

DISEASES AND PESTS

Fungal diseases

Effect of simazine on brown spot tolerance of lupins

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Brown spot (*Pleiochaeta setosa*) is a significant disease of lupins within Western Australia. Brown spot infection can result in development of necrotic lesions on leaves, premature defoliation, reduced plant vigour and in severe cases plant death. Simazine is used for lupin production on most farms in Western Australia. Some varieties show intolerance to triazine herbicides, however the damage is usually minimal and simazine is considered on the whole to be safe for use. It is already known that factors which reduce the growth rate and health of lupins can increase the effects of diseases such as brown spot, therefore in situations where simazine reduces seedling growth (even marginally) there is a possibility that inherent plant resistance to brown spot may be reduced.

During 2003 and 2004 three small plot experiments at Medina Research Station and two glasshouse experiments were conducted to examine interactions between rates of simazine and brown spot severity.

At Medina, all experiments used split plot designs to examine the impact of rates of simazine and presence or absence of brown spot infested soil on the growth and presence of symptoms on 4 lupin varieties. Experiment 3 also examined the interaction between these factors and the efficacy of Rovral seed dressing. Assessments of brown spot severity on leaves were made at regular intervals and plants were removed for dry weight assessment 10 weeks after sowing.

In two glasshouse experiments, lupin seed was sown into moist soil which was either untreated or pre-mixed with simazine to a concentration of 0.25 ppm (Expt. 1) or 0.05 ppm (Expt. 2). Brown spot infection was developed by spray inoculation of a spore suspension onto half of the pots in each experiment. Assessments of brown spot severity on leaves were made 2 weeks after inoculation and plants were removed for dry weight assessment 4 weeks after sowing.

In glasshouse experiment 1 simazine increased the severity of brown spot leaf infection symptoms. Both simazine and brown spot reduced plant dry weight (Table 52) and there was an additive effect of brown spot and simazine on dry weight. In glasshouse experiment 2, brown spot caused leaf necrosis and dry weight reductions but the lower concentration of simazine had no effect on dry weight and no interaction with effects of brown spot occurred.

Table 52. Effect of simazine and brown spot on dry weight of three lupin varieties in grown under glasshouse conditions (4 weeks after sowing, 2 weeks after brown spot inoculation)

Brown spot	Plant dry weight (g/plant)		
	Simazine 0 ppm	Simazine 0.25 ppm	Mean
Nil	0.13	0.11	0.12
Inoculated	0.11	0.08	0.09
Mean	0.12	0.09	0.11

LSD: Brown spot = 0.1, Simazine = 0.01, Interactions = 0.01.

At Medina, in experiments 1 and 2 simazine caused some leaf scorching symptoms, particularly at the 6 L/ha rate and this resulted in reduced dry weight 10 weeks after sowing. Brown spot severity was moderate but still significant enough to cause reduced plant vigour and reduced plant dry weight. Severity of brown spot symptoms of leaves was not affected by any rate of simazine, despite the level of simazine damage that may have occurred. Despite the lack of impact on brown spot leaf infection, there was an additive effect of simazine and brown spot on plant dry weight in all experiments (Table 53). In experiment 3, Rovral seed dressing reduced brown spot severity to a similar degree both in the presence or absence of simazine.

Table 53. Effect of simazine and brown spot on dry weight of lupins (10 weeks after sowing) in two experiments at Medina Research Station

Brown spot	Plant dry weight (g/plant)			
	Simazine 0 L/ha	Simazine 2 L/ha	Simazine 6 L/ha	Mean
Experiment 1				
Nil	4.7	4.0	3.9	4.2
Inoculated	3.8	3.6	3.0	3.5
Mean	4.3	3.8	3.5	3.8
Experiment 2				
Nil	6.5	5.0	5.0	5.5
Inoculated	5.6	4.8	4.6	5
Mean	6.1	4.9	4.8	5.3

Experiment 1 LSD; brown spot = 0.6, Simazine rate = 0.3.

Experiment 2 LSD; brown spot = ns, Simazine rate = 0.6.

Simazine can cause increased severity of brown spot leaf necrosis symptoms, however this occurred in only 1 of 5 experiments. The greatest impact of simazine on brown spot appears to be the additive effect that both have on plant vigour and dry weight rather than increasing leaf necrosis. Under field conditions brown spot is more damaging in less thrifty plants; simazine can reduce plant vigour and therefore has the potential to exacerbate the impact of brown spot on plant growth.

Effect of moisture and temperature on blackspot spore maturation on field pea stubble

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Over the past 5 years research into the epidemiology of the fungus that cause blackspot (*Mycosphaerella pinodes*) on field pea has been carried out in WA. We have significantly increased our understanding of how this pathogen survives on the previous season's stubble and what triggers the production of spores in the following growing season. Temperature and moisture are the two main components that influence the onset of spore maturation and also the potential number of spores to be released by blackspot of field pea. Summer rainfall has been shown to influence the timing of the onset of maturation. In areas receiving summer rainfall, spore maturation tends to occur earlier in the growing season compared with areas that receive no summer rainfall.

