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Resistance to *Phytophthora medicaginis* Hansen and Maxwell in wild *Cicer* species and its use in breeding root rot resistant chickpea (*Cicer arietinum* L.)

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Abstract. Phytophthora root rot caused by *Phytophthora medicaginis* is a major disease of chickpea in Australia. Only partial resistance, derived from chickpea, is available in Australian cultivars. Five wild *Cicer* species were compared with chickpea cv. Jimbour (moderately resistant) in a field experiment. The proportions of accessions with significantly lower ($P < 0.05$) disease scores, where lower scores equate to higher resistance, were 9/9 for *C. echinospermum*, 9/21 for *C. bijugum*, 1/4 for *C. judaicum*, 1/29 for *C. reticulatum*, and 0/3 for *C. pinnatifidum*. The resistance of *C. echinospermum* (7/7 accessions) but not the other *Cicer* species was reproduced in a greenhouse test. Nine out of 30 chickpea × *C. echinospermum*-derived lines were as resistant as the *C. echinospermum* parents in a separate greenhouse experiment. *C. echinospermum* appears to be the best of the sources we examined for breeding chickpea cultivars resistant to *P. medicaginis*.

Additional keyword: *Cicer* spp.

Introduction

Chickpea (*Cicer arietinum* L.) is an important pulse crop in Australia, especially in New South Wales (NSW) and Queensland (Qld) where peak annual crop areas of 156 700 ha and 111 000 ha, respectively, were sown from 1997 to 2006 (Anon. 2004, 2007). Phytophthora root rot (PRR) caused by *Phytophthora medicaginis* emerged as a major constraint to chickpea production in the 1980s, early in the industry's development in these regions. Symptoms are defoliation from the ground up or wilting of entire plants, decay of lateral and tap roots, and dark brown to black tap root lesions sometimes extending above ground level (Vock *et al*. 1980). Up to 100% mortality can occur following a major rainfall event when saturated soil conditions are most conducive to development of this disease. Yield losses were estimated at 50% for individual crops and 20% for a district, based on aerial and ground estimates of dead plants in 1988 (M. Schwinghamer, unpublished results). In contrast, there have been few reports of PRR outside of Australia, which suggests that it is not regarded as a serious problem elsewhere.

Phytophthora isolates reported to cause PRR in Australia were first identified as *P. megasperma* var. *sojae* (Vock *et al*. 1980) and *P. megasperma* f. sp. *medicaginis* (Irwin and Dale 1982), but these and more recent isolates (Liew and Irwin 1997; R. J. Southwell and M. Schwinghamer, unpublished results) have been re-classified as *P. medicaginis* according to Hansen and Maxwell (1991), along with earlier isolates from North America, which were identified as *P. crytogea* and found experimentally to be pathogenic to chickpea (Erwin 1965). *P. medicaginis* appears to have caused PRR of chickpea in Argentina (Frezzi 1950), India (Suryanarayana and Pathak 1968), and Pakistan (Majid *et al*. 1992), based on limited taxonomic evidence. Other *Phytophthora* species, namely *P. citrophthora* in Argentina (Frezzi 1950) and an undescribed species of *Phytophthora* in Spain (Trapero-Casas *et al*. 1992; Liew and Irwin 1994), have reportedly caused root diseases similar to PRR, but their economic importance is unclear. *P. medicaginis* is pathogenic on lucerne (*Medicago sativa* L.) (Hansen and Maxwell 1991) and can infect annual medics (*Medicago* spp.) (De Haan and Sheaffer 1996; De Haan *et al*. 2002), sulla (*Hedysarum* spp.) (Southwell and Crocker 2005), and several other *Fabaceae* species (CAB International 2006).

Resistance is the most promising and practical control option. Field evaluation of more than 200 chickpea breeding lines and germplasm accessions revealed useful field resistance to *P. medicaginis* (Brinsmead *et al*. 1985). One chickpea accession (CPI 56564 = ICC11870) was used as the source of resistance for the cvv. Barwon, Norwin, Jimbour, Moti, Yorker, and Kyabra released in NSW and Qld since 1991. However, mortality and yield loss still occur in these cultivars under high disease pressure and in seasons conducive to the development of PRR. The absence of highly effective resistance in chickpea, despite a presumed co-evolution of pathogen and host in Transcaucasia (Irwin *et al*. 1995), has been attributed to a reduction in genetic variation during domestication (Abbo *et al*. 2003).

