# EVALUATION OF DIFFERENT MEASURES TO CONTROL WILT CAUSING PATHOGENS IN CHICKPEA

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**Abstract:** *Fusarium oxysporum* f. sp. *ciceri, F. solani* and *Rhizoctonia solani* were isolated from the wilted chickpea (*Cicer arietinum*) plants. To manage the wilt complex cultural practices, use of biocontrol agents and fungicides were tried under *in vitro* and *in vivo* conditions. Sowing of chickpea at different dates revealed that early sowing (10th Oct.) resulted in maximum disease incidence (32.20%), whereas, late sowing (24th Nov.) the minimum (13.35%). Twenty and 50 cm row to row spacing resulted in maximum (29.17%) and minimum (17.35%) disease incidence respectively. *In vitro* evaluation of biological control agents revealed the superiority of *Trichoderma viride*. *Trichoderma virens* in controlling the pathogens. Carbendazim at 100, 200, 500 ppm caused maximum per cent inhibition of the pathogens under *in vitro* conditions. Fungicides applied as seed treatment reduced disease incidence significantly. Seed treatment with carbendazim increased seed germination (71.24%), though it was at par with carbendazim + mancozeb (62.21%) and mancozeb (61.46%). Seed coating with *T. viride* resulted in minimum disease incidence (9.24%), however, it was at par with *T. viride* (8.10 q/ha). (10.10 q/ha) was recorded with the application of carbendazim, followed by carbendazim + mancozeb (9.77 q/ha) and *T. viride* (8.10 q/ha).

**Key words:** *Cicer arietinum,* wilt complex, *Fusarium oxysporum* f. sp. *ciceri, Fusarium solani, Rhizoctonia solani,* control, *Trichoderma,* fungicides

## INTRODUCTION

Chickpea (Cicer arietinum) occupies an important place in the pulse cultivation and ranks third in the global farming. About 172 pathogens including fungi, bacteria, viruses and nematodes have been reported to infect the crop, out of which 89 have been reported from India alone (Cother 1977). Ascochyta blight (Ascochyta rabies), wilt (Fusarium oxysporum f. sp. ciceri), black root rot (Fusarium solani) and wet root rot (Rhizoctonia solani) are amongst the serious fungal diseases of chickpea (Nene and Reddy 1987). Wilt complex, which manifests itself by wilting or root rots, is one of the most devastating and challenging diseases, which can damage the crop at any stage. The wilt pathogen is seed-borne (Haware et al. 1978) and can survive in soil in the absence of host for more than six years (Haware et al. 1986). Wilt complex in chickpea is caused by several pathogens however, Sclerotium rolfsii, R. solani, F. oxysporum f. sp. ciceri have been considered as the major pathogens. The disease can appear at any stage of plant growth, symptoms in a highly susceptible cultivar can develop any time between 25 days after sowing till as late as podding stage (Nene 1985). The disease has assumed great importance in Jammu Division of Jammu and Kashmir state during the past few years and being soil borne in nature. It is difficult to manage the disease by the application of chemicals only. Though reports on

different aspects of the disease are available from India and abroad, but very scanty information is available on the disease from Jammu Division, which has congenial agro-climatic conditions for the disease development. Thus there is considerable potential of augmenting the yield of chickpea by minimizing the losses inflicted by the biotic factors such as wilt complex. Keeping in view the importance of disease, socio-economic status of the crop and the inadequate research work carried out on the disease in the state, study was undertaken with an objective of managing the disease through cultural practices, biological agents and fungicides.

## MATERIALS AND METHODS

### **Cultural practices**

## Date of sowing and spacing

The experiments were conducted in Randomized block design (RBD), using 2x1.5 m plot size with three replications. In first experiment the chickpea (cv. C-235) seeds were sown at four dates *viz.*, 10th, 25th Oct., 9th and 24th Nov., whereas, in the second experiment sowing was done on second fortnight of October with four row to row spacings *viz.*, 20, 30, 40 and 50 cm.

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#### Biological control

The antagonistic potential of T. viride and T. virens was evaluated against F. oxysporum f. sp. ciceri, F. solani and R. solani using dual culture technique (Dhingra and Sinclair 1995). Five mm mycelial disc of pathogen taken from 7-day-old culture was placed at one end and same sized mycelial disc of antagonist at the opposite end of Petri plate containing Potato Dextrose Agar (PDA) medium. The pathogen and antagonist discs were placed at equal distances from the periphery of the petriplate. The PDA plates inoculated only with pathogen served as control. The plates were incubated at 25±2°C. The experiment was conducted under Completely Randomized Design (CRD) with ten replications for each treatment. The radial growth of developing colonies was recorded at three day intervals and per cent growth inhibition over control was calculated.

