

Alternative fungicides for controlling husk spot caused by *Pseudocercospora macadamiae* in macadamia

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Abstract. Husk spot, caused by *Pseudocercospora macadamiae* is a major fungal disease of macadamia in Australia. Chemicals to control the disease are limited and frequent failure to control the disease is a major concern to growers. The overall goal of this research was to improve the chemical control strategy of *P. macadamiae* through the provision of fungicides with different modes of action to carbendazim, which is the current industry standard. Husk spot incidence, premature fruit abscission, kernel quality and yield were evaluated following application of different fungicide products in replicated field experiments at three different sites. Results showed significant differences in disease incidence and premature fruit abscission between fungicide treatments, field sites and years. Generally, disease incidence and premature fruit abscission on trees treated with fungicide were significantly ($P < 0.05$) lower than the untreated control. Pyraclostrobin conferred significantly better protection than trifloxystrobin, reducing disease severity by 70% compared with a 50% reduction by trifloxystrobin. The pyraclostrobin treatment had a similar efficacy to the current industry standard (70% reduction cf. 73% reduction by tank-mixed carbendazim and copper). Higher amounts of immature kernels occurred in the untreated control, followed by difenoconazole and trifloxystrobin. Diseased fruit accounted for 78% of premature fruit abscission, which indicates that husk spot enhances fruit abscission in macadamia. Our results suggest that pyraclostrobin provided similar efficacy to the industry standard and could, therefore, play a key role in the management of husk spot.

Additional keywords: disease progress curve, Mycosphaerellaceae, nut, strobilurin.

Introduction

Husk spot of macadamia (*Macadamia integrifolia*, *M. tetraphylla* and their hybrids), caused by *Pseudocercospora macadamiae* affects only the pericarp, or husk, of green macadamia fruit and not the shell enclosing the edible kernel (Beilharz *et al.* 2003). Diseased fruit may abscise prematurely resulting in yield loss because of small kernels with low oil contents. Anecdotal reports indicate that yield losses up to 90% occur in orchards where husk spot is not controlled. Losses of up to 40% have been reported where management practices are not effective (Mayers 1998). Effective control saves the Australian macadamia industry over \$10 million in lost production annually (Jones 2004). When diseased immature fruit are harvested as a mixture with mature fruit, the lots are commonly downgraded at the processing stage, resulting in financial penalties.

Husk spot symptoms develop very slowly, and initial chlorotic symptoms appear between 6 and 8 weeks after penetration of green fruit (Akinsanmi *et al.* 2007). As the fruit matures, from 4 to 5 months after flowering, the husk hardens and the chlorotic spots become tan-brown, necrotic and tougher than the surrounding green tissue. This is when premature fruit abscission usually begins. The chlorotic spots with diffuse pale green halos are easily distinguished from other diseases of the husk such as

husk rot, caused by *Colletotrichum* sp., *Phomopsis* sp. and *Lasiodiplodia* sp. (Akinsanmi *et al.* 2007).

Green fruit are susceptible at any time throughout the season, but yield-limiting infections mostly occur when the husk is soft and green and the fruit is at the 'match head size' (MH) stage of fruit development (Akinsanmi *et al.* 2007). Spores appear to spread from old, diseased husks to the developing fruit by rain splash because abundance of old, diseased husks within the tree canopy influences disease incidence and severity (A. K. Miles, unpubl. data). Disease incidence, which is defined as the number of fruit that are visibly diseased with one or more lesions, and disease severity, defined as the amount of abscised fruit that are visibly diseased relative to the total number of abscised fruit, vary from year to year (Akinsanmi *et al.* 2007). Disease severity and incidence also depend on the age of the tree, variety, time of infection and prevailing weather conditions (Akinsanmi *et al.* 2007).

The efficacy of fungicides commonly used for husk spot control is inconsistent (Akinsanmi *et al.* 2007). This is due to the commencement of fungicide applications at the 'pea size' (PS) stage of the fruit development, using two successive tank-mix applications of carbendazim and copper (Cu) or difenoconazole, followed by two additional Cu-only fungicides treatments. Variation in flowering between seasons and varieties often

creates confusion as to when to spray and whether to still conform to the guidelines of a maximum of two applications of carbendazim per season (Akinsanmi *et al.* 2007).

