

RESEARCH ARTICLE

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

■ R. SOMU<sup>1</sup>, N. THAMMAIAH<sup>2\*</sup>, G.S.K. SWAMY<sup>3</sup>, M.S. KULKARNI<sup>4</sup> AND V. DEVAPPA<sup>4</sup>

<sup>1</sup>Department of Plant Pathology, K.R.C. College of Horticulture, ARABHAVI (KARNATAKA) INDIA

<sup>2</sup>K.R.C. College of Horticulture, ARABHAVI (KARNATAKA) INDIA

<sup>3</sup>Department of Fruit Science, K.R.C. College of Horticulture, ARABHAVI (KARNATAKA) INDIA

<sup>4</sup>Department of Horticultural Plant Pathology, K.R.C. College of Horticulture, ARABHAVI (KARNATAKA) INDIA

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## \*Corresponding author:

Email: nthammaiah@gmail.com

## ABSTRACT

Among the six fungicides evaluated in *in vitro* against *Fusarium oxysporum* f.sp. *cubense*, carbendazim, carboxin, propiconazole and benomyl showed total inhibition of the fungal growth at the concentrations of 500, 1000 and 2000 ppm. However, difenconazole showed total inhibition of the fungal growth at 2000 ppm concentration.

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## INTRODUCTION

*Fusarium* wilt of banana (Panama disease) is caused by the soil-borne fungus, *Fusarium oxysporum* f. sp. *cubense* (E.F. Smith) Snyder and Hansen (Foc) (Stover, 1962). The fungus infects the roots of banana plants, colonising the vascular system of the rhizome and pseudostem, and inducing characteristic wilting symptoms before the plant eventually dies (Wardlaw, 1961; Stover, 1962). Evidence suggests that *Fusarium oxysporum* f. sp. *cubense* originated in Southeast Asia (Ploetz and Pegg, 1997) and from there was disseminated rapidly throughout the world with infected rhizomes (Stover, 1962). During the first half of the 20th century, *Fusarium* wilt caused major destruction in the Central American region, and since Panama was one of the most severely affected countries, the name Panama disease was adopted (Jeger *et al.*, 1996). Due to the destruction Foc race 1 caused in Central America, the international export trade was forced to convert from the susceptible Gros Michel to the resistant Cavendish cultivars (Ploetz *et al.*, 2003). Unfortunately, Cavendish cultivars are highly susceptible to race 4 of Foc (Ploetz and Pegg, 2000).

*Fusarium* wilt of banana is controlled effectively by applying disease-preventive practices such as planting in fields not infested with Foc using disease-free propagation material. Once infested with the pathogen, the only way of continuing banana production is by means of planting cultivars with resistance to the disease (Deacon, 1984; Ploetz and Pegg, 2000; Viljoen, 2002; Ploetz *et al.*, 2003). Cultivars with resistance to Foc have been identified, but these cultivars are not always acceptable to local markets (Viljoen, 2002). *In vitro* evaluation of fungicides provides preliminary information regarding its efficacy against a pathogen within a shortest period of time and therefore, serves as a guide for further field testing. Hence, the present investigation was undertaken to screen a range of fungicides, including new chemical formulations, for their ability to inhibit the mycelial growth of Foc *in vitro*.

## MATERIAL AND METHODS

An experiment was conducted during 2011 at K.R.C. College of Horticulture, Arabhavi to find out the effective fungicide against *Fusarium oxysporum* f. sp. *cubense* in

Sr. No.	Common name	Chemical name	Trade name
1.	Carbendazim	2-methoxycarbamoyl-benzimidazole	Bavistin 50% WP
2.	Benomyl	Methyl-(butylcarbomyl)-2- benzimidazol carbamate	Benomyl 50% WP
3.	Propiconazole	1-[2(2,4-dichlorophenyl)4-propyl-1,3 dioxolon-2-4-1) methyl H-1,2,4 triazole]	Tilt 25% EC
4.	Carboxin (Carboxin 37.5% + Thiram 37.5%)	5,6-dihydro-2-methyl-1,4-oxathi-in-3-carboxnilide	Vitavax 75% WP
5.	Azoxystrobin	Methyl(2E)-2-(2-{[6-(2-cyanophenoxy) pyrimyl]oxy }phenyl)-3 methoxyacrylate	Amistar 23% SC
6.	Difenconazole	cis,trans-3-chloro-4-[4-methyl-2-(1H-1,2,4-triazol-1-ylmethyl)-1,3-dioxolan-2-yl]phenyl 4-chlorophenyl ether	Score 25% EC
7.	Control		

*invitro*. The experiment was designed in Complete Randomized Design (CRD) with three replications and seven treatments.

