

Interaction between wheat varieties and fungicides to control stripe rust for grain yield and quality

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KEY MESSAGES

- Extreme yield loss (87–94%) occurred in a susceptible variety under very high stripe rust disease pressure in a long season environment in 2007 and 2008.
- Yield losses of 27–54% were observed in varieties with partial resistance to stripe rust under high disease pressure.
- Strategic fungicide application provided significant yield benefit in all except the resistant variety, the degree of benefit was greatest in susceptible varieties.
- A fully resistant variety expressed zero yield loss, however major gene resistance has not proved to be durable for stripe rust.
- Partial stripe rust resistance combined with strategic fungicide application can be used to minimise yield losses and restrict epidemic development.

INTRODUCTION

Puccinia striiformis f.sp. *tritici*, the causal fungus of stripe rust in wheat was first detected in Western Australia in the Lake Grace Shire in 2002. Significant yield losses have been observed in susceptible wheat varieties. The use of partially resistant varieties combined with fungicide control remains a poorly understood aspect of disease management.

AIMS

To determine how varieties with different levels of stripe rust resistance respond to fungicide for the control of stripe rust.

METHOD

The interactions between varietal resistance and fungicide application on stripe rust development and crop yield were assessed in experiments in 2008 and 2009 at Manjimup Horticultural Research Station. Varieties with a range of stripe rust resistance, EGA Bonnie Rock (S-VS), Carnamah (MS-S), Wyalkatchem (MS), Janz (MR-MS) and GBA Ruby (R) were tested in combination with 3 fungicide treatments being either nil, partial or full fungicide control. Full control consisted of *Tebuconazole* (Folicur 430SC) @ 290 mL/ha applied at early stem elongation (Z31), flag leaf emergence (Z39/40), ear emergence (Z55) and late flowering (Z68) to provide maximum disease protection to maximise yield. Partial fungicide control consisted of a single application at ear emergence (Z55) in 2007 or two applications commencing with the first sign of the stripe rust (Z32) and again at ear emergence (Z55) in 2008. In 2007, the trial was sown on 5 July, adjacent to susceptible wheat (cv Harrismith) that was inoculated twice on 30 July and 23 August. In 2008 the trial was sown 20 June adjacent to susceptible wheat (cv Westonia) that was inoculated with stripe rust on 24 July 2008.

RESULTS

In both years, the stripe rust pressure was high. In 2007, the stripe rust severity ranged from 4 to 96% in untreated control plots whereas in 2008 the disease severity varied from 6 to 93 per cent among the five wheat varieties tested (Table 1). GBA Ruby (resistant) had no response to fungicide for stripe rust control. Application of fungicides either as single, double or multiple sprays reduced the stripe rust levels in all other wheat varieties, in both years. Under these experimental circumstances, where the varieties were subject to continuous disease pressure from nearby infected wheat, partial fungicide control was less effective than full protection with multiple fungicide sprays in Carnamah, EGA Bonnie Rock, Janz, and Wyalkatchem.

Over two years, extreme yield losses (87–94%) were observed in EGA Bonnie Rock under very high stripe rust disease pressure in a long season environment. In Janz and Wyalkatchem, partial resistance reduced the impact of stripe rust however yield losses of 27–54% were still observed.

Partial fungicide protection combined with partial resistance reduced yield losses to 17–30%, depending on variety. Application of fungicides significantly increased the yield compared to untreated control in all the varieties tested except for GBA Ruby (Table 1).

In 2007, screenings varied from 3.3 to 18.4 per cent among the untreated varieties whereas in 2008, it varied from 0.8 to 7.9 per cent (Table 1). EGA Bonnie Rock had higher screenings in both years. An application of fungicides increased the hectolitre weight in all varieties tested except GBA Ruby.

CONCLUSIONS

Varieties with partial to full resistance significantly out performed susceptible varieties when stripe rust was not controlled or only partially controlled in conditions that were ideal for disease development.

Additional disease control and yield benefits were achieved through strategic fungicide application to all varieties except the resistant variety GBA Ruby. Yield response to fungicide intervention was dependant on variety resistance, with the response being greatest on varieties with stripe rust rating of 'VS' to 'MS' compared to 'MR' to 'R'. Among the varieties tested, Carnamah and EGA Bonnie Rock had high stripe rust levels compared to Janz and Wyalkatchem, which in turn were higher than GBA Ruby in both years. There was no fungicide response in control of stripe rust and yield increase on the resistant variety GBA Ruby. The observed increase in yield was also associated with some reduction in screenings (mainly in EGA Bonnie Rock) and an increase hectolitre weight.

Australian and international experience shows that that single major gene resistance to stripe rust, though fully effective, is not durable in the long term. GBA Ruby carries Yr27, which is currently fully effective in WA even under high disease pressure. Recent reports indicate development of Yr27 virulence in the stripe rust population in eastern Australia.

Some varieties such as Yitpi and Wyalkatchem carry partial stripe rust resistance. Partial resistance can be derived from several genes and generally has proved to be more durable than single major gene resistance against stripe rust.

Partial resistance genes usually express after flag leaf emergence to head emergence stages and are commonly known as adult plant resistance (APR). APR is sensitive to high disease pressure and environmental conditions which favour stripe rust, such as the conditions experienced in these experiments. The first visible stripe rust symptoms were observed at early stem elongation in 2008 and early booting in 2007. The early occurrence of rust and favourable long season environment placed maximum pressure on the varieties with partial resistance in these experiments.

The varieties with partial resistance genes showed high levels of infection, however yield from these varieties was significantly higher than in susceptible types. In general, partial stripe rust resistance combined with strategic fungicide application can minimise yield losses and restrict epidemic development, particularly in conditions that favour prolonged disease development.

KEY WORDS

wheat, stripe rust, foliar fungicide spray

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Table 1. Stripe rust severity, yield and some grain quality parameters in results of interaction between wheat varieties and fungicide program at Manjimup from 2007 to 2008.

Year	Variety	% disease severity at Z75/Z77* (avF nec to avF-1 nec)			Yield (t/ha)			Screening (%)			Density (kg/HL)		
		Nil	Partial control †	Full control #	Nil	Partial control	Full control	Nil	Partial control	Full control	Nil	Partial control	Full control
2007	Bonnie Rock	100a**	66b	19c	0.5a	2.1b	3.9c	18.4a	8.8b	2.1c	57a	70b	78c
	Carnamah	90a	81a	41b	1.6a	2.6b	4.2c	8.4a	6.3ab	3.6b	65a	70b	76c
	Wyalkatchem	53a	48a	26b	2.0a	3.1b	4.1c	3.3a	2.1a	1.3a	68a	76b	80c
	Janz	50a	42a	14b	2.5a	3.1b	4.1c	8.1a	5.4ab	3.1b	73a	78b	80b
	Ruby	4a	3a	2a	3.8a	4.1a	3.9a	3.9a	3.8a	5.5a	80a	80a	80a
2008	Bonnie Rock	93a	53b	14c	0.2a	1.1b	3.4c	7.9a	5.5b	2.6c	62a	70b	75c
	Carnamah	58a	41b	16c	1.8a	2.8b	4.0c	1.8a	1.7a	2.0a	71a	74b	76b
	Wyalkatchem	35a	31ab	21b	2.1a	3.2b	4.6c	1.0a	0.9a	0.6a	71a	77b	79b
	Janz	26a	18a	7b	2.9a	3.3a	4.0b	2.1a	1.6a	1.1a	75a	77ab	78b
	Ruby	6a	5a	4a	4.6a	4.6a	4.6a	0.8a	1.0a	0.9a	80a	80a	80a

† Partial fungicide control consisted of a single application (Folicur @ 290 mL/ha) at ear emergence (Z55) in 2007 or two applications commencing with the first sign of the stripe rust (Z32) and again at ear emergence (Z55) in 2008.