Field pea stubble, naturally infected with blackspot, was weathered at 6 locations within WA in 2004. The ascospore release potential of these stubble samples was determined in the laboratory under repeatable conditions. Obvious differences were found in the maturation process across the different locations (Figure 21a and 1b). In previous years in the Southern Agricultural Region (SAR) the onset of the maturation process and the completion of spore release usually occurred early in the season with the majority of spores having been released by late June. In 2004, spore release was finished by early-June in the Esperance (Scaddan) area (Figure 21a). This coincides well with the time of sowing recommended for this area to reduce the risk of blackspot. In Katanning, spore maturation was delayed in 2004 compared with previous years due to the drier than usual summer conditions experienced in this area. Peak ascospore release occurred in early-June and continued at relatively high levels through the vegetative growth (pre-flowering) stage of the pea crops (Figure 21a). This delayed spore release at Katanning had the potential to cause a high level of blackspot in field pea in 2004.

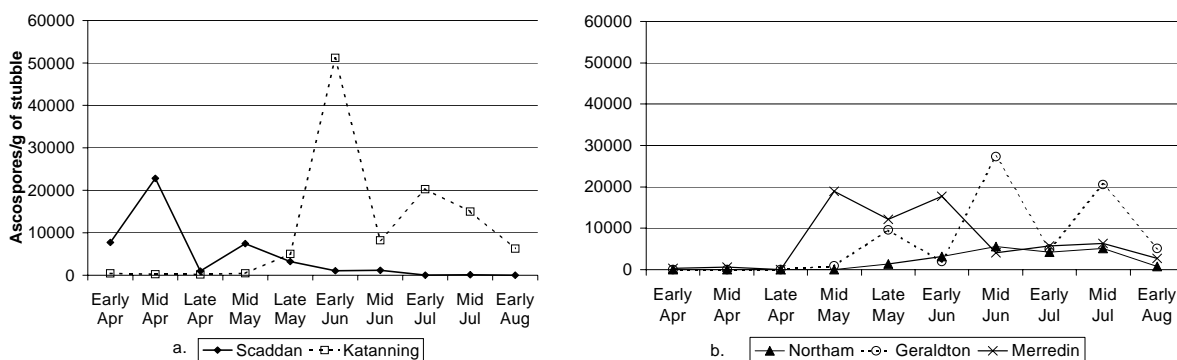


Figure 21. *Mycosphaerella pinodes* ascospore release potential on field pea stubble in 2004. (a) Southern Agricultural Region (Scaddan and Katanning); and (b) Central and Northern Agricultural Region (Northam, Geraldton and Merredin). Data for Three Springs is not shown, but was similar to Geraldton.

In previous years it has been found that in the warmer Central and Northern Agricultural Regions (CAR and NAR) there is a tendency for the blackspot fruiting bodies to mature later than in the SAR. In the NAR the peak spore release period in 2004 was from mid-June onwards (Geraldton and Three Springs), this is similar to previous years. In 2004, the effects of summer rainfall events in the CAR are very evident in the difference in maturation patterns observed between Merredin, where summer rainfall was received in 2004, and Northam, which received no summer rainfall during 2004. At Merredin, spore release occurred from mid-May and the spore shower continued through until August (Figure 21b). In Northam, the spore maturation and release pattern recorded in 2004 was quite different from that typically observed for this area. While fruiting body maturation was delayed as expected by the dry summer conditions, the number of spores that were produced was also significantly reduced. We think that this was due to unfavourably cold conditions that occurred during the maturation process in early winter. Controlled environment experiments conducted in 2004 have shown that blackspot ascospore production is optimal at temperatures around 20°C and decline for lower temperatures.

This data has been used to further validate the 'Blackspot Appraisal' model that predicts blackspot maturation and recommended time of sowing for field pea.

Epidemiology and management of ascochyta blight in improved chickpea varieties

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Immediately after the outbreak of ascochyta blight of chickpea in WA, field experiments were conducted to produce a fungicide management package for the susceptible chickpea cultivars that were grown at the time. That package has been shown to work well, but was expensive, as it relied on the use of the fungicide chlorothalonil applied at about 3 week intervals from 4 weeks after crop emergence.

Since the outbreak of ascochyta, chickpea breeding programs around Australia have identified a number of potential varieties that are much less susceptible to ascochyta, some being moderately resistant. Field trials have been conducted to determine an economic and robust fungicide management package for varieties with improving levels of resistance. In parallel with this work, epidemiology research has also been progressing to ensure that recommended fungicide timings will be protecting the crop from wind-borne spore as well as seed-borne sources.

Chickpea stubble, naturally infected with ascochyta blight (*Ascochyta rabiei*), was weathered at 4 locations within WA in 2004. The sexual stage of this pathogen (*Didymella rabiei*) produces wind borne ascospore that can initiate ascochyta blight in current season's crops. The ascospore release

potential of these stubble samples was determined in the laboratory under repeatable conditions. The timing of the start of ascospore release varied with geographical location (Table 54), but at all sites ascospores were still being released in August at the end of the trapping period.

