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RESEARCH ARTICLE

Exploration of fungicides against *Fusarium oxysporum* f. sp. *ciceri* (Padwick) Snyder and Hansen causing wilt of chickpea

■ **D.S. THAWARE, O.D. KOHIRE, V.M. GHOLVE, S.L. BUDGUJAR AND A.A. CHAVAN**

SUMMARY

Six systemic, six non-systemic and contact fungicides were evaluated, *in vitro* against *Fusarium oxysporum* f. sp. *ciceri*; however, the percentage mycelial growth inhibition was found to be increased with increase in concentrations of the test fungicides. All the systemic, non-systemic and contact fungicides tested, however, among the systemic fungicides, carbendazim, carboxin and benomyl and among non-systemic and contact fungicides, carbendazim + mancozeb, benomyl+ thiram and carbendazim + thiram were found inhibit the growth of the test pathogen completely .

Key Words : Systemic fungicides, Non-systemic fungicides, Contact fungicides

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Chickpea *(Cicer arietinum* L.) is an important pulse crop, which belongs to leguminoceae family, ranking third after dry beans *(Phaseolus vulgaris* L.) and dry peas *(Pisum sativum* L.) The centre of origin of chickpea is in Eastern Mediterranean (Aykoid and Doughty, 1964). The Kabuli and Desi chickpea is grown throughout the world with different names *i.e.*, Chickpea (UK), Garbanzo (Latin America), Bengal gram (India), Hommes Hamaz (Arab world), Shimbra (Ethiopia) and Nohud and Loblebi (Turkey). India is largest producer of chickpea in world sharing 65.25 per cent in area and 65.49 per cent in production. In India, chickpea is grown on 10.23 million ha area with production 9.88 million tonnes and productivity 967 kg/ha. The production of chickpea in Maharashtra is 1.62 million tonnes with productivity 891 kg/ha which covered nearly 1.82 million ha of area. Maharashtra contributes about 16.42 per cent share in total production of country (Anonymous, 2014).

Chickpea grows best as a post-monsoon cool season crop in semi-arid regions of the sub-continent. It takes 80 to 170 days to mature. Optimum conditions for growth include 21 to 29°C nights and 18 to 26°C day's temperature with 600-1000 mm annual rainfall

MEMBERS OF THE RESEARCH FORUM

Author to be contacted :
D.S. THAWARE, Department of Plant Pathology, College of Agriculture, Vasantrao Naik Marathwada Krishi Vidyapeeth, PARBHANI (M.S.) INDIA
Email: sanju.6771@rediffmail.com

Address of the Co-authors:
O.D. KOHIRE, V.M. GHOLVE, S.L. BUDGUJAR AND A.A. CHAVAN, Department of Plant Pathology, College of Agriculture, Vasantrao Naik Marathwada Krishi Vidyapeeth, PARBHANI (M.S.) INDIA

(Muehlbauer *et al.,* 1988 and Duke, 1981). In the dry land areas it fixes atmospheric nitrogen in the soil and helps in the management of soil fertility (Sharma and Jodha, 1984).

It plays a vital role in the diet of poor people which serves as a major source of vegetable protein for nutritionally balanced food. It has highest nutritional composition of dry edible grains containing vitamins, carbohydrates, proteins and minerals. It does not contain any anti-nutritional factor. It has considerable amount of fat contents ranging between 3.8-10.2 per cent in different cultivars. After dehulling chickpea seed is valued for its high nutritive value, with 25.3 to 28.9 per cent protein contents (Muehlbauer and Rajesh, 2008 and Hulse, 1991). In addition to source of proteins it has carbohydrate 38-59 per cent, fibre 3 per cent, oil 4.8-5.5 per cent, ash 3 per cent, calcium 0.2 per cent, and phosphorus 0.3 per cent. Its protein and carbohydrate digestibility varies from 76 to 78 per cent and from 57 to 60 per cent (Hulse, 1991 and Huisman and Vanderpoel, 1994).

