

Original Research Article

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In vitro Bio-Efficacy of Fungicides and Bioagents against Dry Root Rot of Safflower

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ABSTRACT

Keywords

Dry root rot, Safflower, Fungicides, Bioagents, Management, *Macrophomina phaseolina*

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Among the diseases of safflower; dry root rot disease caused by *Macrophomina phaseolina* (Tassi.) Goid is one of the most destructive and wide spread diseases of Safflower (*Carthamus tinctorius* L.) causing accountable losses of about 25-60 per cent. A total of ten fungicides namely: Captan, Iprodione, Copper Oxchloride, Thiram Mancozeb, Difenconazole, Propiconazole, Cymoxanil8%+Mancozeb 64%, Carbandazim, & Metalaxyl 8% + Mancozeb 64% were evaluated *in vitro* against *Macrophomina phaseolina*. All the fungicides (10) tested caused significant inhibition at all the concentration over control. However fungicides *viz.*, Carbendazim, Metalaxyl + Mancozeb and Mancozeb (recorded significantly highest average mycelial growth inhibition of 100.00, 91.42 and 85.86 per cent, respectively. Bioagents (6) evaluated *in vitro* were found fungistatic/antifungal against *M. phaseolina*. However, Fungal antagonists *T. viride*, *T. harzianum* and *T. hamatum* recorded significantly highest mean mycelial inhibition, respectively of 80.00, 77.07 and 66.22 per cent.

Introduction

Safflower (*Carthamus tinctorius* L.) commonly known as *Kardi* (Marathi), *Kusube* (Kannada), *Kusum* (Hindi) and *Kusumba* (Telugu) is one of the important *Rabi* oilseed crop of the country originated from Abyssinia and Afghanistan. It is drought tolerant, self-pollinated crop belonging to the family compositae or asteraceae. The world area under safflower is 8.22 lakh ha with 5.83 lakh tones production and productivity of 709 kg/ha. India is in first place in terms of area

and production of safflower in the world. In India safflower is raised over an area of 296.0lakh ha with a production of 180.0 lakh tones. The important states growing safflower are Maharashtra, Karnataka, Andhra Pradesh, Madhya Pradesh, Gujarat, Orissa and Bihar. In Maharashtra it has occupied an area of 193 lakh ha with a production of 114 lakh tones and productivity of 510 kg/ha (Anonymous 2013-2014).

Among the diseases of safflower; dry root rot disease caused by *Macrophomina phaseolina*

(Tassi.) Goid is one of the most destructive and wide spread diseases of Safflower (*Carthamus tinctorius* L.) causing accountable losses of about 25-60 per cent. It is the major soil borne disease and appears sporadically all over the country (Shambharkar and Indi, 1987). Occurrence of this disease on safflower in India was first reported by Amarsingh and Bhowmik (1979) from IARI, New Delhi and later on by others from different parts of the country (Kore and Deshmukh, (1982) and Lukade, (1992) from Maharashtra; Singh *et al.*, (1987) from M.P).

The pathogen *Macrophomina phaseolina* has been reported to infect safflower at various growth stages of the plant. Affected plants showed grayish black discoloration at collar region from where the plant later broke. The lateral roots were turned black and their skin was easily sloughed off exposing the inner cortical tissues. Affected portion was covered with small, numerous, black sclerotia. These symptoms were more prominent after flowering and their severity increased at maturity. Above ground appearance of disease plant was expressed with yellow, dried leaves, finally wilting the plant completely which could easily be pulled out from soil (Amarsingh and Bhowmik, 1979). It has therefore become highly essential to have a good knowledge of such association, method of detection as well as way to control them. Considering economic importance of the disease, the present investigation was undertaken.

Materials and Methods

In vitro efficacy of fungicides

Efficacy of 10 fungicides viz Captan, Iprodine, Copper Oxochloride, Thiram Mancozeb, Difenconazole, Propiconazole, Cymoxanil 8%+ Mancozeb 64%, Carbandazim, & Metalaxyl 8% + Mancozeb 64% was evaluated systemic (@ 500, 1000

