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STEM-END ROTS -HOW, WHERE AND WHAT DO THEY INFECT?

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SUMMARY

The mechanisms of infection and sources of inoculum of stem-end rot pathogens infecting avocado fruit are reviewed.

KEYWORDS: Avocado, stem-end rots, mechanism of infection, phellophytes, *Persea Americana*

GLOSSARY

Pathogens = fungi that infect avocado fruit to cause a rot.

Names of avocado pathogens are:

C.a.= Colletotrichum acutatum
C.g.= C. gloeosporioides
B.p.= Botryosphaeria parva
B.d.= B. dothidea
P.= Phomopsis sp.

Vasculature = pipes inside the plant for transport of water (xylem) and food (phloem).

Xylem = water carrying pipe.

Phloem = food carrying pipe.

Spores = small egg-like structure from which new colonies of fungi can grow.

Mycelium = thread-like structure of the fungus that in mass can form mushrooms.

Inoculum = spores and/or mycelium by which the fungus can infect the plant.

Phellophytes = fungi that grow just under the bark without causing disease.

INTRODUCTION

'Hass' avocado fruit are detrimentally affected by fungi to cause postharvest rots. There are two types of rots, those that infect through the side of the fruit to cause body rots, and those that infect through the picking wound to cause stem-end rots (Snowden 1990, Hartill 1991). Progress in managing body rots, also called anthracnose or side-rots, has

been reviewed in a previous Australian and New Zealand avocado growers' joint conference proceedings (Everett 1997). However, stem-end rot disease has been less studied, and has not been previously reviewed.

Stem-end rots are estimated to affect 19% to 38% of avocado fruit in New Zealand and are thus considered to be an important problem. Stem-end rots are caused by a number of different fungi, of which the most common is *Botryosphaeria parva*. Also causing stem-end rots are *Colletotrichum acutatum*, *C. gloeosporioides*, *B. dothidea* and *Phomopsis* sp. (Fig. 1).

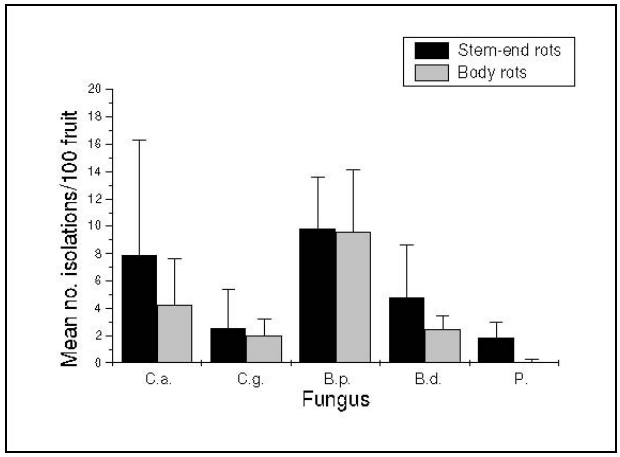

Figure 1: Mean number of isolations from avocado fruit rots +/-standard errors. Numbers are orchard means of isolations from 5100 fruit from 22, 21 and 8 orchards sampled in 1999, 2000 and 2001, respectively. C.a.= *Colletotrichum acutatum*, C.g.= *C. gloeosporioides*, B.p.= *Botryosphaeria parva*, B.d.= *B. dothidea*, P.= *Phomopsis* sp.

WHEN AND HOW DOES INFECTION OCCUR?

There are two mechanisms for infection. Both occur at harvest. The first is by spores landing on the picking wound after or during a period of rain. The fungi that infect avocados can produce spores either asexually or following mating (sexual stage). The two types of spores are different, and can be spread by different mechanisms. For other crops, asexual spores of *Colletotrichum* on beans were clearly spread by rain (Tu 1992). On apples, as well as the rain spreading asexual spores of *Colletotrichum gloeosporioides* within a tree, the sexual spores can be spread by wind from tree to tree (Brook 1977). The theory is that once the sexual spores have spread to each individual tree, infection of fruit is initiated by the asexual spores that are spread by rain. The sexual spores colonise dead fruit and twigs in the canopy, and the asexual spores are produced from embedded fruiting structures within these colonised tissues. On avocados, Fitzell (1987) found that in Queensland, Australia, most spores of *C. gloeosporioides* were washed off both dead leaves and infected fruit that were still hanging in the tree after at least 10 mm of rain. Fewer spores were washed from fruit mummies and dead twigs, and no spores were washed from branch and trunk bark or from green leaves. In New Zealand, Pak et al. (2003), also for avocados, showed that

harvesting fruit immediately after a short period of rain resulted in an increase in the incidence of stem-end rots. In addition, library tray data showed that there was a clear increase in incidence of stem-end rots when rainfall was greater than 10 mm in the 24 hours preceding harvest. Tu (1992) similarly showed that rainfall greater than 10 mm was required for infection of beans to occur, and that infections became apparent 3 to 7 days after each major rainfall, depending on temperature.

