

Original Research Article

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Efficacy of Fungicides and Bioagents against *Pythium aphanidermatum* Causing Rhizome Rot of Turmeric

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ABSTRACT

Rhizome rot (*Pythium aphanidermatum*) is one of the most wide spread, destructive disease of turmeric (*Curcuma longa* L.), which accounts for about 30 to 80 per cent yield losses. All the fungicides tested significantly inhibited mycelial growth of *P. aphanidermatum*, over untreated control. Average mycelial growth inhibition recorded with the test systemic fungicides was ranged from 73.32 (Propiconazole) to 100 (Metalaxyl) per cent. However, it was cent per cent with Metalaxyl (100 %), followed by Carbendazim (97.67 %), Azoxystrobin (94.55 %), Thiophanate methyl (94.15 %), Fosetyl-AL (86.64 %), Hexaconazole (85.76 %) and Difenconazole (82.85). Whereas, it was comparatively minimum with Propiconazole (73.32 %) and Penconazole (81.14 %). Average mycelial growth inhibition recorded with the test non systemic and contact fungicides was ranged from 50.94 (Metalaxyl 8 % WP + Mancozeb 64 % WP) to 100 (Carbendazim 12 WP + Mancozeb 63 WP) per cent. However, Carbendazim 12 WP + Mancozeb 63 WP gave cent per cent (100 %) mycelial inhibition. The next fungicides with significantly least mycelial growth were Copper oxychloride (97.36 %), followed by Chlorothalonil (76.16 %), Mancozeb (70.62 %). However, Metalaxyl 8 % WP + Mancozeb 64 % WP and Cymoxanil 8 % + Mancozeb 64 % WP were found less effective with minimum mycelial inhibition of 50.94 and 55.23 per cent, respectively.

Keywords

Curcuma longa,
Pythium aphanidermatum,
Fungicides,
Bioagents,
Management.

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Introduction

Turmeric (*Curcuma longa* L.) is one of the major spices cultivated for its underground rhizome belongs to family Zingiberaceae. It is originated from Tropical South Asia. This is also called as 'hidden Lilly' or 'golden spice' or 'turmeric of commerce' or 'Indian saffron' or 'Haldi'. Turmeric is the third largest spice produced in the country and it accounts for about 14 % of total spices produced in India. India is the world's largest producer of turmeric and apparently accounts for more than 80 per cent of the world's production,

followed by China, Indonesia, Bangladesh, and Thailand (Selvan *et al.*, 2002). The area, production and productivity of turmeric in India has been reported to be 175.73 and 185.58 thousand hectares, 959.35 and 943.33 thousand tones and 5459 and 5083 kg/ha, respectively, during year 2014-15 and 2015-16 (Anonymous, 2016). The total area in Maharashtra under turmeric was 11.0 thousand hectares, with production 11.0 thousand tones and productivity of 1000 kg/ha, respectively (Anonymous, 2015).

Turmeric is prone to many fungal, bacterial, viral and nematode diseases. Among all diseases rhizome rot caused by *P. aphanidermatum* is most destructive and widespread disease causes very high crop loss under favorable conditions (Rathaiah, 1982). The disease has been reported to causes more than 60 per cent mortality of seedlings both in nursery and field condition and about 50-80 per cent losses during storage (Nirmal, 1992); rhizome rot resulted in yield loss of 50% (Rajalakshmi *et al.*, 2016).

Materials and Methods

In vitro evaluation of fungicides

Efficacy of nine systemic fungicides and six non-systemic / combi fungicides was evaluated *in vitro* at various concentrations against *P. aphanidermatum*, applying Poisoned food technique (Nene and Thapliyal, 1993) and using Potato dextrose agar (PDA) as basal culture medium. Based on active ingredient, requisite quantity of the test fungicides was calculated, mixed separately thoroughly with autoclaved and cooled (40 °C) PDA medium in conical flasks to obtain desired concentrations. This PDA medium amended separately with the test fungicides was then poured (20 ml / plate) aseptically in Petri plates (90 mm dia.) and allowed to solidify at room temperature. For each of the test fungicide and its desired concentrations, three plates / treatment / replication were maintained. After solidification of the PDA medium, all the plates were inoculated aseptically by placing in the centre a 5 mm culture disc obtained from actively growing 7 days old pure culture of *P. aphanidermatum* and incubated in an inverted position at 28 ± 2 °C. Petri plates filled with plain PDA (without any fungicide) and inoculated with the pure culture disc of *P. aphanidermatum* were maintained as untreated control.