#### Chemical control

Five fungicides *viz.*, carbendazim (Bavistin), carbendazim 12% + mancozeb 63% (Companion), captan (Captra), mancozeb (Indofil-M-45) and propicanazole (Tilt), each at 100, 200, and 500 ppm, were evaluated against the pathogens using poisoned food technique (Sinclair and Dhingra 1995), plate without fungicide served as control. Each plate was inoculated with a 5 mm mycelial disc of the pathogen taken from 7 day-old culture and incubated at  $25\pm2^{\circ}$ C. The experiment was conducted under CRD and the treatments were replicated thrice. The colony diameter was recorded when there was complete growth in check and per cent inhibition over check was calculated.

#### Field experiment

The biological control agents and fungicides were tested individually for their efficacy against wilt complex in the field on susceptible chickpea cultivar C-235. The experiment was laid under RBD with ten treatments including control and replicated thrice. The sowing was done on 25 Oct. in 2 m<sup>2</sup> plot with row to row spacing of 30 cm.

## **RESULTS AND DISCUSSION**

## Date of sowing and spacing

Data presented in table 1 reveal that the maximum disease incidence (32.20%) and minimum yield (6.34 q/ha) was observed when chickpea was sown on 10th Oct., whereas, minimum disease incidence (13.35%) and maximum yield (10.62 q/ha) was recorded when sowing was done on 24th Nov. and 9th Nov., respectively. The maximum chickpea seed germination (74.24%) was recorded when sowing was done on 25th Oct. and minimum (61.46%) from 24th Nov. sown crop. Minimum disease incidence in late sown crop is attributed to the environmental factors and less crop canopy which reduces the possibility of moisture buildup and low temperature that favours the development of pathogens. Early sown crops with lush green foliage and excessive canopy helped in conserving moisture, high humidity and low temperature that served as conducive factors for infection and further development and spread of the disease. Our finding corresponds with the earlier studies of Singh and Singh (1984) and Landa *et al.* (2004). The highest yield of 10.62 q/ha with 14.94% disease incidence recorded from 9th Nov. sowing, may be due to better flowering, fruiting and relatively less infection compared to Oct. and late (24th Nov.) sowing. The present findings are in conformity to earlier reports of Singh and Singh (1984). The present investigations conclude that the best time for sowing chickpea was first fort night of Nov., because by doing so the crop escapes heavy infection and subsequent development of the disease and there is maximum seed germination, plant growth and profuse flowering resulting in higher yield.

Table 1. Effect of date of sowing on incidence of chickpea wilt complex under field conditions

Date of sowing	Germination [%]	Percent disease incidence (PDI)	Yield [q/ha]
10th Oct. 2005	65.76 (48.86)	32.20 (34.54)	6.34
25th Oct. 2005	74.24 (57.54)	25.92 (28.48)	7.40
9th Nov. 2005	69.21 (52.04)	14.94 (22.72)	10.62
24th Nov. 2005	61.46 (51.60)	13.35 (21.41)	8.30
CD (p = 0.05)	0.08	1.82	1.99
±SE	0.02	0.56	0.69

\* Figures in parentheses are arc sine transformed values

The maximum (29.17%) and minimum (17.35%) disease incidence was recorded when row to row spacing maintained was 20 and 50 cm, respectively. The highest yield (11.52 q/ha) with corresponding disease incidence of 23.08% was obtained with row to row spacing of 30 cm, whereas, minimum yield (4.96 q/ha) recorded was with 50 cm spacing (Table 2). Maximum disease incidence at row to row spacing of 20 cm could be attributed to the higher crop density and excessive crop canopy which helped in conserving moisture and humidity that favoured the infection and further disease development and secondary spread of disease. The present results corroborates the findings of Singh and Singh (1984) who have reported that larger crop canopy restrict the movement of air which prevents the quick evaporation of moisture conserved by the plants. This also indirectly helps in germination of inoculum, infection of the host plant and further development of the disease. However, maximum yield (11.52 g/ha) was harvested from the crop sown at spacing of 30 cm which is attributed to proper spacing and proper plant population.

