STEM-END ROTS : INFECTION OF RIPENING FRUIT

K.R. EVERETT

The Horticulture and Food Research Institute of New Zealand Ltd. Private Bag 92169, Mt Albert, Auckland

ABSTRACT

Fruit from an unsprayed orchard were harvested at a maturity of >30% dry matter, and placed 10 per box in a room at 20^{o}C. Fifty fruit per day were cut, assessed for rots and all fruit were tested daily for ripeness. When most fruit were ripe, an additional 500 fruit were cut in one day, tested for ripeness by hand and assessed for rots. Stem-end rots were only found in fruit that had reached or passed optimal ripeness. The number of unripe fruit observed with a slight browning at the stem-end was similar to the number of optimally ripe fruit with stem-end rots. However, no fungi were isolated from these symptoms on unripe fruit. It was concluded that because only ripe fruit developed stem-end rots in this study, the hypothesis that stem-end rots are caused by endophytic populations of fungi growing down the stalk to infect fruit is not supported. However, it is possible that host defence mechanisms other than antifungal dienes precluded fungal invasion, or that the dienes may be available in fungitoxic amounts in flesh of 'Hass' fruit in New Zealand.

Keywords: fruit firmness, ripeness, endophytic infection

INTRODUCTION

Johnson et al. (1991, 1992) has suggested that postharvest rots in mango are both seed transmitted and endophytic[1] within the plant. Infection of the stem-end of fruit is via the endophytically infected peduncle[2]. Stem-end rots may also infect avocado trees endophytically (Johnson, unpublished data). Overseas researchers (Prusky et al. 1982) have shown that antifungal dienes are present in the skin of avocados in fungitoxic amounts and prevent infection of immature avocados when the skin is intact. However, these workers also showed that, in the flesh, antifungal dienes were compartmentalised into oil cells and were therefore only available in subfungitoxic levels. Once the skin was breached by peeling, the pathogen (*Colletotrichum gloeosporioides*) was able to rot unripe flesh (Kobiler et al. 1993). If stem-end rots infect avocados by invasion from fungal mycelium[3] growing down the peduncle, then unripe avocado fruit should be able to be infected as there is no skin barrier to invasion. Infection of the stem-end in unripe fruit would provide evidence to support this hypothesis. The following experiments were designed to test this hypothesis to further elucidate the infection mechanism by stem-end rots and therefore enable effective and focussed control strategies to be developed.

Keywords: fruit firmness, ripeness, endophytic infection

[1] Growing in avocado tissue without producing symptoms
[2] Fruit stalk
[3] A mass of fungal strands

METHODS

Fruit from an unsprayed orchard was harvested, placed into boxes (10 per box) and then transported to the Horticulture and Food Research Institute laboratories at Mt Albert Research Centre to a room at 20^{o}C. Fifty fruit per day for 14 days were cut and assessed for stem-end rots using a 0-10 visual rating scale for severity where 0 = no disease and 10 = totally rotten. Fruit were tested for ripeness by hand using a 1-10 scale where 1= hard, 5=optimally ripe and 10= soft, and hand ripeness calibrated with firmometer ripeness (White et al. 1998). When most fruit were ripe (day 10), 500 fruit were cut in one day, each of these fruit tested for ripeness by hand and by firmometer, and assessed for stem-end rots.

RESULTS

Disease progress curves

Cumulative mean severity.

When mean severity was plotted against both ripeness (Fig. 1), and time (Fig. 2), disease increased exponentially. In other words, there was a constantly increasing degree of disease in fruit as ripeness or time increased. Cutting fruit every day indicated that fruit ripened following a sigmoidal pattern. A sigmoidal increase is 'S' shaped, there is an initial rapid increase followed by a tail off to a maximum (in this case a score of 10). When mean severity and ripeness are plotted on the same graph, this indicates that disease started to increase very rapidly after the optimum ripeness value was reached (Fig. 3).

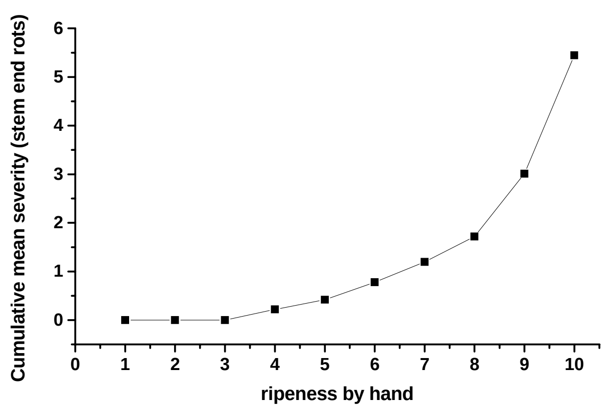

Figure 1: The relationship between ripeness values and cumulative mean severity of stem-end rots. Fruit cut in one day.