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

$$\text{Per cent inhibition} = \frac{C - T}{C} \times 100$$

Where,

C = growth of the test fungus in untreated control plate

T = growth of the test fungus in treated plate

In vitro evaluation of bioagents

Eight fungal and two bacterial bioagents were evaluated *in vitro* against *P. aphanidermatum*, applying Dual Culture Technique (Dennis and Webster, 1971). Seven days old cultures of the test bioagents and test pathogen (*P. aphanidermatum*) grown on PDA were used for the study. Two 5 mm culture discs, one each of the test pathogen and test bioagents were cut out with sterilized cork borer and placed at equidistance, exactly opposite to each other on autoclaved and solidified PDA medium in Petri plates and three plates were incubated at 28 ± 2 °C. PDA plates inoculated alone with pure culture disc (5 mm) of the test pathogen were maintained as untreated control. The experiment is designed in CRD and all treatments replicated thrice.

Observations on linear mycelial growth of the test pathogen and test bioagent were recorded at an interval of 24 hours and continued till untreated control plates were fully covered with mycelial growth of the test pathogen. Per

cent inhibition of the test pathogen with the test bioagent, over untreated control was calculated by applying following formula (Arora and Upadhyay, 1978).

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

Results and Discussion

In vitro evaluation of systemic fungicides

Mycelial inhibition

Results (Table 1) revealed that all the systemic fungicides tested (each @ 500, 1000 and 1500 ppm) significantly inhibited mycelial growth of *P. aphanidermatum*, over untreated control.

Further, per cent mycelial inhibition was increased with increase in concentrations of the fungicides tested (Fig. 1).

At 500 ppm, mycelial growth inhibition was ranged from 62.72 (Propiconazole) to 100 (Metalaxyl) per cent. However, Metalaxyl gave cent per cent (100 %) mycelial inhibition. The next best fungicides found were Carbendazim (93.01 %), followed by Azoxystrobin (90.70 %), Thiophanate methyl (90.35 %), Fosetyl-AL (81.72 %), Hexaconazole (81.18 %) and Difenconazole (74.81 %). However, Propiconazole and Penconazole were found less effective with minimum mycelial inhibition of 62.72 and 73.40 per cent, respectively.

At 1000 ppm, the trend was same as at 500 ppm and mycelial growth inhibition was ranged from 75.06 (Propiconazole) to 100 (Metalaxyl and Carbendazim) per cent. It was cent per cent with the fungicides Metalaxyl

and Carbendazim (each 100 %). In the order of merit the next most effective fungicides with significantly maximum mycelial inhibition were Azoxystrobin (92.96 %), followed by Thiophanate methyl (92.09 %), Fosetyl-AL (86.73%), Hexaconazole (85.53 %), Difenconazole (84.16 %), Penconazole (82.99 %) and Propiconazole (75.06 %).

At 1500 ppm, mycelial growth inhibition was ranged from 82.17 (Propiconazole) to 100 (Metalaxyl, Carbendazim, Azoxystrobin and Thiophanate methyl) per cent. However, it was cent per cent with the fungicides Metalaxyl, Carbendazim, Azoxystrobin and Thiophanate methyl (each 100 %). The next most effective fungicides were Fosetyl-AL (91.46 %), followed by Hexaconazole (90.57 %), Difenconazole (89.59 %), Penconazole (87.02 %) and Propiconazole (82.17 %).

Average mycelial growth inhibition recorded with the test systemic fungicides was ranged from 73.32 (Propiconazole) to 100 (Metalaxyl) per cent. However, it was cent per cent with Metalaxyl (100 %), followed by Carbendazim (97.67 %), Azoxystrobin (94.55 %), Thiophanate methyl (94.15 %), Fosetyl-AL (86.64 %), Hexaconazole (85.76 %) and Difenconazole (82.85). Whereas, it was comparatively minimum with Propiconazole (73.32 %) and Penconazole (81.14 %).