The major limiting factor in chickpea production is Fusarium wilt which is caused by *F. oxysporum* Schlechtend. Fr. f. sp. *ciceris* (Padwick) Matuo and K. Sato (Jalali and Chand, 1992; Haware, 1990 and Nene and Reddy, 1987). It was first reported in Indo-Pak sub-continent (Butler, 1918). McRae (1932) as well as Prasad and Padwick (1939) reported *F. oxysporum* f. sp. *ciceris* pathogenic to chickpea crop which is now accepted worldwide as the causal agent of *ciceri* spp. In general, the disease causes substantial yield losses which may reach even 100 per cent under favourable weather conditions (Jalali and Chand, 1992). The chickpea is cultivated as a rain fed crop in Maharashtra state and yield losses amounted to 10 to 15 per cent (Khilare *et al.,* 2009).

MATERIAL AND METHODS

Six systemic, six non-systemic and contact fungicides belonging to different groups were tested against the test fungus by using 'poisoned food technique' (Nene and Thapliyal, 1993) in the present assay. Potato dextrose agar medium (PDA) was used as basal medium and was distributed in 250 ml sterilized conical flasks each containing 100 ml. The quantity of fungicide per treatment was calculated for 100 ml medium separately. The requisite quantity of the test fungicides was added to each flask at 45°C. The fungicides were thoroughly mixed before solidification and poured into sterilized Petri plates. The mycelial disc of 7 mm diameter of 7 days old culture was cut with the help of sterile cork borer. Each disc was transferred aseptically to the centre of each Petri plate, already poured with poisoned medium. The PDA plates without fungicide were also inoculated and maintained as control. The plates were incubated at room temperature (27 ± 1°C) for 10 days. Three replications per treatment were maintained. The observations on colony growth and sclerotial formation were recorded until Petri plate in control treatment was fully covered with mycelial growth. The per cent inhibition of growth was calculated by the following formula (Horsfall, 1956):

$$X = \frac{Y - Z}{Y} \times 100$$

where,

X = Per cent inhibition

Y = Growth of fungus in control (cm)

Z = Growth of fungus in treatment (cm)

RESULTS AND DISCUSSION

The results obtained from the present investigation as well as relevant discussion have been summarized under following heads :

In vitro the efficacy of systemic fungicides against the pathogen (Poisoned food technique) :

Six systemic fungicides belonging to different groups were tested for their efficacy against *Fusarium oxysporum* f. sp. *ciceri* by employing poisoned food technique. Results (Table 1) revealed that all the systemic fungicides tested exhibited a wide range of radial mycelial growth of *Fusarium oxysporum* f. sp. *ciceri* and was found to be decreased drastically with increase in the concentrations of the fungicides tested.

At 500 ppm, radial mycelial growth of the test pathogen ranged from 00.00 mm (carbendazim, carboxin and benomyl) to 34.30 mm (difenconazole). However, significantly no mycelial growth was recorded with the fungicides *viz*., carbendazim, carboxin and benomyl (each of 00.00 mm), were at par. Minimum radial growth was recorded with propiconazole (25.30 mm), which was followed by captan (28.00 mm) and difenconazole (34.30 mm) as compared to highest growth (90 mm) in untreated control.

At 1000 ppm, all the systemic fungicides tested exhibited similar trend of mycelial growth as that of at 500 ppm and it ranged from 00.00 mm (Carbendazim,

carboxin and benomyl) to 26.00 mm (difenconazole). However, significantly none of the mycelial growth was recorded with the fungicides, carbendazim, carboxin and benomyl (each of 00.00 mm). Minimum radial growth was recorded with propiconazole (20.00 mm), followed by captan (25.60 mm) and difenconazole (26.00 mm) as compared to highest growth (90 mm) in untreated control.