The second mechanism is by inoculum spread onto the wound at harvest. Hartill and Everett (2002) showed that the fungi that cause stem-end rot infections grow in the outer layers of the branches and twigs of the avocado tree canopy. These fungi are called phellophytes, and all the avocado pathogens were isolated from just under the bark. The stem end of harvested avocado fruit was shown to become infected by secateurs dragging the inoculum in twigs across the wound.

There are five important pathogens that cause avocado rots, and each of these pathogens may have a different infection mechanism. Recent work in New Zealand has shown that plucked fruit (picked by removing the stalk completely) are infected by all five of the avocado pathogens (C.a., C.g., B.p., B.d. and P.), but that C.a. was isolated most commonly (Hartill and Everett 2002, Hartill et al. 2002). This suggests that all these fungi can infect by spores landing on the picking wound at harvest, but that C.a. is more successful than the other fungi with this pathway. Spores are probably released from fruiting bodies in dead twigs and fruit in the canopy, either by rain or by shaking when the fruit are harvested.

A clue to when infection by *Phomopsis* takes place was provided by a spray trial in which two applications of benomyl (Benlate®) were made during flowering and early fruit set (Everett et al. 1999). These applications reduced stem-end rots caused by *Phomopsis* in fruit harvested the following January (15 months after the first spray application). When dead branches and green leaves were investigated as sources of inoculum, *Phomopsis* was isolated in low numbers suggesting that these were not important sources of infection by this fungus (Everett et al. 2003). Because of the relationship with flowering, it is possible that *Phomopsis* infections are from dead pieces of flower tissue still attached to the pedicel at harvest, or that phellophytic invasion of the pedicel occurs at flowering.

WHERE DO THEY INFECT?

Both mechanisms result in infection occurring by inoculum on the picking wound. This was further corroborated by harvesting fruit with increasing stalk (pedicel) length (Fig. 2). The longer the stalk, the fewer the rots, suggesting that the fungi were growing down the stalk to infect the fruit. Further evidence for this mode of entry were found by destructively sampling fruit placed in coolstorage at weekly intervals for 9 weeks (Everett and Pak 2002). It was shown that after 28 days in coolstorage the stem-end rot fungus *C. acutatum* had succeeded in growing down the stem-end and into the flesh of the fruit (Fig. 3).

A further trial showed that stem-end rots were fewer in fruit that were coolstored for 28 days (Fig. 4). There are two possible reasons for this, and perhaps others that are less

obvious. We know that spore germination and survival is detrimentally affected by continued exposure to low temperatures (Everett 2003), and that low temperatures reduce the rate of mycelial growth (Hartill, pers. comm.). Rots may be less in coolstored fruit for either of these reasons. However, in an earlier trial that examined development of stem-end rots in fruit that was neither inoculated nor coolstored, there was no evidence of fungi growing down the stem to infect the fruit (Everett 1999). In another trial, in which fruit were inoculated, *C. acutatum* was able to grow down the stalk to infect the flesh of the fruit in unripe fruit during coolstorage (Everett and Pak 2002). In this trial, there was more infection of coolstored fruit than of non-coolstored fruit. This differed from the pattern found by coolstoring fruit from a number of orchards and comparing rot incidence with that in noncoolstored fruit. The reason may be that for inoculated fruit, enough spores remained to cause infection despite attrition due to low temperatures. A simple experiment examining the effect of coolstorage on plucked fruit may help to elucidate this issue.

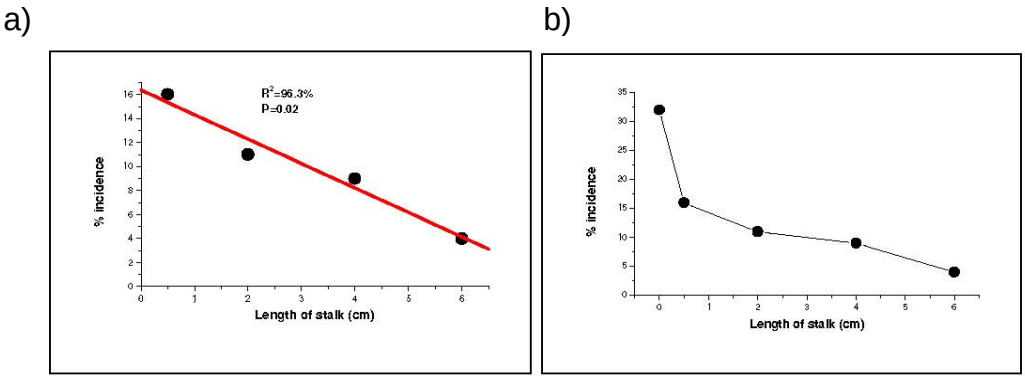

Figure 2: Length of stalk remaining on harvested avocado fruit influences amount of infection by stem-end rots.