and 1500 ppm conc) and non- systemic (@1000, 1500 and 2000 ppm conc) *in vitro* against *M. phaseolina*, by Poisoned food technique (Nene and Thapliyal, 1993), using PDA as basal culture medium. Based on active ingredient, the requisite quantity of each test fungicide was calculated and mixed thoroughly with autoclaved and cooled (40⁰C) Potato Dextrose Agar medium (PDA) separately in conical flasks to obtain desired concentrations of 500, 1000, 1500 and 2000 ppm. Fungicide amended PDA medium was then poured (20 ml/plate) aseptically in Petri plates (90 mm dia.) and allowed to solidify at room temperature. For each test fungicide and its test concentration, three replications were maintained. After solidification of the medium, all the plates were inoculated aseptically with a 5 mm culture disc obtained from a week old actively growing pure culture of *M. phaseolina*. The culture disc was placed on PDA in inverted position in the centre of the Petri plate and plates were incubated at 28±2⁰C. Petri plates filled with plain PDA (without any fungicide) and inoculated with the culture disc of the test pathogen were maintained as control (untreated).

Observations on radial mycelial growth/colony diameter of the pathogen were recorded at 24 hrs. interval and continued till the untreated control plate was fully covered with mycelial growth of the test pathogen. Per cent mycelial growth inhibition of the test pathogen with the test fungicides over untreated control was calculated by applying the following formula (Vincent, 1927).

$$\text{Per cent Inhibition (I)} = \frac{C - T}{C} \times 100$$

Where,

C = Growth (mm) of test fungus in untreated control plate

T = Growth (mm) of test fungus in treated plates

In vitro efficacy of bioagents

Five fungal antagonists viz., *Trichoderma viride*, *T. harzianum*, *T. koningii*, *T. hamatum*, *T. virens* and one bacterial antagonists viz., *Pseudomonas fluorescens* were evaluated *in vitro* against *M. phaseolina*, applying Dual culture technique (Dennis and Webster, 1971). Seven days old cultures of the test bioagents and the test pathogen (*M. phaseolina*) grown on agar media were used for the study. The culture disc (5mm) of the test pathogen and bioagent were cut out with sterilized cork borer, from a week old culture. Then two culture discs, one each of the test pathogen and bioagent were placed aseptically at equidistance and exactly opposite with each other on solidified PDA medium in Petri plates and plates were incubated at $28 \pm 2^{\circ}\text{C}$. Three replications were maintained. PDA plates inoculated only with culture disc of the test pathogen were maintained as untreated control.

Observations on linear mycelial growth of the test pathogen and bioagent were recorded at an interval of 24 hours and continued till untreated control plate was fully covered with mycelial growth of the test pathogen. Per cent inhibition of the test pathogen over untreated control was calculated by applying the following formula (Arora and Updhyay, 1978).

Percent growth Inhibition =

$$\frac{\text{Colony growth in - Colony growth in Control plate intersecting plate}}{\text{Colony growth in control plate}} \times 100$$

Results and Discussion

In vitro evaluation of fungicides

A total of 10 fungicides (systemic @ 500, 1000 and 1500 ppm conc. and non systemic

@ 1000, 15000 and 2000 ppm conc.) evaluated *in vitro* against *M. phaseolina* exhibited a wide range of mycelial growth and inhibition of the test pathogen. The results obtained are presented in the Table 1 Figure 1, 2, 3, 4 and PLATE-I.

Mycelial growth

At 500 ppm, systemic fungicide (Table1,fig. 1 and PLATE-I) radial mycelial growth of the test pathogen was ranged from 00.00 mm (Carbendazim) to 40.00 mm (Cymoxanil + Mancozeb), as against 90.00 mm in untreated control. However, Carbendazim was found with none of the mycelial growth. This was followed by the fungicides viz., Metalaxyl + Mancozeb (11.00mm), Propiconazole (18.33mm), Difenconazole (25.66mm). Fungicide Cymoxanil + Mancozeb were found less effective with maximum mycelial growth of 40.00 mm.

At 1000 ppm, systemic and non systemic (Table 1,fig. 2 and PLATE-I) all the 10 fungicides tested exhibited similar trend of radial mycelial growth as that of observed at 500 ppm, but it was comparatively reduced and was ranged from 00.00 mm (Carbendazim) to 37.50 mm (Copper oxychloride), as against 90.00 mm in untreated control. However, none of the mycelial growth was recorded with Carbendazim (00.00mm). This was followed by the fungicides viz., Metalaxyl + Mancozeb (8.00mm), Propiconazole (15.00mm), Mancozeb (16.00 mm), Difenconazole (20.33mm), Thiram (20.66mm), Captan (28.33mm), Iprodione (35.00mm) and Cymoxanil + Mancozeb (36.66 mm) as against 90.00 mm in untreated control. Copper oxychloride was found comparatively less effective with maximum mycelial growth of 37.50 mm. At 1500 ppm, systemic and non systemic (Table 1,fig. 3 and PLATE-I) all the 10 fungicides tested exhibited similar trend of mycelial growth as that of observed at 500