In vitro evaluation of non-systemic and combi-fungicides

Mycelial inhibition

Results (Table 2) revealed that all the non-systemic and combi fungicides tested (each @ 1500, 2000 and 2500 ppm) significantly inhibited mycelial growth of *P. aphanidermatum*, over untreated control. Further, per cent mycelial inhibition was increased with increase in concentrations of the fungicides tested (Fig. 2).

Table.1 *In vitro* bioefficacy of systemic fungicides against *P. aphanidermatum*

Tr. No.	Treatments	Colony Dia. *(mm) at ppm			Av. (mm)	% Inhibition* at ppm			Av. inhibition (%)
		500	1000	1500		500	1000	1500	
T ₁	Carbendazim 50 WP	6.29	00.00	00.00	2.10	93.01 (74.67)	100.00 (90.00)	100.00 (90.00)	97.67 (81.22)
T ₂	Metalaxyl 50 WP	00.00	00.00	00.00	00.00	100.00 (90.00)	100.00 (90.00)	100.00 (90.00)	100.00 (90.00)
T ₃	Hexaconazole 5 EC	16.94	13.02	8.49	12.82	81.18 (64.29)	85.53 (67.64)	90.57 (72.12)	85.76 (67.83)
T ₄	Difenconazole 25 EC	22.67	14.26	9.37	15.43	74.81 (59.87)	84.16 (66.55)	89.59 (71.18)	82.85 (65.54)
T ₅	Penconazole 10 EC	23.94	15.31	11.68	16.98	73.40 (58.95)	82.99 (65.64)	87.02 (68.88)	81.14 (64.26)
T ₆	Thiophanate methyl 70 WP	8.68	7.12	0.00	5.27	90.35 (71.90)	92.09 (73.67)	100.00 (90.00)	94.15 (76.00)
T ₇	Azoxystrobin 23 SC	8.37	6.34	0.00	4.90	90.70 (72.24)	92.96 (74.61)	100.00 (90.00)	94.55 (76.50)
T ₈	Fosetyl-AL 80 WP	16.45	11.94	7.69	12.03	81.72 (64.69)	86.73 (68.64)	91.46 (73.01)	86.64 (68.56)
T ₉	Propiconazole 25 EC	33.55	22.45	16.05	24.02	62.72 (52.37)	75.06 (60.04)	82.17 (65.02)	73.32 (58.90)
T ₁₀	Control	90.00	90.00	90.00	90.00	00.00 (00.00)	00.00 (00.00)	00.00 (00.00)	00.00 (00.00)
	S.E.±	0.23	0.21	0.17	0.20	0.25	0.24	0.20	0.23
	C.D.(P=0.01)	0.75	0.70	0.59	0.68	0.83	0.75	0.75	0.78

*: Mean of three replications, Dia: Diameter, Av.: Average Figures in Parentheses are angular transformed values

Table.2 *In vitro* evaluation of non-systemic/contact fungicide

Tr. No.	Treatments	Colony Dia. *(mm) at ppm			Av. (mm)	% Inhibition* at ppm			Av. inhibition (%)
		1500	2000	2500		1500	2000	2500	
T ₁	Chlorothalonil 75 WP	26.38	21.25	16.74	21.46	70.69 (57.22)	76.39 (60.93)	81.40 (64.45)	76.16 (60.77)
T ₂	Copper oxychloride 50 WP	7.12	00.00	00.00	2.37	92.09 (73.67)	100.00 (90.00)	100.00 (90.00)	97.36 (80.66)
T ₃	Cymoxanil 8 % + Mancozeb 64 % WP	48.97	39.86	32.04	40.29	45.59 (42.47)	55.71 (48.28)	64.40 (53.37)	55.23 (48.00)
T ₄	Mancozeb 50WP	31.54	26.35	21.44	26.44	64.96 (53.70)	70.72 (57.24)	76.18 (60.79)	70.62 (57.18)
T ₅	Metalaxyl 8 % WP + Mancozeb 64 % WP	53.64	43.68	35.14	44.15	40.40 (39.47)	51.47 (45.84)	60.96 (51.33)	50.94 (45.54)
T ₆	Carbendazim 12% WP + Mancozeb 63 % WP	00.00	00.00	00.00	00.00	100.00 (90.00)	100.00 (90.00)	100.00 (90.00)	100.00 (90.00)
T ₇	Control (untreated)	90.00	90.00	90.00	90.00	00.00 (00.00)	00.00 (00.00)	00.00 (00.00)	00.00 (00.00)
	S.E.±	0.30	0.20	0.24	0.25	0.33	0.23	0.24	0.27
	C.D.(P=0.01)	0.89	0.60	0.73	0.74	0.98	0.67	0.73	0.79