Wild *Cicer* species have more diversity than chickpea in response to a range of biotic and abiotic stresses (Croser *et al*. 2003). Singh *et al*. (2005) used an accession of *Cicer reticulatum* Ladizinski, the presumed progenitor of chickpea, as the source of resistance to 4 fungal root pathogens in crosses with chickpea. In

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Australia, genotypes moderately to highly resistant to the root-lesion nematodes *Pratylenchus thornei* and *P. neglectus* have been identified in *C. reticulatum* and another closely related annual, *Cicer echinospermum* PH Davis (J. P. Thompson, unpublished data). A preliminary investigation of a small set of *C. echinospermum* accessions also found this species to be a superior source of resistance to *P. medicaginis* (Singh *et al*. 1994). This paper reports the reactions of 5 annual, wild *Cicer* species to *P. medicaginis* and the feasibility of exploiting these species as novel sources of resistance. A preliminary report appeared in a Conference Proceedings (Knights *et al*. 2003).

Materials and methods

2001 Field experiment

The experiment included 182 genotypes. Chickpea entries (68 'desi' types and 48 'kabuli' types) from Australia and overseas comprised 7 commercial cultivars including cv. Jimbour, 101 breeding lines, and 8 bulk populations (F_3 or F_4). Wild *Cicer* entries included 29 *C. reticulatum*, 21 *C. bijugum* K.H. Rechinger, 9 *C. echinospermum*, 4 *C. judaicum* Boissier, and 3 *C. pinnatifidum* Jaubert & Spach. Most of these (63) were provided by the International Center for Agricultural Research in the Dry Areas (ICARDA) and 3 were from the International Center for Research in the Semi-Arid Tropics (ICRISAT).

The experimental site was at the Tamworth Agricultural Institute in northern NSW. The soil varied from a brown Dermasol with surface crusting to a red Dermasol with a self-mulching surface and was naturally infected with *P. medicaginis* from a preceding lucerne stand. Early in 2000 a mixture of 4 *P. medicaginis* isolates from chickpea and lucerne was used to generate a dried soil inoculum as follows: 4 × 10-kg batches of brown Dermasol soil were pasteurised, each mixed with chopped agar cultures of 1 *P. medicaginis* isolate, placed in separate plastic tubs in the glasshouse, planted with 3 generations of chickpea seedlings (each flooded once) over 7 months; the soil from all tubs was then pooled, air-dried, crushed, and sieved to give a particle size of ~1 mm. This inoculum was raked into a chickpea field site at the seedling stage in mid 2000 to provide a uniform distribution of disease. The site was left fallow over summer and the experiment sown on 19 July 2001. Seeds were treated with P-Pickel T® (3.6 g thiram/kg plus 2 g thiabendazole/kg) to provide early season control of some common seed- and soil-borne seedling diseases. Plots were single 1.5-m rows, 45 cm apart, sown with 30 seeds of chickpea or 25 seeds of other species. There were 4 replicates arranged in randomised blocks to accommodate the variation in soil type, except for some accessions of the wild *Cicer* species where only 2 or 3 replicates were possible due to insufficient seed. Chickpea cultivars with known reaction (Jimbour, moderately resistant; Howzat, moderately susceptible; Tyson, highly susceptible) were randomly allocated as check plots every fourth row. In-crop rainfall received until plants were scored for resistance was 204 mm and included 7 rain events >10 mm. This resulted in severe PRR infection in susceptible genotypes.

A disease score was assigned to each plot on 16 November when plants were at the late podding stage. This was the visual integration of disease incidence and severity was scored on a 1–9 scale (1, no symptoms; 3, 21–50% plants symptomatic and/or 0–10% plants dead; 5, 51–100% plants symptomatic and/or 21–40% plants dead; 7, 100% plants symptomatic and/or 61–80% plants dead; and 9, all plants dead). Disease scores were analysed in a linear mixed-model framework using residual maximum likelihood (REML). Genotype was fitted as a random effect and spatial techniques (Cullis and Gleeson 1991) were used to account for changes in disease resulting from natural trends in the field. The replicate effect was similar for the 3 check cultivars, indicating that it was probably caused by field effects and not due to the replicates having differing sets of entries. The program ASREML (Gilmour *et al*. 1999) was used to give predicted disease scores (hereafter referred to as DSs) and their standard errors. The assumption of normally distributed residuals was met and no transformation was necessary.