ppm and 1000 ppm and it was ranged from 00.00 mm (Carbendazim) to 33.00 mm (Copper oxychloride), as against 90.00 mm untreated control. However, with the fungicide Carbendazim growth was observed. This was followed by the fungicides Metalaxyl + Mancozeb (4.16mm), Propiconazole (11.66mm), Mancozeb (13.50mm), Difenconazole (17.33mm), Thiram (17.66mm), Captan (22.33mm), Iprodine (31.00mm), Cymoxanil + Mancozeb (33.00). Copper oxychloride was found comparatively less effective with maximum mycelial growth of 34.00 mm.

At 2000 ppm, non systemic fungicide (Table 1, figure 4 and PLATE I) radial mycelial growth of the test pathogen was ranged from 8.66 mm (Mancozeb) to 31.33 mm (Copper oxychloride), as against 90.00 mm in untreated control. However, Mancozeb was found with 8.66mm of the mycelial growth. This was followed by the fungicides *viz.*, Thiram (14.00mm), Captan (19.66mm) and Iprodine (28.00mm). Fungicides Copper oxychloride was found comparatively less effective with maximum mycelial growth of 31.33 mm.

Mycelial growth inhibition

Results (Table 1, fig. 1 and PLATE-I) revealed that all the 10 fungicides tested (@ 500, 1000, 1500 and 2000 each) significantly inhibited mycelial growth of *M. phaseolina*, over untreated control (00.00%). Further, the percentage mycelial growth inhibition was increased with increase in concentrations of the fungicides tested

At 500 ppm, systemic fungicide (Table 1,fig. 1 and PLATE-I) mycelial growth inhibition of the test pathogen was ranged from 55.55 (Cymoxanil + Mancozeb) to 100.00 per cent (Carbendazim). However, fungicide Carbendazim was found best inhibited cent

per cent (100.00%) mycelial growth. The second and third best fungicides found were Metalaxyl + Mancozeb (87.77%) and Propiconazole (79.62%) was followed by Difenconazole (71.85%) Cymoxanil was found least effective (55.55%).

At 1000 ppm, systemic and non systemic (Table 1,fig. 2 and PLATE-I) mycelial growth inhibition was increased compared to 500 ppm and it was ranged from 58.32 (Copper oxychloride) to 100.00 (Carbendazim) per cent. However, fungicide Carbendazim caused cent per cent (100.00%) mycelial inhibition was followed by the fungicides, *viz.*, Metalaxyl + Mancozeb (91.11%) and Propiconazole (83.33), and Mancozeb (82.22%). This was followed by Thiram (77.03%), Difenconazole (77.40%), Captan (68.51%), Iprodine (61.11%), Cymoxanil + Mancozeb (59.62%). Copper oxychloride was found least effective (58.32%).

At 1500 ppm, systemic and non systemic (Table 1, figure 3 and PLATE-I) mycelial growth inhibition was increased compared to 500 ppm, 1000 ppm and it was ranged from 62.22 (Copper oxychloride) to 100.00 (Carbendazim) per cent. However, fungicide Carbendazim caused cent per cent (100.00%) mycelial inhibition was followed by the fungicides, *viz.*, Metalaxyl + Mancozeb (95.39%), Propiconazole (87.58), Mancozeb (85.00%), Difenconazole (80.74%), Thiram (80.37%). This was followed by Captan (75.18%), Iprodine (65.55%), Cymoxanil + Mancozeb (63.33%). Copper oxychloride was found least effective (62.22%).

At 2000 ppm, non systemic (Table 1, figure 4 and PLATE-I) fungicides tested exhibited comparatively increased mycelial growth inhibition than that of at 500, 1000 and 1500ppm and it was ranged from 65.18 (Copper oxychloride) to 90.37 (Mancozeb) per cent inhibition of mycelial growth of the

test pathogen. However, fungicide Mancozeb caused cent per cent (90.37%) mycelial inhibition was followed by the fungicides, viz., Thiram (84.44%), Captan (78.14%), Iprodine (68.88%), and Copper oxychloride (65.18%). Thus, all the fungicides tested were found fungistatic against *M. phaseolina* and

significantly inhibited its mycelial growth over untreated control. However, fungicides found most effective in the order of merit were Carbendazim, Metalaxyl + Mancozeb, Mancozeb, Propiconazole, Thiram, Difenconazole, Captan, Cymoxanil, Iprodine and Copper oxychloride.

