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TITLE: Effect of fungicide treatment timing on postharvest decay of Anjou pear fruit

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Cooperators: Blue Star Growers, Inc.
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ACCOMPLISHMENTS:

Procedures

Three single replicate bins of Anjou pear fruit from each of two growers were treated with Mertect 340-F (TBZ, 16 oz/100 gal) applied with a chain driven cascading drencher. In 1997, the fungicide was applied either at 0 wk (at harvest), 1 wk, 2 wk, 4 wk, or 5 wk after harvest. In 1998, TBZ was applied at 0 wk, 1 wk, 2 wk, 3 wk, and 4 wk after harvest. In each year, an additional 6 bins of fruit, three from each grower lot, were left untreated as controls (UTC). All treatments were made prior to the appearance of visible symptoms of gray mold.

All bins of fruit were received at the Research Controlled Atmosphere (RCA) facility at Stemilt Growers, Inc., Wenatchee, WA, and immediately placed in cold storage at 31 C and standard CA conditions (1.5% O_2, < 1.0% CO_2) were imposed within 24 hr. At each treatment time, rooms were aired, bins of fruit were removed and treated, then replaced in the room and CA reimposed. In 1997, fruit were stored at the RCA for 4 mo at which time one grower lot was assessed and the other was placed in a commercial storage in regular atmosphere for an additional 1.5 mo. In 1998, fruit were stored for about 5 mo at the RCA. Decay was assessed by removing all fruit with symptoms from each bin and the type of decay (e.g., gray mold, blue mold, etc.) determined.

In each year, decay data were combined and blocked by grower for analysis. In 1998, incidence of cork was high throughout the growing district, therefore, cork incidence was used as a covariate in all analyses. Data were transformed ($\log_e + 1$) before analysis of variance. Means were separated using Fisher's protected LSD ($P = 0.05$). Means presented are original, untransformed, values.

Results and Discussion

In 1997, incidence of decay in fruit from grower 2 was considerably higher than that in fruit from grower 1 and accounts for the high decay incidence observed in that year (Table 1). Much of the fruit was small and light colored and appeared to have a smoother, thinner skin than normal Anjou fruit. In addition, it appears likely that during the additional 10 wk of storage at regular atmosphere fruit became sufficiently senescent that it became highly susceptible to infection by *B. cinerea*. Much of the fruit was soft and becoming yellow at the time the trial was assessed. Decay control probably would have been better with more rigorous storage conditions. In 1998, fruit was more consistent, both between and within grower lots, but incidence of cork

was relatively high compared to most years and was a significant covariate in analysis of gray mold and total decay incidence.

In untreated fruit, the largest single loss factor was gray mold, which accounted for about 73% of decayed fruit in 1997 and 87% of decayed fruit in 1998. Losses due to blue mold (caused by *Penicillium expansum* and *P. solitum*) were slight (<1% of decay) in untreated fruit in 1997, but accounted for about 5% of decay in 1998. However, about 25% of decay was due to other fungal diseases in 1997, especially bull's-eye rot (caused by *Pezicula malacorticis*) and a disease with symptoms similar to Coprinus rot or fisheye rot (caused by *Coprinus psycromorbidus* and *Corticium centrifugum*, respectively) and associated with an unidentified basidiomycete. The majority of losses by these other fungi were in fruit from grower 2. In 1998, losses from these fungi accounted for only about 3% of decay in untreated fruit.

Gray mold. Postharvest treatment of fruit with TBZ up to 5 wk after harvest significantly reduced the total incidence of decay in both years (Table 1). In 1997, gray mold incidence in fruit treated at harvest was reduced by 95% of that which occurred in the UTC. The incidence of gray mold increased with time of treatment after harvest up to 4 wk after harvest. Numbers of fruit decayed following treatment either 4 wk or 5 wk after harvest were not significantly different and decay was reduced by only about 50% (42% and 57 % at 4 wk and 5 wk, respectively) of that which occurred in the UTC.

A similar pattern was observed in 1988, but the extent of differences between treatment at harvest and subsequent treatments was not as great as occurred in 1997. Gray mold incidence in fruit treated at harvest was reduced by only 76% of that which occurred in the UTC. By 2 wk after harvest, decay control was about 50% of that in the UTC treatment (Table 1) and there was no significant difference in numbers of fruit with gray mold in bins of fruit treated either 2 wk, 3 wk, or 4 wk after harvest.

Although these observations are indirect, it appears likely that TBZ treatments made in the first or second week after harvest prevented primary infections. Likewise, those treatments made about 3 wk after harvest probably were not effective against primary infections and served as prophylactic treatments against secondary infections. In both years, the later fungicide applications reduced gray mold incidence to about 50% of that in the UTCs. This may indicate the proportion of fruit that can be expected to develop secondary gray mold infections from fruit to fruit spread.

