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RESEARCH PAPER

In vitro bioefficacy of fungicides against *Fusarium oxysporum* f.sp. ciceri causing chickpea wilt

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Abstract: Chickpea (*Cicer arietinum* L.) holds significant importance as a pulse crop cultivated throughout India. *Fusarium oxysporum* f. sp. *ciceri*, the causal agent of chickpea wilt, stands as a major affliction in northern Karnataka. This disease is transmitted via soil and seeds, with abundant pathogenic presence in the soil coupled with favourable environmental conditions leading to plant fatality and subsequent yield depreciation. An experiment was carried out to identify effective fungicides for managing chickpea wilt. Five combinations of fungicides andfive systemic fungicides were tested against *Fusarium oxysporum* f. sp. *ciceri* under laboratory conditions, employing three concentrations for each. Systemic fungicides, specifically carbendazim 50% WP, displayed significant hindrance of mycelial growth, registering 83.61%. Among the combination fungicides, carbendazim 25% + mancozeb 50% demonstrated remarkable complete inhibition of mycelial growth.

Key words : Chickpea, Fungicides, *Fusarium oxysporum* f. sp. *ciceri,* Inhibition

Introduction

Chickpea (*Cicer arietinum* L.) is an important pulse crop grown in tropical, subtropical and temperate regions of the world. It is the world's third most important grain legume after common bean and pea (Anwar *et al.,* 2009). Globally chickpea is grown on 150.04 lakh ha area, with a total production of 158.71 lakh tonnes and withan average productivity of 1057.8 kg/ha (Anon., 2022). During 2021-22, India contributed 86% of total global bengal gram production, with 137.50 lakh tonnes grown on 102.65 lakh hectares with a productivity of 1447 kg/ hectare (agricoop.nic.in).Chickpea is grown as a post monsoon (*rabi*) crop and it occupies very important position in semi-arid farming system both for human nutrition and restoring the soil fertility (Singh and Sirohi, 2003). The estimated yield loss due to insects and diseases varied from 5 to 10% in temperate and 50 to 100% in tropical regions (Van Emden *et al.,* 1988). So far, over 172 pathogens have been documented to infect chickpea in various regions of the world (Nene *et al.,* 1996), although only a handful have the capacity to damage the crop. Chickpea wilt complex is the most significant, destructive, and difficult to control, causing seed rot, seedling blight, root rot, and mature plant wilt, resulting in a 60-70% production loss (Tewari and Mukhopadhyay, 2001). Multipathogenic disease with a complexityand is caused by two or more pathogens.In general, *F. oxysporum* f. sp. *ciceri* (Padwick) Snyd. and Hans, frequently referred to as FOC, is blamed for chickpea wilt.The disease has been recorded from 23 different nations and is significant between the latitudes of 30°N and 30°S of the equator, where the chickpea growing season is dry and warm (Nene *et al.,* 1989).The fungus can attack a number of different crops, such as lentil, pea, pigeonpea, alfalfa, and broad bean, even though it is primarily pathogenic to chickpea (Haware and Nene, 1982; Trapero-Casas and Jimenez-Diaz, 1985), without showing any overt symptoms.Characteristic symptoms of wilt are drooping of leaves and petioles, no external rotting of roots and black internal discoloration involving xylem and pith (Dubey and Singh, 2004). The disease is characterized by two syndromes, namely vascular wilt and yellowing that can be distinguished by both symptomology and chronological development. The wilt syndrome results in a rapid flaccidity and desiccation of the leaves and stems within 20 days after inoculation. Whereas yellowing syndrome results in a progressive foliar yellowing followed by necrosis on 30-40 days after inoculation (Trapero-Cases and Jimenez-Diaz, 1985).

Material and methods

Isolation of *Fusarium oxysporum* f. sp. *ciceri*

The disease specimens collected from different areas of northern Karnataka during survey used for isolation of pathogen associated with chickpea wilt. *Fusarium oxysporum* f. sp. *ciceri* was isolated from infected tissues. The *Fusarium oxysporum* f. sp. *ciceri* was identified, purified and preserved in PDA medium and confirmation of *F. oxysporum*. sp. *ciceri*by Koch's postulation and based on the morphological characters described by Booth (1971).

