

Effect of antagonists on the growth of *Colletotrichum capsici* causing anthracnose of yam (*Dioscorea alata* L.) by dual culture technology



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SUMMARY

The yam crop was found infected with anthracnose disease caused by *Colletotrichum capsici*. In present investigation nine antagonists viz., *Trichoderma viride*, *Trichoderma harzianum*, *Trichoderma longibrachyatum*, *Gliocladium virens*, *Chaetomium globosum*, *Pseudomonas fluorescens*, *Aspergillus niger*, *A. flavus* and *Bacillus subtilis* were tested against *C. capsici* by dual culture technology. *In vitro* studies on interaction of antagonists revealed strong antagonism of *T. viride*, *P. fluorescens* and *Aspergillus flavus*.

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Key words :

Colletotrichum capsici, Growth, Sporulation, Antagonist, Yam

The yam is an important tuber crop originated from the Indo-Burmese region of South East Asia. The yam is a common name for some species in the genus *Dioscorea* (Family: Dioscoreaceae). The major yam producing states in India include Gujarat, Maharashtra, Orissa, Rajasthan, Kerala, West Bengal, Bihar and Assam. Two Asiatic yams, viz., *Dioscorea alata* Linn (greater yam) and *Dioscorea esculenta* (Lour.) Burkill (lesser yam) are the major food of the Indians. In the year 2007, the yam crop was found to be severely affected by anthracnose disease resulting in severe losses on the Horticulture farm of Navsari Agricultural University, Navsari, Therefore, present investigation was carried out to study the interaction of nine different antagonists against *C. capsici*.

The test organism and the pathogen were grown on PDA and from 10 days old culture, a 5 mm disc of the test organism (antagonist) was cut aseptically from the periphery of the colony and placed at one end of the Petri plate

containing 20 ml solidified PDA. In the opposite place an approximately 70 mm away from the first, a similar disc of the pathogen was aseptically placed. Three repetitions of each were kept and the plates with only pathogen served as control. The plates were incubated at room temperature ($27 \pm 2^\circ\text{C}$) and the radial growth of the test organism and pathogen was measured after 6 days. The per cent growth inhibition (PGI) was calculated as per Sundar *et al.* (1995).

$$\% \text{ Inhibition} = \frac{X-Y}{X} \times 100$$

where,

X= Growth of pathogen in control plate (mm)

Y= Growth of pathogen in treated plate (mm)

The results presented in Table 1 revealed that all the antagonists screened against *C. capsici* were significantly superior over the control. Out of these, *Trichoderma viride*

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Table 1 : Testing of antagonists against *C. capsici* under dual culture method

Sr. No.	Test organism	Average colony diameter (mm)	Per cent growth inhibition
1.	<i>Trichoderma viride</i>	6.33	86.24
2.	<i>Trichoderma harzianum</i>	20.33	55.80
3.	<i>Trichoderma longibrachyatum</i>	21.67	52.89
4.	<i>Aspergillus niger</i>	24.67	46.37
5.	<i>Gliocladium virens</i>	8.33	81.89
6.	<i>Pseudomonas fluorescens</i>	6.67	85.50
7.	<i>Aspergillus flavus</i>	7.67	83.33
8.	<i>Bacillus subtilis</i>	26.33	42.76
9.	<i>Chaetomium globosum</i>	25.00	45.65
10.	Control	46.00	-
	S.E. ±	0.54	-
	C.D. (P=0.05)	1.59	-
	C.V. %	4.82	-

Pers. (6.33 mm) significantly reduced the growth of the pathogen which was statistically at par with *Pseudomonas fluorescens* Migula (6.67 mm) and *Aspergillus flavus* Link (7.67) which were statistically at par with *Gliocladium virens* Miller (8.33 mm) followed by *Trichoderma harzianum* Rifai (20.33 mm) and *Trichoderma longibrachyatum* Rifai (21.67 mm) and rest of the antagonists inhibited comparatively least growth of *C.capsici*.

Trichoderma viride Pers. (86.24%) showed maximum growth inhibition and appeared to be the most superior over all other antagonists tested which was at par with *Pseudomonas fluorescens* Migula (85.50 %) and *Aspergillus flavus* Link (83.33 %) which was statistically at par with *Gliocladium virens* Miller (81.89 %) followed by *Trichoderma harzianum* Rifai (55.80 %) and *Trichoderma longibrachyatum* Rifai (52.89 %) and rest of the antagonists showed comparatively least growth inhibition.

These results are in harmony with the findings of earlier workers viz., Hegde *et al.* (2002), Chirame and Padule (2005), Kaur *et al.* (2006) and Anand and Bhaskaran (2009).

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