There are several possibilities that would explain the development of the infections observed following the treatments at harvest. Infections could have occurred either preharvest (in the field), or postharvest due to failure to get complete coverage of fungicide especially in the calyx end.

Blue mold. None of the fungicide treatments had an effect on the incidence of blue mold in either year (Table 1). Resistance to benzimidazole fungicides by species of *Penicillium* is widespread. The sensitivity of the strains of *Penicillium* that grew in fruit in these experiments was not tested. Therefore, it is not possible to determine when infections might have initiated. The relatively high numbers of fruit infected with blue mold following the 0 wk and 1 wk treatments in 1988 may be due to contamination of the drencher used in this study, which was used for commercial hydro-cooling of cherries.

Other. Although treatment of fruit with TBZ at each timing significantly reduced the incidence of decay from other fungi compared to the UTC in 1997, the treatment at harvest (0 wk) resulted in the greatest reduction (Table 1). Differences among treatments applied after that

initial one (i.e., 1-5 wk) were not significant. Unlike that observed in the case of gray mold, disease incidence did not sharply increase after a period of time following harvest. Rather disease incidence appeared to plateau at about 27% to 37% of that in the untreated control. It would appear, therefore that application of fungicide did prevent fruit to fruit spread of these fungi, in particular those that caused Coprinus rot or fisheye rot-like symptoms.

Total decay. In both years, the majority of decay was caused by gray mold and the effects of postharvest treatments on total decay mirrored those on gray mold. Postharvest treatment of fruit with TBZ up to 5 wk after harvest significantly reduced the total incidence of decay in both years (Table 1). In 1997, decay incidence in fruit treated at harvest and 1 wk after harvest was reduced by almost 90% from that which occurred in the UTC. Although decay incidence in fruit treated 2 wk after harvest was about twice that which occurred in fruit treated at 0 wk and 1 wk after harvest, the level of control was not significantly different from the treatment at 1 wk. Significantly less decay control was achieved at 4 and 5 wk after harvest than in the earlier treatments. In 1998, total decay incidence was relatively high in 0 and 1 wk treatments, largely because of the high numbers of fruit with blue mold and the effect of treatment timing on the total amount of decay was not as great as in 1997.

Conclusions

A large proportion of primary infections (75-90%) by *B. cinerea* appear to occur in the first two weeks after harvest. These infections are likely initiated by inoculum deposited on fruit at or before harvest. Primary infections can be controlled with fungicides in the first two weeks after harvest. Only secondary infections will be controlled after that. Secondary infections may double the number of fruit lost to gray mold. Other postharvest pathogens, *P. malicorticis* in particular, may infect fruit at harvest and those infections can be effectively controlled by postharvest applications of fungicides.

PUBLICATIONS:

Sanderson, P. G. and Bennett, D. L. 1999. Effect of treatment timing on postharvest decay of Anjou pear fruit. Proc. Wash. State Hortic. Assoc. 94:(in press).

Table 1. Effect of fungicide treatment timing on number of decayed fruit recovered from bins of Anjou pear fruit that were stored in controlled atmosphere storage

Treatment timing (wk)	Gray mold	Blue mold	Other[x]	Total
1997				
0	9.7 a[y]	3.8 n.s[z]	16.3 a	30.0 a
1	13.5 ab	2.3	19.5 ab	35.5 ab
2	36.7 b	1.0	26.0 ab	63.8 b
4	122.5 c	1.3	34.3 b	158.7 c
5	90.2 c	4.0	23.8 b	118.0 c
Untreated	211.2 d	2.8	73.2 c	287.7 d
	$P < 0.001$	$P = 0.298$	$P = 0.001$	$P < 0.001$
1998				
0	17.8 a	11.0 n.s.	0.3 n.s.	33.2 a
1	22.3 ab	15.3	0.7	43.8 a
2	36.3 bc	3.2	0.5	42.3 a
3	36.2 c	4.3	1.3	48.3 a
4	33.8 c	6.0	0.5	43.8 a
Untreated	75.2 d	4.0	2.2	86.2 b
	$P < 0.001$	$P = 0.052$	$P = 0.147$	$P = 0.018$

[x] Category includes bull's-eye rot and Coprinus rot.

[y] Data were transformed to $\log_e$ values before analysis of variance. Means presented are original data. Means in each column for each year followed by a common letter are not significantly different according to Fisher's protected LSD ($P = 0.05$).

[z] n.s. = F statistic not significant ($P \geq 0.05$).