Full fungicide control consisted of Folicur 430SC @ 290 mL/ha applied at early stem elongation (Z31), flag leaf emergence (Z39/40), ear emergence (Z55) and late flowering (Z68)

* per cent leaf area under necrosis due to stripe rust infection at approximately milk development stage (Z75/77) on the top two leaves Flag to Flag-1.

** Means followed by the same letter in the same row for each parameter are not significantly different at the $p = 0.05$ level.

Findings of canola disease survey 2008 and its implications for better disease management in 2009

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KEY MESSAGES

- The survey results indicated that levels of *Sclerotinia* stem rot were substantially high in the northern agricultural region and some crops were also badly affected in the southern areas.
- Levels of blackleg were low in northern areas, moderate in central and high in southern areas.
- Incidence of club root was generally low in all areas except for a few crops in the northern region that had high incidence of mild forms of club root.
- Powdery mildew levels were high in the northern region, low in the southern region and no powdery mildew was observed in the central region.
- Survey results highlight that *Sclerotinia* and blackleg were the two predominant diseases affecting canola yields in 2008. Growers need to carefully manage both these diseases in order to minimise losses.

AIMS

To monitor root and stem diseases in canola crops to provide timely feedback to the industry about the fungal diseases which need to be considered in establishing and managing canola crops.

METHOD

Samples were collected from canola crops across the Western Australian grainbelt. About 100 stems were collected for each sample along a 200 m transect. Stems were washed and rated for severity of internal infection of blackleg on a 0–4 scale (0 = no disease, 4 = more than 75% stem cross section showing internal necrosis). Disease incidence (% plants with crown cankers) and disease severity expressed as percent disease index (PDI) for each sample were calculated. Plants were also assessed for the incidence of *Sclerotinia* stem rot (SSR) caused by *Sclerotinia sclerotiorum*, club root caused by *Plasmodiophora brassicae* and powdery mildew caused by *Erysiphe cruciferarum*.

RESULTS

A total of 74 samples were collected from canola crops. About 16% samples were cultivar (cv) Beacon, 15% cv Thunder, 14% cv Cobbler and 12% each cv Bravo and cv Tornado. A majority of the crops were sown in paddocks where there had been at least a three year break since the previous canola crop. Prevalence of disease (percentage crops affected) was highest (100%) for blackleg followed by that of *Sclerotinia* stem rot, club root and powdery mildew respectively (Figure 1). When averaged across all samples, the incidence of blackleg was highest compared with that of other diseases (Figure 2). Average severity of internal infection of blackleg was 23%. The range of incidence within a sample, across all samples, was 6–95% for blackleg, 0–65% for *Sclerotinia* stem rot, 0–44% for club root and 0–98% for powdery mildew. Average incidence of various canola diseases in different agricultural regions is presented in Figure 3.

Implications of findings of 2008 canola disease survey

Although blackleg levels were lower particularly in the northern agricultural region than reported from previous surveys (conducted during 2001–2004). Low levels of blackleg in the northern region were expected due to continuous dry conditions during the past few years and very little canola being grown in the region. As a result the 2008 canola crop was subjected to low blackleg inoculum levels. This year, canola growers in all areas need to carefully manage blackleg in their canola crops given there is sufficient blackleg inoculum around. The wetter than normal conditions over summer may cause the early spore production from the 2008 stubble therefore, posing the risk of major spore showers coinciding with the susceptible seedling stage of the crop.

Sclerotinia stem rot incidence appears to have risen in the past few years. High levels of SSR in some crops indicate growers need to manage the risk of Sclerotinia in their current season's crops. They should avoid sowing close to paddocks that had high incidence of Sclerotinia in 2008. In high risk situations, growers should use fungicides recommended for the control of Sclerotinia in canola.

Although club root didn't emerge as a serious problem, its widespread incidence means that growers are required to maintain good hygiene practices and be extra cautious in order to prevent the spread of this pathogen with machinery to club root free paddocks. Other options to prevent the spread of club root are the control of Brassica weeds, long rotations and sow club root free seed. This disease is recognised as a key disease in Alberta, Canada as it has spread rapidly and caused significant losses of yield in canola crops.

High levels of powdery mildew were also found in some crops, however, the impact of powdery mildew on canola yields is not known. Further work is required to determine the effect of powdery mildew on canola production.

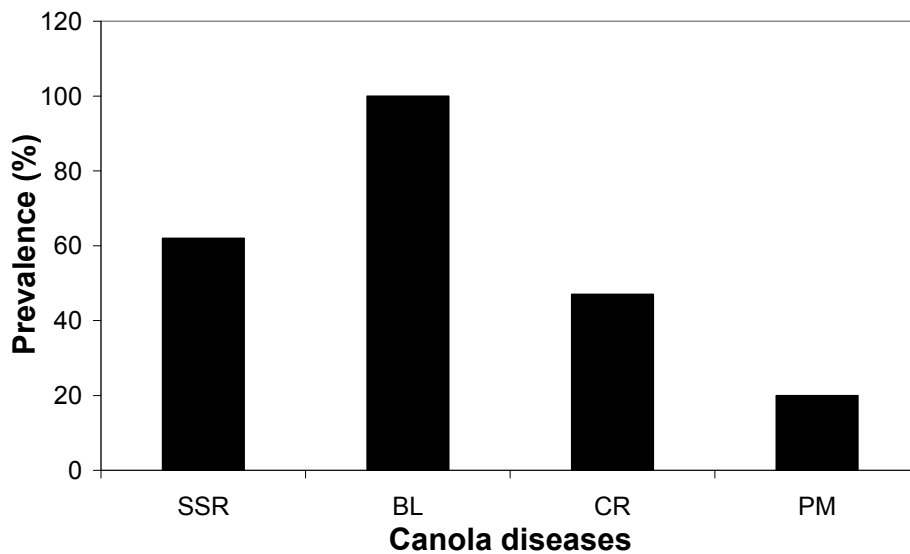


Figure 1 Prevalence (percentage crops affected) of various canola diseases in Western Australia during 2008. (SSR = Sclerotinia stem rot, BL = Blackleg, CR = club root, PM = powdery mildew.)

