

Interaction between *Ascochyta* intensity and sowing arrangement with chickpea

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Abstract

A field experiment was conducted in 2001 to determine the interaction between *Ascochyta* blight intensity and sowing arrangement with chickpea under 3 fungicide regimes. The experiment was a 3 x 2 factorial (row spacing x seeding rate) design with strips (fungicide treatments). The experiment found wider row spacing did reduce the disease intensity, but was limited to treatments with the higher seeding rate. Whilst the reduction in disease due to agronomic management was small when compared to yield benefits obtained from fungicide use (eg. yield increases of 300%), the benefits were significant and important in overall crop management.

Media summary

Whilst fungicide still provides the best opportunity for reduction of *Ascochyta* in chickpea, this study also shows some potential associated with agronomic management.

Key words

Ascochyta blight, chickpea, canopy management

Introduction

Ascochyta blight, caused by the fungus *Ascochyta rabiei*, is a devastating foliar disease of chickpea (*Cicer arietinum* L.). For example, its presence in southern Australia has reduced chickpea sowings in South Australia from 12,000 ha in the 1997/98 season to less than 1,500 ha in 2000. The fungus survives on infected plant residue and is also transmitted through the seed (Nene et al. 1981). *Ascochyta* infection is influenced by both humidity and ambient temperature, particularly at flowering. The majority of chickpea crops worldwide are grown in India where traditionally the chickpea is sown in wide row spacings and at low densities (Jettner et al. 1999). No chickpea cultivar has been found to be highly resistant to *Ascochyta* blight, although lines with some resistance have been identified from overseas screening programs and are currently being crossed and evaluated for Australian environments (Hawthorne, 2002). Fungicide treatments, both foliar (controlling the spread of the disease in the crop) and seed dressings (controlling seed-borne inoculum of *Ascochyta rabiei*) are used in controlling *Ascochyta* in chickpea crops. Whilst there is much work done in Australia on selecting *Ascochyta* resistant chickpea varieties and developing effective fungicides and application strategies, there has been little work done on the use of agronomic management (such as sowing arrangement) to help manage *Ascochyta* blight in chickpea. In the study reported here we evaluate various sowing configurations of chickpea and their impact on chickpea's ability to withstand the pressure of *Ascochyta* infection.

Methods

The experiment was undertaken in 2001-02 at the Roseworthy Campus in the lower-north region, South Australia (34°32'S, 138°41'E). The soil is a Brown Earth soil (Hypercalcic, Red, Chromosol; Isbell, 1998) and mean rainfall is 442 mm per annum. The experiment was a 3 x 2 factorial (row spacings: 178 mm, 356 mm spaced single rows and 1000 mm spaced paired (150 mm) rows; and chickpea seeding rate: 75 and 150 kg/ha), split with 3 strips applied across the plots consisting of 0, 4 and 8 foliar fungicide applications (Bravo² (chlorothalonil, at a rate of 1 l/ha) was applied at 20-24 day intervals when 4 sprays were made and at 10-12 day intervals when 8 sprays were made. All treatments were randomly allocated

in 4 replicates. Sub-plots were 8 x 4 m (two seeder passes with an AGROW-DRILL tow-behind cone-seeder, no-till seeding with 38 mm spear points) for 178 mm and 356 mm row-spacing treatments and for the 1000 mm paired row treatments were 8 x 6 metres (three seeder passes).

The chickpea seed (Kaniva) was disease screened, cleaned, and graded to 8 mm diameter and treated with P-Pickel-T (36% Thiram, 20% Thiabendazole, The Lentil Company, Horsham), and inoculated (group N rhizobia) prior to seeding on 3rd August, 2001. Two rows of Desavic chickpea, known to be highly sensitive to *Ascochyta* blight, were sown as a disease spreader at the ends of the sub-plots. Barley buffers (4 m wide) were sown to separate fungicide treatments and replicates. The experimental area was exposed to *Ascochyta rabiei* by spreading chickpea residue from an infected crop. The infected debris (inoculum) was supplied by the Plant Pathology Unit, SARDI. The inoculum was applied on the 18th September 2001 (47 Days After Sowing, DAS).

The site was sprayed with 2L/ha of glyphosate on 20th July 2001. Three applications of Cypermethrin were applied late in the season to control Native Budworm (*Heliothis punctiger*).

Chickpea seedling counts were taken on 22nd August and 4th September 2001. Disease observations consisted of six visual assessments over time; 17th October (75 DAS), 25th October (83 DAS), 2nd November (91 DAS), 11th November (100 DAS), 19th November (108 DAS), and 30th November 2001 (119 DAS). The disease assessment started when the disease first appeared in the spreader rows (75 DAS). The visual assessment consisted of a 1-9 scale (Nene *et al.* 1981; 1 = no disease visible; 9 = plot killed by *Ascochyta*). Plots were harvested on 1st February 2002, using a K.E.W. plot harvester.

The data were analysed using the Genstat 5 statistical computer package. LSD values in the Tables are given for the $P=0.05$ probability level, and lower case letters are used to indicate significant differences. Linear regression was also carried out with some visual assessment data.

Results

The numbers of plants emerging differed with the row spacing treatments and with seeding rate. For the 75 kg/ha seeding rate the number of emerged plants were 41 plants/m² with 178 mm, 35 plants/m² with 356 mm spacing and 28 plants/m² with 1000 mm (paired) row spacing. The number of emerged plants were approximately double with the higher seeding rate. There was a significant interaction ($P = 0.05$) between row spacing and seeding rate on chickpea *Ascochyta* rating with all the times of observation (Table 1). The increased seeding rate in the two wider row spacings resulted in lower chickpea *Ascochyta* rating, but only with the 150 kg/ha seeding rate. At no observation time did the row spacing treatments differ for *Ascochyta* rating with the lower seeding rate (75 kg/ha).