2002 Greenhouse experiment

A greenhouse test was used to evaluate resistance under controlled conditions to an isolate of *P. medicaginis* from chickpea (NSW Scientific Collections Unit, DAR 66705). There were 39 genotypes (29 wild *Cicer* and 10 chickpea) in a randomised complete block design with an experimental unit of 1 plant per cup and 10 replicates. Seed lots known to be free of seed-borne diseases were used. The seed coats of wild *Cicer* species were nicked with a scalpel on the opposite side to the hilum to facilitate imbibition and synchronise germination with chickpea. The seeds were washed for 2 h in running tap water, pre-germinated on germination pads at 22°C in an incubator, and planted in plastic cups containing 134 g of soil-sand (10% w/w) and 0.4 g of dried soil artificially infested with the *P. medicaginis* isolate. Seedlings were grown to a mean plant height of 5 cm, and then subjected to cycles of flooding (40 h) and draining (56 h) and examined daily to determine time of death, i.e. irreversible wilting or cessation of growth. The experiment was terminated after 29 days at 24°C. The survival times (days after planting) were analysed using the Kaplan–Meier estimator in failure time analysis (Kalbfleisch and Prentice 1980).

2004 Greenhouse experiment

The reaction of 30 F_4 and F_5 lines, derived randomly from 4 interspecific crosses of *C. echinospermum* accessions ILWC245 and ILWC246 × chickpea cvv. Howzat and Jimbour, were compared with those of the 2 parent *C. echinospermum* accessions and cv. Tyson. The experimental procedure and analysis were the same as in the 2002 greenhouse experiment, except that seedlings were grown for 35 days and there were only 5 replicates.

Results

Wild Cicer *spp. and chickpea: 2001 field experiment*

Bar plots of the number of genotypes in each DS category (frequency distribution, Fig. 1) showed resistance superior to that of chickpea in individual accessions of all wild *Cicer* spp. except *C. pinnatifidum.* All *C. echinospermum* (9/9) and almost half of the *C. bijugum* accessions (9/21) had DSs significantly lower ($P < 0.05$) than the moderately

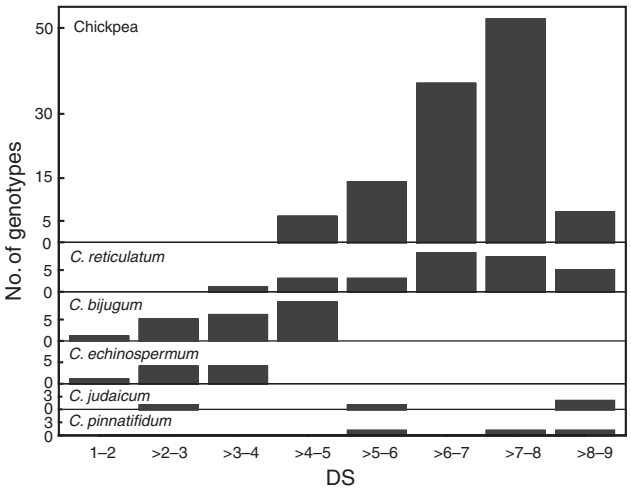

Fig. 1. Comparison of frequency distributions of disease scores (DSs from linear model) of wild *Cicer* spp. and chickpea genotypes in the field experiment at Tamworth in 2001. The DS for the moderately resistant chickpea cv. Jimbour was 5.1.

resistant chickpea cv. Jimbour. Fewer accessions of *C. judaicum*, *C. reticulatum*, and *C. pinnatifidum* (1/4, 1/29, and 0/3, respectively) had DSs significantly lower ($P < 0.05$) than Jimbour. DSs for a subset of chickpea and wild *Cicer* genotypes, i.e ones tested subsequently in the greenhouse, including Jimbour chickpea (DS 5.1), are shown in Fig. 2*a*.