Table 2. Effect of row to row spacing on incidence of chickpea wilt complex under field

Row to row spacing	Per cent disease incidence (PDI)	Yield [q/ha]
20 cm	29.17 (29.86)	8.10
30 cm	23.08 (28.46)	11.52
40 cm	20.49 (26.58)	6.04
50 cm	17.35 (24.56)	4.96
CD (p = 0.05)	3.45	0.37
<u>+</u> SE	1.08	0.10

\* Figures in parentheses are arc sine transformed values

	Fusarium oxysp	orum f.sp. ciceri	i Fusarium solani Rhizoctonia sol			nia solani
Treatment	colony diameter [mm]	inhibition [%]	colony diameter [mm]	inhibition [%]	colony diameter [mm]	inhibition [%]
Trichoderma viride	13.40	84.79	12.15	86.34	24.10	73.05
Trichoderma virens	14.25	83.83	13.70	84.60	25.60	71.38
Check	88.15	_	89.00	-	89.45	-
CD (p = 0.05)	1.24		1.18		2.09	
±SE	0.43		0.40		0.72	

Table 3. In vitro evaluation of antagonists against wilt causing pathogens

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	2	F.usarium oxysporum		Fusarium solani		Rhizoctonia solani	
Fungicide	Conc. [ppm]	colony diameter [mm]	inhibition [%]	colony diameter [mm]	inhibition [%]	colony diameter [mm]	inhibition [%]
Captan 50% WP	100	22.33	74.07	90.00	0	90.00	0
(Captra)	200	17.33	80.74	64.33	28.52	73.66	18.15
	500	12.00	86.66	40.66	54.82	10.66	88.15
Carbendazim 50% WP	100	0.00	100	0.00	100	0.00	100
(Bavistin)	200	0.00	100	0.00	100	0.00	100
	500	0.00	100	0.00	100	0.00	100
Carbendazim 12% WP	100	1.33	98.52	6.66	92.60	01.1	89.00
+ Mancozeb 63% WP	200	0.00	100	2.33	97.41	0.00	100
(Companion)	500	0.00	100	0.00	100	0.00	100
Mancozeb 75% WP	100	57.66	35.93	64.56	28.26	52.66	67.44
(Indofil-M-45)	200	50.66	43.71	42.33	52.96	40.66	59.44
	500	39.00	56.66	26.15	70.94	30.00	70.00
Propiconazole 25% EC	100	27.33	69.63	17.66	80.37	0.00	100
(Tilt 25 EC)	200	24.33	72.96	14.66	83.71	0.00	100
	500	22.66	74.82	13.33	85.18	0.00	100
Control	_	90.00	-	90.00	_	90.00	_
CD (p = 0.05)		2.71		5.41		1.04	
±SE		0.94		1.87		0.36	

## **Biological** control

The data presented in table 3 indicate that both *T. viride* and *T. virens* were effective against the isolated pathogens. *T. viride* inhibited the mycelial growth of *F. oxysporum* f. sp. *ciceri*, *F. solani* and *R. solani* by 84.79, 86.34 and 73.05%, respectively, and was statistically at par with *T. virens*. The antagonistic effect can be attributed to diffusible substances (antibiosis) secreted by the antagonists or due to their direct effect on the target pathogens. Effectiveness of *T. viride, T. harzianium* and *T. (Gliocladium) virens* against chickpea wilt complex has also been reported by Sonawane and Pawar (2001), Tewari and Mukhopadhyay (2001) and Gupta *et al.* (2003).

#### Chemical control

The data presented in table 4 revealed that carbendazim was highly effective resulting in 100% inhibition against all the three pathogens even at lowest concentra-

Treatment	Dose [g/kg]	Germination [%]	Per cent disease incidence (PDI)	Disease reduction over control [%]	Yield [q/ha]
Captan 50% WP	3.0	43.23 (41.09)	13.26 (20.72)	53.00	6.20
Carbendazim 50% WP	2.0	71.24 (57.54)	6.48 (14.53)	77.00	10.10
Carbendazim 12% WP + Mancozeb 63% WP	2.0	62.21 (52.04)	8.56 (16.84)	70.00	9.77
Mancozeb 75% WP	3.0	61.46 (51.60)	9.02 (17.15)	68.00	6.98
Propicanazole 25% EC	2 ml	49.43 (44.65)	12.36 (20.29)	56.00	4.22
Gliocladium virens	5.0	58.99 (50.15)	9.72 (18.05)	66.00	7.23
Trichoderma viride	5.0	66.76 (54.77)	9.24 (17.37)	67.00	8.10
Control	_	35.33 (36.45)	28.64 (32.12)	_	4.02
CD (p = 0.05)		0.03	7.2		1.36
<u>+</u> SE		0.01	2.42		0.44