Table.1 In vitro efficacy of fungicides against mycelial growth and inhibition of *M. phaseolina*

Tr. No.	Treatments	Col. dia. *(mm)at Conc.			% Inhibition*		
		1000 ppm	1500 ppm	2000 ppm	1000 ppm	1500 ppm	2000 ppm
Non-systemic fungicides							
T ₁	Captan 50WP	28.33	22.33	19.66	68.51 (43.23)	75.18 (48.74)	78.14 (51.38)
T ₂	Iprodine	35.00	31.00	28.00	61.11 (37.66)	65.55 (40.96)	68.88 (43.53)
T ₃	Copper oxychloride 50WP	37.50	34.00	31.33	58.32 (35.67)	62.22 (38.47)	65.18 (40.67)
T ₄	Thiram 75 WP	20.66	17.66	14.00	77.03 (50.38)	80.37 (53.48)	84.44 (57.61)
T ₅	Mancozeb 75WP	16.00	13.50	8.66	82.22 (55.29)	85.00 (58.23)	90.37 (64.65)
Tr. No.	Treatments	500 ppm	1000 ppm	1500 ppm	500 ppm	1000 ppm	1500 ppm
Systemic / Combi fungicides							
T ₆	Difenconazole	25.66	20.33	17.33	71.85 (45.93)	77.40 (50.74)	80.74 (53.83)
T ₇	Propiconazole	18.33	15.00	11.16	79.62 (52.77)	83.33 (56.44)	87.58 (61.16)
T ₈	Cymoxanil 8%+ Mancozeb 64% (72WP)	40.00	36.33	33.00	55.55 (33.74)	59.62 (36.59)	63.33 (38.47)
T ₉	Carbendazim 50WP	00.00	00.00	00.00	100.00 (89.98)	100.00 (89.98)	100.00 (89.98)
T ₁₀	Metalaxyl 8% + Mancozeb64% (72WP)	11.00	8.00	4.16	87.77 (61.42)	91.11 (65.64)	95.39 (72.48)
T ₁₁	Control (Untreated)	90.00	90.00	90.00	00.00 (00.00)	00.00 (00.00)	00.00 (00.00)
	S.E.±	0.32	0.36	0.27	0.58	0.62	0.50
	C.D. (P=0.05)	0.96	1.06	0.79	1.70	1.82	1.47

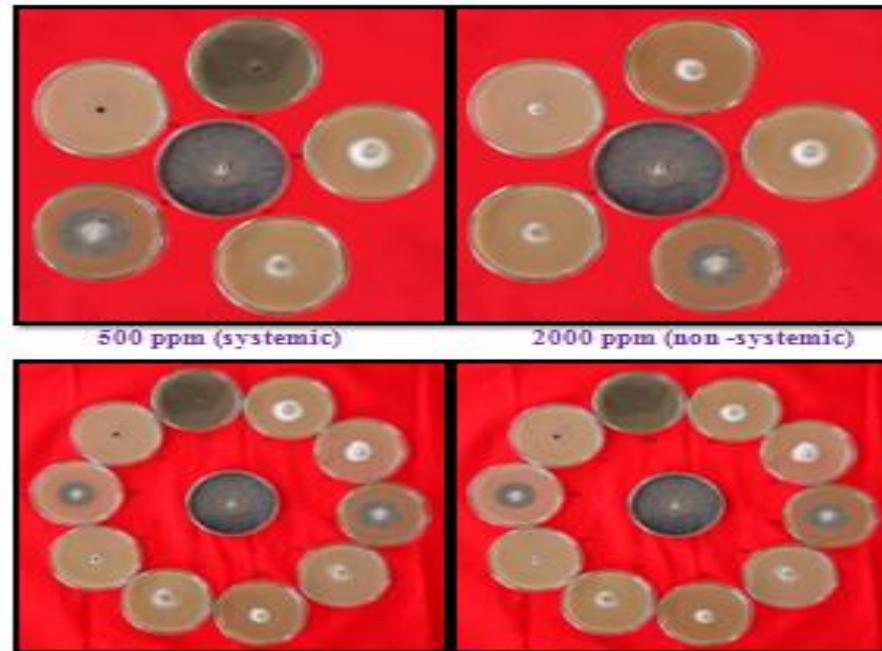
*-Mean of three replications, Col. = Colony, Dia. = Diameter, Conc. = Concentration, Av. =Average, Figures in parenthesis are arc sine transformed value