At 1500 ppm, all the systemic fungicides tested exhibited somewhat similar trend of mycelial growth as that of at 500 ppm and 1000 ppm in the range of 00.00 mm (carbendazim, carboxin and benomyl) to 23.00 mm (captan and difenconazole). However, significantly least mycelial growth was recorded with the fungicides *viz*., carbendazim, carboxin and benomyl (each of 00.00 mm), followed by the fungicide propiconazole (15.30 mm). Minimum radial growth was recorded with captan and difenconazole (each of 23.00 mm) were at par to each other.

Average radial mycelial growth recorded with all the systemic fungicides tested ranged from 00.00 mm (carbendazim, carboxin and benomyl) to 27.70 mm (difenconazole). However, highest average radial mycelial growth was recorded with difenconazole (27.70 mm), followed by captan (25.50 mm) and propiconazole (20.20 mm). The least mean radial mycelial growth was recorded with carbendazim, carboxin and benomyl (each of 00.00 mm). The fungicides carbendazim, carboxin and benomyl, all of three were at par with each other.

Mycelial inhibition :

Results (Table 1) revealed that all the systemic fungicides tested (@ 500, 1000 and 1500 ppm each) significantly inhibited mycelial growth of *Fusarium oxysporum* f. sp. *ciceri* over untreated control (00.00%). Further, the percentage mycelial inhibition of the test pathogen was increased with the increase in concentrations of the fungicides tested.

At 500 ppm, percentage mycelial growth inhibition ranged from 61.88 per cent (difenconazole) to 100 per cent (carbendazim, carboxin and benomyl). However, significantly highest mycelial inhibition was recorded with the fungicides, carbendazim, carboxin and benomyl (each of 100%), all of these were at par, followed by propiconazole (71.88%) and captan (68.88%). The fungicide difenconazole was found less effective with 61.88 per cent inhibition of the test pathogen.

At 1000 ppm, similar trend of mycelial growth inhibition observed and it ranged from 71.11 per cent (difenconazole) to 100 per cent (carbendazim, carboxin and benomyl). However, significantly highest mycelial inhibition was recorded with the fungicides, carbendazim, carboxin and benomyl (each of 100%) all of three were at par, followed by propiconazole (77.77%). The fungicides captan and difenconazole were found less effective with 71.55 per cent and 71.11 per cent inhibition, respectively, to the test pathogen; both were at par.

At 1500 ppm, all the systemic fungicides tested exhibited somewhat similar trend of mycelial growth inhibition as that of at 500 and 1000 ppm and it ranged from 74.44 per cent (difenconazole and captan) to 100 per cent (carbendazim, carboxin and benomyl). However, significantly highest mycelial inhibition was recorded with the fungicides, carbendazim, carboxin and benomyl (each of 100%); all of three were at par, followed by propiconazole (83.00%). The fungicides captan and difenconazole were found less effective with 74.44 per cent and 74.44 per cent inhibition, respectively, of the test pathogen; both were at par.

Average mycelial inhibition with all the systemic fungicides tested ranged from 69.14 per cent

Table 1 : *In vitro* efficacy of systemic fungicides at various concentrations on mycelial growth and inhibition of *Fusarium oxysporum* f. sp. *ciceri*

Treatments No.	Treatments	*Colony diameter (mm) at ppm			Average (mm)	*Per cent inhibition at ppm			Average (%)
		500	1000	1500		500	1000	1500	
T_1	Propiconazole (Tilt 25% EC)	25.30	20.00	15.30	20.20	71.88 (45.92)	77.77 (51.06)	83.00 (56.11)	77.55
T_2	Carbendazim (Bavistin 50% WP)	00	00	00	00	100 (89.99)	100 (89.99)	100 (89.99)	100
T_3	Captan (Captaf 75% WP)	28.00	25.60	23.00	25.50	68.88 (43.55)	71.55 (45.62)	74.44 (48.12)	71.62
T_4	Carboxin (Vitavax 75% WP)	00	00	00	00	100 (89.99)	100 (89.99)	100 (89.99)	100
T_5	Difenconazole (Score 25% EC)	34.30	26.00	23.00	27.70	61.88 (38.31)	71.11 (45.31)	74.44 (48.10)	69.14
T_6	Benomyl (Benlate 50%WP)	00	00	00	00	100 (89.99)	100 (89.99)	100 (89.99)	100
T_7	Control	90.00	90.00	90.00	90.00	00 (00.00)	00 (00.00)	00 (00.00)	00
	C.D. (P=0.01)	0.20	0.17	0.14	0.17	2.25	1.86	3.12	2.41
	S.E. ±	0.05	0.04	0.03	0.04	0.53	0.44	0.74	0.57