Table 5. Effect of seed treatment with fungicides/bio-control agents on incidence of wilt complex in chickpea under field conditions

\* Figures in parentheses are arc sine transformed values

PDI - Per cent disease incidence

tion (100 ppm), followed by carbendazim + mancozeb which resulted in 98.52% inhibition in case of *F. oxysporum* f. sp. *ciceri*, 92.60% in *F. solani* and 89.00% in *R solani* at 100 ppm. The lowest (35.93%) inhibition was observed with mancozeb at 100 ppm against *F. oxysporum* f. sp. *ciceri*. Propiconazole was found effective against *R. solani* at all the concentrations exhibiting cent per cent inhibition. These findings are in conformity to the earlier results of Sugha *et al.* (1995), Singh and Sindhan (1998), Prajapati *et al.* (2002) and Poddar *et al.* (2004), who have also reported the effectiveness of carbendazim in inhibiting the mycelial growths of wilt and root rot pathogens.

The data regarding effect of seed treatment with different fungicides viz., carbendazim, carbendazim + mancozeb, captan, mancozeb, propiconazole, and biological control agents (T. viride and T. virens) on the incidence of wilt complex tabulated in table 5 revealed that treatment with carbendazim resulted in significantly higher germination (71.24%), followed by carbendazim + mancozeb (62.21%) and mancozeb (61.46%). Minimum disease incidence (6.48%) occurred with carbendazim followed by carbendazim + mancozeb (8.56%). The seed coating with biological control agents indicated that minimum (9.24%) wilt disease incidence was recorded with T. viride, however, it was at par with T. virens (9.72%). Disease reduction due to T. viride (67.00%) and T. virens (66.00%) was less than that of carbendazim and carbendazim + mancozeb. The highest yield of 10.10 q/ha was recorded with the application of carbendazim, followed by the treatment with carbendazim + mancozeb. The average yield obtained in case of T. viride (8.10 q/ha) and T. virens (7.23 q/ha) was higher than that of captan (6.20 q/ha) and propiconazole (4.22 q/ha). Disease management studies with the use of fungicides as seed treatment revealed that the application of carbendazim (0.2%) provided better protection against the disease and resulted in 77.0% disease control over check, followed by 70.0% in carbendazim + mancozeb. This may be attributed to the fact that seed treatment at the preliminary stages might have reduced the initial inoculum present in soil, thereby reducing the secondary

spread of the disease. Our findings on the effectiveness of seed treatment with carbendazim are similar to the results obtained by Sugha *et al.* (1995), Singh and Sindhan (1998) and Poddar *et al.* (2004). The present results regarding use of different biological control agents through seed treatment are in conformity with the findings of Prasad *et al.* (2002) who also reported that application of *T. viride* into soil through seed treatment could be an effective and useful approach. Singh *et al.* (1998) also found that seed treatment of chickpea with bio-control agents like *T. harzianium* and *G. virens* gave excellent results against *R. solani* and *F. oxysporum* f. sp. *ciceri.* 

## REFERENCES

- Cother E.J. 1977. Identification and control of root rot fungi in *Cicer arietinum* (Chickpea). Plant Dis. Rep. 61: 736–740.
- Dhingra O.D., Sinclair J.B. 1995. Basic Plant Pathology Methods. CRS Press, Inc. Boca Raton, Florida, 335 pp.
- Gupta S.B., Thakur M.P., Tedia K., Singh A., Bachkaiya K.K., Kapil S. 2003. Studies on local isolates of *T. viride* and their relationship with wilt/root rot causing fungi of chickpea (*Cicer arietinum* L.). p. 182–188. In: "Chickpea Res. for Mellenium". Proc. Int. Chickpea Conf. Raipur, Chhattisgarh, India, 20–22 January 2003, 449 pp.
- Haware M.P., Nene Y.L., Natrajan M. 1986. The survival of *Fusarium oxysporum* f. sp. *ciceri* in the soil in the absence of chickpea. Phytopathol. Mediterrian 35: 9–12.
- Haware M.P., Nene Y.L., Rajeshwari R. 1978. Eradication of *Fusarium oxysporum* f. sp. *ciceri* transmitted in chickpea seed. Phytopathology 68: 1364–1367.
- Landa B.B., Juan A.N.C., Rafael M.J.D. 2004. Integrated Management of Fusarium wilt of chickpea with sowing date, host resistance, and biological control. Phytopathology 94: 946–960.
- Nene Y.L. 1985. Opportunities for research on disease of pulse crops. Indian Phytopathol. 38: 1–10.
- Nene Y.L., Reddy M.V. 1987. Chickpea disease and their control. p. 233–270. In: "The Chickpea" (M.C. Saxena, K.B. Singh, eds.). Common Wealth Agricultural Bureau Int, Oxon, Eng.