Carbendazim-based products may be withdrawn from use in Australia and the development of resistance to sterol dimethylase inhibitor (DMI) fungicides by fungi similar to *P. macadamiae* (Karaoglanidis and Bardas 2006; Karaoglanidis and Karadimos 2006) highlights the need for additional control options. The mode of actions of carbendazim, a Group A fungicide that interferes with the nuclear division of fungi, and difenoconazole, a DMI product classified as Group C, differ from the strobilurins, which are classified as Group K (Australian Pesticides and Veterinary Medicines Authority 2006). Strobilurin fungicides were considered as an option as this new class of chemicals are effective against a broad range of diseases (Reuveni 2000, 2001; Leskovar and Kolenda 2002; Bubici *et al.* 2006; Karaoglanidis and Bardas 2006; Karaoglanidis and Karadimos 2006).

The fungicidal activity of the strobilurins stems from their ability to inhibit mitochondrial respiration by binding at the Q_o site of cytochrome *b*, which is part of the cytochrome bc₁ complex, located in the inner mitochondrial membrane of fungi and other eukaryotes (Bartlett *et al.* 2002). Two strobilurins – trifloxystrobin and pyraclostrobin – were available for comparison in this study. The trifloxystrobin product (Flint), released for commercial use in 1999, and the pyraclostrobin product (Cabrio), released in 2002, provide good control of many fungal diseases in numerous crops including grapes, pears, pome fruit and apples (Reuveni 2001; Bartlett *et al.* 2002). However, information on the efficacy of the fungicides on macadamia crops under field conditions is needed before either of these compounds is introduced into spray programs. The long incubation period of *P. macadamiae* makes laboratory studies of the effects of these products on different stages of pathogenesis *in planta* difficult.

Thus, the overall goal of this study was to improve the chemical control strategy of *P. macadamiae* through the provision of fungicides with different modes of action to carbendazim. The specific aims of the study were: (i) to test the effectiveness of strobilurin products in reducing husk spot incidence and severity; (ii) to compare the efficacy of the strobilurins to a tank-mix of carbendazim and Cu and a difenoconazole-only spray program; and (iii) to test the effect of the fungicides on macadamia yield and kernel maturity. Avoidance of continual use of the same product or chemicals of the same mode of action to control fungal pathogens will provide more sustainable ways of managing fungicide resistance risk in macadamia.

Methods

Experimental design

Experiments were conducted on 9-year-old macadamia trees in three commercial orchards at Bangalow, New South Wales (28°69'S, 153°52'E), Beerwah, Queensland (26°85'S, 152°95'E) and Bundaberg, Queensland (24°87'S, 152°35'E). The height of the trees was ~4 m with an average canopy volume of 83 m³, and most of the fruit were within 1 and 3 m from the ground. The macadamia variety A16, which is widely grown in Australia and susceptible to husk spot, was used at all sites. An additional variety, HAES 246, also widely grown in

New South Wales, was included at Bangalow. Six different treatments, including an untreated control, were assigned to plots in a randomised complete block design with three replicates at each site. Each plot in Beerwah and Bundaberg contained 15 trees, arranged in three rows of trees, while each plot in Bangalow contained 20 trees arranged in four rows with varieties A16 and HAES 246 in the middle rows. Data were recorded separately from the three middle trees of each plot for each variety, while the other trees served as buffer trees to reduce spray drifts between treatments. The experiments were conducted on a total of 225 trees in Beerwah and 300 trees with parallel rows of A16 and HAES 246 in Bangalow. At Bundaberg, rows contained mixed varieties and a total of 297 trees with at least three A16 trees together within each data row were used. The total number of data trees was 54 in Beerwah and Bundaberg and 108 in Bangalow. All trees received similar agronomic practices and insect control such as endosulfan. Insect populations were monitored by pest scouts and controlled by the application of insecticide sprays. The experiments were conducted in the 2004–05 macadamia production season (October–August) and repeated in the 2005–06 season.