* Mean of three replications

* Figures in parenthesis are arc sine transformed value

(Difenconazole) to 100 per cent (Carbendazim, carboxin and benomyl). However, significantly highest average mycelial inhibition was recorded with the fungicides carbendazim, carboxin and benomyl (each of 100%); all of three were at par, followed by propiconazole (77.55%) and captan (71.62%). The fungicide difenconazole was found comparatively less effective with 69.14 per cent inhibition, of the test pathogen.

Thus, all the systemic fungicides tested were found fungistatic against *Fusarium oxysporum* f. sp. *ciceri* and significantly inhibited its mycelial growth over untreated control. However, systemic fungicides found most effective in the order of merit were carbendazim, carboxin, benomyl, propiconazole, captan and difenconazole.The present finding are in closed agreement with finding of Kapoor and Kumar (1991); Kude (1991); llyas *et al.* (1992); Gupta *et al.* (1997); Mayur *et al.* (2001); Singh and Jha (2003); Poddar *et al*. (2004); Kishore and Kulkarni (2008); Mulik (2009) and Subhani *et al.* (2011).

Similar, fungistatic effects of the systemic fungicides against *Fusarium oxysporum* f. sp. *ciceri* infecting chickpea were reported earlier by several workers. Quaiser *et al*. (1989) also reported the complete inhibition of radial growth of *Fusarium oxysporum* f. sp. *ciceri* by carbendazim and benomyl. Sharma and Dohroo (1991) reported that the carbendazim completely inhibited growth of *Fusarium oxysporum* f. sp. *ciceri*. Dikkar *et al*. (2001) reported that carbendazim was effective against chickpea wilt and inhibiting the radial mycelial growth by 100 per cent even at the lowest concentration followed by difenconazole. Andrabi *et al.* (2011) revealed that carbendazim at different concentrations completely inhibited the growth of the pathogens. Korde (2011) also reported that carbendazim and difenconazole were most effective fungicides for fungus.

In vitro the efficacy of non-systemic and contact fungicides against the pathogen (Poisoned food technique) :

Radial mycelial growth :

Six non-systemic and contact fungicides belonging to different groups were tested for their efficacy against *Fusarium oxysporum* f. sp. *ciceri* by employing poisoned food technique. Results (Table 2) revealed that all the non-systemic and contact fungicides tested exhibited a wide range of radial mycelial growth of *Fusarium oxysporum* f. sp. *ciceri* and was found to be decreased drastically with increase in the concentrations of the fungicides tested.

At 2000 ppm, radial mycelial growth of the test pathogen ranged from 00.00 mm (carbendazim + mancozeb, benomyl + thiram and carboxin + thiram) to 37.30 mm (mancozeb). However, significantly none mycelial growth was recorded with the fungicides *viz*., carbendazim + mancozeb, benomyl + thiram and carboxin + thiram (each of 00.00 mm), all of three were at par. Minimum radial growth was recorded with thiram (29.30 mm), which was followed by cymoxnil + mancozeb (34.30 mm) and mancozeb (37.30 mm) as compared to highest growth (90 mm) in untreated control.