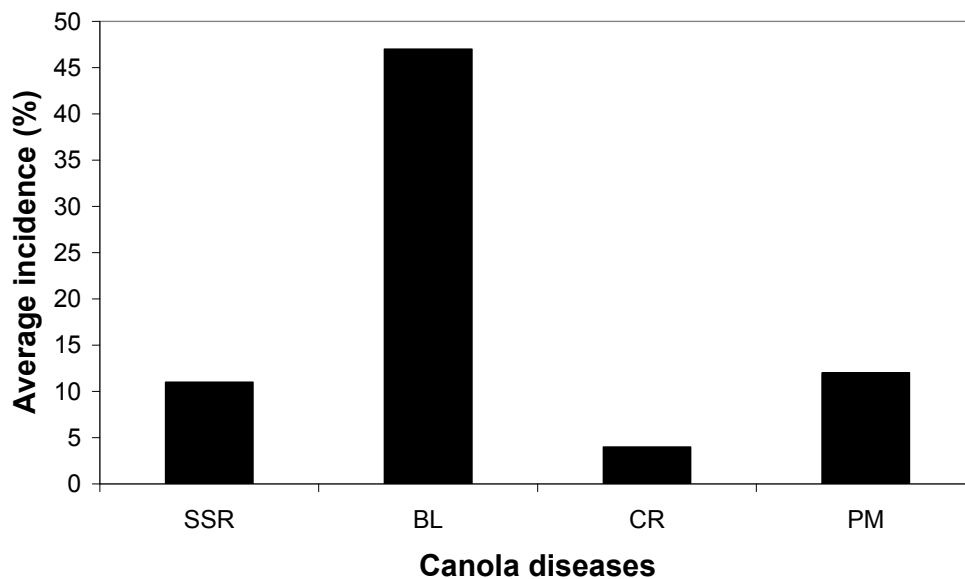


Figure 2 Average state-wide incidence of various canola diseases in Western Australia during 2008. (SSR = Sclerotinia stem rot, BL = Blackleg, CR = club root, PM = powdery mildew.)

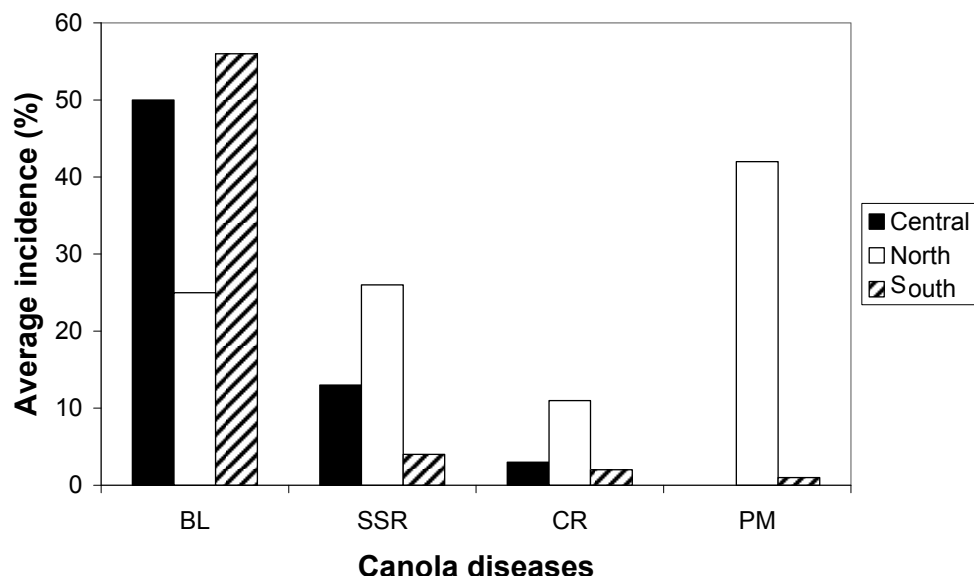


Figure 3 Average incidence of various canola diseases in different regions of Western Australia during 2008. (SSR = Sclerotinia stem rot, BL = Blackleg, CR = club root, PM = powdery mildew.)

CONCLUSION

Growers need to minimise the risk of infection by blackleg and SSR, in particular, avoid sowing their 2009 crop close to paddocks badly affected with Sclerotinia in 2008. Club root levels were low in most crops but this disease was widely distributed across canola growing regions of WA. Powdery mildew was also detected for the first time particularly in the northern agricultural region, however, its impact on canola yields is not known.

In 2009, seasonal conditions appear to be conducive for blackleg, growers need to reassess their risk from blackleg, especially if growing moderately resistant varieties, in order to minimise yield penalties. Growers are also encouraged to use fungicides to protect crops in which the susceptible seedling stage will coincide with the peak of spore showers. The risk from spore showers in different regions will be updated regularly and available through the Department of Agriculture’s website (www.agric.wa.gov.au/cropdiseases) or Pestfax.

KEY WORDS

canola, blackleg, Sclerotinia stem rot, club root, canola powdery mildew, disease survey

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Combating wheat leaf diseases using genome sequencing and functional genomics

Richard Oliver, Australian Centre for Necrotrophic Fungal Pathogens, Murdoch University

KEY MESSAGE

Detailed studies of fungal pathogen genes based on the genome sequences of *Stagonospora nodorum* (cause of stagonospora/septoria nodorum/glume blotch) and *Pyrenophora tritici-repentis* (cause of yellow spot) suggest that wheat cultivars can be differentiated on the basis of their genetic predisposition to sensitivity to an important fungal toxin and this information will assist growers in selecting lower risk varieties and assist breeders in selecting improved resistance in breeding. These developments should contribute significantly to integrated disease management to reduce losses to these diseases.

BACKGROUND

Two of the major diseases of wheat are *Stagonospora* (*Septoria*) *nodorum* blotch (SNB) resulting in leaf and glume blotch (caused by *Stagonospora nodorum*) and yellow spot (YS) (caused by *Pyrenophora tritici-repentis*). Both can cause losses of up to 20–40% in affected crops. SNB is mainly a problem in WA whilst YS is common in most wheat growing areas in Australia. Many diseases caused by similar fungi in related genera have stubble survival stages and have been exacerbated by the adoption of reduced tillage. These include the wheat diseases SNB and YS, the barley net-blotch diseases, canola black-leg and the legume black spot and ascochyta blights.

No wheat cultivars are completely resistant to either SNB or YS and many currently grown and newly released cultivars are classified as moderately susceptible or susceptible. As resistance is incomplete and genetically complex, the development of molecular tools that assist breeding for resistance (marker-assisted selection) has been very challenging and few have been adopted by the breeders.

RESULTS

The Australian Centre for Necrotrophic Fungal Pathogens (ACNFP) has undertaken a study of *Stagonospora nodorum* based on the analysis of the entire fungal genome sequence of a Western Australian isolate. The 38 million base pair sequence is the largest genomics project undertaken and analysed by any group in the Southern hemisphere. The identification of the genes encoded by this pathogen has revolutionised our understanding of the interaction with wheat. In collaboration with groups in North Dakota and Zurich, we have shown that *Stagonospora nodorum* produces a series of toxins that interact with specific wheat receptors. One of these toxins is also produced by *Pyrenophora tritici-repentis*, the YS pathogen. The fungal isolate produces a toxin and if the wheat cultivar possesses the relevant receptor gene, the interaction produces a leaf spot that enables disease to flourish. Without the relevant receptor gene the disease is slowed, but not eliminated. This revelation has opened up an improved pathway for the control of these diseases.

We have so far obtained detailed evidence for the expression of three toxins by Australian isolates of SNB. The toxin we know most about is called ToxA. Both SNB and YS produce this toxin and the wheat receptor gene has been identified and designated *Tsn1*. Cultivars that lack this receptor gene are easily identified using either molecular markers or the reaction to the ToxA protein and are more resistant to SNB and YS. We therefore suggest that growers avoid using cultivars that contain the receptor gene and are therefore more susceptible to SNB/YS. There appears to be no deleterious effect if the receptor gene is absent. We are also working with the breeders so that they can remove the gene from breeding lines being used to create the next generation of varieties.