Table 54. The release of wind dispersed ascospores from ascochyta blight infected chickpea stubble in WA in 2004

Geographic location	Start of ascospore release	Peak ascospore release
Esperance (Scaddan)	Late-March	Mid-April to early-July
Katanning	Late-May	Mid-June
Geraldton	Late-May	Mid-June
Northam	Early-July	Early-July

This data will be used to further refine and validate the 'Chickpea Blight Appraisal' model. The concept of this model is to provide growers with a prediction of when ascospore showers are likely to occur, so that time of sowing and fungicide spray applications can be optimised to reduce the risk of yield limiting levels of ascochyta blight.

The relatively dry conditions experience during the 2004 growing season, and the improved resistance of the varieties use, both limited the build-up of ascochyta in fungicide trials in the central and northern agricultural regions. These trials received the standard inoculation of 2 pieces of infected stubble per plot which has resulted in severe disease in trials with the varieties Sona and Howzat in previous trials.

The four trials reported here compared the disease control achieved in improved desi and kabuli lines only. The susceptible varieties such as Sona or Kaniva were not included because, without frequent fungicide sprays, these varieties become heavily infected and act as an on-going source of disease within the trial. This constant re-inoculation of the improved varieties would lead to more fungicide sprays being recommended than would be required for a sole crop of that variety.

Kabuli chickpea trials were established at 2 high rainfall sites, Beverley (Avondale Research Station; H3/M3) and Irwin River flats 5 km north of Dongara (H1). The sowing date and fungicide application rates and dates are given (Table 55). All seed was treated with P-Pickel-T. The disease inoculated rate of 2 infected pieces of stubble per plots is equivalent to a seed transmission rate of about 0.15%.

The three varieties (2 at each site) included in these trials have been developed by CLIMA through international collaborative projects. They are all moderately resistant to ascochyta blight and produce bold, bright seed, and are currently being bulked for release. This is the first year replicated fungicide management trials have been conducted with these varieties.

Due to the level of resistance of these varieties, a 3 weekly fungicide spray program was not necessary to estimate potential yield. All varieties received 4 spray programs of chlorothalonil (Bravo®), each of which started with an early spray 4 weeks after emergence followed by one, two or three follow-up sprays (Table 55). Each variety also received a two spray program of 2 kg/ha of mancozeb. The 4 fungicide application time was late at Beverley and probably had little effect.

Table 55. Final disease score and yield of 3 kabuli chickpea varieties in response to 4 chlorothalonil and 1 mancozeb fungicide application regimes for experiments conducted at Mingenew and Merredin 2004

Treatment	Dongara		Beverley	
	Final disease (score 0-9)	Yield (t/ha)	Final disease (score 0-9)	Yield (t/ha)
Variety				
Flip97-503	0.9	2.56		
Flip97-530	1.0	2.57	0.8	0.71
Flip97-537D			1.5	1.06
<i>LSD 5%</i>	<i>NS (1.0)</i>	<i>NS (0.05)</i>	<i>NS (1.2)</i>	<i>0.68</i>
Fungicide programs				
4 Sprays (1-4 Bravo)	0.2	2.63	1.0	0.95
2 sprays (1 and 2 Bravo)	0.8	2.52	0.9	0.88
2 sprays (1 and 2 mancozeb)	1.1	2.52	1.4	0.82
2 sprays (1 and 3 Bravo)	1.2	2.57	1.3	0.96
2 sprays (1 and 4 Bravo)	1.6	2.57	1.3	0.82
<i>LSD 5%</i>	<i>0.7</i>	<i>NS (0.23)</i>	<i>0.5</i>	<i>NS (0.27)</i>

* Details of foliar fungicide applications.

Spray timing	Dongara	Beverley
	Sown - 27 May	Sown - 30 May
4 weeks PE	5 July, 1.5 L/ha Bravo or 2 kg mancozeb	9 July, 1.5 L/ha Bravo or 2 kg mancozeb
7 weeks PE	26 July, 1.5 L/ha Bravo or 2 kg mancozeb	30 July, 1.5 L/ha Bravo or 2 kg mancozeb
Flowering	30 August, 1.5 L/ha Bravo	20 August, 1.5 L/ha Bravo
Podding	8 October, 1.5 L/ha Bravo	13 October, 1.5 L/ha Bravo

The most conspicuous result from the two kabuli trials is the very good yields achieved at Dongara from both varieties. The yields were much lower at Beverley and Flip 97-537D appears to be better adapted to the dryer conditions experienced there during 2004. There were no yield differences between the fungicide programs at either site, although there is a suggestion that spray 3 (flowering) has been effective in protecting yield, while the mancozeb program was less effective than chlorothalonil.

Satellite plots were also sown at both sites, these received no fungicide sprays. At both sites, significant disease patches developed in plots of both varieties used in that trial. These satellite plots illustrated that even with the level of resistance present in the kabuli varieties, a fungicide program of at least 2 strategically times fungicide sprays will be required to maximise yield and prevent an increase in disease pressure which will threaten both the current crop and the following year's crop.