At 2500 ppm, all the non-systemic and contact fungicides tested exhibited similar trend of mycelial growth as that of at 2000 ppm and it ranged from 00.00

Table 2 : ***In vitro*** **efficacy of non-systemic and contact fungicides at various concentrations on mycelial growth and inhibition of** ***Fusarium oxysporum*** **f. sp.** ***ciceri***

Treatments No.	Treatments	*Colony diameter (mm) at ppm			Average (mm)	*Per cent inhibition at ppm			Average (%)
		2000	2500	3000		2000	2500	3000	
T_1	Mancozeb (Indofil M-45 75% WP)	37.30	36.60	32.30	35.40	58.55 (35.81)	59.33 (36.33)	64.11 (37.47)	60.66
T_2	Thiram (Thiram 75% WP)	29.30	27.00	2.530	27.20	67.44 (42.37)	70.00 (44.42)	71.88 (46.08)	69.77
T_3	Cymoxnil+Mancozeb (Curzate M 68% WP)	34.30	30.30	20.00	28.20	61.88 (38.20)	66.33 (41.52)	77.77 (51.08)	68.66
T_4	Carbendazim+Mancozeb (Saff 75% WP)	00	00	00	00	100 (89.99)	100 (89.99)	100 (89.99)	100
T_5	Benomyl + Thiram	00	00	00	00	100 (89.99)	100 (89.99)	100 (89.99)	100
T_6	Carbendazim + Thiram	00	00	00	00	100 (89.99)	100 (89.99)	100 (89.99)	100
T_7	Control	90.00	90.00	90.00	90.00	00 (00.00)	00 (00.00)	00 (00.00)	00
	C.D. (P=0.01)	0.21	0.18	0.25	0.21	2.29	1.96	1.32	1.86
	S.E.±	0.05	0.04	0.12	0.07	0.54	0.46	0.31	0.44

*Mean of three replications Figures in parenthesis are arcsine transformed value

mm (carbendazim + mancozeb, benomyl + thiram and carboxin + thiram) to 36.60 mm (mancozeb). However, significantly none of the mycelial growth was recorded with the fungicides *viz*., carbendazim + mancozeb, benomyl + thiram and carboxin+thiram (each 00.00 mm), all of three were at par. Minimum radial growth was recorded with thiram (27.00 mm), followed by cymoxnil + mancozeb (30.30 mm) and mancozeb (36.60 mm) as compared to highest growth (90 mm) in untreated control.

At 3000 ppm, all the non-systemic and contact fungicides tested exhibited somewhat similar trend of mycelial growth as that of at 2000 ppm and 2500 ppm in the range of 00.00 mm (carbendazim + mancozeb, benomyl + thiram and carboxin + thiram) to 32.30 mm (mancozeb). However, significantly least mycelial growth was recorded with the fungicides *viz*., carbendazim + mancozeb, benomyl + thiram and carboxin + thiram (each of 00.00 mm), all of three were at par. Minimum radial growth was recorded with cymoxnil + mancozeb (20.00 mm) which was followed by thiram (25.30 mm) and mancozeb (32.30 mm) as compared to highest growth (90 mm) in untreated control.

Average radial mycelial growth recorded with all the fungicides tested ranged from 00.00 mm (carbendazim + mancozeb, benomyl + thiram and carboxin + thiram) to 35.40 mm (mancozeb). However, highest average radial mycelial growth was recorded with mancozeb (35.40 mm) which was followed by cymoxnil + mancozeb (28.20 mm) and thiram (27.20 mm). The least mean radial mycelial growth was recorded with carbendazim + mancozeb, benomyl + thiram and carboxin + thiram (each of 00.00 mm), all of three were at par.

Mycelial inhibition :

Results (Table 2) revealed that all the non-systemic and contact fungicides tested (@ 2000, 2500 and 3000 ppm each) significantly inhibited mycelial growth of *Fusarium oxysporum* f. sp. *ciceri* over untreated control (00.00%). Further, the percentage mycelial inhibition of the test pathogen was increased with the increase in concentrations of the fungicides tested.

