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NOTA CIENTIFICA

INFECTION OF UNRIPE AVOCADO FRUIT BY STEM END ROT FUNGI IN NEW ZEALAND

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INTRODUCTION

Johnson *et al.* (1991, 1992) has suggested that fungi that cause postharvest rots in mango are both seed transmitted and endophytic within the plant. lnfection of the stem end of fruit is via the endophytically infected pedicel. Stem end rots might also infect avocado trees endophytically (Johnson 1997). 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). Avocado fruit that had been sprayed by benomyl twice during flowering had less stem end rots than unsprayed control fruit at harvest (Everett *et al.,* 1999). These results suggest infection of avocado fruit by stem end rot fungi may occur at flowering. lf stem end rots infect avocados by invasion from fungal mycelium growing down the pedicel, then unripe avocado fruit should be able to be infected as there is no skin barrier to invasion. lnfection of the stem end in unripe fruit would provide evidence to support this hypothesis. The following experiment was designed to test this hypothesis to further elucidate the infection mechanism by stem end rots and therefore enable effective and focused control strategies to be developed.

METHODS

Fruit from an unsprayed orchard was harvested 14 months after fruit set at a dry matter content of >30%. Fruit were placed in boxes, 10 per box at 20^0C. Fifty fruit per day for 16 days were cut (the first 50 two days after harvest) and assessed for stem end rots using a 0-10 visual rating scale for severity where O = 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.

RESULTS

Ripeness was assessed by hand each day as fruit were cut. ln some unripe fruit a small amount of browning was noted at the stern end. ln unripe fruit, this browning remained localized and did not develop further. When disease progress curves of cumulative mean severity score were examined, disease increased exponentially with increasing

ripeness (Figure 1).

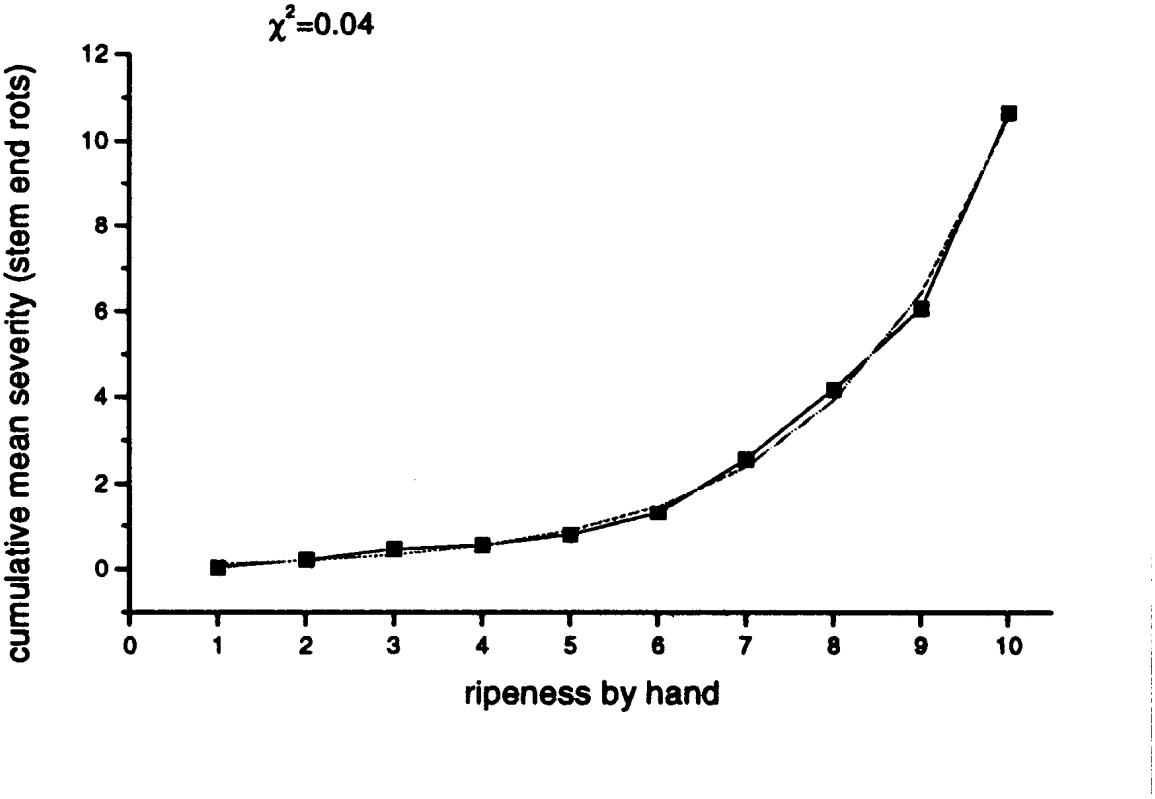

Figure 1: Disease progress curve (cumulative mean severity) compared to ripeness of fruit determined by gentle hand squeezing.

The incidence of the browning symptom in unripe fruit at a ripeness of 1 was the same as the incidence of disease in fruit at optimal ripeness (Fig. 2 (a), dotted one). The incidence of the browning symptom declined in fruit at a ripeness of 2, 3 and 4. When fruit with this browning symptom were excluded disease followed a sigmoidal curve (Figure 2 (b). With the browning symptom in unripe fruit included the curve followed two trends, one decreasing (ripeness of 1-4) and the other increasing (ripeness of >4). Attempts to isolate fungi from the browning symptom in unripe fruit were not successful.

Ripening followed a sigmoidal pattern of increase over time, whilst rots, when severity values were used instead of incidence, followed an exponential increase over time. When the two were plotted on the same graph (Figure 3), there was no apparent point after which disease increased more rapidly. However, before optimal ripeness was reached at day 8 (arrowed in Figure 3) disease increased less rapidly than the fitted exponential curve. After day 8, disease increased at the same rate as the fitted exponential curve.