Desi chickpea trials were established at 2 medium rainfall sites, Merredin Research Station (L3) and Yandanooka (15 km south east of Mingenew; M1). The sowing date and fungicide application rates and dates are given (Table 56). As with the kabuli trials, all seed was treated with P-Pickel-T and the disease inoculation equivalent to a seed transmission rate of about 0.15% was used.

Compared to desi chickpea trials conducted during the past 4 years, the level of disease in these trials was low. This was partly a result of omitting the susceptible varieties, Sona and Howzat, from the comparison and also partly due to the weather conditions being less conducive to ascochyta spread. Nonetheless, Flip94-508C was demonstrated to be more resistant to ascochyta blight than Sonali, although it was also shown to be not well adapted to the low rainfall, short season environment at Merredin.

Table 56. Final disease score and yield of 2 desi chickpea varieties in response to 4 chlorothalonil and 1 mancozeb fungicide application regimes for experiments conducted at Mingenew and Merredin 2004

Treatment	Mingenew		Merredin	
	Final disease (score 0-9)	Yield (t/ha)	Final disease (score 0-9)	Yield (t/ha)
Variety				
Sonali	0.8	1.15	0.4	0.64
Flip94-508C	0.1	1.11	0.1	0.37
<i>LSD 5%</i>	0.2	<i>NS (0.22)</i>	<i>NS (0.7)</i>	<i>NS (0.44)</i>
Fungicide programs				
4 sprays (1-4 Bravo)	0.2	1.19	0.1	0.54
3 sprays (1,2 and 3 Bravo)	0.1	1.20	0.1	0.52
3 sprays (1,2 and 3 mancozeb)	0.7	1.05	0.5	0.42
3 sprays (1, 3 and 4 Bravo)	0.6	1.14	0.6	0.57
2 sprays (1 and 2 Bravo)	0.4	1.07	0.0	0.50
<i>LSD 5%</i>	0.3	0.12	0.4	0.10

* Dates and details of sowing and foliar fungicide applications.

Spray timing	Mingenew	Merredin
	Sown - 27 May	Sown - 4 June
4 weeks PE	5 July, 1.5 L/ha Bravo or 2 kg mancozeb	12 July, 1.5 L/ha Bravo or 2 kg mancozeb
7 weeks PE	26 July, 1.0 L/ha Bravo or 2 kg mancozeb	5 August, 1.0 L/ha Bravo or 2 kg mancozeb
Flowering	30 August, 1.5 L/ha Bravo or 2 kg mancozeb	Sonali only 7 Sept, 1.5 L/ha Bravo or 2 kg mancozeb
Flowering		Flip94-508C only 24 September, as above
Podding	8 October, 1.5 L/ha Bravo	Not applied

It is evident from the disease scores that both early sprays (4 weeks and 7 weeks after sowing) are required for effective management of *Ascochyta* blight, the 3 spray program that omitted the spray 7 weeks after emergence were less effective than the two early sprays. While the two early sprays were as effective as the 4 spray program under the low disease conditions of 2004.

As in the kabuli trials, 3 sprays of mancozeb were less effective than two early sprays of chlorothalonil in minimising disease development. In years favourable to *ascochyta* development, the differences in control provided by chlorothalonil and mancozeb observed in these trials may become greater with a commensurately larger impact on yield.

Satellite plots were also sown at both the desi chickpea trial locations, these did not receive the early fungicide spray. At both sites, significant disease developed in plots of both varieties. These satellite plots illustrated that even under the relatively low disease conditions of 2004, omitting the first fungicide spray will allow the disease to develop to a level that cannot be reined in by later sprays.

The results from these trials supports the fungicide package we proposed last year for these improved desi varieties, i.e. the first 2 fungicide sprays, approximately 4 weeks and 7 weeks after emergence, will need to be applied. The requirement for subsequent sprays will be determined by whether or not disease develops in the crop, if *ascochyta* is observed then a fungicide spray during flowering or early podding is strongly recommended.

To achieve the cheapest fungicide management program, chickpea growers must also observe other aspects of the chickpea establishment package. That is, selecting a paddock that is remote from where chickpeas were grown last year (both on their own and their neighbour's properties) and which has not grown chickpeas for at least 3 years, then sowing during the recommended time determined by yield and disease risk considerations.

Botrytis grey mould of chickpea

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Botrytis Grey Mould (BGM) of chickpea is caused by *Botrytis cinerea*. It becomes apparent in spring when the chickpea crop flowers and warm, humid conditions prevail. Chickpeas with vigorous seedling growth, early canopy (mid-winter) closure and early flowering (late winter) are most likely to develop BGM. Serious epidemics of BGM occurred in desi and kabuli chickpea crops during 1997 and 1998. During 1999, BGM developed in some chickpea crops late in the growing season and caused significant damage, this was also the first season that ascochyta blight (*Ascochyta rabiei*) occurred in WA crops. From 2000 to 2004, the disease has been observed infrequently, probably due to a combination of the drier seasons in 2000-2002, extensive fungicide spraying for ascochyta blight control and the low frequency of chickpeas crops.