- Poddar R.K., Singh D.V., Dubey S.C. 2004. Management of chickpea wilt through combination of fungicides and bioagents. Indian Phytopathol. 57: 39–43.
- Prajapati R.K., Gangwar R.K., Srivastava S.S.L., Rashmi H.J. 2002. Biological control of wilt and root rot of chickpea under field conditions. Ann. Plant Protect. Sci. 10: 72–75.
- Prasad R.D., Rangeshwaran R., Anuroop C.P., Rashmi H.J. 2002. Biological control of wilt and root rot of chickpea under field conditions. Ann. Plant Protect. Sci. 10: 72–75.
- Sinclair J.B., Dhingra O.D. 1995. Basic Plant Pathology Method. CRS Press, Inc. Boca Raton, Florida, 355 pp.
- Singh R., Sindhan G. 1998. Effect of fungicides on the incidence of dry root rot and biochemical status by chickpea plants. Plant Dis. Res. 13: 35–37.
- Singh R., Sindhan G.S., Parashar R.D., Hooda I. 1998. Application of antagonist in relation to dry root rot and biochemical status of chickpea plants. Plant Dis. Rep. 13: 35–37.
- Singh U.P., Singh R.B. 1984. Effect of date of sowing on the incidence of *Sclerotinia* stem rot and wilt of gram (*Cicer arietinum*). J. Phytopathol. 109: 254–260.
- Sonawane S.S., Pawar N.B. 2001. Studies on biological management of chickpea wilt. Maharashtra Agric. Univ. 26: 215–216.
- Sugha S.K., Kapoor S.K., Singh B.M. 1995. Management of chickpea wilt with fungicides. Indian Phytopathol. 48: 27–31.
- Tewari A.K., Mukhopadhyay A.N. 2001. Testing of different formulations of *Trichoderma virens* against chickpea wilt complex. Indian Phytopathol. 54: 37–71.

## **POLISH SUMMARY**

# OCENA RÓŻNYCH METOD ZWALCZANIA UWIĄDU CIECIERZYCY POSPOLITEJ POWODOWANEGO PRZEZ PATOGENICZNE GRZYBY

Z roślin ciecierzycy pospolitej (Cicer arietinum L.) z objawami uwiądu wyizolowano następujące patogeny: Fusarium oxysporum f.sp. ciceri, Fusarium solani oraz Rhizoctonia solani. W celu zwalczania kompleksu sprawców uwiądu porównywano skuteczność zabiegów agrotechnicznych, czynników biologicznego zwalczania oraz fungicydów zarówno w warunkach in vitro jak też in vivo. Dokonując analizy różnych terminów siewu roślin stwierdzono, że przy wczesnym siewie (10 października) wystąpiło maksymalne nasilenie choroby (32,20%), a w przypadku późnego terminu siewu (24 października) nasilenie choroby było słabe i wynosiło 13,35%. Przy rozstawie międzyrzędzi 20 cm obserwowano silne występowanie choroby, natomiast zwiększenie rozstawu do 50 cm powodowało ograniczenie jej nasilenia, a procentowe wartości wynosiły odpowiednio 29,17 i 17,35%. Ocena czynników biologicznego zwalczania wykazała lepszą skuteczność zwalczania sprawców uwiądu przy wykorzystaniu grzyba Trichoderma viride w porównaniu do Trichoderma virens. Fungicyd karbendazym zastosowany w ilości 100, 200, 500 ppm powodował najsilniejszą inhibicję patogenów w warunkach in vitro. Fungicydy zastosowane jako zaprawy nasienne istotnie obniżały nasilenie choroby. Zaprawianie nasion karbendazymem powodowało wzrost ich kiełkowania (71,24%), chociaż dobre wyniki uzyskano stosując też karbendazym + mankozeb (62,21%) i mankozeb (61,46%). Otoczkowanie nasion przy pomocy grzyba T. viride przyczyniało się ograniczenia występowania choroby (9,24%) i było porównywalne ze skutecznością grzyba T. virens (9,72%). Porównując oceniane metody zwalczania sprawców uwiądu, najwyższy plon uzyskano po zastosowaniu fungicydu karbendazym (10,10 q/ha), a w dalszej kolejności mieszaniny karbendazym + mankozeb (9,77 q/ha) oraz grzyba T. viride (8,10 q/ha).