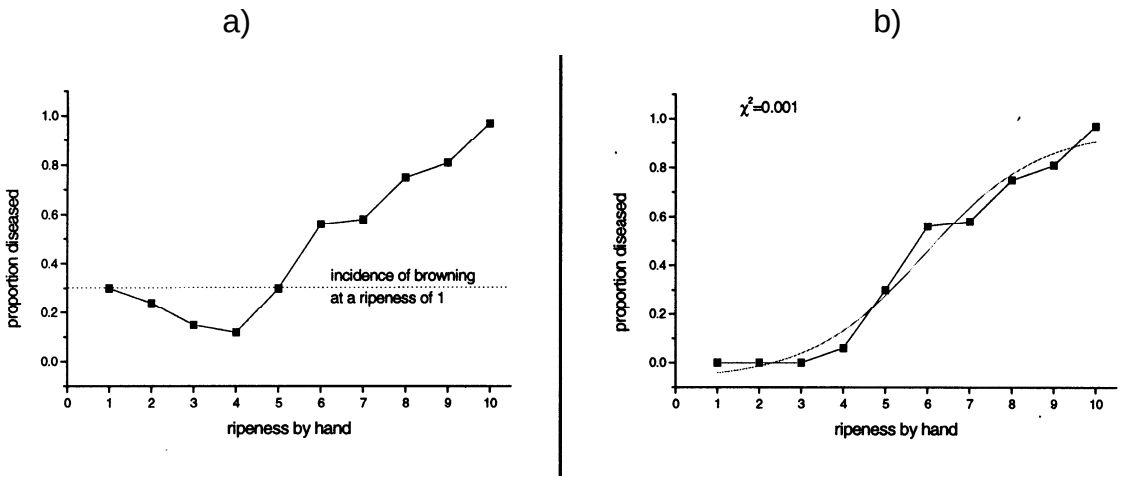

Figure 2. Disease progress curve (incidence) compared to ripeness of fruit determined by gentle hand squeezing. (a) including fruit with initial browning symptom, and (b) excluding fruit with initial browning symptom. Dotted line in (a) represents the incidence of the initial browning symptom at a ripeness of 1.

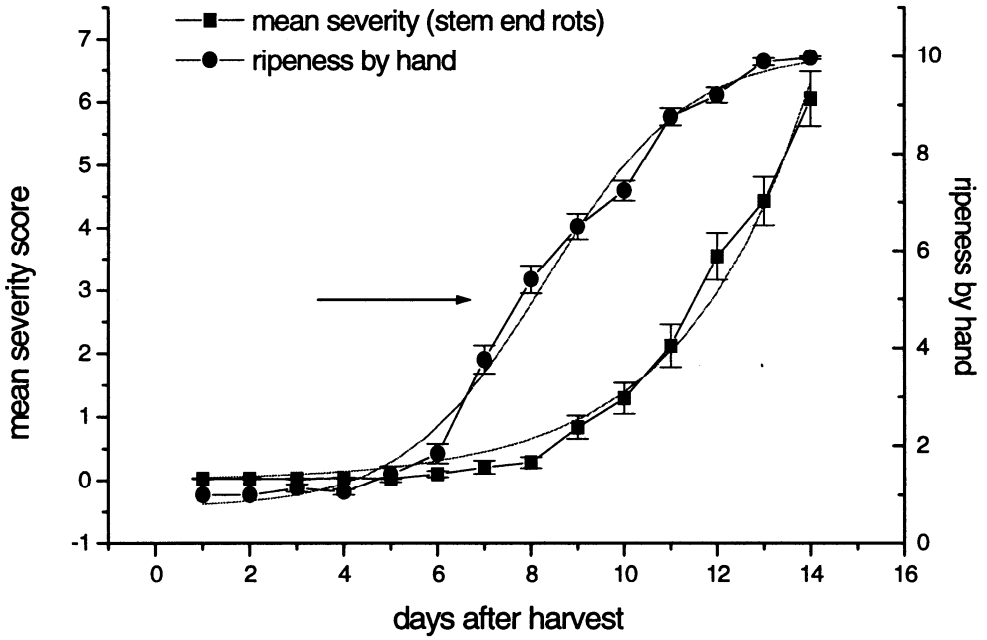

Fig. 3: Change in mean severity of stem end rots and ripeness (as determined by gentle hand squeezing) in time (days) after harvest.

DISCUSSION

Results of this study do not support the hypothesis that stern end rots are caused by endophytic populations of fungi growing down the pedicel to infect fruit. No inhibition of endophytic invasion of stem end rot fungi through the pedicel by antifungal dienes is expected because of compartmentalization of antifungal dienes in oil cells in the flesh of avocado (Kobiler, 1993). Slight browning was occurring, but no fungi were able to be isolated. This indicates either that other host defense mechanisms, such as suberisation and hypersensitivity, preclude the fungus from growing any further in unripe fruit and kill the fungus, or that this symptom of physiological and not pathological. ln support of a physiological cause, as fruit ripen (Figure 2 (a)), the slight browning symptom declines, and rot symptoms increase. Even if the slight browning symptom is caused by a host response to fungal invasion of the fruit through the pedicel, these results show that host defense mechanisms preclude invasion of unripe avocado fruit through the stem end.

ln conclusion, the pedicel 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 wound at harvest. If the outer layers of the stem tissue are infected, cutting with scatters could disperse the inoculums over the stem end wound. Reduction in stem end rots by applying benomyl at flowering (Everett *et al.,* 1999) is probably achieved by reducing infection of the pedicel.

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