Fungicide products

Four fungicides, SpinFlo (50% carbendazim; Bayer CropScience, Australia), Score (25% difenoconazole; Syngenta, Australia), Flint (50% trifloxystrobin; Bayer CropScience, Australia) and Cabrio (25% pyraclostrobin; BASF, Australia) were used. A tank-mix of carbendazim and Nordox (83.9% cuprous oxide; Swift and Co. Ltd, Australia) was used as the industry standard treatment and applied at the MH (~2 mm diameter) and PS (2–5 mm diameter) fruit stages to allow for a meaningful comparison of the different products (Table 1). Pyraclostrobin and trifloxystrobin were applied when fruit were at MH stage. Based on the current industry recommendation, difenoconazole was applied when fruit were at PS stage (Table 1). The spray regime was conducted at intervals of 4 weeks, based on the existing industry application protocol (Table 1). The fungicides were applied at recommended rates, 0.1 g a.i./L for carbendazim and trifloxystrobin and 0.125 g a.i./L for difenoconazole and pyraclostrobin, while cuprous oxide was applied at 4 g/L. Control plots received no fungicide. All fungicides were applied using an airshear sprayer (Silvan Supaflo, Australia, 2000 L), which uses very low pressure (20–30 kPa) to distribute spray with a low volume of high-speed air through 16 twin non-drip jet nozzles at ~3 L per tree. No surfactant or spreader was added to the chemicals.

Disease incidence and preharvest fruit abscission

To evaluate the efficacy of the different fungicides, the number of diseased fruit on the tree was recorded from 25 arbitrarily selected fruit from each of the four canopy aspects (within and between rows) at intervals of 2 weeks between December and March. The data were recorded as percentage of husk spot from which the area under disease progress curve (AUDPC) was determined (Akinsanmi *et al.* 2007). Disease severity was recorded between December and March on fruit collected from the ground on the same day as the disease incidence data were recorded. The fruit were classified into two categories: diseased (with lesions caused by *P. macadamiae*) and others (undamaged

Table 1. Timing and frequency of different fungicide products applied on macadamia varieties in the 2004–05 and 2005–06 seasons in Australia

WPA indicates approximate weeks post anthesis when the majority of the racemes in the orchard were fully open. Spin + Cu, tank-mix of carbendazim (SpinFlo) and copper oxide (Cu); Flint, trifloxystrobin; Cabrio, pyraclostrobin; Score, difenoconazole

Treatments (time of application) ^A	3 WPA	5 WPA	7 WPA	9 WPA	11 WPA	13 WPA	15 WPA	17 WPA
Carbendazim (MH)	Spin + Cu	–	Spin + Cu	–	Cu	–	Cu	–
Pyraclostrobin (MH)	Cabrio	–	Cabrio	–	Cu	–	Cu	–
Trifloxystrobin (MH)	Flint	–	Flint	–	Cu	–	Cu	–
Carbendazim (PS)	–	Spin + Cu	–	Spin + Cu	–	Cu	–	Cu
Difenoconazole (PS)	–	Score	–	Score	–	Cu	–	Cu
Control	–	–	–	–	–	–	–	–

^ALetters in parentheses indicate the stage of fruit development when treatment commenced. MH, match head size; PS, pea size.

and husk spot free, insect and rat damaged or damaged by pathogens other than *P. macadamiae*). The weight of fruit in the two categories was recorded. Thereafter, fruit were dehusked, pooled for each plot and the weight of wet nuts (kernel in shell) was recorded.

Harvest yield and kernel maturity

The harvest yield was determined by harvesting the fruit monthly starting from mid March (start of normal harvest period) in both years. All fruit were harvested from the ground for each individual data tree and dehusked separately using commercial dehuskers that exclude non-commercial-sized nuts (<15 mm diameter). Weights of the nuts were recorded at each harvest and the harvest yield per tree was determined as the total weight of nuts harvested from mid March until the end of fruit drop each year. A kernel quality parameter (kernel maturity) was determined from the nuts dried in a fan-forced laboratory oven for 2 days each at 38, 45 and 55°C, to ~1.5% moisture content. About 100 dried nuts from each plot were cracked using a table-top cracking machine from which the percentage of immature kernels containing oil levels below 72% was determined using a standard flotation method (Mason and Wills 1983).

