CONTROL OF STORAGE ROT BY INDUCTION OF PLANT DEFENSE MECHANISMS USING JASMONIC ACID AND SALICYLIC ACID

Karen Klotz Fugate[1], Jocleita Peruzzo Ferrareze[2], and Melvin D. Bolton[1]

[1]USDA-ARS, Northern Crop Science Laboratory, Fargo, ND
[2]Department of Plant Biology, Federal University of Viçosa, Viçosa, MG, Brazil

Storage rots contribute to sugarbeet postharvest losses by consuming sucrose and producing carbohydrate impurities that increase sugar loss to molasses. Storage rots also increase root respiration, a process that degrades sucrose and generates heat, contributing to pile warming. As piles warm, the incidence and severity of storage rots increase, and the respiration of both healthy and rotted roots increase. Storage rots, therefore, increase storage and processing losses, initiate events that escalate the rate of storage loss, and if sufficiently severe, can cause rapid deterioration of storage piles (Campbell and Klotz, 2006).

Storage rots are currently controlled by pile management techniques. Since low temperatures reduce the growth rate of many rot-causing organisms, cooling piles and timely removal of 'hotspots' in piles reduces loss to storage rots. Pile management techniques, however, require favorable weather conditions for storage, continuous monitoring of piles, and are limitedly effective in controlling those rot-causing fungi that are capable of growth at low temperatures. Although genetic resistance and chemical fungicides can be used to reduce storage rot losses (Miles et al., 1977; Bugbee and Cole, 1979), neither control mechanism is utilized since introduction of additional traits into breeding programs slows progress toward other desirable characteristics and fungicides generally have deleterious effects on sugarbeet root storage properties in the absence of disease (Akeson et al., 1979; Campbell, 2005). Application of jasmonic acid (JA), salicylic acid (SA), or derivatives or analogs of JA and SA has been shown to be effective in reducing storage diseases in other crops (Tripathi and Dubey, 2004; Asghari and Aghdam, 2010). These compounds are endogenous plant hormones that induce a plant's native defense mechanisms and have been shown to protect against a wide variety of pathogens. The ability of JA and SA to control storage rot pathogens common to sugarbeet roots, however, has not been investigated.

Objective

Research was conducted to determine the feasibility of treating sugarbeets with JA or SA to induce plant defense mechanisms and reduce the incidence and severity of storage rots. Both JA and SA were used in these studies since the two compounds induce different sets of plant defense mechanisms and are expected to differ in their ability to control rot-causing organisms. Initial research determined the effectiveness of postharvest treatments in controlling storage rots. Current research investigates the efficacy of preharvest treatments for storage rot control.

Materials and methods

Plants of sugarbeet hybrid VDH66156 were greenhouse grown using supplemental light. Plants were watered as needed for all JA experiments and for SA experiments identified as well-watered. For SA experiments where a water-stress was applied, plants were watered as needed for the first 5 weeks after planting then severely water stressed for the subsequent 11–12 weeks by allowing plants to wilt prior to rewatering. For all experiments, roots were harvested 16 – 18 weeks after planting, all leaf and petiole tissue was removed, and roots were gently washed to remove adhering soil. Postharvest JA and SA treatments were administered by submerging roots for 1 h at room temperature in 0, 0.01, 0.1, 1, 10 or 100 μM JA or 0, 0.01, 0.1, 1, or 10 mM SA, using at least 6 roots per treatment. Preharvest JA treatments were made by spraying foliage to runoff with 0, 0.01, or 10 μM JA 7 d prior to harvest. For postharvest treatments, roots were stored 3 d at 20°C and 90% relative humidity prior to inoculation with *Botrytis cinerea*, *Penicillium claviforme*, or *Phoma betae*. For preharvest treatments, roots were inoculated on the day of harvest. Prior to inoculation, fungi were cultured on potato dextrose agar (PDA) plates by placing mycelia at the center of the plate and incubating at 25°C until fungal growth covered the plate. Roots were inoculated by drilling two holes, approximately 12 mm in diameter and 10 mm deep, into opposite sides of the root at the widest portion of the root and inserting a 10 mm diameter plug of fungal-covered PDA into each hole. Inoculated roots were stored at 20°C and 90% relative humidity until severe disease symptoms were evident on control roots. Root

rot severity was assessed by removing and weighing the rotted tissue from each infection site. Each experiment was repeated at least once.