*: Mean of three replications, Dia.: Diameter, Av.: Average Figures in parentheses are angular transformed values

Table.3 *In vitro* bioefficacy of bioagents against *P. aphanidermatum*

Tr. No.	Treatments	Colony Dia. of test pathogen * (mm)	% Inhibition
T ₁	<i>Trichoderma viride</i>	12.77	85.81 (67.87)
T ₂	<i>T. harzianum</i>	17.64	80.40 (63.72)
T ₃	<i>T. hamatum</i>	23.94	73.40 (58.95)
T ₄	<i>T. longibrachiatum</i>	21.86	75.71 (60.47)
T ₅	<i>T. (Gliocladium) virens</i>	19.58	78.24 (62.20)
T ₆	<i>T. koningii</i>	15.22	83.09 (65.72)
T ₇	<i>Aspergillus niger</i>	19.34	78.51 (62.38)
T ₈	<i>T. lignorum</i>	32.14	64.29 (53.30)
T ₉	<i>Pseudomonas fluorescens</i>	51.71	42.54 (40.71)
T ₁₀	<i>Bacillus subtilis</i>	47.66	47.04 (43.31)
T ₁₁	Control (untreated)	90.00	0.00 (0.00)
	S.E. ±	0.55	0.61
	C.D. (P=0.01)	1.56	1.73

*-Mean of three replications, Dia.: Diameter, Figures in Parentheses are angular transformed values