Wild Cicer *spp. and chickpea: 2002 greenhouse experiment*

The resistances of 28 wild *Cicer* accessions that showed low DSs in the field experiment (7 *C. echinospermum*, 3 *C. reticulatum*, 16 *C. bijugum*, and 2 *C. judaicum*, Fig. 2*a*) plus 1 *C. echinospermum* untested in the field experiment were assessed by survival time in the greenhouse test (Fig. 2*b*), together with 4 chickpeas that had been included in the field experiment and 6 previously untested chickpeas of varying resistance (only 1 of which, cv. Norwin, is shown in Fig. 2*b*). All 8 *C. echinospermum* accessions survived significantly ($P < 0.05$) longer than all chickpea genotypes including Jimbour. Only 1 of the other 21 wild *Cicer* accessions (*C. bijugum* ILWC69) and 1 chickpea cultivar (Norwin) survived significantly ($P < 0.05$) longer than Jimbour.

Interspecific lines: 2004 greenhouse experiment

All replicates of 9 chickpea × *C. echinospermum*-derived interspecific lines (out of 30 of these lines tested) and their *C. echinospermum* parents (either ILWC245 or ILW2C46) survived for the duration of the experiment, i.e. 35 days

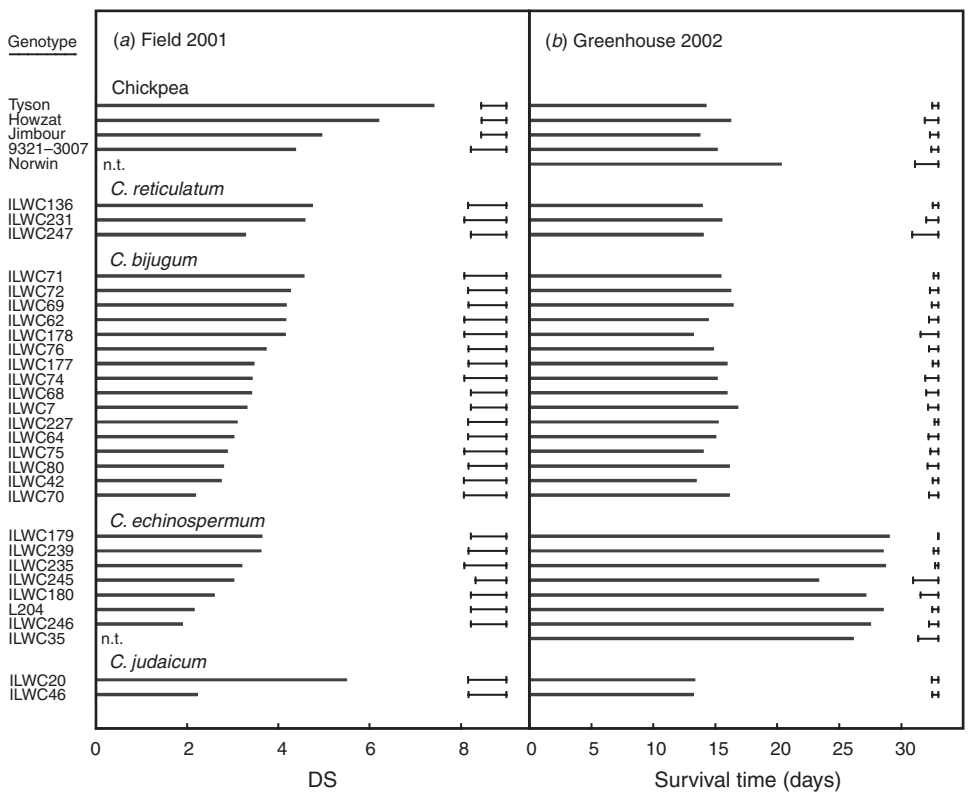

Fig. 2. Comparison of resistance in the 2001 field experiment and the 2002 greenhouse experiment. Left-hand bars in each frame show DSs (disease scores from the linear model) and survival times from analyses and right-hand bars the standard errors. (*a*) DSs for 32 of the 182 genotypes tested in the 2001 field experiment (Fig. 1) in which chickpea cv. Norwin and *C. echinospermum* line ILWC 35 were not tested (n.t.). (*b*) Survival times for all genotypes in the 2002 greenhouse experiment apart from 5 chickpea genotypes (survival times not shown). The greenhouse experiment was terminated 29 days after planting.

(Table 1). Survival time for these and 13 other interspecific lines was significantly ($P < 0.05$) longer than for chickpea cv. Tyson, which was as susceptible as the chickpea parents cv. Jimbour and cv. Howzat in the 2002 greenhouse experiment (Fig. 2*b*).