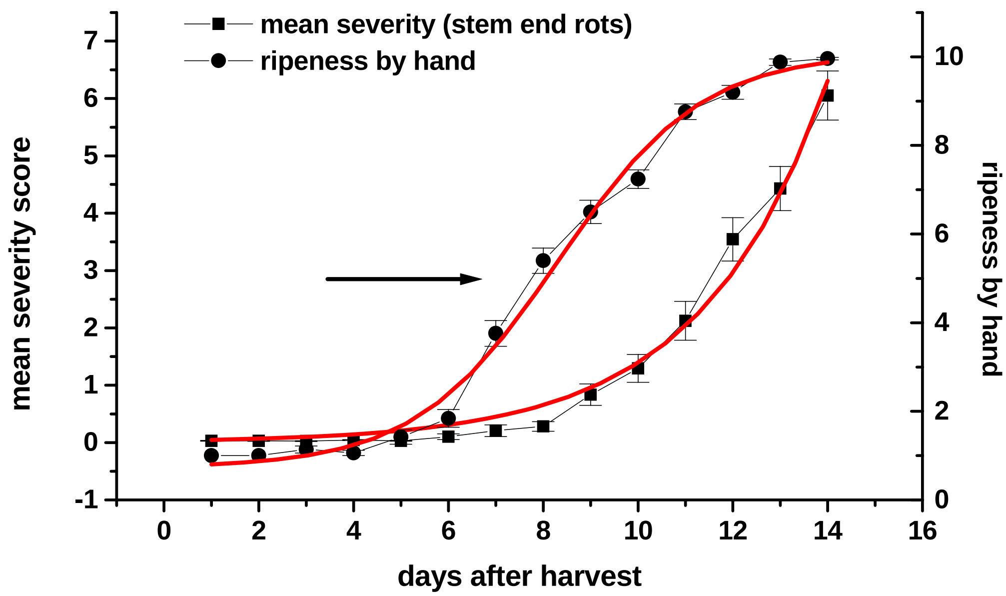

Figure 2: Change in mean severity of stem-end rots and ripeness as determined by hand in time (days) after harvest. Separate samples of fruit were cut each day. Arrow indicates optimal ripeness of 5.

Proportion of fruit diseased.

Both for fruit cut in one day and for fruit cut each day for 16 days, when proportion of fruit with disease was plotted against ripeness values, the numbers of fruit with disease initially increased in a sigmoidal ('S'-shaped) manner. However, after a ripeness of 7 or 8, the increase appears to remain linear until 100% of fruit are diseased (Fig. 3). It seems that at this point the fruit became senescent and lost any resistance to invasion by the rot fungi. When the initial sigmoid shaped first part of this disease progress curve was logit transformed (Fig. 4), the formula for the fitted straight line can be used to calculate the ripeness value at which any proportion of the fruit used in these experiments was diseased i.e. 10% of fruit would be diseased at a ripeness value of 4.54, 20% at a ripeness value of 4.97, 30% at a ripeness value of 5.33. When ripeness was 5.75, the number of diseased fruit rose to 40%.

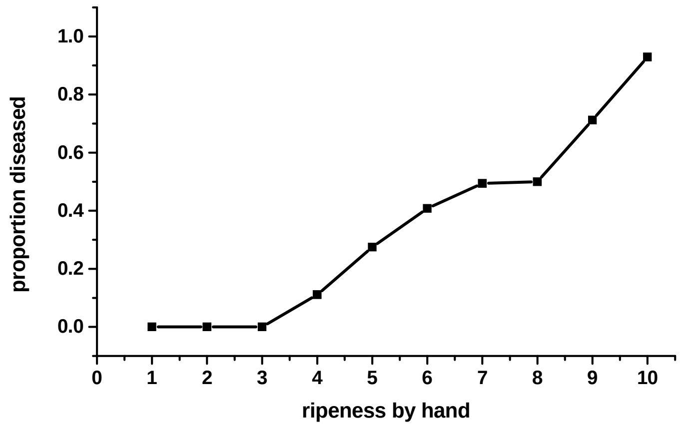

Figure 3: Cumulative numbers of diseased fruit. Proportion out of 500 fruit cut in one day

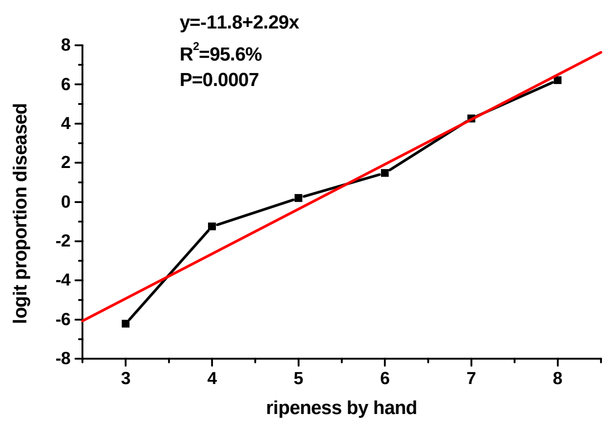

Figure 4: Logit transformation of sigmoid part of curve in Figure 3 where logit = ln(p/0.5-p).