With the release of ascochyta blight resistant varieties of chickpea, and the associated reduction in fungicide sprays, BGM may again become a problem in the northern agricultural region during favourable seasons. Research to understand the survival and spread of this disease and to identify varieties that are more resistant to BGM is in progress. This should lead to the development of improved management packages for BGM that will compliment the improved ascochyta blight management packages.

The possible role of latent infection in BGM (*Botrytis cinerea*) of chickpea was examined in a field trial in Northam and glasshouse trials at Shenton Park Field Station (UWA). In Northam, trap plants placed next to the previous season's stubble on a weekly basis from April to September 2004 showed that viable *Botrytis cinerea* spores were spread from the stubble every week from early May to early August, and then sporadically from August to September. This indicates that infection of the plants can be occurring at any time during the growing season and is not necessarily restricted to the period when BGM disease expression occurs. Despite infection occurring, the dry finish to the season resulted in no BGM symptoms developing at the Northam trial site in 2004. At the Shenton Park Field Station site no BGM was seen until flowering, but Botrytis could be isolated from apparently healthy tissue before flowering. Severe stem girdling lesions developed on some plants, but leaf and flower lesions were more frequent. However, leaf and flower infections are easily overlooked, as they are ephemeral and readily drop to the ground.

Since the occurrence of Ascochyta blight in WA, it has become impossible to do BGM field trials without the complication of fungicides for Ascochyta management, or indeed an Ascochyta outbreak in the trial. This problem has been overcome through a collaborative project with Bangladesh funded by Australian Centre for International Agricultural Research (ACIAR). Bangladesh is an ideal partner in this project as they have annual and severe infections of BGM in chickpea crops, but do not have Ascochyta blight. The project has achieved two years of field screening of chickpea germplasm at two sites in Bangladesh in addition to screening similar lines in growth-rooms at ICRISAT in India

Screening of chickpea genotypes in Bangladesh in 2002-03 identified a substantial number of genotypes with potentially useful resistance to BGM, including two groups of genotypes with *Cicer echinospermum* parentage. Field screening was again carried out at two sites in Bangladesh in the 2003-04 growing season. The 208 genotypes screened included 101 new entries from the chickpea breeding program at Tamworth, NSW. Both BGM nurseries were exposed to natural disease nonetheless differentiation among entries for both yield and disease score was achieved with 77 of the entries yielding in excess of 100 g/plot (single row 2 m long).

There was general agreement between the BGM disease scores and plot yields observed in both screening nurseries, therefore the two sites are considered here collectively as average data. A relationship is apparent for entries with higher disease scores to have lower plot yields, however, this relationship only explains about 30% of the variability, the majority of this variability being dictated by relative adaptation to the environment (Figure 22). This trend is also illustrated when the 10 highest yielding and 10 lowest yielding lines are considered. The average disease score for the ten highest yielding lines is 4.6 (on the 0-9 scale) and the average yield 116 g/plot, whereas for the ten lowest yielding lines the disease score is 5.8 and the average yield 30 g/plot (Figure 23). Furthermore, selection for reduced BGM severity has been effective as the mean scores of 4.3 for the 106 lines retained from the 2002/03 screening was lower than the mean score of 5.3 for the 101 new entries screened for the first time in 2003/04.

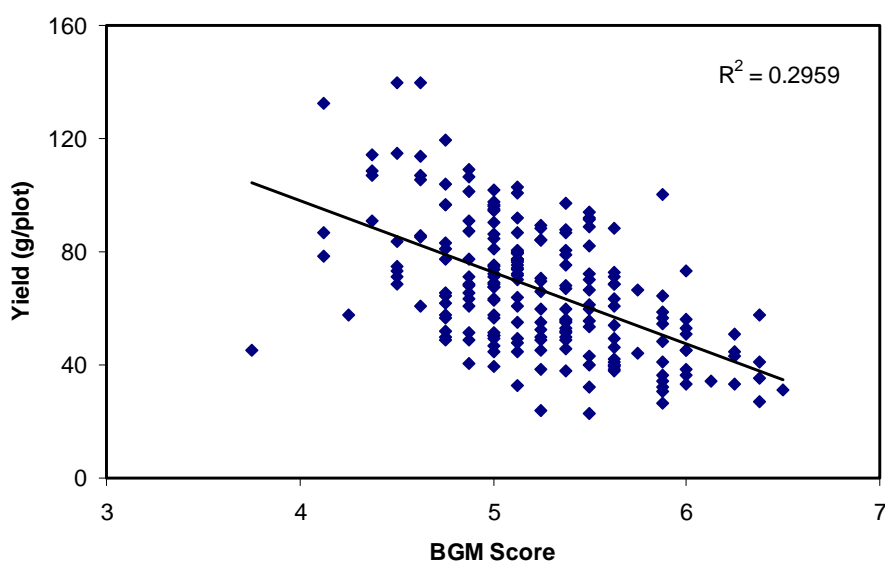


Figure 22. Relationship between average plot yield (g/plot) and Botrytis Grey Mould disease score (on 0-9 scale) when averaged across screening nurseries at both Regional Agricultural Research Station, Jessore and Regional Agricultural Research Station Ishurdi, Bangladesh, 2003-04.