Discussion

C. echinospermum was the only one of the 5 wild *Cicer* species apart from one accession of *C. bijugum*, which showed superior resistance to *P. medicaginis* in both the field experiment and 2002 greenhouse experiment, i.e. significantly lower DS in the field and significantly longer survival time in the greenhouse compared with chickpea cv. Jimbour.

Table 1. Survival of *C. echinospermum* × chickpea-derived (inter specific) F_4 and F_5 lines compared with their *P. medicaginis*-resistant parents and chickpea cv. Tyson in the 2004 greenhouse experiment

Genotype	Survival time (days)[A]	s.e.
	Chickpea × *C. echinospermum*[B]	
00297–1014	35.0	0
00335–1006	35.0	0
00335–1016	35.0	0
00335–1023	35.0	0
00347–1004	35.0	0
00347–1006	35.0	0
00347–1007	35.0	0
00347–1060	35.0	0
00347–1071	35.0	0
00347–1072	34.6	0.4
00347–1045	32.8	1.4
00297–1010	32.0	2.7
00335–1008	30.6	3.9
00335–1011	30.2	4.3
00335–1030	30.2	4.3
00347–1055	30.2	4.3
00347–1020	30.0	3.8
00347–1051	30.0	4.5
00335–1037	29.4	3.8
00347–1059	29.2	4.2
00347–1014	28.8	4.3
00347–1024	28.0	4.2
00297–1004	25.8	5.1
00335–1007	25.8	5.1
00335–1014	25.6	5.2
00347–1050	25.6	5.1
00347–1022	23.8	4.5
00297–1002	22.5	8.8
00347–1034	16.0	2.8
00304–1001	13.7	2.6
	C. echinospermum parents	
ILWC245	35.0	0
ILWC246	35.0	0
	Chickpea cultivar	
Tyson[C]	17.6	2.8

[A]The experiment was terminated at 35 days.

[B]Crosses were chickpea cv. Jimbour × ILWC246 (genotypes with prefix 00297), cv. Jimbour × ILWC245 (prefix 00304), cv. Howzat × ILWC245 (prefix 00335), and cv. Howzat × ILWC246 (prefix 00347).

[C]Survival not significantly ($P < 0.05$) different from chickpea cvv. Jimbour and Howzat (parents of interspecific lines) in the 2002 greenhouse experiment.

C. echinospermum therefore appeared to be a reliable source of superior resistance.

Individual accessions of *C. reticulatum*, *C. judaicum*, and particularly *C. bijugum* showed superior resistance in the field experiment, but this was not reproduced in the greenhouse experiment (cf. Fig. 2*a* and *b*). The discrepancy suggests that some of the field results were artefactual or that the greenhouse test failed to reveal useful resistance. There was evidence for the latter explanation, in that the 2002 greenhouse experiment did not reveal differences in resistance among chickpea cvv. Tyson (highly susceptible), Howzat (moderately susceptible), and Jimbour (moderately resistant) (Fig. 2*b*), which have been demonstrated repeatedly in field disease nurseries at Tamworth (E. J. Knights and R. J. Southwell, unpublished data). In unpublished greenhouse experiments we previously demonstrated significant differences between field resistant and field susceptible chickpea lines, but these differences were small compared with those between chickpea and *C. echinospermum*. A seedling test in the greenhouse is different in many ways from a field test covering all growth stages and may be less sensitive in resolving differences in resistance. For example, Dale and Irwin (1991) found that resistance to *P. medicaginis*, which was effective in chickpea roots, was not expressed if infection occurred through stomata near the soil surface, as could occur in our greenhouse test that involved repeated flooding.

The 2004 greenhouse experiment demonstrated that the resistance of *C. echinospermum* could be transferred to the progeny of crosses with chickpea. The practicality of using *C. echinospermum* in chickpea improvement programs has been previously established (Singh and Ocampo 1997). This species is considered to be in the primary gene pool (Croser *et al*. 2003), and our experience with many crosses indicates that fertile interspecific progeny can be readily obtained. Although it has many of the primitive features of wild legume species (e.g. spreading growth habit, dehiscent pods), these traits are under simple genetic control and can be easily culled from segregating populations. Moreover, the seed size of *C. echinospermum* is similar to that of many chickpea cultivars, which eliminates one of the main difficulties in recovering domesticated phenotypes in backcross progeny. Using *C. echinospermum* as a parent should not compromise yield potential and the recovery of domesticated features in hybrid or backcross progeny. In Syria the mean yield of 12 F_7 lines was not significantly different from that of the kabuli-type chickpea parent (Singh and Ocampo 1997), and in northern NSW and southern Qld, analysis of 39 trials did not reveal a significant difference between the mean yield of first backcross derivatives and chickpea cultivars and breeding lines (Knights *et al*. 2002). In both studies, the seed quality of interspecific lines matched that of their chickpea counterparts for a range of parameters.