Varieties without the toxin receptor gene *Tsn1* have a lower than average risk of severe YS and SNB diseases. These include Blade, Brookton, Calingiri, Carnamah, Cascades, Cunderdin, EGA blanco, EGA bonnie rock, EGA eagle rock, GBA ruby, H45, Machete, Nyabing, Perenjori, Westonia, and Wyalkatchem.

Varieties with the toxin receptor gene *Tsn1* have a higher than average risk of severe YS and SNB diseases. These include Amery, Arrino, Binnu, Bullaring, Cadoux, Camm, Carinya, Catalina, Chara, Clearfield JNZ, Clearfield STL, Corrigin, Datatine, Drysdale, EGA2248, EGA Gregory, EGA Jitarning, Eradu, Frame, GBA Sapphire, Gladius, Halberd, Harrismith, Janz, Kalannie, Kennedy, Kukri, Kulin, Mira, Mitre, Schomburgk, Sentinel, Silverstar, Spear, Stiletto, Sunco, Tammarin Rock, Tincurrin and Yitpi.

CONCLUSION

The identification of toxins in the SNB/YS system has identified a step-by-step breeding strategy to reduce the incidence of these diseases. We expect that implementation of this and related knowledge of major toxin genes in breeders' selections will lead to a reduction in the impact of this disease progressively over successive breeding cycles. We emphasise that these pathogens produce a number of toxins and so *Tsn1* will be responsible for only part of the resistance response. The next research question is whether other similar pathogens—the barley net-blotch diseases, canola black-leg and the legume black spot and ascochyta blights—can be attacked in the same manner.

KEY WORDS

Stagonospora, Septoria nodorum blotch, glume blotch, yellow spot or tan spot, barley net-blotch diseases, canola black-leg, pea black spot, ascochyta blights

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Distribution and survival of wheat curl mite (*Aceria tosichella*), vector of *Wheat Streak Mosaic Virus*, in the WA grainbelt during 2008

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KEY MESSAGES

Volunteer wheat collected during April to July, which was at the heading growth stage, were 100% infested with wheat curl mite (*Aceria tosichella*; WCM). Samples collected from sown wheat from seedling to flowering growth stage had no detected infestation, however, sown wheat in the grain fill growth stage (October-November) were 67% infested.

Field trials conducted near Merredin and Wongan Hills indicated that the presence of an infested slowly dying host in the immediate vicinity of a wheat crop, could contribute considerably to the eventual populations of WCM present on the wheat crop.

These investigations confirm the wide distribution of the mite over the WA grain belt and confirm the value of practising some form of control of mite numbers in high *Wheat Streak Mosaic Virus* (WSMV) risk areas. For such situations we propose the control of volunteer wheat and other grass hosts by ensuring this material is dead or removed by grazing by at least two weeks prior to germination of the sown wheat crop.

INTRODUCTION

WCM may have been in WA for a long time. However, the first documented record of its presence dates from a survey conducted in 1993. WCM breed rapidly in warm weather, with the optimum temperature range for reproduction between 24–27 °C. The mite is well adapted for aerial dispersal, and relies on this method for its survival, requiring the continuous presence of a living, green host. WCM is the only known vector for WSMV and direct damage by feeding results in minor and mostly undetectable yield loss in WA field conditions.

In autumn 2006, WSMV was detected for the first time in WA. Surveys for the virus in spring 2006 found it to be widespread throughout the WA grainbelt with incidences in crops ranging from < 1–100%. The potential impact of WSMV infection on yields was unknown under WA conditions as hot, dry summers and cool winters were unlikely to favour WCM populations and virus spread. However, in 2006 a commercial crop of wheat growing near Merredin was found to be heavily infected (incidence >95%) with WSMV. The crop was infected at the early seedling growth stage by mites transferring the virus from advanced self sown, herbicide treated wheat plants within the paddock. The WSMV infection resulted in an estimated 80% yield reduction compared to nearby healthy crops. Furthermore, WSMV has been shown to be seed borne in wheat at low levels (< 1%).

As there are no in-season control options for WCM or WSMV, control of volunteer wheat prior to germination of sown wheat crops is considered the most effective strategy. Previous cases of high WCM and WSMV infection resulting in yield loss have occurred in situations where wheat crops were germinating in the presence of a large source of infected wheat volunteers or other grasses that followed summer and autumn rains. Where these host plants are sprayed with herbicides such as glyphosate, their gradual death encourages the movement of viruliferous mites onto the young wheat crop.

AIMS

- To investigate the presence and distribution of WCM throughout the WA grainbelt at various times of the year, and various growth stages of wheat and other grasses.
- To determine the risk of WSMV infection to wheat crops where the virus may be present from viruliferous mites or seed-borne sources.
- To investigate the ability of WCM to build up and disperse onto healthy plants in field conditions within the central wheatbelt of WA.

METHOD

WCM surveys

Volunteer wheat was sampled opportunistically during April-July and multiple samples were taken from the 14 locations found with volunteers. Approx. 50 locations were sampled throughout the wheatbelt during May-September which ranged from seedling to flowering growth stages. Sampling was limited at these growth stages given the high time and labour inputs of whole-plant inspections under stereo microscopy. Each site consisted of randomly sampling 20 whole seedlings or 30 tillers depending on available growth stages. Wheat leaves were pinned on foam boards and inspected for mites under stereo microscopes.

During October-November, samples were taken along crop edges (within a 50 m length) at 92 locations situated throughout the WA grainbelt at grain fill growth stage. For samples at the ear emergence growth stage or later, 20 ears were trimmed of awns, placed on strips of black sticky contact and allowed 7–9 days to dry. Mites crawled out of the ear as it commenced to dry and were trapped on the uniform black contact film, allowing subsequent counting of the light coloured mites under a stereo microscope. The black sticky contact method provided a simple and reliable means of assessing mite numbers in wheat ears, especially where numbers were too low to detect consistently directly on the intact head.

Subsamples of mites found were confirmed as the wheat curl mite species (*Aceria tosichella*) using morphological characteristics. As mites could not be removed intact from the sticky contact surface, 30 ears were sampled at sites and 10 of them left in reserve under refrigeration. These reserved heads were used to extract mites from locations where the mites were in high numbers (i.e. 10 or more per ear). Ears were dissected under stereo microscope and mites slide mounted for identification under 1000x oil immersion and phase contrast compound microscopy.

Leaf samples from the 64 locations sampled during April-September were tested for WSMV by ELISA using WSMV-specific antibodies. Samples during October-November could not be tested due to lack of green leaf material.

Field trials (Wongan Hills and Merredin)

Identical field trials were done at Merredin and Wongan Hills research stations. WSMV infected/WCM infested wheat plants were transplanted into plots of healthy wheat cv Calingiri at crop emergence so as to simulate 0%, 0.2%, 0.5% and 1% seed infection (0, 4, 18 and 36 'infectors' plants per plot). The trials were a randomised complete block design with 6 replicates; plots were 5.4 x 10 m with 20 m buffer between plots. The 'infectors' plants were infected with WSMV by sap inoculation and then manually infested with WCM from a reared colony in glasshouse prior to being transplanted into plots.

Samples of wheat leaves, whole plants or heads were taken throughout the season to determine the level of WCM abundance and spread. Random leaf samples were taken from each plot every four weeks to determine WSMV incidence. The leaves were tested by ELISA using WSMV-specific antibodies.