Table.2 In vitro efficacy of bioagents against mycelial growth and inhibition of *M. phaseolina*

Tr. No.	Treatments	Colony diameter* (mm)	% Inhibition
T ₁	<i>Trichoderma viride</i>	18.00	80.00 (53.12)
T ₂	<i>Trichoderma harzianum</i>	20.66	77.07 (50.40)
T ₃	<i>Trichoderma koningii</i>	34.33	61.85 (38.20)
T ₄	<i>Trichoderma hamatum</i>	34.00	66.22 (38.47)
T ₅	<i>Trichoderma virens</i>	42.00	53.33 (32.23)
T ₆	<i>Pseudomonas fluorescens</i>	38.33	57.04 (35.03)
T ₇	Control (untreated)	90.00	00.00 (00.00)
	S.E. ±	0.56	0.78
	C.D. (P=0.05)	1.72	2.37

*Mean of three replications, Figures in parenthesis are arc sine transformed values

PLATE I



- | | |
|-------------------------------------|---|
| T ₁ : Captan | T ₇ : Propiconazole |
| T ₂ : Iprodione | T ₈ : Cymoxamil + Mancozeb |
| T ₃ : Copper oxychloride | T ₉ : Carbendazim |
| T ₄ : Thiram | T ₁₀ : Metalaxyl 8% + Mancozeb 64% |
| T ₅ : Mancozeb | T ₁₁ : Control (untreated) |
| T ₆ : Difenconazole | |

In vitro efficacy of the systemic (500, 1000, 1500 ppm) and non systemic (1000, 1500, 2000 ppm) fungicides against mycelial growth and inhibition of *M. phaseolina*

PLATE-II



T₁ : *Trichoderma viride*

T₅ : *T. virens*

T₂ : *T. harzianum*

T₆ : *Pseudomonas fluorescens*

T₃ : *T. koningii*

T₇ : Control

T₄ : *T. hamatum*

In vitro* efficacy of the bioagents against mycelial growth and inhibition of *M. phaseolina