C. echinospermum, *C. bijugum*, *C. judaicum*, *C. reticulatum*, and *C. pinnatifidum* are represented by only 110 original accessions in world germplasm collections. Of the accessions we tested, only 6/9, 6/21, 3/4, 6/29, and 3/3, respectively, represented original accessions (Berger *et al*. 2003); the others were re-selections. Testing of additional original accessions of these species is warranted, particularly for *C. reticulatum*,

which is the progenitor of the cultivated chickpea (Ladizinsky and Adler 1976) and hybridises readily with it.

In our study, *C. echinospermum* resistance was clearly superior to chickpea-derived resistance examined previously by Brinsmead *et al*. (1985) and Dale and Irwin (1991). No other superior source of resistance has been reported. We therefore regard *C. echinospermum* as the best source of resistance to *P. medicaginis* at present and suitable for use in widening the genetic base of chickpea without significantly detracting from the elite nature of the domesticated material.

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References

Anon. (2004) Australian area sown, yield and production of selected pulses by state. In 'Australian Commodity Statistics 2004'. p. 195. (The Australian Bureau of Agricultural and Resource Economics (ABARE): Canberra, ACT)

Anon. (2007) Australian area sown, yield and production of selected pulses by state. In 'Australian Commodity Statistics 2007'. p. 195. (The Australian Bureau of Agricultural and Resource Economics (ABARE): Canberra, ACT)

Abbo S, Berger J, Turner NC (2003) Evolution of cultivated chickpea: four bottlenecks limit diversity and constrain adaptation. *Functional Plant Biology* **30**, 1081–1087. doi: 10.1071/FP03084

Berger J, Abbo S, Turner NC (2003) Ecogeography of annual wild *Cicer* species: the parlous state of the world collection. *Crop Science* **43**, 1076–1090.

Brinsmead RB, Rettke ML, Irwin JAG, Ryley MJ, Langdon PW (1985) Resistance in chickpea to *Phytophthora megasperma* f. sp. *medicaginis. Plant Disease* **69**, 504–506. doi: 10.1094/PD-69-504

CAB International (2006) *Phytophthora medicaginis* (text revised 2006 by R. Southwell). In 'Crop protection compendium'. (CAB International: Wallingford, UK) Available at: www.cabicompendium.org/cpc/home.asp.

Croser JS, Ahmad F, Clarke HJ, Siddique KHM (2003) Utilisation of wild *Cicer* in chickpea improvement—progress, constraints and prospects. *Australian Journal of Agricultural Research* **54**, 429–444. doi: 10.1071/AR02157

Cullis BR, Gleeson AC (1991) Spatial analysis of field experiments—an extension to two dimensions. *Biometrics* **47**, 1449–1460. doi: 10.2307/2532398

Dale ML, Irwin JAG (1991) Stomata as an infection court for *Phytophthora megasperma* f. sp. *medicaginis* in chickpea and a histological study of infection. *Phytopathology* **81**, 375–379. doi: 10.1094/Phyto-81-375

De Haan RL, Sheaffer CC (1996) First report of *Phytophthora medicaginis* causing phytophthora root rot on annual *Medicago* spp. *Plant Disease* **80**, 710.

De Haan RL, Scheaffer CC, Samac JM, Moynihan JM, Barnes DK (2002) Evaluation of annual *Medicago* for upper midwest agroecosystems. *Journal of Agronomy & Crop Science* **188**, 417–425. doi: 10.1046/j.1439-037X.2002.00591.x

Erwin DC (1965) Reclassification of the causal agent of root rot of alfalfa from *Phytophthora cryptogea* to *P. megasperma. Phytopathology* **55**, 1139–1143.

Frezzi MJ (1950) Las especies de "*Phytophthora*" en la Argentina. *Revista de Investigaciones Agricolas (Ministerio de Agricultura y Gansderia, Argentina)* **4**, 47–133.