RESULTS

Autumn survey (April-July)

WCM were found in relatively high numbers in wheat at all 14 sites sampled throughout the WA grainbelt from April to July where wheat was in head (Figure 1). The volunteer plants were surviving in pockets (often less than 10 plants) of heading volunteer wheat and grasses in roadside ditches and paddock edges where they had not been controlled by herbicide or grazing. WSMV was confirmed to be present with WCM near Doodlakine, Munglinup and Mount Madden in wheat volunteers, at the heading growth stage, originating from spilled grain in roadside ditches. Opportunistic sampling of wheat volunteers prior to ear emergence growth stage revealed no presence of WCM (40 locations). WCM were also found on feathery windmill grass (*Chloris virgata*) and paspalum (*Paspalum* sp.) bordering infected wheat in ear at two sites.

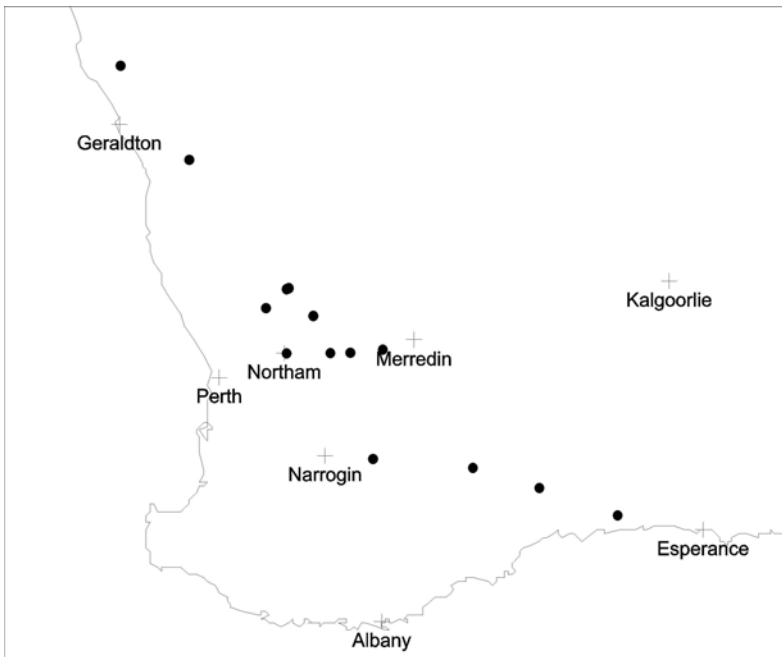


Figure 1 Locations of volunteer autumn wheat in ear found between April and July 2008. WCM present at all locations.

Seedling to flowering (May-September)

WCM were not detected on any of the 50 samples collected from the edges of sown wheat paddocks throughout the grainbelt prior to early grain fill growth stage. WSMV was also not detected.

Grain fill growth stage (October-November)

A survey of wheat ears at the soft dough growth stage during October-November revealed varying levels of WCM at 62 of 92 sites sampled (67%) (Figure 2).

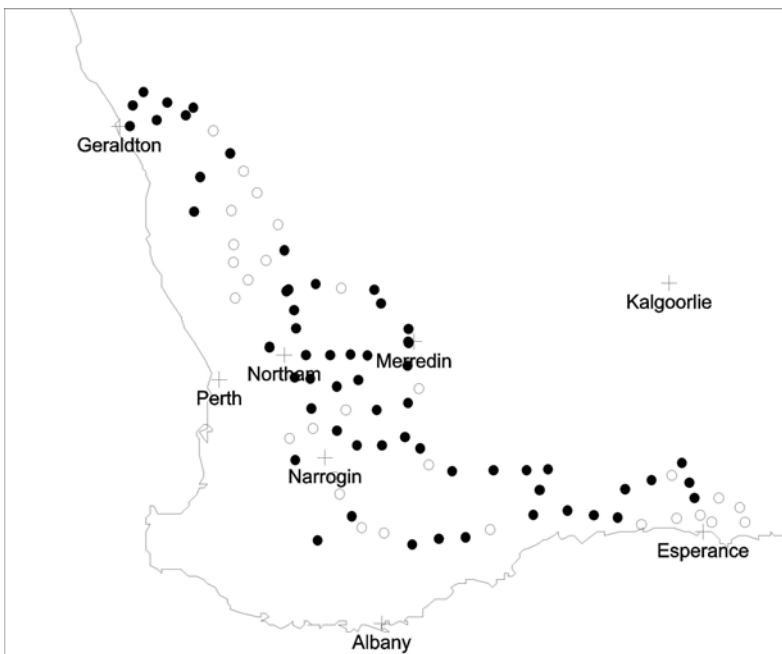


Figure 2 Sampling locations indicating presence (filled circle) and absence (open circle) of WCM within 20 randomly sampled wheat heads at soft dough growth stage (October-November).

Field trials (Wongan Hills and Merredin)

Very low numbers of WCM were found at Wongan Hills and Merredin when sampling seedlings, tillers and flowering heads, even in the plots with high numbers of 'infector' plants introduced (36 infector plants/plot to simulate 1% WSMV-infected seed).

At Wongan Hills, the treatments 0, 4, 18 and 36 infector plants introduced into plots resulted in the means of 52.5, 68.3, 65.0 and 71.7% of sown wheat plants infected with WCM, respectively, when sampled at the soft dough growth stage.

At Merredin, the treatments 0, 4, 18 and 36 infector plants resulted in the means of 9.2, 11.7, 21.7 and 63.3% of sown wheat plants infected with WCM, respectively, when sampled at the soft dough growth stage.

WSMV was detected in plots at Merredin on the final sampling date (14 October) only. The virus was detected in plots with 4, 18 and 36 'infector' plants at incidences of 1–2%. At Wongan Hills, WSMV was not detected on any sampling date in any plot.

DISCUSSION

Field survey

Although no outbreaks of WSMV were reported in 2008, the field surveys have shown that large populations of WCM are present during April-July. Therefore the risk of WSMV epidemics exists in all regions of the WA wheatbelt, particularly at locations with a history of WSMV. The presence of WSMV and spread by WCM will be more significant where summer/autumn rainfall allows for a build-up of WSMV-infected host plants and subsequent infection of wheat crops during early plant growth.

The abundance of WCM and WSMV in the WA grainbelt is considered to depend on pre-season rainfall encouraging a germination of self sown wheat and other grasses that act as hosts to the virus and mite that coincides with autumn temperatures above 24 °C.

The summer/early autumn of 2007/08 was dry and few samples were available during the autumn sampling from April-July 2008. It was therefore surprising to find high numbers of WCM on the few heading wheat volunteers at this time. Some pockets of WCM-infested wheat were even found isolated from other host plants by distances greater than 500 m (e.g. next to bush reserves). These finds demonstrate the mite's strong survival ability and dispersal over widespread areas. WCM populations are anticipated to be in much higher numbers in seasons where a wetter summer/autumn and widespread wheat volunteers are present.

The extremely low populations of WCM found during the May-September survey period on young wheat crops appears to be related to a number of possible causes including:

- low initial population sources;
- late dispersal of mites from green autumn host plants; and
- cool winter temperatures not conducive to mite build-up.

However, in spite of the low winter population, build-up and dispersal during early spring is evident from the heading samples taken during October-November.

