South African Avocado Growers' Association Yearbook 1987. 10:113-116
Proceedings of the First World Avocado Congress

Epidemiology of anthracnose disease of avocados

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SYNOPSIS
*The Fuerte avocado is very susceptible to anthracnose disease (*C. gloeosprioides *var* minor) *when grown along the east coast of Australia. This disease caused both field and post-harvest losses to growers, retailers and consumers. In addition to ripe-rots, lesions also appear in immature, unripe fruit up to three months before harvest and these fruit are generally shed. Losses of 71 per cent of pre-harvest fruit have been recorded. Spore trap studies show that the disease spreads from dead leaves caught in the canopy and the accumulations of this inoculum leads to higher losses in older trees. Two types of lesions occur in immature fruit, one is large (up to 4 cm diameter) and develops rapidly, resulting in fruit fall within 5 to 10 days. Another is small (0,1-0,5 cm) and occurs in groups over undamaged skin and lenticels. The larger lesions were associated with skin injuries (insect or mechanical) and were easily reproduced artificially. A mechanism for these wound infections of unripe fruit is proposed. The mode of infection of the smaller lesions in apparently undamaged fruit remains unclear. Control of anthracnose disease in cv Fuerte relies on orchard management to prevent long periods of fruit surface wetness. This involves frequent pruning to remove dead leaves and twigs, removal of short-term windbreaks, timely applications of fungicides to achieve best possible coverage and control of insect pests such as fruit sucking bugs and caterpillars.*

INTRODUCTION

The three main cultivars of avocado grown along the east coast of Australia are Fuerte, Sharwil and Hass. Anthracnose disease *(Colletotrichum gloeosporioides* Penz var *minor* Simonds) DAR 37820^{+} can infect all three, although Fuerte is the most susceptible. Fuerte flowers during the drier months (August to October), carries its fruit through the wet summer months (January to March) and is harvested from April onwards. Anthracnose disease is predominantly a ripe-rot problem; infections remain latent (Binyamini & Schiffman-Nadel, 1972) possibly because of the presence of an antifungal diene in unripe fruit (Prusky *et al,* 1982). However, lesions also develop in immature, unripe fruit still on the tree. Fruit can display symptoms from up to three months before harvest and these fruit generally fall. Fruit abscission is caused by premature ripening resulting from fungal infection, This phase of the disease is known locally as pre-harvest anthracnose fruit drop. This is distinct from natural fruit shedding which occurs one to two months after set or during extremely dry conditions. Growers apply orchard fungicide sprays in an attempt to prevent both the pre-harvest fruit-drop phase and the

latent ripe-rot phase of anthracnose disease. These sprays are applied in addition to post-harvest fungicide treatments applied before packaging.

Little work has been published on the epidemiology of anthracnose disease to aid control in the orchard. Peterson (1978) found that fruit infections were related to rainfall incidence and could occur from fruit-set until harvest. Peterson& Inch (1980) reported that immature fruit lesions developed around injuries to fruit and claimed that damage from insect pests such as Queensland fruit fly (*Dacus tryoni* Froggatt) and fruit spotting bug *(Amblypelta nitida* Stal) could break the latency of dormant infections.

Work reported on in this paper consists of field studies aimed at identifying sources of disease inoculum, orchard conditions which favour disease, types of immature fruit lesions, their mode of infection and chemical control.

MATERIALS AND METHODS

Field work was done at Alstonville, New South Wales (29°S, 153°E, mean annual rainfall 1 650 mm with a 65 per cent incidence from November to April) using 4 to 14-year-old cv Fuerte trees.

Inoculum sources

Spore traps similar to those reported by Fitzell & Peak (1984) were used to trap water-borne conidia and air-borne ascospores *(Glomerella cingulata* Stonem) from different sites within the tree canopy. Traps were positioned within trees in February and removed in April, 1981.

Orchard survey

Six Fuerte orchards were surveyed for the incidence of immature fruit infection. The age, canopy density, copper fungicide spraying history and the current season's spray schedule were recorded. One hundred and fifty fruit from 15 randomly selected trees in each orchard were tagged in the first week of March, 1981. Fruits were inspected every seven days and the percentage of tagged fruit which developed anthracnose lesions and/or fell prior to harvest (mid-May, 1981) was recorded. Fallen fruits were examined and reasons attributing to their abscission also recorded.