In vitro evaluation of fungicides against *Fusarium oxysporum* f. sp. *ciceri*

Individual fungicides and combi products (Table 1 and Table 2) were added separately into sterilised molten and cooled potato dextrose agar to achieve the appropriate concentration of fungicides using the poisoned food technique (Nene and Thapliyal, 1973).

Following that, 20 ml of the poisoned medium was poured into sterilised Petriplate. A disc or mycelia of five mm size from actively growing zone of seven days old culture was cut by a sterile cork borer and one such disc was placed at the centre of

each agar plate. The control treatment was kept without any fungicide added to the medium. Three replications were kept for the combi product and systemic fungicides. The plates were then incubated at room temperature, and radial growth was evaluated when the fungus reached maximum growth in the control plates. The percentage inhibition of mycelial growth

$$I = \frac{C - T}{C} \times 100$$

above control was estimated using the Vincent (1947) formula.

Where as, I = Per cent mycelial inhibition C = Radial growth in control T = Radial growth in treatment.

Results and discussion

Among the five systemic fungicides evaluated against *Fusarium oxysporum* f.sp. *ciceri* carbendazim 50% WP and tebuconazole 5.36% w/w FS (in all concentration) were found best with mean 83.61and 82.49 per cent mean mycelial inhibition which were on par with each other and significantly superior to all other treatments in inhibiting growth of *F. oxysporum* f.sp. *ciceri*. The least inhibition of mycelial growth was observed in azoxystrobin 23% SC (30.42%) (Fig. 1.). The carbendazim 50% WP and tebuconazole 5.36% w/w FS were effective @ 0.1% with 86.39 and 84.67 per cent inhibition respectively and significantly superior to all fungicides and their concentrations. The least inhibition was found in the azoxystrobin at @ 0.025%

Table 1. Details of systemic fungicides used at 0.025, 0.05 and 0.1 per cent

Chemical name	Trade name
Tebuconazole 5.36 % w/w FS	Raxil
Carbendazim 50% WP	Bavistin
Difenoconazole 25% Ec.	Score
Azoxystrobin 23%SC	Amistar
Propiconazole 25% EC	Propikon

Table 2. Details of combi product fungicides used at 0.15, 0.20 and 0.25 per cent

Chemical name	Trade name
Carboxin 37.5%+ Thiram 37.5% WS	Vitavax power
Mancozeb 50% + Carbendazim 25% WS	Sprint
Penflufen 13.28% w/w + Trifloxystrobin 13.28% w/w FS	EverGol Xtend
Tricyclazole 18% + Mancozeb 62% WP	Merger
Thiophanate methyl 45% + Pyraclostrobin 5% FS	Xelora