Field trials (infector plants)

The Merredin and Wongan Hills field trials have shown that WCM can eventually infest a high proportion of wheat plants in a paddock from an initial low number of infested plants. The levels of infestation will generally increase relative to abundance of nearby infested host plants, with levels having reached 63.3% at Merredin and 71.7% infestation at Wongan Hills from 36 infector plants scattered amongst 54 m² plots of wheat (1% initial infection).

Despite the high level of WCM found during late spring, no WSMV was detected in collected leaf samples from Wongan Hills and very low levels (1–2%) were detected at Merredin. The absence of mites in samples taken at the seedling stages suggests that there was very little mite movement from

the transplanted infector plants until late in the season. It appears that the mites were content to stay on the infector plants until the plants commenced to die naturally, which eventually encouraged the mites to migrate off to the nearby crop plants. At this stage the sown plants were advanced and commencing to form ears.

CONCLUSION

The survey and field trial results have confirmed certain aspects of WCM dispersal behaviour. Since the presence of WCM is essential in the transmission and widespread infection of WSMV, known hot spots of mites should be targeted for timely control. High populations of viruliferous mites need to be dispersing and landing on new seedlings within the seedling growth stages for WSMV to have a significant impact on crop yield. This information should be taken into account in the development of a management strategy. Later infection periods are likely to be of little consequence on yield. Yield loss has generally been minimal or not detected where wheat crops are infected post-tillering. The mass dispersal of WCM from host plants will be delayed until conditions on the host are unfavourable for the mites. This could be the premature death of wheat host plants by herbicides used for weed control or the natural aging and eventual death of plants. The abundance of nearby WCM source plants during dispersal will generally determine the mite and disease transmission level. Prevailing winds accompanying the mite dispersal phase will also influence population spread and direction.

Higher risk WCM/WSMV situations occur in seasons with high summer/autumn rainfall promoting host plants, and warmer autumn temperatures favouring WCM reproduction and dispersal. As there are no in-season control options for WCM or WSMV, control of volunteer wheat and grasses prior to crop germination remains essential for an effective management strategy.

Based on the current observations as well as literature on the population dynamics of WCM, it follows that WCM could survive for some time after the death of the host. Thus, after herbicide spray, a break of at least two weeks between complete death of host plants and crop germination is important to ensure the death of WCM present in the paddock.

KEY WORDS

Wheat Curl Mite, WCM, *Aceria tosichella*, Wheat Streak Mosaic Virus, WSMV

ACKNOWLEDGMENTS

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Paper reviewed by: Bill MacLeod

Partial resistance to *Stagonospora* (*Septoria*) Partial resistance to *Stagonospora* (*Septoria*) *nodorum* blotch and response to fungicide in a severe epidemic scenario

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KEY MESSAGES

1. Use of varietal resistance to *Stagonospora nodorum* blotch (SNB) reduces the requirement for fungicide in control of this disease.
2. In severe epidemics fungicide can reduce the impact of SNB on both susceptible and partially resistant varieties.
3. The highest level of disease control on the upper two leaves (using a single fungicide application) is achieved when fungicide is applied at early booting.
4. Timing of fungicide application may be less critical for partially resistant varieties, enabling the optimisation of fungicide use for this disease together with other diseases, such as stripe rust.

INTRODUCTION

Stagonospora nodorum is a major fungal pathogen of wheat and other cereals in many parts of the world. It induces *stagonospora nodorum* blotch (SNB) of leaves and glumes and is one of the most severe fungal diseases affecting wheat production in Western Australia. A 30–50% reduction in grain yield has been reported with an estimated loss to grain growers in excess of \$58 million annually in wheat growing regions with high disease pressure. These studies were conducted as part of a broader study on disease assessment with SNB to distinguish varietal and fungicide effects at various stages of plant growth.

AIMS

To determine the optimum timing of fungicide application for the control of SNB in wheat varieties varying in resistance.

METHOD

Cultivars Millewa (susceptible) and EGA Blanco (moderately resistant) were used in this study. The experiment was sown in 2007 at South Perth in an irrigated field nursery in a split-plot design with three replications. An artificial epidemic of SNB was created by spreading infected straw around the plots and by infecting a pre-grown buffer of the susceptible variety Tincurrin planted around the plots. Treatments consisted of Folicur (Tebuconazole) application @ 125 g/ha after disease measurement on the second highest leaf showing infection at various stages of plant growth {6 weeks after sowing (WAS) (tillering), 8 WAS (stem extension), 10 WAS (early booting), 12 WAS (heading), 14 WAS (anthesis) and 16 WAS (soft dough)} as infection moved up the canopy. Treatments with no Folicur application and total protection (Folicur application at fortnightly intervals) were also included. A final assessment of disease on the flag leaf (F) and the leaf below (F-1) was made at the 16 WAS on all treatments.

RESULTS

High levels of disease were observed on plots exposed to infection and not protected with fungicide (Figure 1). Leaf disease severity of Millewa was significantly higher than EGA Blanco throughout the experiment indicating the effectiveness of partial resistance in EGA Blanco in reducing leaf disease development.

When fungicide application timing was compared as final leaf disease severity at 16WAS variety Millewa was more sensitive to variation in fungicide timing than EGA Blanco (Figure 2). The highest level of disease control on the upper two leaves (using a single fungicide application) was achieved when fungicide was applied at early booting (10WAS). Inferior disease control was observed in Millewa with 2–4 weeks earlier or later application of fungicide. Disease control on EGA Blanco was less sensitive to fungicide timing and application of fungicide over a 4 week period (8, 10 or 12 weeks) resulted in similar levels of disease control.

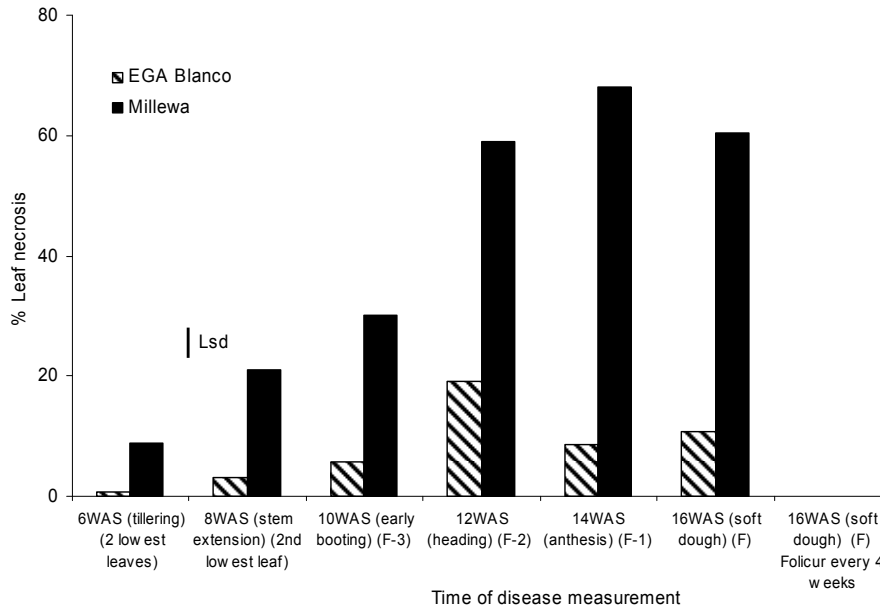


Figure 1 Disease progress of *Stagonospora nodorum* blotch up the plant canopy at various stages of plant growth in wheat varieties Millewa and EGA Blanco. WAS = Weeks after sowing; F = Flag leaf; F-1 = Leaf below the flag leaf; F-2 = Second leaf below the flag leaf; F-3 = Third leaf below the flag leaf.