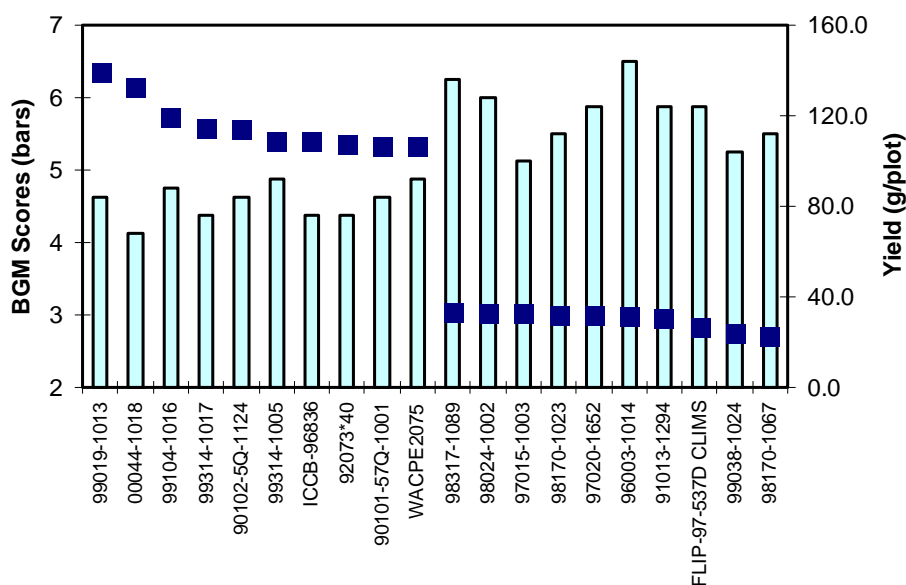


Figure 23. Botrytis Grey Mould (BGM) severity score (on 0-9 scale, bars) and yields (g/plot, squares) for the 10 highest and 10 lowest yielding lines when averaged across screening nurseries at both RARS Jessore and RARS Ishurdi, Bangladesh, 2003-04.

Four hundred and twenty four lines evaluated for resistance to BGM and Ascochyta blight in controlled environment at ICRISAT, Patancheru, India. Among these lines, none was observed to be immune or resistant, however, 108 entries were scores as moderately resistant (3.1-5 rating), 261 were susceptible (5.1-7 rating) and 39 highly susceptible (>7.0 rating).

In addition to screening for resistance to BGM in chickpea the ACIAR project between Bangladesh and Australia is examining the variability within *B. cinerea*, the fungus which causes the disease. Isolates have been collected in Bangladesh in 2003 and 2004 and from various locations around Australia for genetic analysis by Dr Taylor at Melbourne University. Microsatellite analysis revealed a total of 26 haplotypes or putative genotypes among the 59 isolates from Bangladesh. These results have indicated the lack of population differentiation and revealed potential gene flow, especially

between individual sites within Bangladesh. BGM of chickpea can therefore be considered a pathogen of 'moderate-high risk' to durable resistance breeding as it showed high gene diversity, evidence of gene flow between regions, and has a mixed reproductive system. Isolates collected in 2004 are yet to be included in this analysis and an alternative molecular analysis is currently being conducted by DAWA.

Chocolate spot of faba bean

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Chocolate spot of faba bean is caused by *Botrytis fabae*. It can occur throughout the WA agricultural area and is most severe in areas that experience warm, moist spring conditions. Major crop losses occurred as a result of this disease in 1997 and 1998 resulting in a decline in the area planted to faba beans in subsequent years. In 1999, severe chocolate spot was recorded on early sown crops but from 2000 to 2002, the disease was hard to find. In 2003 and 2004 disease was reported in some areas but remained at low levels in fungicide treated crops. Few faba bean crops have been grown since 2000 due in part to grower reluctance to grow faba beans until more resistant varieties and an improved chocolate spot management package are available. The area of faba bean sown has also been limited by the lack of sowing opportunities in recent years.

The sporadic nature of this disease makes it difficult to work with under natural conditions. A chocolate spot nursery has been established at the Centre for Cropping Systems in Northam where trial work is conducted. A small (5 x 5 m) irrigated area is sown into the previous seasons infected stubble in mid-April each year, to ensure there is an area in which chocolate spot develops from which spores can infect the remainder of the disease nursery. The remainder of the area is sown when conditions suitable for planting occur. In 2004, the faba beans plots were sown on the 9 June.

The possibility that latent, or symptomless, infections which occur during the pre-flowering stage are playing a role in the development of chocolate spot was examined in 2004. Infection opportunities were monitored on trap plants placed within the disease nursery on a weekly basis throughout the growing season. Spore release and deposition of *B. fabae* occurred from early May onwards. This spore release was initially from the previous season's stubble (primary infection) and once disease expression occurred post-flowering, spore production and release was from chocolate spot lesions in the bottom of the canopy (secondary infection). Marker strains of *B. fabae* were used to prove that symptomless infection could occur during the vegetative stage and disease expression occur after flowering.