Fig.1

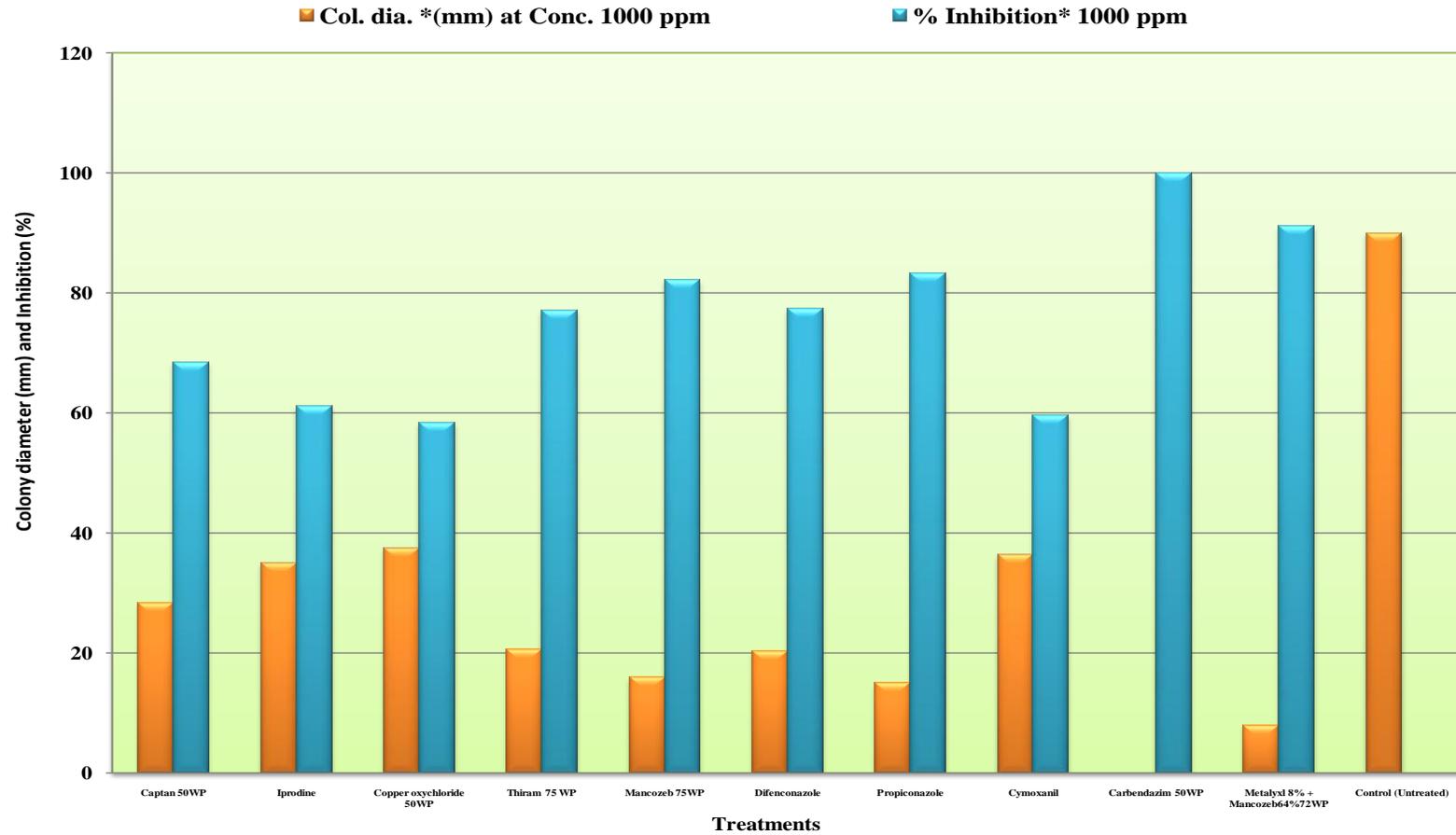


Fig 2. *In vitro* efficacy of systemic and non- systemic fungicides at 1000 ppm against mycelial growth and inhibition of *M. phaseolina*