Data analysis

The AUDPC and the yield data were log-transformed [$\log_{10}(x+1)$]. Any significant difference between years, field sites, fungicides and their interactions was determined by a

factorial ANOVA performed using GENSTAT Release 8.1 (Lawes Agricultural Trust, Rothamsted Experimental Station, UK). Because the strobilurin and difenoconazole products were applied at different stages of fruit development, their data were compared with the tank-mixed carbendazim + Cu treatment applied at the same time. Significant factors were separated and tested using Ryan/Elliot-Gabriel/Welsh Multiple Range tests in GENSTAT Release 8.1.

Results

Husk spot incidence

Although the climatic conditions of seasonal mean maximum temperatures of 25–33°C and monthly rainfall of 16–212 mm were favourable for infection (Mayers 1998), almost no husk spot was observed on HAES 246 in both years, while husk spot was consistently observed on A16. Therefore, only A16 data are presented. Husk spot symptoms were first observed as small chlorotic spots on the fruit in January in the 2004–05 season and in December in the 2005–06 season at all field sites. Although the number of diseased fruit increased with time throughout the season, the ANOVA of the AUDPC data showed a significant variation among the field sites, fungicide treatments and between years (Table 2). All the 2-way and 3-way interactions of the main effects (site, fungicide treatments and year) except the site × year interactions were also significant (Table 2). The AUDPC was highest in the untreated control plots at all sites in both seasons compared with all other fungicide treatments (Fig. 1). When the first

Table 2. ANOVA of the area under disease progress curve (AUDPC), yield (weight of nuts) and kernel quality (weight of immature kernels) of the macadamia variety A16, treated with different fungicide products at three field sites in the 2004–05 and 2005–06 seasons in Australia

Source of variation	d.f.	AUDPC		Preharvest yield ^A		Harvest yield ^B		Immature kernel ^C	
		Mean square	P- value	Mean square	P- value	Mean square	P- value	Mean square	P- value
Site	2	37.12	<0.001	2.64	0.04	0.57	<0.001	2.57	0.01
Year	1	145.26	<0.001	3.40	0.04	0.99	<0.001	2.87	0.03
Treatment	5	177.45	<0.001	10.15	<0.001	0.06	0.45	9.97	<0.001
Site × year	2	2.33	0.26	1.01	0.30	0.02	0.83	0.90	0.22
Site × treatment	10	9.70	<0.001	0.90	0.39	0.01	0.97	0.75	0.24
Year × treatment	5	12.90	<0.001	0.16	0.97	0.08	0.16	0.14	0.94
Site × year × treatment	10	3.75	0.02	0.56	0.76	0.06	0.12	0.46	0.64

^APreharvest yield relates to fruit harvested before end of March.

^BHarvest yield relates to fruit harvested from the end of March to August each season.

^CBased on preharvest yield data.