Table 1. *Ascochyta* rating (score 1-9) on chickpea for row spacing and seeding rate (mean of 6 observation times)

Row Spacing	Seeding Rate	
	75 kg/ha	150 kg/ha
178 mm	3.51 <i>b</i>	3.54 <i>b</i>
356 mm	3.51 <i>b</i>	3.17 <i>a</i>
1000 mm (paired)	3.50 <i>b</i>	3.21 <i>a</i>

LSD (row spacing x seeding rate) = 0.18

With the wider row spacing there was less disease with the 0 and 4 foliar fungicide application treatments respectively, with the trend to the two wider row spacings reducing chickpea Ascochyta rating (Table 2).

Table 2. Ascochyta rating (score 1-9) for chickpea at 119 days after sowing for row spacing and number of foliar fungicide applications

Row Spacing	Fungicide		
	0 foliar sprays	4 foliar sprays	8 foliar sprays
178 mm	7.08 <i>c</i>	5.08 <i>bc</i>	1.00 <i>a</i>
356 mm	6.76 <i>bc</i>	4.61 <i>b</i>	1.00 <i>a</i>
1000 mm (paired)	6.69 <i>bc</i>	4.66 <i>b</i>	1.00 <i>a</i>

LSD (row spacing x foliar fungicide applications) = 2.19

Ascochyta blight development in chickpea with the different foliar fungicide treatments is shown in Table 3. No disease was found when 8 foliar fungicide sprays were applied. Both the control and 4-time fungicide treatments showed an increase in Ascochyta development with time. Regression analysis showed the slopes of the Ascochyta progression to be parallel with the control and the 4-time fungicide applications, with the difference being the 25 days advanced infection (determined by points of intercept) where no spraying was involved.

Table 3. Disease progression with the different foliar spray treatments

Spray schedule	Rating score (scale 1-9) days post sowing					
	75	83	91	100	108	119
0	3.40 <i>b</i>	4.93 <i>c</i>	5.76 <i>c</i>	6.52 <i>c</i>	6.58 <i>c</i>	6.84 <i>b</i>
4	1.53 <i>a</i>	3.18 <i>b</i>	3.59 <i>b</i>	3.58 <i>b</i>	4.44 <i>b</i>	4.78 <i>b</i>
8	1.00 <i>a</i>	1.10 <i>a</i>	1.00 <i>a</i>	1.09 <i>a</i>	1.00 <i>a</i>	1.00 <i>a</i>
LSD	0.89	1.16	1.22	1.44	1.34	2.19

The largest grain yield differences between treatments occurred (range 0.46-1.46 t/ha) between fungicide treatments. With the other treatments the yield differences were small, but nonetheless significant. With the row spacing treatments, the 178 mm and 356 mm row spacings had similar grain yields, but the 1000

mm (paired) spacing resulted in a lower yield. There were no interactions between the number of foliar fungicide applications, row spacing and seeding rate for chickpea yield ($P = 0.05$).

Table 4. Main effects of seeding rate (kg/ha), row spacing (mm) and foliar application treatments (LSD in parenthesis) on grain yield (t/ha)

Seeding rate (0.09)		Row spacing (0.11)			Foliar application schedule (0.38)		
75	150	178	356	1000	0	4	8
0.93 a	1.03 b	1.00 a	1.06 ab	0.89 a	0.46 a	1.03 b	1.46 c

Discussion

Yields in this experiment were comparable to chickpea yields at the SARDI trial plots, Turretfield (23 km east of Roseworthy), where Kaniva yielded 1.10 t/ha in 2001 with a long-term average of 1.57 t/ha (Hawthorne, 2002). The disease pressure in the experiment was quite high in the no fungicide treatment, with mean disease scores close to 7 and an associated three-fold reduction in yield compared with the disease free crop. This disease pressure is likely to be similar to that experienced when sowing chickpea back into (or near) where there was a chickpea crop previously infected with *Ascochyta* blight.

Higher plant densities have been shown to increase the severity of various fungal diseases in pulse crops, and high relative humidity with favourable temperatures is reported to encourage *Ascochyta* blight on chickpea (Jettner *et al.* 1999; Siddique *et al.* 1998). Thus the logic is that by reducing humidity with a more open canopy, such as is obtained with lower seeding rates and wider row spacing, and then this could restrict fungal disease development. In the current trial, seeding rate only affected *Ascochyta* levels with the two widest row spacings (significant interaction), where the increased seeding rate resulted in lower disease scores. In this experiment the inter-row space was mostly bare ground and it is possible the higher intra-row density (from higher seeding rate), together with the wider row spacings gave other benefits such as a slowing of the spread of *Ascochyta* by reducing the effects of rain splash (Sweetingham *et al.* 1993).

The use of 4 foliar fungicide sprays delayed the onset of *Ascochyta*, compared with the non-sprayed treatment and resulted in a two-fold yield increase. Possibly better control with such a spray regime would be obtained if the fungicide had been applied strategically just prior to significant rainfall events (Craddock *et al.* 2000), rather than the structured calendar regime used for this experiment. The chickpea in this experiment was sown in late winter; this is consistent with recommendations of later sowing dates for chickpeas in South Australia due to the risk of damage from *Ascochyta* (Lamb and Poddar 1996). Lamb and Poddar (1996) also suggest that the later sowing of chickpeas can allow the number of foliar fungicide applications necessary to control *Ascochyta* to be reduced.

Conclusion

At this stage the impact of agronomic management upon *Ascochyta* control may be seen as marginal, when compared to fungicide use and the potential benefits from the breeding of resistant varieties. However these incremental benefits due to agronomic influences are likely to be important particularly when *Ascochyta* is less of a constraint.

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