Gilmour AR, Cullis BR, Welham J, Thompson R (1999) ASREML, Reference manual. Biometric Bulletin 3, NSW Agriculture.

Hansen EM, Maxwell DP (1991) Species of the *Phytophthora megasperma* complex. *Mycologia* **83**, 376–381. doi: 10.2307/3759999

Irwin JAG, Cahill DM, Drenth A (1995) *Phytophthora* in Australia. *Australian Journal of Agricultural Research* **46**, 1311–1337. doi: 10.1071/AR9951311

Irwin JAG, Dale JL (1982) Relationships between *Phytophthora megasperma* isolates from chickpea, lucerne and soybean. *Australian Journal of Botany* **30**, 199–210. doi: 10.1071/BT9820199

Kalbfleisch JD, Prentice RL (1980) 'The statistical analysis of failure time data.' (John Wiley & Sons: New York)

Knights EJ, Brinsmead RB, Fordyce M, Wood JA, Kelly A, Harden S (2002) Use of the wild relative *Cicer echinospermum* in chickpea improvement. In 'Plant Breeding for the 11th Millenium. Proceedings of the 12th Australasian Plant Breeding Conference'. Perth, WA, 15–20 September 2002. (Ed. JA McComb) pp. 93–111. (Australasian Plant Breeding Association Inc.: Perth, WA)

Knights EJ, Southwell RJ, Schwinghamer MW (2003) Evaluation of wild *Cicer* species for resistance to *Phytophthora* root rot. In 'Chickpea Research for the Millennium. Proceedings of the International Chickpea Conference'. (Eds RN Sharma, GK Shrivastava, AL Rathore, ML Sharma, MA Khan) pp. 54–57. (Indira Gandhi Agricultural University: Raipur, India)

Ladizinsky G, Adler A (1976) The origin of chickpea *Cicer arietinum* L. *Euphytica* **25**, 211–217. doi: 10.1007/BF00041547

Liew ECY, Irwin JAG (1994) Comparative studies on *Phytophthora megasperma* isolates from chickpea collected in Australia and Spain. *Mycological Research* **98**, 1284–1290.

Liew ECY, Irwin JAG (1997) Differential disease reactions on lucerne genotypes inoculated with *Phytophthora medicaginis* isolates from lucerne and chickpea. *Australian Journal of Agricultural Research* **48**, 545–551. doi: 10.1071/A96143

Majid K, Aslam M, Saleem A (1992) Root rot of chickpea caused by *Phytophthora megasperma* var. *sojae* Dreschler: a new record for Pakistan. *Pakistan Journal of Phytopathology* **4**, 71.

Singh KB, Malhotra RS, Halila MH, Knights EJ, Verma MM (1994) Current status and future strategy in breeding chickpea for resistance to biotic and abiotic stresses. *Euphytica* **73**, 137–149. doi: 10.1007/BF00027190

Singh KB, Ocampo B (1997) Exploitation of wild *Cicer* species for yield improvement in chickpea. *Theoretical and Applied Genetics* **95**, 418–423. doi: 10.1007/s001220050578

Singh S, Gumber RK, Joshi N, Singh K (2005) Introgression from wild *Cicer reticulatum* to cultivated chickpea for productivity and disease resistance. *Plant Breeding* **124**, 477–480. doi: 10.1111/j.1439-0523.2005.01146.x

Southwell RJ, Crocker GJ (2005) *Hedysarum*—a new susceptible host for *Phytophthora medicaginis. Australasian Plant Pathology* **34**, 265–267. doi: 10.1071/AP05029

Suryanarayana D, Pathak SR (1968) Foot blight—a new disease of gram. *FAO Plant Protection Bulletin* **16**, 71–73.

Trapero-Casas A, Rodriguez-Tello A, Kaiser WJ, Jimenez-Diaz RM (1992) Seedling blight and root rot of chickpea caused by *Phytophthora megasperma* in Spain. In 'Second International Food Legume Research Conference: Program and Abstracts'. 12–16 April 1992, Ramses Hilton, Cairo, Egypt. (Ministry of Agriculture: Cairo, Egypt)

Vock NT, Langdon PW, Pegg KG (1980) Root rot of chickpea caused by *Phytophthora megasperma* var. *sojae* in Queensland. *Australasian Plant Pathology* **9**, 117. doi: 10.1071/APP9800117a

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