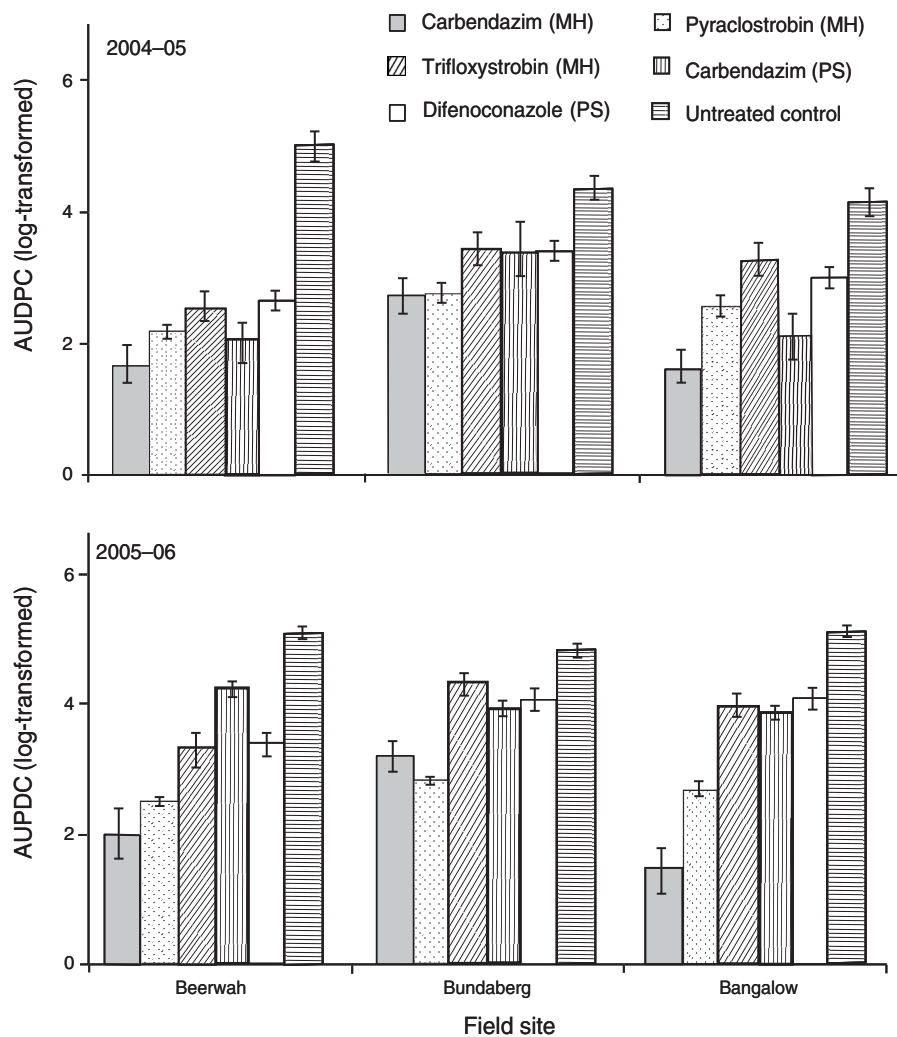


Fig. 1. Area under the disease progress curve of husk spot on macadamia treated with different fungicide products at the match head size and pea size stages of fruit development at three locations in the 2004–05 and 2005–06 seasons. Bars indicate standard errors.

spray was applied at MH stage, the AUDPC was higher in the trifloxystrobin treatment than in the pyraclostrobin and carbendazim (MH) treatments (Fig. 1). When the first spray was applied at PS stage, the AUDPC for difenoconazole treated trees was similar to that of carbendazim-treated (PS) trees except at Bangalow in the 2004–05 season and at Beerwah in both seasons (Fig. 1). Pyraclostrobin significantly ($P < 0.001$) reduced husk spot incidence more than trifloxystrobin at all sites and in both seasons. Pyraclostrobin significantly reduced disease severity by reducing preharvest abscission by more than 70%, while trifloxystrobin reduced abscission by 50% over the untreated control.

Preharvest fruit abscission and preharvest yield

Generally, abscission of premature fruit started in December in both years. However, the abscission of fruit infected with husk spot commenced in January of both years. The effect of the fungicide treatments on the level of premature fruit abscission

was significantly different between treatments (Table 2). Although site and year effects were significant ($P = 0.04$), their interactions with treatments were not significant ($P = 0.76$) (Table 2). When premature fruit were analysed by category of fruit that abscised (with husk spot and without husk spot), there were no significant differences between years for both categories (Table 3). The weight of fruit that abscised without husk spot was similar between treatments, except difenoconazole and the untreated control (Table 4), and this natural fruit abscission varied significantly between sites, treatments and their interactions with other factors (Table 3), reflecting the effect of environmental conditions on macadamia fruit abscission. Diseased fruit accounted for more than 78% of the preharvest fruit drop in all the treatments (Table 4). The mean weight of diseased fruit that abscised before harvest was significantly higher in the untreated control than in the treatments. The mean weight of diseased fruit that abscised before harvest was significantly lower in the trees treated with pyraclostrobin or

