Activating Mango Fruit Defence to Anthracnose Disease

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INTRODUCTION

The fungus causing anthracnose disease in mango, *Colletotrichum gloeosporioides, (C.g.)*, infects immature fruit early in the season, then enters a long latent phase. After harvest, when fruit start to ripen, the latency breaks and the fungus ramifies through the peel and pulp tissues causing black disease lesions. The breaking of pathogen latency in ripening mango fruit has been correlated with decreasing concentrations of the endogenous antifungal resorcinol compounds (Droby et al., 1986). The level of these antifungal resorcinols vary among mango cultivars (Droby et al., 1986). Controlling diseases by managing natural resistance of fruit to fungal attack could minimize the use of pesticides, which have become of major public concern on health and environmental grounds.

The plant resistance activator benzo(1,2,3)thiadiazole-7-carbothioic acid S-methyl ester (trade name Bion®) has been widely reported as an effective inducer of systemic resistance. For example, Bion® was reported to induce pathogenesis-related proteins (PR proteins) and stimulate plant defence in peas (Dann and Deverall, 2000) and roses (Suo and Leung, 2001). However, until now, there is no information about the role of Bion® in activation of mango (cv. Kensington Pride) fruit resistance to anthracnose disease. The aim of this research is to determine the effect of resistance activators on defence responses of mango fruit to anthracnose disease.

METHODS

Fruit of the mango cv. 'Kensington Pride' from a commercial orchard near Gatton (Long. =152°17', Lat. =27°34') Queensland, were used in this experiment. The fruit were treated on the trees with the plant activator Bion® (100 mg a.i./L) or *C. g.* (10^5 spores/mL) or water as a control treatment. A wetting agent, Tween 20 (0.01 % v/v) was added to the Bion®, fungus and water solutions. After dipping in the *C.g.* spore suspension, the fruit were covered with a humid plastic bag and a brown paper bag to optimize conditions for spore germination and infection. After 48 h incubation, the bags were removed. The fruit were harvested 7 days after treatment. Harvested fruit were divided into two lots, one lot for the antifungal activity assay and another lot for the disease and ripening assessments. For the antifungal assay, skin of fruit samples were cut into small parts and then stored at -80°C until ready for extraction. The solvent extraction, thin layer chromatography (TLC) and plate bioassay were carried out following the methods of Droby et al. (1996) and Zainuri et al. (2001) with modification. For the disease and ripening samples, the fruit were either challenge-inoculated with the anthracnose pathogen by applying a droplet of *C.g.* spore suspension (10^6 spores/mL) or water to the fruit surface. The fruit were then incubated in humid plastic trays for 48 h at 25°C. Thereafter, the mango fruit were allowed to ripen at 22-23°C and 80 % relative humidity for approximately 14 days. Disease severity was recorded as mm lesion diameter for the postharvest challenge-inoculated fruit. Disease severity caused by natural field infection was also assessed, as % fruit surface area affected by anthracnose lesions. Disease incidence representing the percentage of infected fruit in the samples for each treatment was also assessed. In addition, fruit physiology including visual skin colour and hand firmness ratings (Shorter and Joyce, 1998) was assessed daily.

RESULTS AND DISCUSSION

The results indicate that both Bion® and field inoculation with *C.g.* were able to delay postharvest disease progression significantly up to the eating-ripe-stage. Fewer Bion®-treated fruit were covered by disease lesions compared with the control (water-treated) fruit. However, after reaching the eating-ripe-stage, disease lesions had appeared on all fruit. Similarly, Bion® was also able to reduce fungal growth due to the natural infections. The disease lesions did not appear until day 11 after harvest. Bion® may have been able to trigger the defence mechanism in the fruit. The fact that each fruit started to get diseased once they ripened could be related to the decrease in the concentration of preformed antifungal compounds as the fruit ripen. Although not yet quantitatively assessed, our results show that the level of antifungal compounds, indicated by the area of fungal growth inhibition on the TLC plates sprayed with the assay organism *Cladosporium sp.*, was higher in the fruit sampled 7 days before harvest compared to those fruit sampled 3 days before harvest. This result supports the findings of previous studies (Droby et al., 1986) that green mature fruit have higher levels of the endogenous antifungal compounds compared to ripe fruit. On the other hand, the fruit treated preharvest with *C.g.* and not subsequently challenged with the pathogen, were more severely covered by disease lesions compared to the the Bion®-treated and control fruit. In addition, both *C.g.* and Bion® did not significantly affect fruit skin colour or firmness, and all of the fruit reached the eating ripe stage at the same time.

CONCLUSION

There was an indication that Bion® treatment, in the absence of postharvest challenge inoculation with the anthracnose pathogen *C. gloeosporioides*, stimulated the fruit natural defence mechanism and reduced anthracnose disease severity. Future research will repeat and extend these preliminary results.

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REFERENCES

Dann, E.K., and B.J. Deverall, Activation of systemic disease resistance in pea by an avirulent bacterium or a benzothiadiazole, but not by a fungal leaf spot pathogen, *Plant Pathology, 49,* 324-332.

Droby, S., D. Prusky, B. Jacoby, and A. Goldman, Presence of antifungal compounds in the peel of mango fruits and their relation to latent infections of *Alternaria alternata, Physiological and Molecular Plant Pathology, 29,* 173-183, 1986.

Shorter, A.J., and D.C. Joyce, Effect of partial pressure infiltration of calcium into 'Kensington' mango fruit, *Australian Journal of Experimental Agriculture, 38,* 287-294, 1998.

Suo, Y., and W.M. Leung, Elevation of extracellular β-1,3-glucanase and chitinase activities in rose in response to treatment with acibenzolar-s-methyl and infection by *D. rosae, Journal of Plant Physiology, 158,* 971-976, 2001.

Zainuri, D.C. Joyce, A.H. Wearing, L.M. Coates, and L. Terry, Effects of phosphonate and salicylic acid treatments on anthracnose disease development and ripening of 'Kensington Pride' mango fruit, *Australian Journal of Experimental Agriculture, 41,* 805-813, 2001.