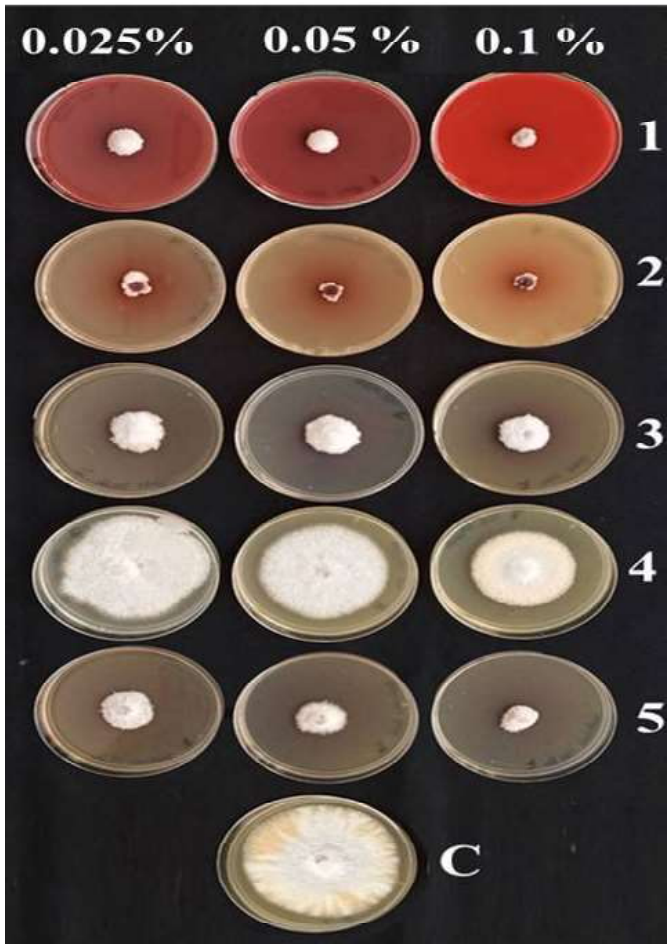

Fig.1. *In vitro* evaluation of systemic fungicides against *Fusarium oxysporum* f.sp. *ciceri*
1. Tebuconazole 5.36 % w/w FS
2. Carbendazim 50% WP
3. Difenoconazole 25% Ec.
4. Azoxystrobin 23%SC
5. Propiconazole 25% EC
C Control

Table 3. *In vitro* evaluation of systemic fungicides against *Fusarium oxysporum* f. sp. *ciceri*

Fungicides	Inhibition of mycelial growth (%)			
	Concentrations (%)			Mean
	0.025	0.05	0.1	
Tebuconazole 5.36% w/w FS	80.03 (63.46)*	82.78 (65.48)	84.67 (66.95)	82.49 (65.26)
Carbendazim 50% WP	80.00 (63.43)	84.44 (66.77)	86.39 (68.35)	83.61 (66.12)
Difenoconazole 25% Ec.	66.67 (54.74)	70.28 (56.96)	72.22 (58.19)	68.47 (55.84)
Azoxystrobin 23%SC	19.44 (26.16)	41.39 (40.04)	44.44 (41.81)	30.42 (33.47)
Propiconazole 25% EC	67.78 (55.42)	72.50 (58.37)	76.94 (61.30)	70.14 (56.88)
Mean	62.74 (52.38)	66.51 (54.64)	70.28 (56.96)	66.51 (54.64)
	S.Em ±	CD at 1%		
Fungicides (F)	0.39	1.45		
Concentrations (C)	0.33	1.28		
FXC	0.68	1.93		

* Angular transformed values

Table 4. *In vitro* evaluation of combi product fungicides against *Fusarium oxysporum* f. sp. *ciceri*

Fungicides	Inhibition of mycelial growth (%)			
	Concentrations (%)			Mean
	0.15	0.2	0.25	
Carboxin 37.5%+ Thiram 37.5% WS	81.08 (64.22)*	98.52 (83.01)	100.00 (90.00)	93.20 (74.88)
Mancozeb 50% + Carbendazim 25% WS	80.42 (63.74)	97.25 (80.45)	100.00 (90.00)	92.55 (74.16)
Penflufen 13.28% w/w + Trifloxystrobin 13.28% w/w FS	50.00 (45.00)	62.00 (51.94)	72.56 (58.41)	61.52 (51.66)
Tricyclazole 18% + Mancozeb 62% WP	86.33 (68.30)	81.33 (64.42)	84.63 (66.92)	84.10 (66.50)
Thiophanate methyl 45% + Pyraclostrobin 5% FS	45.00 (42.13)	62.30 (52.12)	71.93 (58.01)	59.74 (50.62)
Mean	68.57 (55.90)	80.28 (63.64)	85.82 (67.88)	78.22 (62.18)
	S.Em ±	C.D at 1%		
Fungicides (F)	0.424	1.52		
Concentrations (C)	0.306	1.177		
FXC	0.507	1.632		