Table 3. ANOVA of macadamia fruit that abscised with and without husk spot at preharvest period following application of different fungicide products in the 2004–05 and 2005–06 seasons at three field sites in Australia

Source of variation	d.f.	Abscised fruit with husk spot		Abscised fruit without husk spot	
		Mean square	P-value	Mean square	P-value
Site	2	1.3	0.22	8.4	<0.001
Year	1	2.0	0.12	1.1	0.13
Treatment	5	15.8	<0.001	2.0	0.001
Site × year	2	0.5	0.56	8.7	<0.001
Site × treatment	10	1.2	0.16	1.2	0.01
Year × treatment	5	0.8	0.41	1.2	0.04
Site × year × treatment	10	0.9	0.38	0.9	0.08

Table 4. Mean weight of fruit (wet nut-in-shell) of the macadamia variety A16 that abscised with or without husk spot during the preharvest period following application of different fungicide products in the 2004–05 and 2005–06 seasons at three field sites in Australia

Means within the same column followed by the same letter are not significantly different ($P > 0.05$) according to Ryan/Einot-Gabriel/Welsch multiple range test

Treatments (time of application) ^A	Weight of fruit with husk spot (kg)	Weight of fruit without husk spot (kg)	Total weight of fruit that abscised (kg)	Fruit with husk spot (%)
Carbendazim (MH)	0.82ab	0.20ab	1.02a	80.4
Pyraclostrobin (MH)	0.80ab	0.22ab	1.02a	78.4
Trifloxystrobin (MH)	1.55c	0.21ab	1.76bc	88.6
Carbendazim (PS)	1.13bc	0.32bc	1.45bc	77.9
Difenoconazole (PS)	1.29c	0.34c	1.63c	79.1
Untreated control	3.13d	0.37c	3.47d	90.2

^ALetters in parentheses indicate the stage of fruit development when treatment commenced. MH, match head size; PS, pea size.

carbendazim (MH) than in those with the other fungicide treatments (Table 4). In comparison with the untreated control (3.13 kg/tree), pyraclostrobin and trifloxystrobin reduced abscission of diseased fruit by 74 and 50%, respectively. Abscission of diseased fruit was reduced by 73% in tank-mixed carbendazim + Cu treated trees and 69% in difenoconazole-treated trees (adjusted for time of application).

Harvest yield and kernel maturity

Average harvest yield (wet nuts) was highest in Bangalow (11.7 kg/tree), followed by Bundaberg (10.9 kg/tree) and Beerwah (9.5 kg/tree), while the yield of wet nuts averaged over all sites in the 2005–06 season (11.2 kg/tree) was higher than in the 2004–05 season (10.2 kg/tree). The weight of fruit that abscised during the first normal harvest was higher in the untreated control than in the treated trees at all the locations and in both seasons (Fig. 2). Therefore, a large percentage of the fruit were harvested between April and June in all the treated trees compared with March in the untreated control (Fig. 2). This early fruit abscission in the untreated control was due to the effect of husk spot on fruit abscission in macadamia, as more than 90% of the fruit had husk spot symptoms. Harvest yield varied significantly between sites and years, but not between the treatments ($P = 0.45$) (Table 2). However, the weight of immature kernels in the preharvest yield was higher in the untreated control, followed by difenoconazole, trifloxystrobin and carbendazim (PS) treatments, while pyraclostrobin and

carbendazim (MH) treatments had very few immature kernels. At normal harvest, kernel maturity was similar for all the treatments (data not shown), which showed that husk spot influenced fruit abscission and not kernel maturity.

Discussion

The main goal of this study was to evaluate alternative fungicides for the control of husk spot in macadamia. This study showed that the strobilurin fungicide pyraclostrobin is effective against husk spot in macadamia. Pyraclostrobin was more effective than trifloxystrobin at all sites and in both seasons. The efficacy of pyraclostrobin against husk spot was comparable to the tank-mixture of carbendazim + Cu and better than difenoconazole, the treatments that are currently registered for husk spot control in macadamia. Only very few immature kernels occurred in pyraclostrobin and carbendazim-treated (MH) trees at the preharvest stage. Even though the kernel maturity at normal harvest was similar for all treatments, the amount of immature kernels was significantly higher in the preharvest yield of the untreated control than in the fungicide treated trees. This confirmed that infection by *P. macadamiae* promotes premature fruit abscission in macadamia.