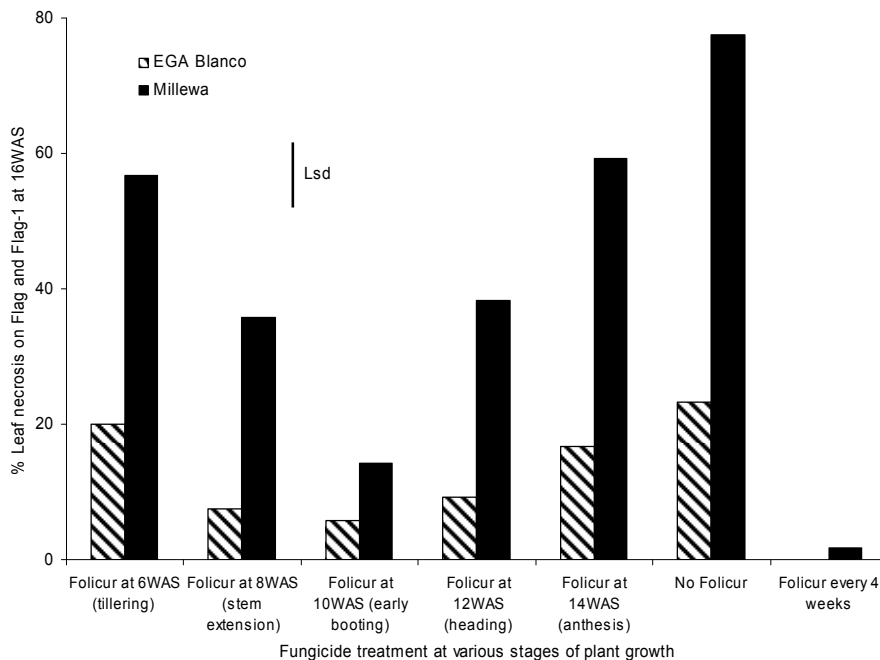


Figure 2 Final assessment of *Stagonospora nodorum* blotch on the flag leaf and the leaf below (Flag-1) at 16 WAS (weeks after sowing) after a single application of fungicide at various stages of plant growth in wheat varieties Millewa and EGA Blanco.

CONCLUSION

The response of EGA Blanco compared to Millewa indicates that the development and use of variety resistance to SNB will reduce the requirement for fungicide in control of this disease. In severe epidemics fungicide can also reduce the impact of SNB on a variety that is partially resistant to this disease, with the added benefit that timing of fungicide application may be less critical than for more susceptible variety, thus enabling the optimisation of fungicide use for this disease together with other diseases, such as stripe rust.

KEY WORDS

partial resistance, Tebuconazole, timing of fungicide application

ACKNOWLEDGMENTS

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Paper reviewed by: Geoff Thomas

Black pod syndrome in lupins can be reduced by regular insecticide sprays

Peter White and Michael Baker, Department of Agriculture and Food, Western Australia.

KEY MESSAGES

Competition for assimilates between pods on the main stem and lateral branches does not seem to be the cause of black pod syndrome.

Some evidence indicates that Bean Yellow Mosaic Virus (BYMV) may be one cause of black pod syndrome in lupins, but this still needs to be confirmed.

BACKGROUND

Lupins grown in mild high rainfall environments, particularly in the southern wheatbelt, sometimes produce yields considerably below their potential. Many pods on these plants appear black and contain seeds that are flat and underdeveloped. We have termed this disease black pod syndrome; its cause is unknown.

Jenabillup was released in 2007 because it showed some resistance to black pod syndrome. In trials where black pod syndrome was severe, Jenabillup produced 30% more yield than Mandelup (White et al. 2007). Despite its improved resistance, however, the yield of Jenabillup was still reduced by black pod syndrome in these trials. A better understanding of the causes of black pod syndrome will enhance yields further by improving crop management and breeding for resistance.

Observations by farmers and results from trials indicate that BYMV may have some role in causing black pod syndrome, but evidence is scant and there may be other causes.

AIMS

Identify further avenues of research into the causes of black pod syndrome in lupins by determining if the severity of symptoms of black pod syndrome can be increased or decreased by either: regular applications of insecticide, supplying a full complement of nutrients to plants, or removing some pods from plants.

METHOD

A field trial was conducted at the Esperance Downs Research Station. It was sown on 17 May and located on a paddock that had grown wheat the year before. Four genotypes (Quilinoch, Jenabillup, Mandelup, Tallerack) received three types of treatment (regular insecticide sprays, full nutrient application, control (untreated)) replicated four times. A split-plot design was used with the treatment as the main plot and genotype as the sub plot. Treatments were designed to cause differences in the severity of black pod syndrome and were not designed to investigate possible management options for black pod syndrome.

The regular insecticide treatment contained a mixture of Confidor (200 mL/ha) (plus a pulse penetrant) and Fastac (500 mL/ha) and was sprayed onto plants every two weeks starting from the emergence of the lupin seedlings. Buffers were placed in the trial to separate the sprayed from the unsprayed treatments. The full nutrient treatment contained a commercial granulated micronutrient fertiliser plus urea. Urea was applied at 180 kg/ha, split into three equal doses (60 kg/ha each) broadcast at sowing, flowering and podding. The commercial fertiliser was drilled with the seed at sowing. Plots received the following rates (kg/ha): 6, Cu₂SO₄; 4, ZnSO₄; 17, MnSO₄; 0.2, CoSO₄; 25, MgSO₄; 120, K₂SO₄; 0.25, Na₂MoO₄. Nutrients applied in this treatment were in addition to the basal fertiliser rates applied to all treatments across the trial. Basal fertiliser was applied as a sulphur coated superphosphate with additional MnSO₄. Rates of nutrients applied were (kg/ha): 14, P; 12, S; 10, Ca; 8 Mn.

About 13 weeks after sowing plants were at the open flower point on the main stem and some had reached the budding point on the primary lateral branches. At this stage five plants were selected at random from plots of Mandelup and Jenabillup and the top three primary lateral branches were

removed from the plant. Similarly five more plants were selected from the same plots and all branches and leaves were removed below the top three primary lateral branches. All of these plants were tagged and harvested separately at maturity.

RESULTS

Symptoms of black pod syndrome

Some plants in all treatments showed symptoms of black pod syndrome. When plants were untreated about 20 plants/m² of Mandelup and Tallerack had black pod syndrome (Figure 1). This equated to about 60% of the plants in the plots. Only about 10 plants/m² of Jenabillup and Quilinock had black pod syndrome when plants were untreated. This equated to about 25% of the plants in the plot.

Applying insecticide reduced the number of plants with black pod syndrome for all genotypes. Applying fertiliser increased the number of plants of Mandelup and Tallerack with black pod syndrome but did not affect Jenabillup or Quilinock.

