches	Berms
951	A	10 May	57.9 a ^x	37.6 b
953	A	1 June	6.3 a	2.9 b
954	B	1 June	56.3 a	19.4 b ^y
954	B	30 June	60.3 a	20.5 b ^y
955	B	1 June	24.2 a	13.9 b ^y
962	A	18 June	13.0	ND ^z
962	A	3 July	4.7	ND

^x Values, followed with different letters within a row, differed significantly according to Fisher's least significant difference test at $P = 0.05$.

^y On which naturally fallen fruit was assessed for *M. fructicola* conidial sporulation.

^z Not determined.

Fig. 2. Incidence of thinned fruit sporulating with *Monilinia fructicola* after thinning of nectarine (cv. Summer Grand) fruit in orchard 961, Parlier, California, 1996. Green fruit were thinned on 13 May. Vertical bars are the standard errors of the means.

Fig. 3. Pre- and postharvest brown rot by *Monilinia fructicola* on nectarine fruit from A1 plots, from which thinned fruit were completely removed, and from A2 plots, from which thinned fruit were not removed and were supplemented by an equal amount of thinned fruit obtained from A1 plots, in orchard 951 (cv. Fantasia) at the Kearney Agricultural Center, and orchards 952 (cv. August Red) and 953 (cv. Fantasia), near Reedley, California, 1995. Arrows indicate the dates of fruit harvest for determining postharvest diseases. Only bars to the right of the arrow represent postharvest assessment (in the lab). Bars topped by different letters within a date differed significantly according to Fisher's least significant difference test at $P = 0.05$.

assessment were greater ($P < 0.05$) than those in orchard 962 (1,000 thinned fruit/tree and 9.7% postharvest brown rot).

DISCUSSION

Epidemiologically, brown rot of stone fruit is a polycyclic disease (5), and its fruit rot phase is characterized by latent infections (8,15,16,19,26,35,37,39, and T. J. Michailides, unpublished data). In California, latent infections are facilitated by semi-arid weather conditions, particularly in drier seasons such as 1996. This conclusion is supported by the fact that brown rot was much less severe on fruit in trees before harvest than after harvest. Additionally, more fruit had sporulation of *M. fructicola* in orchards in 1995 (4.3 to 14.6%)

than in 1996 (0.0 to 0.5%), but the incidences of postharvest brown rot were comparable for 1995 (15.1 to 56.7%) and 1996 (29.5 to 45.5%). Therefore, local weather conditions may have reduced the number of infection cycles per year. It is not unusual to have dry weather in the San Joaquin Valley during the spring and summer. Thus, conidia of *M. fructicola* produced on maturing fruit in trees are less important to the secondary spread of fruit brown rot under the semi-arid weather conditions, such as that of California, than in more humid climates, such as those of Canada (1) and South Carolina (22).

Dry weather conditions also limit some of the other sources of the secondary inoculum for fruit brown rot. Over the last 2 years, we observed that most aborted fruit

dropped from trees before thinning. Non-abscised, aborted fruit were either very rarely observed in trees or mostly dried up following thinning, because of low humidity and poor rainfall during the seasons (C. X. Hong and T. J. Michailides, unpublished data). This dry weather also prevented sporulation of *M. fructicola* on blighted flowers. Therefore, non-abscised, aborted fruit and blossoms blighted by *M. fructicola* are unlikely to be important sources of the secondary inoculum for fruit brown rot in California orchards. This situation also differs from observations made in more humid areas (1,22).

Extensive and intensive sporulation of *M. fructicola* on thinned fruit was observed in orchards both in South Carolina and Canada, so that thinned fruit was considered an important source of secondary inoculum in those areas (1,22). Our study further quantified the importance of this inoculum source. Pre- and postharvest fruit brown rot was significantly reduced by completely removing the thinned fruit from 1,600 to 2,300 m² plots in all five orchards in 1995 and 1996. The importance of thinned fruit as a source of the secondary inoculum might have been reduced by applying fungicides in orchards 951, 952 and 953, and by covering part of thinned fruit with reflective plastic film beginning in early June in orchards 961 and 962. Even under these circumstances, removal of thinned fruit resulted in significant reduction of brown rot at harvest (Figs. 3 and 4). Conidia of *M. fructicola* are airborne (14,17,21) and also can be transported by various vectors (9,21,25,27,36). Consequently, some interplot interference occurred due to airborne conidia that traveled across the plot borders from plots A2 to A1, and from neighboring orchards. Therefore, the significance of thinned fruit as a source of secondary inoculum for fruit brown rot may have been greater if fungicides had not been applied and the experimental plot had been larger in size.