Although harvest yield was comparable for all of the treatments and the untreated control, the high amount of immature kernels, a measure of kernel quality, in the untreated control at the preharvest stage, would cause a considerable reduction in the value of the produce if the preharvest nuts

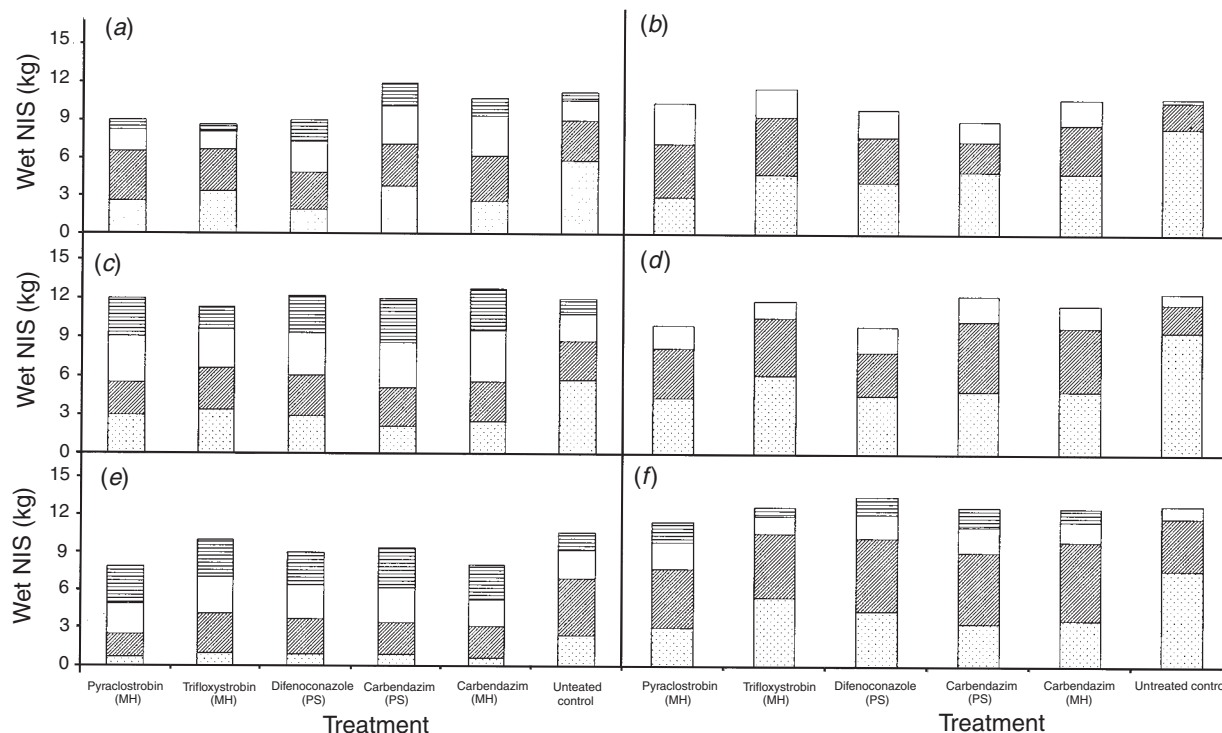


Fig. 2. Average yield of nuts-in-shell (wet NIS/kg) of macadamia trees after treatment of the fruit with different fungicides at the match head size and pea size stages of fruit development at three locations (*a–b*) Beerwah, (*c–d*) Bangalow and (*e–f*) Bundaberg in the 2004–05 (left side) and 2005–06 (right side) seasons. Sections of each bar with dots, diagonal lines, open and horizontal lines indicate yields at monthly harvests from March to June, respectively.