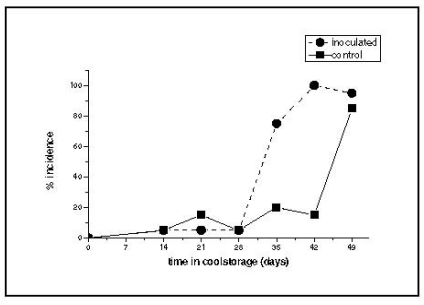

Figure 3: Progress of stem-end rots in coolstored fruit.

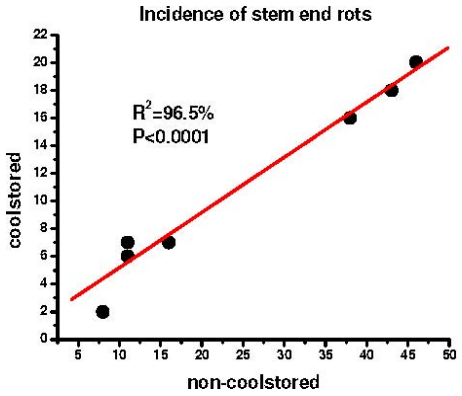

Figure 4: Incidence of stem-end rots in fruit from 8 orchards that was coolstored at 5.5°C or stored at 20°C.

For the trial in which fruit were picked with an increasing length of stalk still attached, plucked fruit were infected at a much higher level than for fruit that still had a stalk attached (Fig. 2b). Also problematic is the lack of total control of infections in plucked fruit after treatment with Benlate®, which has systemic activity. Both these results, and the results discussed above for fruit that had not been inoculated or coolstored, suggest that there may be another mechanism of infection other than growth through the stalk. Currently there are two possibilities to explain some of these differences. The first is that the stalk tissue is more difficult for the fungus to invade than flesh tissue, and thus the growth rate through stalk tissue is slower than through flesh tissue. Another theory is that some spores are sucked deep into the vasculature after withdrawal of the phloem/xylem contents immediately after cutting. These spores may be beyond the reach of the systemic fungicides applied later. Alternatively, even under the best of circumstances Benlate® does not kill all spores, and enough remain viable to cause the infections that occur.

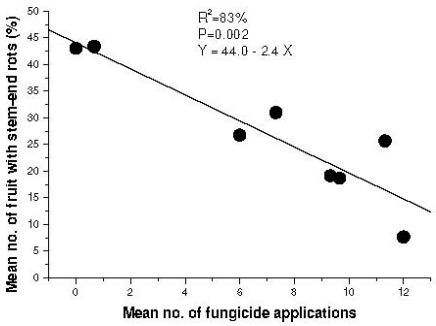

Figure 5: Stem-end rot incidence is linearly related to fungicide usage.

Analysing three years of fungicide data on 8 orchards showed a linear relationship between fungicides and stem-end rots, suggesting that incidence was directly related to inoculum availability (Everett and Pak 2001) (Fig. 5). It is also possible that, as for *Colletotrichum* infecting beans, success of infection may be influenced by temperature. It has been shown that for *Phomopsis* and *Colletotrichum* that there may be a threshold

temperature that is required for infection to take place (Everett 2002). This suggests that for these fungi infection occurs mainly in the summer months (Fig. 6), but that for *B. parva* infection probably occurs throughout the year.

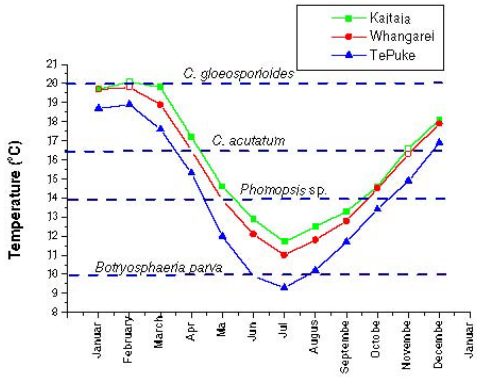

Figure 6. Temperature thresholds required for infection by avocado rot fungi.