Results

Postharvest JA treatments reduced rot due to *Botrytis cinerea*, *Penicillium claviforme*, and *Phoma betae* (Table 1). JA treatments of 0.01 to 100μM reduced rot due to *B. cinerea* by 36 – 62%. All JA treatments reduced rot to a statistically similar extent, and on average, JA treatment reduced rot due to *B. cinerea* by 51%. JA treatments of 0.01 to 100 μM reduced rot due to *P. claviforme* by 34 – 65%. JA concentrations of 0.01 to 10 μM provided statistically similar protection against *P. claviforme* and reduced the weight of rotted tissue by an average of 44%. Additional protection against *P. claviforme* was provided by an increase in JA concentration to 100 μM, which reduced the weight of rotted tissue by 65%. JA treatments of 0.01 to 100μM also reduced rot due to *P. betae*. JA treatments reduced the weight of rotted tissue due to *P. betae* by 58 – 81%, although all concentrations of JA provided statistically similar protection. On average, the weight of rotted tissue due to *P. betae* was reduced by 71% by JA treatment. Research is fully described in a recent publication (Fugate et al., 2012).

Table 1. Relative weight of rotted tissue in roots after postharvest jasmonic acid (JA) treatment and inoculation with *Botrytis cinerea*, *Penicillium claviforme*, or *Phoma betae*.

JA concentration	rotted tissue (relative weight)[1]		
(μM)	*Botrytis cinerea*	*Penicillium claviforme*	*Phoma betae*
0	1.00 a[2]	1.00 a	1.00 a
0.01	0.46 b	0.54 b	0.19 b
0.1	0.52 b	0.55 b	0.28 b
1	0.38 b	0.66 b	0.34 b
10	0.44 b	0.50 bc	0.21 b
100	0.64 b	0.35 c	0.42 b

[1]Weight of rotted tissue as a fraction of the weight of rotted tissue of the control
[2]Treatments with different letters within a column are statistically different (Fisher's LSD; $\alpha = 0.05$)

Postharvest SA treatments reduced rot due to *B. cinerea*, *P. claviforme*, and *P. betae* in roots of water-stressed plants, but had no effect on rot severity in roots from unstressed plants (Table 2). When plants were well watered, SA treatments of 0.01 to 10 mM had no effect on the weight of rotted tissue in roots inoculated with *B. cinerea*, *P. claviforme*, or *P. betae* relative to water-treated controls. However, when roots were harvested from plants that were severely water stressed prior to harvest, SA treatments of 0.01 to 10 mM reduced rot due to *B. cinerea* by 49 – 58%, due to *P. claviforme* by 30 – 53%, and due to *P. betae* by 57 – 79%. Statistically, all SA treatments provided similar reductions in rot for the three disease-causing organisms used to inoculate roots. On average, SA reduced rot due to *B. cinerea*, *P. claviforme*, and *P. betae* by 54, 45, and 67%.

Table 2. Relative weight of rotted tissue in roots of well-watered (unstressed) or water-stressed (stressed) plants after postharvest salicylic acid (SA) treatment and inoculation with *Botrytis cinerea*, *Penicillium claviforme*, or *Phoma betae*.

SA concentration	rotted tissue (relative weight)[1]					
	Botrytis cinerea		*Penicillium claviforme*		*Phoma betae*[3]	
(mM)	unstressed	stressed	unstressed	stressed	unstressed	stressed
0	1.00 a[2]	1.00 a	1.00 a	1.00 a	1.00 a	1.00 a
0.01	1.02 a	0.46 b	0.92 a	0.49 b	0.91 a	0.27 b
0.1	1.16 a	0.51 b	0.89 a	0.70 b	1.18 a	0.21 b
1	0.97 a	0.44 b	0.97 a	0.51 b	0.71 a	0.43 b
10	0.96 a	0.42 b	1.21 a	0.47 b	0.79 a	0.42 b

[1]Weight of rotted tissue as a fraction of the weight of rotted tissue of the control
[2]Treatments with different letters within a column are statistically different ($\alpha = 0.05$)
[3]Preliminary data.