Fig.1 *In vitro* bioefficacy of systemic fungicides against *P. aphanidermatum*

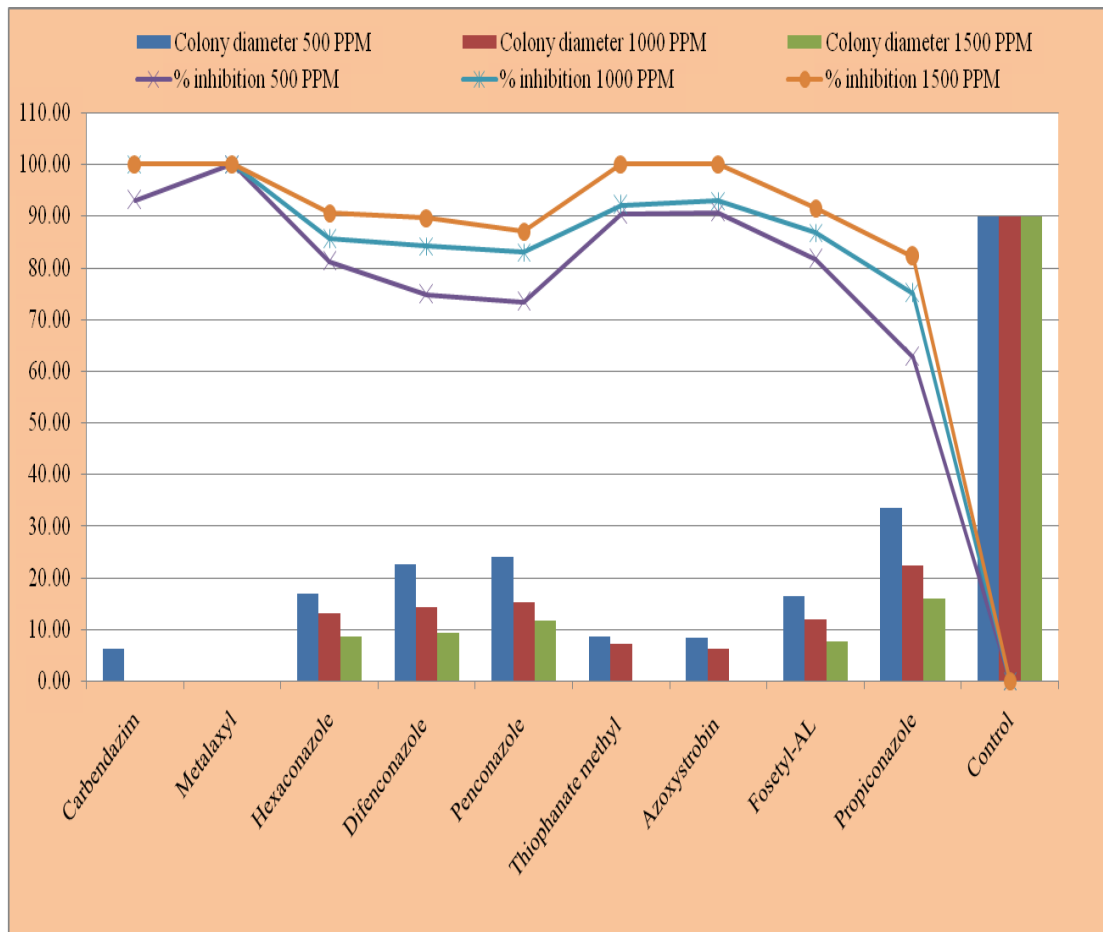


Fig.2 *In vitro* bioefficacy of non-systemic and combi-fungicides against *P. aphanidermatum*