Fig.2

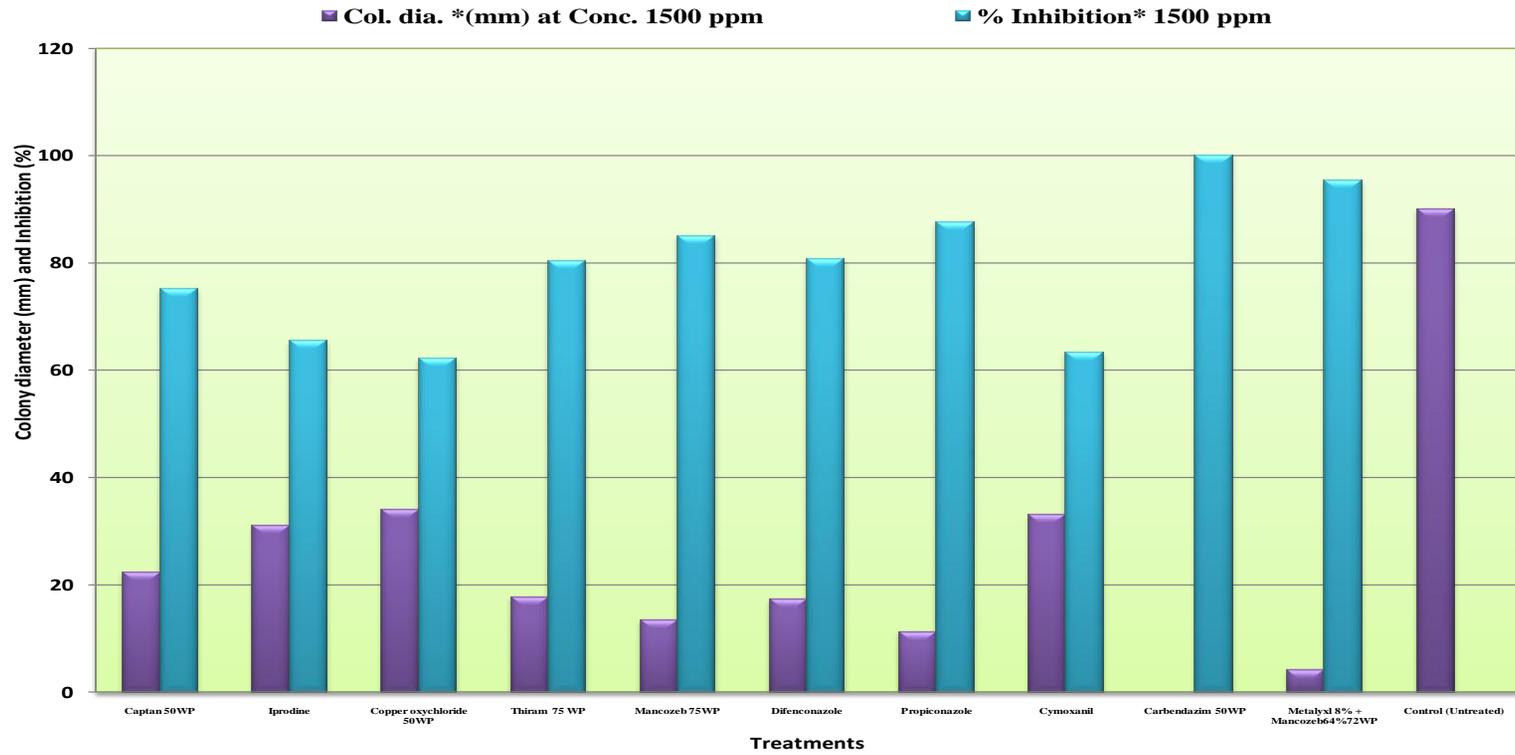


Fig 3. *In vitro* efficacy of systemic and non- systemic fungicides at 1500 ppm against mycelial growth and inhibition of *M. phaseolina*

Similar fungistatic effects of the test fungicides against *M. phaseolina* infecting safflower and many other crops were reported earlier by several workers. Fungicides viz., Carbendazim, Metalaxyl + Mancozeb, Mancozeb, Propiconazole, Thiram, Difenconazole, Captan, Cymoxanil, Iprodione and Copper oxychloride. Were reported to cause significant mycelial growth inhibition of *M. phaseolina*, earlier by several workers (Srivastava and Tripathi, 1998; Lambhate *et al.*, 2002; Malathi and Doraisamy, 2003; Meena *et al.*, 2006; Prajapati *et al.*, 2004; Verma and Ram, 2006; Suryawanshi *et al.*, 2008; Tandel *et al.*, 2010; Magar *et al.*, 2011; Moradia, 2011; Kumari *et al.*, 2012; Chaudhari and Chaudhari, 2012 and Arora *et al.*, 2013).

***In vitro* evaluation of bioagents**

The results obtained on mycelial growth and inhibition of *M. phaseolina* with five fungal and one bacterial antagonists are presented in Table 2 and depicted in Fig.5 and PLATE II. Results revealed that all the bioagents evaluated exhibited fungistatic / antifungal activity against *M. phaseolina* and significantly inhibited its growth over untreated control.

Amongst the bioagents/antagonists tested, *T. viride* was found most effective with significantly least mycelial growth (18.00mm) and highest mycelial growth inhibition (80.00%) of the test pathogen as compared to control. The second and third best antagonists were found *T. harzianum* and *T. hamatum* with second and third least mycelial growth of 20.66 mm and 34.00 mm and inhibition of 77.07 and 66.22 per cent, respectively. These were followed by *T. koningii* and *P. fluorescens* (34.33 and 38.33mm) and (61.85mm and 57.04%) of mycelium growth and its inhibition, respectively. *T. virens* was found comparatively less effective with

maximum mycelial growth (42.00mm) and minimum mycelial inhibition (53.33%).

Results of the present study on antifungal activity of the *T. viride*, *T. harzianum*, *T. hamatum*, *T. koningii* and *T. virens* one bacterial antagonists viz., *P. fluorescens* against *M. phaseolina* are in conformity with those reported earlier by several workers (Hazarika and Das, 1998; Jahagirdar *et al.*, 2001; Kaur *et al.*, 2008; Lokesh and Benagi, 2007; Pan and Bhagat, 2007; Das *et al.*, 2008; Suryawanshi *et al.*, 2008; Meena and Sangit, 2010; Kumari *et al.*, 2012; Shrishail *et al.*, 2012 and Amreen and Sanathkumar, 2013).

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