Dry weather conditions suppress the sporulation of *M. fructicola* on infected tissue in trees in California orchards. These dry weather conditions, however, require growers to irrigate their orchards for maximum fruit production. Regular irrigation provides an ideal microclimate for infection by and sporulation of *M. fructicola* to occur on thinned fruit, and expedites the dispersal of conidia from sporulating thinned fruit to those that had been uninfected. The fact that more fruit initially sporulated in the irrigation trenches than on the dry berms provides direct evidence that irrigation water enhances sporulation of *M. fructicola* on thinned fruit. This fact also indirectly supports our observations that the sporulation was very rarely seen on non-abscised aborted fruit and blighted blossoms in trees during fruit maturing.

Irrigation water also disturbs insect

Fig. 4. Pre- and postharvest brown rot by *Monilinia fructicola* on nectarine fruit from A1 plots, from which thinned fruit were completely removed, from A2 plots, from which thinned fruit were not removed and were supplemented by an equal amount of thinned fruit obtained from A1 plots, and from A3 plots, in which thinned fruit were left undisturbed, in orchard 961 (cv. Summer Grand), Parlier, and orchard 962 (cv. Fantasia), Easton, California, 1996. Arrows indicate the dates of fruit harvest for determining postharvest diseases. Only bars to the right of the arrow represent postharvest assessment (in the lab). Bars topped by different letters within a date differed significantly according to Fisher's least significant difference test at $P = 0.05$.

vectors and drives them away from the sporulating thinned fruit in the irrigation trenches (18,34). Some of these insects (nitidulid beetles, thrips, and vineyard flies) are not only able to transport the conidia from infected thinned fruit to the surface of healthy fruit on trees, but also able to facilitate infection of fruit by creating wounds (34). The latter situation is particularly important to the fruit rot phase of brown rot because it increases fruit susceptibility (38), and is extremely important to the epidemiology of brown rot under dry weather conditions (18). The high efficiency of dispersal and subsequent infection by the conidia of *M. fructicola* produced on thinned fruit might also have contributed to the importance of this substrate as a major secondary inoculum supply in comparison with other possible sources for fruit brown rot epidemics in California orchards.

The high incidence of thinned fruit with sporulation of *M. fructicola* and their high density per tree further signify their importance as a major source of inoculum. In addition, preliminary experiments to quantify sporulation on sampled sporulating thinned fruit indicated that a minimum of 3.5 to 11.7 million conidia of *M. fructicola* were produced per fruit, although the pro-

ductivity of individual thinned fruit might differ from one another, depending on the concentration of glucose and temperature conditions (6,7,23,24,31–33).

Our results suggest that a reduction of fruit brown rot can be achieved by removing thinned fruit from orchards immediately after thinning. Unfortunately, removing thinned fruit is a very time-consuming and costly process. It took approximately 100 to 160 man-hours of labor per hectare to remove the thinned fruit from plot A1 and place it in A2, depending largely on the density of thinned fruit on the orchard floor. An alternative to this procedure could be spraying chemicals (22) or biological agents to suppress sporulation of *M. fructicola* on or to expedite the decomposition of thinned fruit through cultural practices, such as disking or rototilling. Thinning of excess fruit prior to pit-hardening can reduce the infection of thinned fruit by conidia of *M. fructicola* (1,22). This practice also can be an alternative to amend the source of inoculum, provided that it is justified epidemiologically and economically. The applicability of our results to orchards of other stone fruit species, and possible ways of destroying thinned fruit in orchards are currently under investigation.

Fig. 5. The relationship between preharvest fruit brown rot caused by *Monilinia fructicola* and thinned fruit in four experiment orchards of different cultivars of the same maturity (early to middle July), 1995. Disease incidence data from A2 plots in orchards 951 and 953 and B2 plots in orchards 954 and 955 were pooled. The density of thinned fruit in the A2 plots was calculated by doubling the orchard average numbers of thinned fruit per tree in the same orchards, since A2 plots received equal amounts of thinned fruit from A1 plots.

ACKNOWLEDGMENTS

This research was supported in part by a grant from the University of California Integrated Pest Management Project, the California Tree Fruit Agreement, and California Prune Board. We thank G. Bergen in Parlier, Surabian Packing in Reedley, and H. A. Kaprielian in Easton for allowing us to conduct the experiments in their orchards. Our thanks extend to D. Rice for sharing the experimental orchard and to J. Chevalier for the support in the field experiments.

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