* Angular transformed values

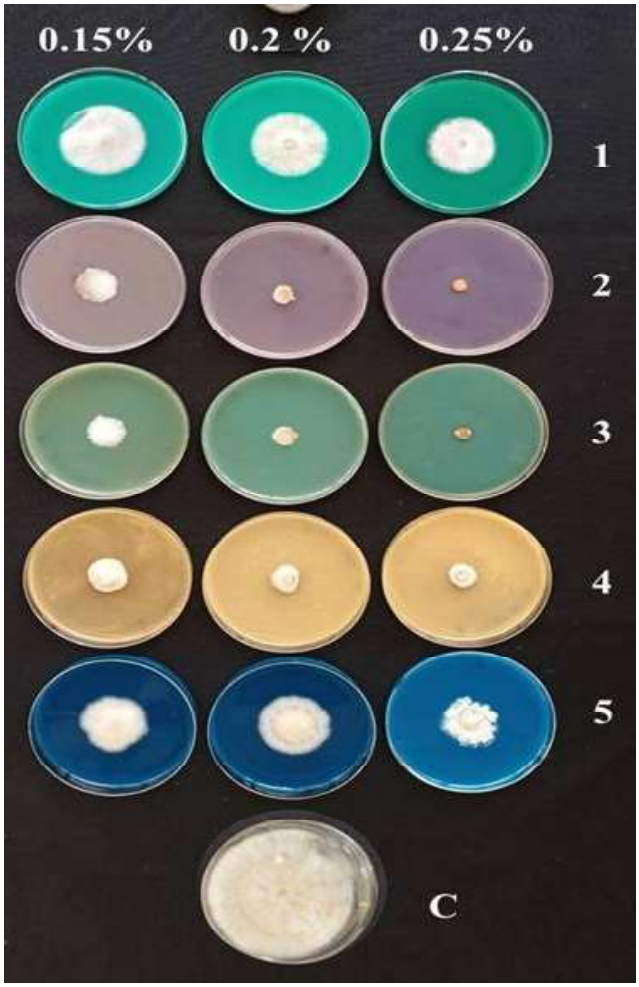

Fig.2. *In vitro* evaluation of combi product fungicides against *Fusarium oxysporum* f.sp. *ciceri*
1. Thiophanate methyl 45% + Pyraclostrobin 5% FS
2. Carboxin 37.5%+ Thiram 37.5% WS
3. Mancozeb 50% + Carbendazim 25% WS
4. Tricyclazole 18% + Mancozeb 62% WP
5. Penflufen 13.28% w/w + Trifloxystrobin 13.28% w/w FS
C. Control

with inhibition of 19.44 % (Table 3). Results depicted in Table 4. indicated that among five combi products evaluated, carboxin 37.5%+ Thiram 37.5% WS)was found to be most effective and significantly superior over control, which inhibited 93.20 cent per cent growth of *F. oxysporum* f.sp. *ciceri*. This was followed by mancozeb 50% + carbendazim 25% WS with 92.55 per cent inhibition which was on par with the first best fungicide vitavax power and the least inhibition of mycelial growth (59.74%) was observed in thiophanate methyl 45% + pyraclostrobin 5% FS. Among different concentrations tested carboxin 37.5%+ thiram 37.5% WS and mancozeb 50% + carbendazim 25% WS at 0.25 per cent concentration caused cent per cent inhibition which was significantly superior all other treatments andthe least inhibition (45 %) was recorded in thiophanate methyl 45% + pyraclostrobin 5% FS at 0.15 per cent (Fig. 2.). Similar results were reported by Ravichandran (2015).

Systemic fungicides primarily operate by disturbing the electron transport chain, thereby impacting the cell's energy allocation, diminishing the synthesis of essential cell components vital for growth and developmental processes, and causing disturbances in cell architecture alongside the permeability of cell membranes. Combination product fungicides tackle the issue of pathogen resistance to systemic fungicides by targeting a limited number of functions within fungal physiology. This limitation makes it susceptible to being overcome either through a single mutation or the selection of resistant individuals in a population.

Conclusion

In the assessment of combi product fungicides against *Fusarium oxysporum* f. sp. *ciceri*, Carboxin 37.5%+ Thiram 37.5% WS and Mancozeb 50% + Carbendazim 25% WSdemonstrated the most favorable outcome, achieving a remarkable inhibition rate. Similarly, in the evaluation of systemic fungicides targeting the same pathogen, carbendazim 50% WP exhibited the highest efficacy, with the maximum inhibition of mycelia.

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