DISCUSSION

Because of the compartmentalisation of antifungal dienes in oil cells in the flesh of avocado (Kobiler 1993) no barrier to endophytic invasion of stem-end rot fungi through the peduncle is expected. Therefore, unripe fruit should be able to be infected via the stem-end. Results of this study do not support the hypothesis that stem-end rots are caused by endophytic populations of fungi growing down the peduncle to infect fruit because no stem-end rots were found in unripe fruit. However, these results suggest that other host defence mechanisms preclude invasion of unripe avocado fruit through the stem-end. If indeed antifungal dienes are not active in the flesh then other host defences such as suberisation[4] and hypersensitivity are probably preventing any further fungal spread. Another explanation is that in avocados, unlike mangos (Johnson et al. 1991, 1992) postharvest pathogens do not infect endophytically. However, this requires further investigation.

Invasion through the peduncle could be an important source of infection, but probably not by systemic invasion. It is more likely that infection occurs by contamination of the stem-end picking wound at harvest. If the outer layers of the stem tissue are infected, cutting with secateurs could disperse the inoculum over the stem-end wound (Hartill and Everett 1997). Application of benomyl at flowering (Everett et al. 1999), which reduced stem-end rots, may reduce infection of the outer stem tissue, and hence stem end rots.

An increase of ripeness value from 4.54 to 4.97 (just under-ripe) resulted in doubling the proportion of fruit that were diseased from 10% to 20%, and a further increase in ripeness to 5.33 (just over-ripe) increased proportion of fruit that were diseased to 30%. A further increase to 40% diseased fruit follows an increase of the ripeness value to 5.75. From the ripening curves fruit increases from a ripeness value of 5 (optimal) to 6 (slightly overripe) in just one day. The consumer would probably still assess this fruit as ripe to eat, and may not realise that it was overripe. In other words, if the consumer or the supermarket left the fruit for a day past optimal eating, then the chances of the fruit being rotten would increase from 20% to 40%. Because of the dark skin, it is not obvious from the external appearance that 'Hass' avocados one day overripe have rots. It is therefore very important for fruit quality that avocados are not sold or consumed even a day past optimal ripeness.

REFERENCES

Everett, K.R., Stevens, P.S. and Cutting, J.G.M. (1999). Postharvest fruit rots of avocado reduced by benomyl applications during flowering. *Proceedings of the New Zealand Plant Protection Society* **52**: 153-156.

Johnson, G.I., Mead, A.J., Cooke, A.W. and Dean, J.R. (1991). Mango stem end rot pathogens- Infection levels between flowering and harvest. *Annals of Applied*

[4] Deposition of a layer of suberin, which is a waxy impregnable substance, in the cell wall.

Biology **119**: 465-473.

Johnson, G.I., Mead, A.J., Cooke, A.W. and Dean, J.R. (1992). Mango stem end rot pathogens- Fruit infection by endophytic colonisation of the inflorescence and pedicel. *Annals of Applied Biology* **120**: 225-234.

Hartill, W.F.T. and Everett, K.R. (1997). An investigation of the mode of entry of pathogens causing post-harvest rots in ripening avocados. III. Effect of different harvesting techniques. *HortResearch Client Report No. 97/51*.

Kobiler, I., Prusky, D., Midland, S., Sims, J.J. and Keen, N.T. (1993). Compartmentation of antifungal compounds in oil cells of avocado fruit mesocarp and its effect on susceptibility to *Colletotrichum gloeosporioides*. *Physiological and Molecular Plant Pathology* **43**: 319-328.

Prusky, D., Keen, N.T., Sims, J.J. and Midland, S.L. (1982). Possible involvement of an antifungal diene in the latency of *Colletotrichum gloeosporioides* on unripe avocado fruits. *Phytopathology* **72**: 1578-1582.

White, A., Woolf, A.B., Harker, F.R. and Davy, M.W. (1998). Measuring avocado firmness: Assessment of various methods. *HortResearch Client Report No. 98/138*.