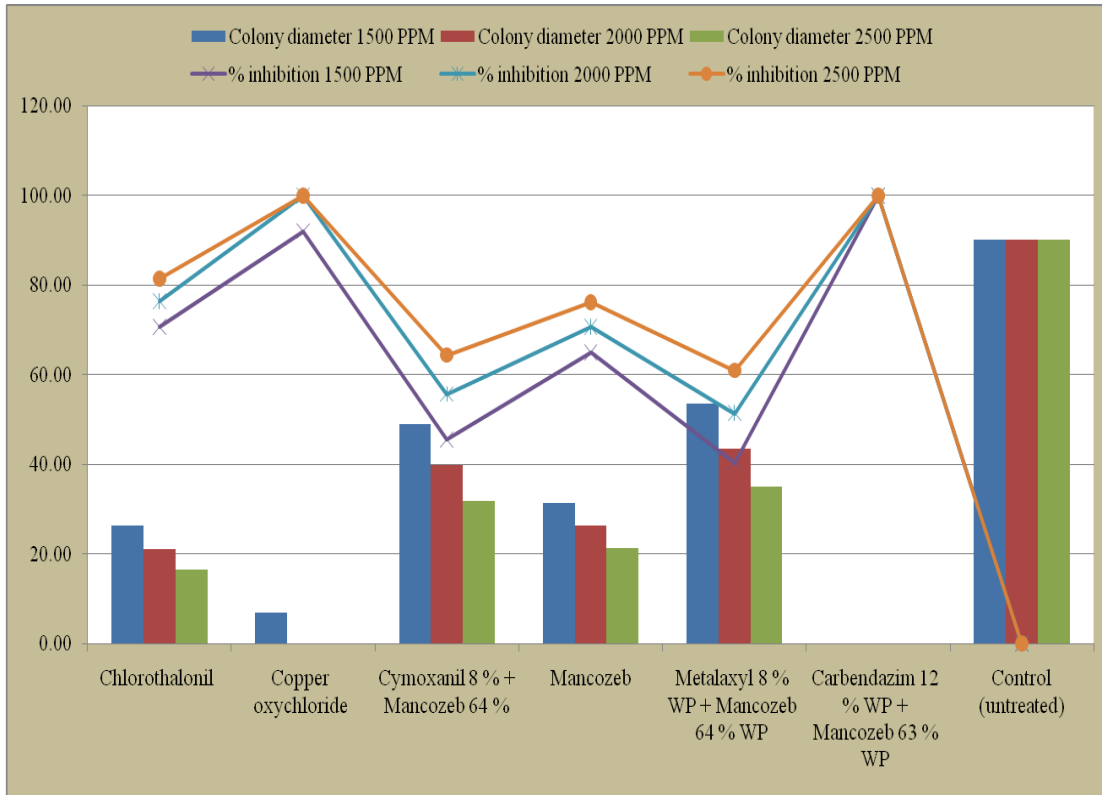
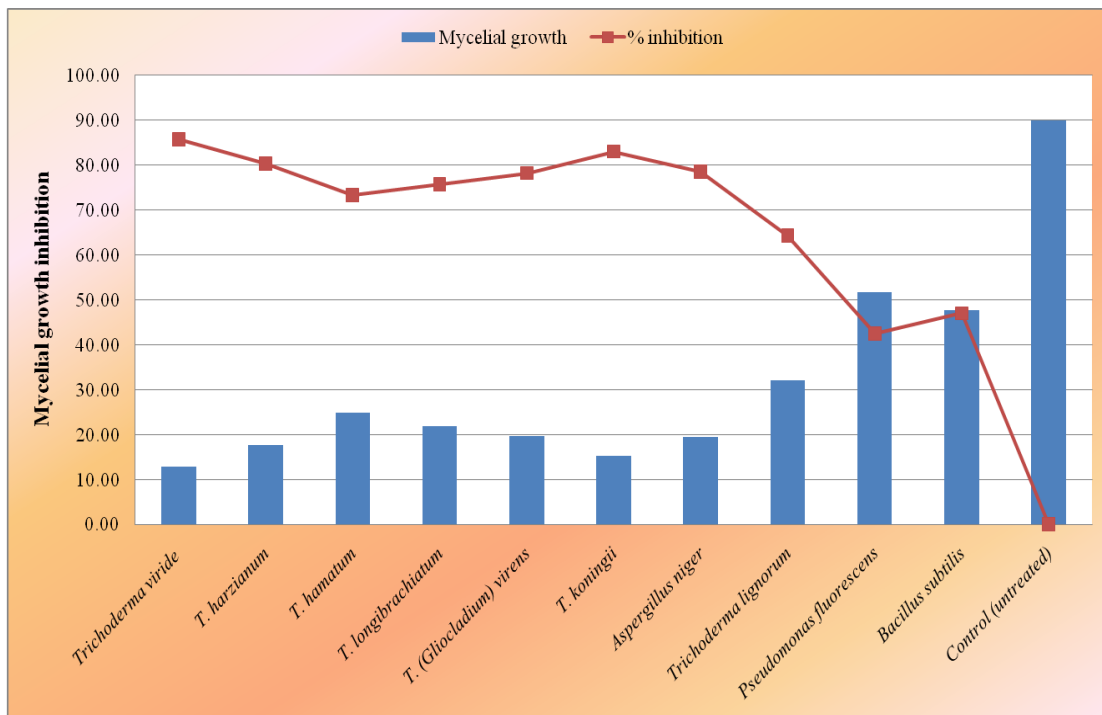


Fig.3 *In vitro* bioefficacy of bioagents against *P. aphanidermatum*



At 1500 ppm, mycelial growth inhibition was ranged from 40.40 (Metalaxyl 8 % WP + Mancozeb 64 % WP) to 100 (Carbendazim 12 WP + Mancozeb 63 WP) per cent. However, Carbendazim 12 WP + Mancozeb 63 WP gave cent per cent (100 %) mycelial inhibition.

The next best fungicides found were Copper oxychloride (92.09 %), followed by Chlorothalonil (70.69 %) and Mancozeb (64.96 %). However, Metalaxyl 8 % WP + Mancozeb 64 % WP and Cymoxanil 8 % + Mancozeb 64 % WP were found less effective with minimum mycelial inhibition of 40.40 and 45.59 per cent, respectively.

At 2000 ppm, mycelial growth inhibition was ranged from 51.47 (Metalaxyl 8 % WP + Mancozeb 64 % WP) to 100 (Carbendazim 12 WP + Mancozeb 63 WP and Copper oxychloride) per cent. However, Carbendazim 12 WP + Mancozeb 63 WP and Copper oxychloride gave cent per cent (100 %) mycelial inhibition. The next best fungicides found were Chlorothalonil (76.39 %), followed by Mancozeb (70.72 %) and Cymoxanil 8 % + Mancozeb 64 % WP (55.71 %). However, Metalaxyl 8 % WP + Mancozeb 64 % WP was found less effective with minimum mycelial inhibition of 51.47 per cent.