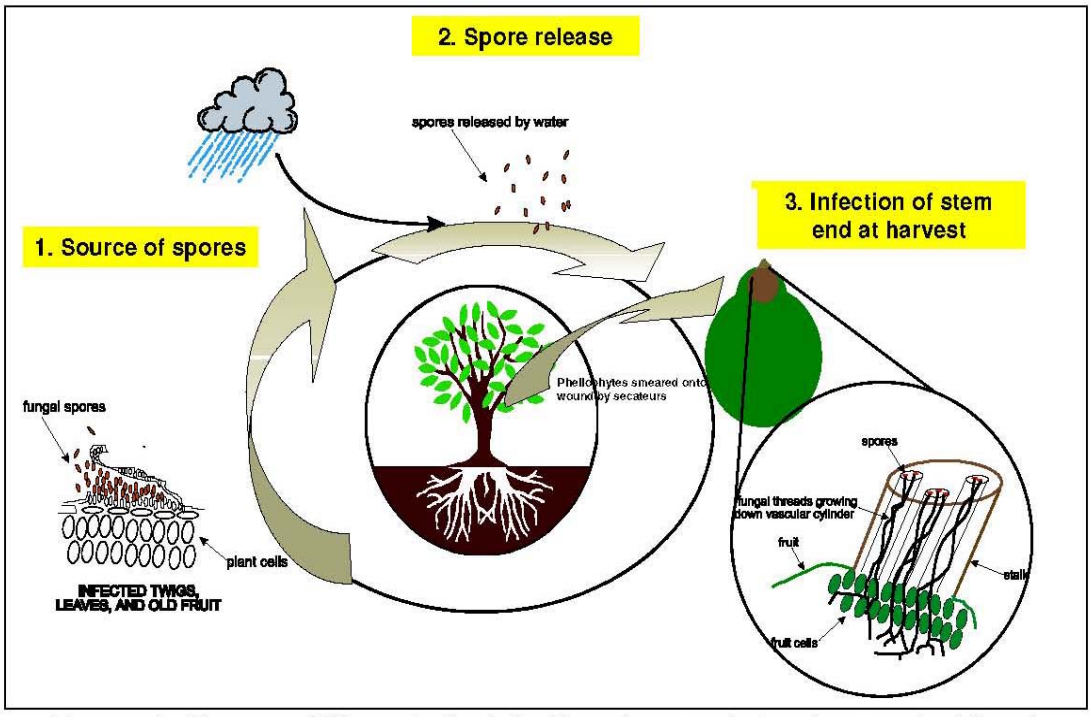

Figure 7: Proposed life cycle for infection of avocado by stem-end rot fungi.

WHAT DO THEY INFECT?

Inoculation experiments with *Botryosphaeria parva* mycelium and spores have shown that mycelium grows down the vasculature (Fig. 7). Vasculature is the food carrying (phloem) and water carrying (xylem) pipes inside a plant. Following inoculation with

spores and with mycelial plugs of *B. parva* at the stem-end wound, avocado fruit tissue was invaded by the fungus to cause an obvious rot, but preceding this rotten tissue was brown streaking extending down the vasculature of the fruit. This is often observed in purchased avocados as brown stringy strands that start at the stem end and finish at varying distances down the length of the fruit. The fungus was isolated from rotten tissue, but was not always able to be isolated from brown stained vascular strands. Microscopical examination of this tissue confirmed the results of isolations, with fungal structures not always being seen in the browned vasculature. The fungus was seen growing through the xylem elements of browned vasculature on occasion. It was concluded that the browning of the vascular tissue was a defensive host response to fungal invasion, similar to scarring of a wound. This result also showed that for *B. parva*, spread of the fungus through avocado fruit is by means of mycelial growth, and that spores were not transported in the vasculature following the methods of inoculation that were used. Further work would be useful to examine the method of infection by other fungi that cause stem-end rots.

CONCLUSIONS

From experimental evidence it is clear that infection of avocado fruit by stem-end rot fungi occurs at harvest. There are three possible mechanisms of infection, by smearing fungal tissue present just under the bark over the picking wound with secateurs, spores landing on the wound from inoculum released from dead fruit and twigs in the canopy by rain or shaking, and possibly by spores in small pieces of dead flower tissue attached to the junction of the fruit and the stalk. Reducing inoculum by applying fungicides as sprays throughout the season was an effective means of control, as was coolstoring fruit for 28 days. Further work is recommended to optimise fungicide timing for maximum effectiveness, and to enable alternative control strategies to be devised.

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