Foliar JA treatments applied 7 days before harvest had variable effects on the severity of disease symptoms in roots inoculated with *B. cinerea*, *P. claviforme*, and *P. betae*. At a concentration of 0.01 µM, JA reduced rot in roots inoculated with *B. cinerea* and *P. betae* by 55 and 65%, respectively, but had no effect on rot due to *P. claviforme*. At a concentration of 10 µM, JA reduced rot due to *P. betae* by 33%, but had no statistically significant effect on rot due to *B. cinerea* or *P. claviforme*.

Table 3. Relative weight of rotted tissue in roots of plants that received a 7 day preharvest jasmonic acid (JA) treatment and were inoculated with *Botrytis cinerea*, *Penicillium claviforme*, or *Phoma betae* after harvest.

JA concentration	rotted tissue (relative weight)[1]		
(µM)	*Botrytis cinerea*	*Penicillium claviforme*	*Phoma betae*
0	1.00 a[2]	1.00 a	1.00 a
0.01	0.45 b	0.94 a	0.35 b
10	0.62 ab	1.15 a	0.67 b

[1]Weight of rotted tissue as a fraction of the weight of rotted tissue of the control
[2]Treatments with different letters within a column are statistically different ($\alpha = 0.05$)

Conclusions

- Jasmonic acid, applied as a postharvest treatment at concentrations of 0.01 – 100 µM, reduced rot due to *Botrytis cinerea*, *Penicillium claviforme* and *Phoma betae* by an average of 51, 44, and 71%, respectively.

- JA reduced storage rot at extremely low concentrations. A postharvest JA treatment of 0.01 µM reduced rot due to *B. cinerea*, *P. claviforme*, and *P. betae* by 54, 46, and 81%, respectively. This same concentration of JA, applied to foliage 7 days prior to harvest, reduced storage rot due to *B. cinerea* and *P. betae* by 55 and 65%, but had no effect on rot due to *P. claviforme*.

- Salicylic acid, applied as a postharvest treatment at concentrations of 0.01 – 10 mM, had no effect on root rot due to *B. cinerea*, *P. claviforme*, and *P. betae* when roots were harvested from unstressed plants.

- When roots were harvested from plants that were severely water stressed prior to harvest, postharvest SA treatment at concentrations of 0.01 – 10 mM reduced rot due to *B. cinerea*, *P. claviforme* and *P. betae* by an average of 54, 45, and 67%, respectively.

References

Akeson, W.R., Yun, Y.M., Sullivan, E.F., 1979. Effect of chemicals on sucrose loss in sugarbeets during storage. J. Am. Soc. Sugar Beet Technol., 20, 255-268.

Asghari, M., Aghdam, M.S., 2010. Impact of salicylic acid on post-harvest physiology of horticultural crops. Trends Food Sci. Technol., 21, 502-509.

Bugbee, W.M., Cole, D.F., 1979. Comparison of thiabendazole and genetic resistance for control of sugar beet storage rot. Phytopathology 69, 1230-1232.

Campbell, L.G., 2005. Post-harvest storage traits. In: Biancardi, E., Campbell, L.G., De Biaggi, M., Skaracis, G.N. (eds.) Sugar Beet Breeding and Genetics. Science Publishers, Inc, Enfield, NH. pp. 122-126.

Campbell, L.G., Klotz, K.L., 2006. Storage. In: Draycott, A.P. (ed.) Sugar Beet. Blackwell Publishing, Oxford, UK. pp. 387-408.

Fugate, K.K., Ferrareze, J.P., Bolton, M.D., Deckard, E.L., Campbell, L.G., 2012. Postharvest jasmonic acid treatment of sugarbeet roots reduces rot due to *Botrytis cinerea*, *Penicillium claviforme*, and *Phoma betae*. Postharvest Biol. Technol. 65, 1-4.

Miles, W.G., Shaker, F.M., Nielson, A.K., Ames, R.R., 1977. A laboratory study of the ability of fungicides to control beet rotting fungi. J. Am. Soc. Sugar Beet Technol. 19, 288-293.

Tripathi, P., Dubey, N.K., 2004. Exploitation of natural products as an alternative strategy to control postharvest fungal rotting of fruit and vegetables. Postharvest Biol. Technol. 32, 235-245.