were harvested and mixed with the first normal harvest. The significant reduction in disease incidence following fungicide application indicated that the fungicides prevented or delayed infection of the fruit early in the season. However, *P. macadamiae* still infected green and mature fruit, thus causing more of the mature diseased fruit to abscise earlier. Consequently, more fruit were harvested in March in the untreated control than in other treatments. The similar cumulative amount of fruit obtained at harvest during each season, despite extensive preharvest fruit abscission in the untreated control, could be due to a compensation effect by the tree for the early crop loss. Compensation effects have been observed in studies on the impact of rodents on macadamia, where similar yields were obtained from rat-damaged trees and undamaged trees (Tobin *et al.* 1993, 1997). However, if higher levels of husk spot-induced fruit abscission occur immediately before the normal harvest, significant yield loss due to husk spot may occur. This yield loss could be further accentuated if fruit development is impaired by poor tree nutrition, water stress and high temperatures (Stephenson and Gallagher 1986, 1987; Trueman and Turnbull 1994).

In this study, trees were infected naturally with high disease incidence (A16 trees only), showing that an abundant level of natural inoculum was available within the macadamia orchards used in this study. However, the low disease incidence in HAES 246 trees, which were adjacent to A16 trees at Bangalow, showed that other factors besides inoculum availability influence husk

spot incidence. Preliminary studies have revealed that HAES 246 is as susceptible to husk spot as A16 (Mayers and Giles 2001); based on these studies, it is unclear why HAES 246 trees were less affected by husk spot than A16 trees in this study. One hypothesis is that HAES 246 trees were less affected because they contained few or no old, diseased husks that had failed to abscise in preceding seasons, whereas these husks were abundant in trees of A16. The abundance of old diseased husks within tree canopy has been observed to translate to a higher husk spot incidence in A16 (A. K. Miles, unpubl. data). In general, conidia of *Pseudocercospora* spp. (teleomorph *Mycosphaerella* spp.) are known to be spread by water splash, while long-distance spread is attributable to airborne ascospores (Stover 1962; Meredith 1970; Mourichon and Fullerton 1990). Therefore, the presence of old, diseased husks within the tree canopy could allow *P. macadamiae* conidia to be easily rain-splashed from the husks onto new developing fruit. Since the sexual stage of *P. macadamiae* is unknown, information on its long-distance airborne dispersal is unknown.

Although we used the existing strategy of fungicide application at intervals of 4 weeks, more information is required to determine efficacy and/or tenacity of fungicide deposits at varying frequency of application. Determining the effect of the fungicides on lesion appearance, expansion and sporulation on individual fruit or in a detached fruit assay is difficult because of the long latent period. The efficacy of the products in reducing disease incidence and severity in field

conditions implies that the products affect infection. Applying the strobilurins before infection or in the early stages of disease development should capitalise on their preventive and curative activities against spore germination (Bartlett *et al.* 2002). The consistently low level of husk spot incidence in trees treated with carbendazim at MH stage compared with treatment at PS stage confirmed our earlier findings that fungicide application started at MH stage is most efficacious (Akinsanmi *et al.* 2007). Since disease incidence in the untreated control was significantly higher than in the treated trees at all sites and in both years, fungal control treatments should not be neglected.

While husk spot is adequately controlled by treatment with a tank-mixture of carbendazim + Cu, new fungicides are desirable so that replacements are available in the event of resistance or removal of carbendazim-based products from the market. The results presented in this study suggest that strobilurin fungicides, particularly pyraclostrobin, could play a significant role in the management of husk spot in macadamia. Practical differences like translaminar activity, redistribution in the vapour phase and mobility in the plant, could have influenced the effectiveness of trifloxystrobin against husk spot (Bartlett *et al.* 2002). Introduction of pyraclostrobin into the spray programs, alternating with the DMI and/or carbendazim products will minimise the risk of developing fungicide-resistant strains of the pathogen. The more specific mode of action of strobilurin increases the risk of rapid occurrence and selection of resistant genotypes in the pathogen populations (Karaoglanidis and Karadimos 2006). Therefore, strategies such as limiting the number of applications, alternating with compounds from different cross-resistance groups and the use of mixtures with effective partner fungicides should be considered (Karaoglanidis and Karadimos 2006). Long-term strategies also need to account for the tolerance and resistance of different varieties when applying fungicides and planning new orchards.

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