Pre-harvest fruit lesions

Seventeen trees in two adjoining rows of a 14-year-old orchard were selected on the basis of similar canopy density and crop load. The pedicel of 60 fruit in each tree were tagged in February, 1986. Each fruit was inspected every seven days until the orchard was harvested (mid-May). Anthracnose lesions which developed in immature fruit were categorised as follows: Type 1; large (1-4 cm diameter) lesions generally developing singularly. Type 2; small (0,1-0,5 cm diameter) lesions occurring in groups. Records were made of percentage infected fruit having Type 1 or 2 lesions, percentage of fruit drop attributed to both lesion types and the time interval between lesion development and fruit drop.

Artificial inoculation studies

Two hundred symptom-free fruit were tagged in one 11 -year-old Fuerte tree in March, 1983. One hundred fruit were artificially inoculated *in situ* by spraying one side with a solution of 10 x 10^4 conidia mf^{-}t collected by washing the sporulating surface of fruit lesions, or from that of pure cultures (potato dextrose agar) of *C. gloeosporioides* var *minor. A* moist piece of tissue paper was immediately placed over the sprayed area. Half of the fruit was wounded prior to inoculation. The skin of wounded fruit was punctured to a depth of 3 mm using a 0,25 mm entomology pin. Wounds were designed to artificially represent damage which results from activity of either Queensland fruit fly, or fruit-spotting bug. Treated and untreated control fruit were then enclosed in plastic polyethylene bags for two days. An attached branchlet, having three leaves, was also enclosed in each bag to generate high humidity and prevent conidia from dessicating. Fruit were inspected daily and records made of the percentage which developed lesions and the time taken to develop them. Fruit which remained free of symptoms on the tree, were harvested 40 days later and monitored for incidence of anthracnose riperot.

Effect of insect injury on latent infections

During March, 1984 and 1985, a plastic rain shelter (similar to that described by Fitzell & Peak, 1984) was built over one 12-year-old Fuerte tree. The shelter kept fruit completely dry after set and thus prevented natural field infections during the study. One side of 15 fruit were artificially inoculated with conidia from pure cultures, on dates shown in Tables 5 and 6. Artificial inoculations were performed from one to 50 days prior to having live fruit-spotting bugs or Queensland fruit flies caged around them. Insects were removed after three or more wound sites were visible inside the inoculated area on each fruit. Fruit were monitored for development of anthracnose lesions and those remaining after 40 days, were harvested and checked for incidence of ripe-rot.

Pesticide management programme to control anthracnose

An orchard spray trial was conducted during the 1984 season in a 12-year-old orchard, Treatments were (1) unsprayed control, (2) prochloraz (0,65 g ai f^{-}t) applied strategically (after wet periods lasting two days) from October to December then copper oxychloride (2 g ai f^{-}t) monthly from January to April, (3) copper oxychloride (2 g ai f^{-}t) monthly from October to April, (4) fenthion (0,04 per cent) mixed with endosulfan (0,05 per cent) applied twice per month from November to April, (5) treatments (2) plus (4), (6) treatments (3) plus (4). Trees were sprayed with a radial flow, as fan applying 10[spray per tree. Treatments were replicated six times using single tree plots. Two hundred fruit were tagged in each tree in February. Trees were harvested in mid-May and the percentage tagged fruit remaining was recorded at harvest.

RESULTS

Inoculum sources

Results in Table 1 show that the majority of conidia of *C. gloeosporioides* var *minor* came from dead leaves entangled in the tree canopy and from infected fruit which had not fallen. Few conidia were trapped in water splash from dead twigs or bark. No air-borne ascospores of *G. cingulata* were detected in a volumetric air sampling trap operating over leaf litter.

TABLE 1 Average number of conidia caught per water trap after wet periods of greater than 10 mm rainfall.

Source of disease	Conidia caught x 10^6
Dead leaves	2,18
Infected fruit (still hanging)	2,07
Fruit mummies	0,77
Dead twigs	0,08
Branch/trunk bark	Nil
Green leaves	Nil

Orchard survey

Anthracnose infections were considered to be responsible for 92 per cent of tagged fruit abscission in the six orchards from March until May. Stemend rot *(Dothiorella aromatica* (Sacc) Petr (Syd)) and damage by monolepta beetle *(Monolepta australis* Jacoby), fruit-spotting bug, Queensland fruit fly and hairy caterpillar *(Olene mendose* Huber) was judged to be solely responsible for 8 per cent of total fruit drop. More immature fruit infections were recorded in unsprayed orchards containing older trees with dense canopies (Table 2) than in orchards having younger trees.