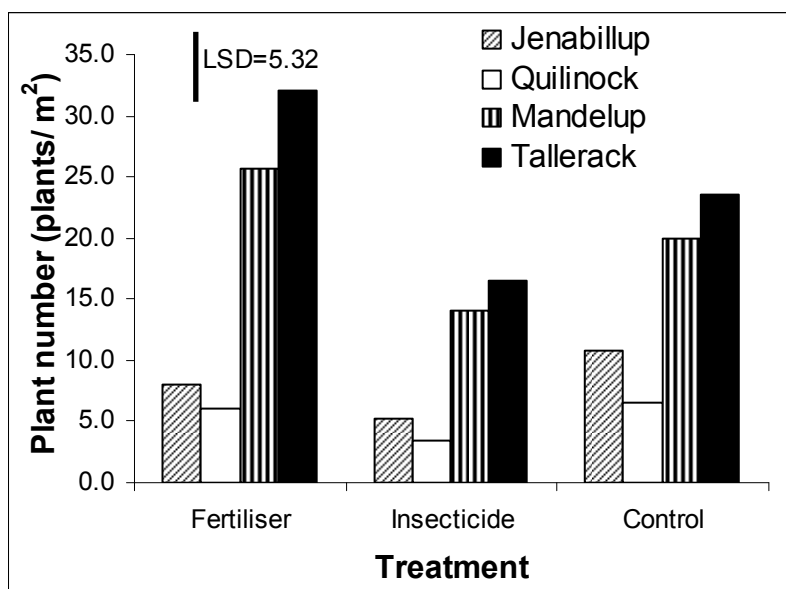


Figure 1 Number of plants showing symptoms of black pod syndrome when Jenabillup, Quilinock, Mandelup or Tallerack, were left untreated (control), received regular applications of insecticide or received a full complement of nutrients as fertiliser.

Removing the top three primary lateral branches from plants or removing all branches and leaves below the top three primary lateral branches did not affect the proportion of plants that showed black pod syndrome (data not presented). Mandelup still showed a greater number of plants with black pod syndrome than Jenabillup and the number of plants were either decreased or increased by application of insecticide or fertiliser in the same proportion as plants that had not had branches removed.

Grain yield

Grain yields were closely related to the number of plants showing black pod syndrome. Jenabillup and Quilinock produced higher grain yields than Mandelup or Tallerack (Figure 2). Similarly, applying insecticide increased grain yields while applying extra fertiliser decreased grain yields. These responses mirrored the effect treatments had on the number of plants with black pod syndrome. Yields were about 3 t/ha when less than five plants per m² had black pod syndrome, and only 1.5 t/ha when about 20 plants per m² had black pod syndrome (Figure 3). Total shoot biomass showed similar responses as grain yields (data not shown).

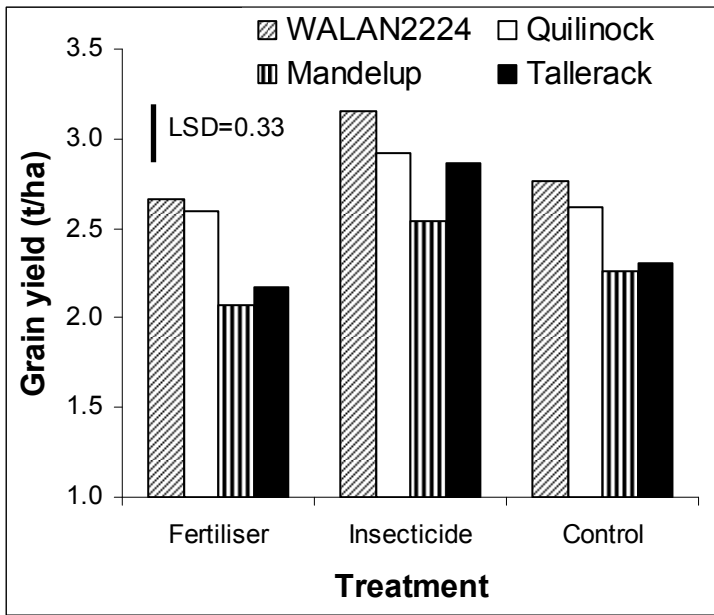


Figure 2 Grain yield of plants showing symptoms of black pod syndrome when Jenabillup, Quilinock, Mandelup or Tallerack, were left untreated, received regular applications of insecticide or received a full complement of nutrients as fertiliser.

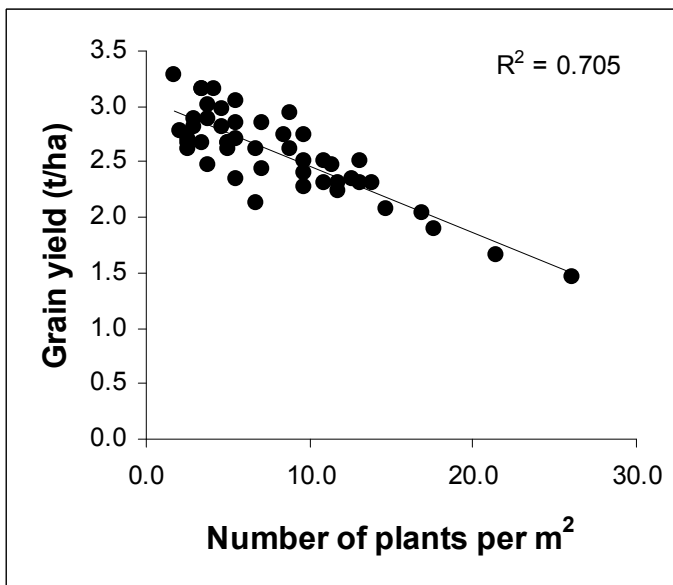


Figure 3 Correlation between the number of plants with black pod syndrome and grain yield. Each point represents an individual genotype x amendment x replicate combination.

DISCUSSION

Regular application of insecticide was aimed at reducing the transmission of BYMV by reducing the number of aphids entering the plots to feed on plants. However it was recognised that insecticide application would have also had a wide range of additional effects unrelated to transmission of BYMV. Results from this trial, nevertheless are consistent with the theory that a link between BYMV and black pod syndrome exists and more detailed research into this possible link is required. Other possible causes of black pod syndrome in lupins cannot be ruled out, however, the results from this trial are not consistent with the hypothesis black pod syndrome is cause by a nutrient deficiency or through competition between pods for limited assimilates.

The increased incidence of black pod syndrome when extra fertiliser was applied was unexpected. If black pod syndrome was related to a nutrient deficiency then its severity would be expected to be decreased by fertiliser application rather than increased. Total biomass production was not increased by fertiliser application so it is unlikely that the high rates of some nutrients may have induced, through a dilution effect, a deficiency of other nutrients that were in marginal supply (particularly boron which was not applied). Possibly the very high levels of nitrogen applied to plants made them more attractive to aphids causing a higher incidence of BYMV related black pod syndrome.

Removing branches from plants was aimed at altering the competition between pods forming on the main stem and lateral branches for assimilates. Removing lateral branches may be expected to reduce competition for assimilates, while removing leaves and basal branches from the main stem may be expected to reduce assimilate supply to pods on the main stem. It is unlikely that black pod syndrome is related to assimilate supply because removing branches or leaves had no effect on the number of plants showing black pod syndrome. This, in fact, is not surprising given that lateral branches of lupins become autonomous in their carbon supply early on in their development (Palta et al. 2007).

KEY WORDS

narrow-leafed lupin, black pod syndrome, BYMV

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