At 2500 ppm, mycelial growth inhibition was ranged from 60.96 (Metalaxyl 8 % WP + Mancozeb 64 % WP) to 100 (Carbendazim 12 WP + Mancozeb 63 WP and Copper oxychloride) per cent. However, Carbendazim 12 WP + Mancozeb 63 WP and Copper oxychloride gave cent per cent (100 %) mycelial inhibition. The next fungicides with significantly least mycelial growth were Chlorothalonil (81.40 %), followed by Mancozeb (76.18 %), Cymoxanil 8 % + Mancozeb 64 % WP (64.40 %) and Metalaxyl 8 % WP + Mancozeb 64 % WP (60.96 %).

Average mycelial growth inhibition recorded with the test non systemic and contact fungicides was ranged from 50.94 (Metalaxyl 8 % WP + Mancozeb 64 % WP) to 100 (Carbendazim 12 WP + Mancozeb 63 WP) per cent. However, Carbendazim 12 WP + Mancozeb 63 WP gave cent per cent (100 %) mycelial inhibition. The next fungicides with significantly least mycelial growth were Copper oxychloride (97.36 %), followed by Chlorothalonil (76.16 %), Mancozeb (70.62 %). However, Metalaxyl 8 % WP + Mancozeb 64 % WP and Cymoxanil 8 % + Mancozeb 64 % WP were found less effective with minimum mycelial inhibition of 50.94 and 55.23 per cent, respectively.

In vitro* evaluation of bioagents against *P. aphanidermatum

Results (Fig. 3 and Table 3) revealed that all the bioagents evaluated exhibited fungistatic / antifungal activity against *P. aphanidermatum* and significantly inhibited its growth, over untreated control. Of the antagonists tested, *T. viride* was found most effective with highest mycelial growth inhibition (85.81%) of the test pathogen. The second and third inhibitoriest antagonists found were *T. koningii* and *T. harzianum* with and inhibition of 83.09 and 80.40 per cent, respectively. These were followed by *Aspergillus niger* (78.51 %), *T. (Gliocladium) virens* (78.24 %), *T. longibrachiatum* (75.71 %), *T. hamatum* (73.40 %), *T. lignorum* (64.29 %). However, *P. fluorescens* and *Bacillus subtilis* were found less effective with minimum mycelial inhibition of 42.54 and 47.04 per cent, respectively.

These results are in conformity with the earlier findings of those workers who reported systemic fungicides viz., Carbendazim, Metalaxyl, Hexaconazole, Difenconazole, Penconazole, Thiophanate methyl, Azoxystrobin, Fosetyl-AL and Propiconazole

non systemic and contact fungicides viz., Chlorothalonil, Copper oxychloride, Cymoxanil 8 % + Mancozeb 64 % WP, Mancozeb, Metalaxyl 8 % WP + Mancozeb 64 % WP and Carbendazim 12% WP + Mancozeb 63 % WP at various concentrations had significantly inhibited mycelial growth of *P. aphanidermatum* infecting turmeric (Rekha, 2006), *P. myiotylum* infecting turmeric (Shaikh and Kareappa, 2008), *P. aphanidermatum* infecting ginger (Sagar, 2006; Ram and Thakore, 2009; Rekha, 2013; Kadam 2014).

These results are in conformity with the earlier findings of those workers who reported bioagents viz., *T. viride*, *T. koningii*, *T. harzianum*, *Aspergillus niger*, *T. (Gliocladium) virens*, *T. longibrachiatum*, *T. lignorum*, *Pseudomonas fluorescens* and *Bacillus subtilis* had significantly inhibited mycelial growth of *P. aphanidermatum* infecting turmeric (Sagar, 2006; Ushamalini et al., 2008; Anoop and Bhai, 2014), *P. aphanidermatum*, *P. myriotylum* and *Pythium* spp. infecting ginger (Bhai et al., 2005; Sagar, 2006; Kadam, 2014; Dhroo et al., 2015).

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