A marker strain (Nit 1) of *B. fabae* was grown on sterilised grain. Once colonised, the grain was placed in a mesh bag in the field at 6 weeks after sowing (6 WAS). This primary inoculum source was left in the field for 3 weeks and removed at 9 WAS, 3 weeks prior to the start of flowering. This marker strain was subsequently recovered from symptomless plants prior to flowering and throughout the reproductive stage until the end of October. As the only possible infection period for this marker strain was between 6-9 WAS, this proved that latent infections are occurring in chocolate spot in faba beans.

Strategically timed fungicide sprays were used to further examine the possibility that latent infections were occurring with chocolate spot and to determine if this symptomless infection was having any effect on yield:

Pre-flowering fungicide sprays were applied from 3 WAS and thereafter at 3 week intervals until 9 WAS. The function of the pre-flowering spray was to control any primary infections that may have been occurring during the pre-flowering period. At 9 WAS when the last pre-flowering fungicide spray was applied, there were no visible symptoms of chocolate spot in the faba bean crop. Three weeks later, at 12 WAS, the crop was beginning to flower and the first symptoms were becoming apparent. Five weeks after the last pre-flowering spray was applied (14 WAS) there was still less disease in plots that had received early protection compared with plots that were not treated during the pre-flowering period (Figure 1a). Due to the high level of inoculum produced on diseased plants post-flowering the level of secondary infection was very high. This resulted in a steady build up of disease in the later part of the growing season in the plots that only received the pre-flowering fungicide sprays (i.e. inter-plot interference (Figure 24a). Consequently, there was only a 40 kg/ha yield difference between the pre-flowering fungicide treatment and untreated plots at harvest (Figure 24b).

Post-flowering fungicide sprays were applied when the crop was just beginning to flower (12 WAS) and 3 weeks later at 15 WAS. The function of the post-flowering spray was to control secondary infections that occurs during the post-flowering or reproductive period when *B. fabae* sporulation is evident on chocolate spot lesions within the current season's crop. Where post-flowering sprays were applied, less chocolate spot developed and there was a yield increase of 150 kg/ha compared with the untreated plots (Figure 24).

Pre- and post-flowering fungicide sprays were applied at 3 week intervals from 3 WAS until 15 WAS. The function of this regime was to control both primary infections that may have been occurring during the vegetative period and secondary infections that occurred during the reproductive period. The plots that received both pre- and post-flowering sprays developed the least amount of disease (Figure 1a) and there was a yield increase of 350 kg/ha compared with the untreated plots (Figure 24b).

If the yield increase for the pre- and post-flowering sprays are looked at on their own, it would appear that pre-flowering control of primary infection results in a 40 kg/ha yield increase and post-flowering control of secondary infection results in a 150 kg/ha yield increase. If the two treatments were combined this should result in a yield increase somewhere in the region of 190 kg/ha, however the yield increase component for the pre- and post-flowering fungicide sprays was 350 kg/ha – nearly double the expected yield increase (Figure 24b). In the absence of an ongoing source of spores, from unsprayed plots and the highly infected early sown area, the pre-flowering sprays alone would have been expected to have yields similar to the post-flowering spray treatment.

This data further supports the idea that chocolate spot infection is occurring during the vegetative stage and is remaining latent or symptomless in the plant until disease expression is triggered around about the time of flowering. Symptomless infection is therefore impacting on the final disease expression and yield.

Please note that these sprays were used as a tool to understand the infection process and were not evaluated for disease management. No consideration was given to the economic benefits of applying sprays according to this schedule. The current recommendations for the economic control of chocolate spot in faba beans are unchanged and should be used commercially (Farmnote No. 81/2002), however it is expected that confirmation of this research will lead to a change in the recommended management strategy for control of chocolate spot.

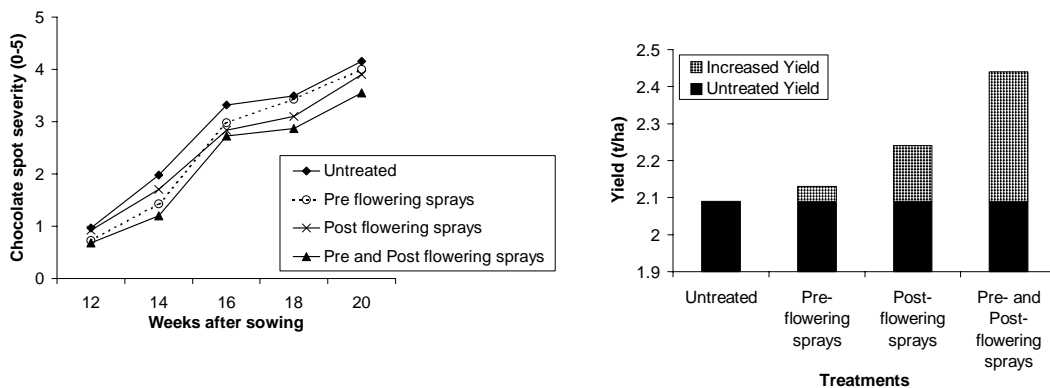


Figure 24. The impact of pre- and post-flowering sprays on the development of chocolate spot and yield loss in faba bean. Pre-flowering sprays were applied at 3, 6 and 9 WAS. Post-flowering sprays were applied at 12 and 15 WAS.