At 2000 ppm, percentage mycelial growth inhibition ranged from 58.55 per cent (mancozeb) to 100 per cent (carbendazim + mancozeb, benomyl + thiram and carboxin + thiram). However, significantly highest mycelial inhibition was recorded with the fungicides carbendazim + mancozeb, benomyl + thiram and carboxin + thiram (each 100%) all of three were at par, followed by thiram (67.44%) and cymoxnil + mancozeb (61.88%). The fungicide mancozeb was found less effective with 58.55 per cent inhibition of the test pathogen.

At 2500 ppm, similar trend of mycelial growth inhibition as that of at 2000 ppm was observed and it ranged from 59.33 per cent (mancozeb) to 100 per cent (carbendazim + mancozeb, benomyl + thiram and carboxin + thiram). However, significantly highest mycelial inhibition was recorded with the fungicides *viz.*, carbendazim + mancozeb, benomyl + thiram and carboxin + thiram (each of 100%); all of three were at par, followed by thiram (70.00%) and cymoxnil + mancozeb (66.33%). The fungicide mancozeb was found less effective with 59.33 per cent inhibition of the test pathogen.

At 3000 ppm, all the non-systemic and contact fungicides tested exhibited somewhat similar trend of mycelial growth inhibition as that of 2000 and 2500 ppm and it ranged from 64.11 per cent (mancozeb) to 100 per cent (carbendazim + mancozeb, benomyl + thiram and carboxin + thiram). However, significantly highest mycelial inhibition was recorded with the fungicides *viz.*, carbendazim + mancozeb, benomyl + thiram and carboxin + thiram (each of 100%); all of three were at par, followed by cymoxnil + mancozeb (77.77%) and thiram (71.88%). The fungicide mancozeb was found less effective with 64.11 per cent inhibition of the test pathogen.

Average mycelial inhibition with all the non-systemic and contact fungicides tested ranged from 60.14 per cent (mancozeb) to 100 per cent (carbendazim + mancozeb, benomyl + thiram and carboxin + thiram). However, significantly highest average mycelial inhibition was recorded with the fungicides carbendazim + mancozeb, benomyl + thiram and carboxin + thiram (each of 100%); all of three were at par. Thiram (69.77%) and cymoxnil + mancozeb (68.66%), both were at par. The fungicide mancozeb was found comparatively less effective with 60.66 per cent inhibition, of the test pathogen.

Thus, all the non-systemic and contact fungicides tested were found fungistatic against *Fusarium oxysporum* f. sp. *ciceri* and significantly inhibited its mycelial growth over untreated control. However, non-systemic and contact fungicides found most effective in the order of merit were carbendazim + mancozeb, benomyl + thiram, carboxin + thiram, thiram, cymoxnil + mancozeb and mancozeb.

Similar, fungistatic effects of the non-systemic and

contact fungicides against *Fusarium oxysporum* f. sp. *ciceri* infecting chickpea were reported earlier by several workers. Sharma and Dohroo (1991) reported that the carbendazim (bavistin) and carbendazim + dithane M-45 (mancozeb) completely inhibited growth of *F. oxysporum.* Aghnoom *et al.* (1999) stated that iprodione + carbendazim, carboxin + thiram and captan reduced Fusarium mycelial growth. Singh and Jha (2003) proved that thiram was the most suitable fungicide in inhibiting the growth of *F. oxysporum* f. sp. *ciceri*. Mulik (2009) reported mancozeb and thiram were effective for *Fusarium oxysporum* f. sp. *ciceri*. The above finding are in closed agreement with finding of Agrawal *et al*. (1974); Mirkova (1988); Mehta *et al.* (1990); Kapoor and Kumar (1991); Tomar *et al*. (1996); Gupta *et al.* (1997); Mayur *et al*. (2001); Chavan (2004); Nikam *et al.* (2007); Kishore and Kulkarni (2008); Patil (2010) and Taskeen-Un-Nisa *et al.* (2011).

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