Regular spraying with copper fungicides reduced the incidence of anthracnose in immature fruit, particularly where the programme had been applied over previous seasons.

TABLE 2 Survey of immature fruit infection in Fuerte orchards near Alstonville, New South Wales.

Orchard Number	Age (yrs)	Canopy Condition	Previous years orchard sprayed	Sprays applied 1980/81 season	Percentage of tagged fruit infected
1	8	++(+)	0	0	58
2	9	++(+)	1	6	34
3	5	+++	2	9	7
4	4	+	0	1	16
5	5	+	0	1	13
6	6	+	1	8	8

+ foliage not touching within or between rows
+++ canopy completely closed over

Immature fruit lesions

Table 3 shows the percentage of fruit within a close group of trees having Type 1 or 2 lesions.

Both lesion types were present in similar numbers. The majority of fruit which developed Type 1 lesions fell in 8,3 days compared to less than half those with Type 2 lesions which took much longer to drop. Injury to skin, either insect or mechanical, was found in 91,8 per cent of Type 1 lesions but in only 6,2 per cent of Type 2. Sixty four per cent of Type 2 lesions was associated with raised lenticels on the skin, but it was not possible to detect if these had been injured prior to infections.

TABLE 3 Characteristics of anthracnose lesions in immature Fuerte fruit

Lesion Type	% Fruit infected	% Infected fruit which fell before harvest	Average time to drop (days)	% Lesions associated with skin injury	% Lesions associated with lenticels
1	47,4	89,7	8,3	91,8	N D+
2	52,5	36,5	15,6	6,2	69,3

+ No data, unable to detect due to skin breakdown around lesion.

Artificial inoculations and effects of insect damage on latent infections All fruit wounded at the time of artificial inoculation developed anthracnose within five days (Table 4). Uninoculated wounded fruit also developed anthracnose lesions, presumably as a result of field infections of the wound site. Disease symptoms did not develop in immature fruit which were not wounded prior to inoculations. Conidia collected from diseased fruit were more successful in establishing latent infections than those from pure culture.

The effect of damage by fruitspotting bug on latent infections is presented in Table 5. The number of stylet feeding sites and the resulting severity of skin damage varied between fruit. Severe damage (30 or more stings) resulted in some fruit drop. However, anthracnose lesions did not develop around damage occurring in the inoculated area. Post-harvest anthracnose ripe-rot developed in all but one inoculated fruit, thus confirming the presence of established latent infections at the time insects were feeding. The effect of Queensland fruit fly damage on latent infections is shown in Table 6. Damage by this insect was less severe to the skin of fruit and similarly, damage did not break the latency of artificial infection.

TABLE 4 Disease levels in immature and ripe fruit after inoculation in the orchard

Inoculation Treatment	% Fruit diseased on the tree		Days to first lesion in unripe fruit		% Healthy fruit remaining which developed disease when ripe	
1. Inoculated/ wounded	100*	100**	4*	5**		
2. Inoculated/ unwounded	0	0			100	31
3. Uninoculated/ wounded	87	34	10	8		14
4. Uninoculated/ unwounded	0	0			48	39

*Exp 1 Conidia washed from infected fruit.
**Exp 2 Conidia from pure culture

TABLE 5 Effects of damage by fruit-spotting bug on latent fruit infections

Date inoculated	Date fruit-spotting bugs caged with fruit (av 2/fruit)	Days bugs left feeding on fruit	Av number of stings per entire fruit` surface	Number of fruit developing lesions under stings /fruit caged	Number of ripe fruit developing lesions /fruit harvested
1-2-84	29-2-84	12	15	0/3	3/3
	5-3-84	8	25	0/3	1/1
	15-3-84	4	23	0/3	1/2
	19-3-84	3	18	0/3	0/3
	22-3-84	4	19	0/3	2/3
6-2-84	29-2-84	11	22	0/7	5/6
	15-3-84	4	40	0/8	5/5
28-2-84	29-2-84	13	15	0/5	4/5
	19-3-84	5	10	0/10	10/10
Uninoc	22-3-84	4	16	0/10	0/10
control	26-3-84	4	21	0/10	0/10

TABLE 6 Effect of damage by Queensland fruit fly on latent fruit infections.