Viruses

Cultural control of AMV in lentil

By Rohan Prince and Roger Jones, Department of Agriculture and CLIMA, The University of Western Australia

A field trial was done at Avondale in 2004 to examine the effects of plant density and straw groundcover in limiting the spread of *Alfalfa mosaic virus* (AMV) in lentil. AMV spread was very late due to exceptionally late arrival of aphid vectors. High plant density suppressed virus spread significantly (by 56%) but straw had no effect on spread as, by the time aphids arrived and spread started, plant growth had covered the ground. Groundcover effects on aphid landing rates and virus spread only occur when aphids arrive while plants are still small. In the generic integrated management strategy produced for AMV control in pulses, these results help validate inclusion of high seeding rates to generate high plant densities and so reduce virus spread but not deployment of stubble retention to diminish aphid landing rates.

Screening pulses for virus resistance and seed quality defects

By Rohan Prince and Roger Jones, Department of Agriculture and CLIMA, The University of Western Australia

In our 2004 Pulse Crop Update article, we described a large-scale screening trial done at Medina Research Station in 2003 which evaluated 81 different pulse lines for resistance/susceptibility to PSbMV and to seed staining defects caused by the virus. The results were processed in 2004. Generally, the desi chickpea had the lowest percentage infection with PSbMV (5-36%), followed by the kabuli chickpea (14-47%), field pea (18-94%), lentil (60-92%) and, finally, faba bean had the highest (72-86%). Two potentially PSbMV-resistant lines of field pea were found. None of the seven lupin species present became infected.

Plants of all faba bean genotypes were severely symptomatic, while the lentil and pea genotypes were the least severely symptom-affected. Chickpeas ranged in sensitivity. Faba bean plant symptoms included leaf distortion, shoot tip distortion, varying degrees of mottle and varying degrees of stunting. Lentil symptoms were leaf distortion and a mild chlorosis. Peas displayed a mild stunt, mild mottle and a shoot tip distortion. The symptoms in the chickpeas were tip distortion, leaf distortion, stunt, and mild chlorosis (kabuli) or reddening (desi).

The most severely affected seeds were those of faba beans (3-5 sensitivity rankings), with peas the next most severely damaged (3-4). Least affected were lentils (all ranked 2). With chickpea, kabuli were the most affected because the seed was darkened and the necrotic markings showed up more clearly (2.5-3.5). Desi seeds were affected by line markings at an intermediate level (2-3). The different types of seed staining symptoms found in each species were: lentil - necrotic rings, malformation and reduced size; pea - necrotic rings, malformation, reduced size and splitting; kabuli chickpea - pronounced darkening of colour, necrotic rings, malformation, reduced size and splitting; desi chickpea - necrotic rings, malformation, reduced size and some darkening; faba bean - necrotic rings, malformation, splitting and reduced seed size.

Thus, with seed quality defects caused by PSbMV, field pea and faba bean were the most severely compromised. Chickpea seed symptoms were more severe than previously thought while lentil seeds were the least compromised by infection. Darkening of kabuli chickpea seed by PSbMV is of concern in regards to sale price (in addition to malformation and reduced seed size) because of high priority put on colour in the market. Pea lines and chickpea lines with potentially high resistance levels were identified. Lentils all proved highly susceptible to infection by PSbMV. Chickpea proved intermediate in susceptibility, but kabuli tended to be somewhat more susceptible than desi. Lupins were not susceptible under these conditions, so PSbMV infection seems not of concern for this grain legume.

A similar screening trail to that in 2003 was done in 2004, but there were fewer genotypes and more replicates of each. Results will be reported at the 2005 Crop Updates.

Survey for viral seed staining in pulse seed lots

By Rohan Prince and Roger Jones, Department of Agriculture and CLIMA, The University of western Australia

In 2003-2004, a total of 150,966 seeds from 116 different pulse seed lots harvested from plots at Carnarvon, Dongara, Frankland, Merredin, Mingenew, Scaddan and Wilgoyne were examined for virus staining symptoms. The numbers of seed lots examined were (numbers of seed lots/total numbers of seeds in brackets): kabuli chickpea (5/3,752), faba bean (18/39,557), field pea (70/84,657) and lentil (23/23,000). All seed lots of kabuli chickpea and field pea inspected contained some seeds with viral seed staining, while the figures were 10/18 and 1/23 for faba bean and lentil respectively. The proportions of seeds within individual affected seed lots with viral staining were 3-12% (chickpea), 1-7% (faba bean), < 1-32% (field pea) and < 1% (lentil). Tests with antisera to Pea seed-borne mosaic (PSbMV), Broad bean stain and Broad bean true mosaic viruses on germinated seed samples from some of the worst-affected seed lots of faba bean (16) and field pea (7) detected only PSbMV virus.

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