Date inoculated	Date fruit flies caged around fruit (Av 10/fruit)	Days flies left to lay eggs	Av number stings/entire fruit surface	Number of fruit developing lesions around stings/fruit caged	Number of ripe fruit developing lesions/number harvested
13-3-85	22-3-85	4	1	0/15	12/15
20-3-85	22-3-85	8	3	0/15	8/15
23-3-85	26-3-85	8	7	1/15'	11/15
29-3-85	30-3-85	12	5	0/15	9/15

*Anthracnose lesion developed over pen-marking.

Disease control by pesticide management

Insecticide alone reduced pre-harvest fruit loss when compared with unsprayed trees (Table 7). Addition of insecticides in fungicide sprays also reduced pre-harvest fruit drop, but did not alter the level of post-harvest riperot. Trees which received copper or copper plus insecticides, yielded significantly more disease-free ripe fruit than unsprayed or insecticide-treated trees.

TABLE 7 1984 Alstonville spray trial on Fuerte avocado

Spray treatments	No fungicide applied	Prochloraz applied strategically then copper fungicide	Copper fungicide throughout season
	tagged fruit fallen or infected at harvest (10-5-84)		
No insecticides	71,4 d*	51,4 c	36,1 a
Insecticide added	40.4 ab	47,1 b	33.9 a
	% disease-free ripe fruit		
No insecticides	10.0 d	26,6 be	32,0 ab
Insecticide added	15.8 cd	19.8 bcd	42.6 a

Similar superscripts indicate no significant difference (P = 0,10)

DISCUSSION

Spore trapping for water-borne conidia of *C. gloeosporioides* var *minor* showed that dead leaves entangled in the canopy were the main source of primary inoculum. Conidia are spread by rain water to fruit which develop sporulating lesions and become a source of secondary inoculum. Few conidia were trapped from green leaves and lack of vegetative symptoms makes the disease difficult for growers to comprehend. Older trees soon develop dense canopies and the higher incidence of disease found in these unsprayed trees during the orchard survey, can be attributed to both an accumulation of inoculum and a more favourable micro-climate. Orchards of similar age receiving regular copper fungicide sprays, had less disease, particularly where spraying had been conducted in previous seasons. Darvas (1981), Kotze *et al* (1982) and Peterson & Inch

(1980) all reported similar effects with copper fungicide. Regular spraying would gradually reduce inoculum, as well as protect developing fruit from infection.

Anthracnose was the principal cause of premature fruit abscission; however, not all fruit lesions were associated with injuries to the skin as indicated by Peterson (1978) and Peterson & Inch (1980). In an intensive survey of a close group of trees in similar vegetative conditions, two types of lesion were found: Type 1 lesions were associated with insect damage and caused the majority of fruit infected to fall. Smaller, Type 2 lesions, however, were associated with raised lenticels. Type 1 lesions were easily reproduced by artificial wounding prior to inoculation. This indicates that the *Colletotrichum* fungus acted as a wound parasite as claimed by others (Wolfe *et al,* 1934; Stevens & Piper, 1941; Nilda de Ramallo & Zabala, 1966). A possible explanation for this mode of infection is that wounds cause localised ripening resulting in a fall in concentration of antifungal diene in the affected part of the skin. This enables conidia, washed into the damaged tissue, to germinate and grow into the flesh, causing fruit to ripen prematurely and fall. Injury to the skin of fruit by two common avocado pests, however, did not break the latency of infections artificially established in fruit.

The mode of infections resulting in the formation of Type 2 lesions remains unclear. It is not possible to readily reproduce these lesions artificially. Type 2 lesions develop without apparent damage to skin. They may occur as a result of an unexplained decline or inherited low level of antifungal diene, which allows either direct lenticel infections or normal latent infections to proceed prematurely. Field spray trials involving fungicides scheduled with insecticides, gave some but not completely adequate control of pre-harvest anthracnose. This indicates that something other than insect damage predisposes immature fruit to anthracnose infection. Attempts by the author to compare antifungal diene levels in fruit from trees having high and low levels o1 Type 2 lesions, have been unsuccessful.

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