Six systemic fungicides with three concentrations *viz.*, 500, 1000 and 2000 ppm were evaluated against Panama wilt pathogen, *Fusarium oxysporum* f. sp. *cubense* under laboratory conditions by following poisoned food technique (Flack, 1907). The details of the fungicides used are given in Table A.

The pathogen was grown on Potato dextrose agar medium prior to the setting of the experiment. The fungicide suspension was made by adding required quantity of fungicides to the melted Potato dextrose agar medium to obtain the desired concentration on the basis of active ingredient present in the chemical. Thirty ml of poisoned medium was poured in each sterilized Petriplate and suitable checks were maintained without addition of fungicides. Five mm of ten days old fungal disc was taken from the periphery of the culture and was placed in the centre of the poisoned medium aseptically and incubated at 26±1°C for seven days. Three replications were maintained for each treatment and the diameter of the colony was measured in 2 directions and the average was recorded after incubation for seven days. Per cent inhibition of the fungus was calculated by using the following formula :

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

where,

I = Per cent inhibition.

C = Growth of the pathogen in control plate.

T = Growth of the pathogen in dual culture plate.

## RESULTS AND DISCUSSION

*In vitro* evaluation of different chemicals against *Fusarium oxysporum* f. sp. *cubense* was done as described in “material and methods” using by following poisoned food technique. The fungicides were tested at 500, 1000 and 2000 ppm concentrations each and the observations on colony diameter and per cent inhibition of colony growth over control are presented in Table 1 and Plate 1.

Carbendazim, carboxin, propiconazole and benomyl completely inhibited the mycelial growth of the fungus (100 %) at 500 ppm followed by difenconazole (81.62 %). While azoxystrobin recorded the least inhibition of the fungus (41.46 %).

Soma *et al.* (2008) reported that carbendazim and carboxin were highly fungitoxic and showed 100 per cent inhibition in case of *Fusarium oxysporum* at 100 ppm and 200 ppm

Sr. No.	Fungicides	Colony diameter (mm)			Per cent inhibition of colony growth over control		
		Concentrations			Concentrations		
		500 ppm	1000 ppm	2000 ppm	500 ppm	1000 ppm	2000 ppm
1.	Carbendazim	0.00 (1.00)	0.00 (1.00)	0.00 (1.00)	100.00	100.00	100.00
2.	Carboxin	0.00 (1.00)	0.00 (1.00)	0.00 (1.00)	100.00	100.00	100.00
3.	Propiconazole	0.00 (1.00)	0.00 (1.00)	0.00 (1.00)	100.00	100.00	100.00
4.	Difenconazole	14.33 (3.91)	11.33 (3.50)	0.00 (1.00)	81.62	85.95	100.00
5.	Azoxystrobin	45.66 (6.83)	41.33 (6.50)	21.33 (4.72)	41.46	48.76	73.55
6.	Benomyl	0.00 (1.00)	0.00 (1.00)	0.00 (1.00)	100.00	100.00	100.00
7.	Control	78.00 (8.88)	80.66 (9.03)	80.66 (9.03)	–	–	–
S.E.±		0.04	0.05	0.03			
C.D. (P=0.01)		0.19	0.20	0.15			

Figures in the parenthesis are the square root transformed values



Plate 1 : In vitro evaluation of fungicides against *Fusarium oxysporum* f. sp. *cubense*

concentrations. Carbendazim and carboxin completely inhibited the growth of *Fusarium oxysporum* f.sp. *lentis* (Vinit *et al.*, 2010).

At 1000 ppm carbendazim, carboxin, propiconazole and benomyl showed highest inhibition of 100 per cent on mycelial growth of fungus followed by difenconazole (85.95 %) and azoxystrobin showed least effectiveness at 1000 ppm by inhibiting 48.76 % of fungus growth compared to 500 ppm.

The result obtained at 2000 ppm concentration of different fungicides clearly showed that carbendazim, carboxin, propiconazole and benomyl and also difenconazole were quite effective in inhibiting the mycelial growth of the fungus. Whereas, azoxystrobin showed effectiveness at 2000 ppm by inhibiting 73.55 per cent of mycelial growth compared to 500 and 1000 ppm.

In the present study, laboratory testing of fungicides by food poison technique revealed that all the fungicides showed effectiveness in decreasing the fungal growth at increased concentrations of the fungicides. The fungal growth was totally inhibited in all the three concentrations of carbendazim, carboxin, propiconazole and benomyl. Whereas, difenconazole inhibited mycelial growth completely only at 2000 ppm. Azoxystrobin showed moderate inhibitive effective at 2000 ppm compared to other chemicals tested. However, Nel *et al.* (2007) reported that prochloraz and propiconazole significantly inhibited the mycelial growth of *Fusarium oxysporum* f.sp. *cubense* at concentration of 1 and 5 mg ml<sup>-1</sup>.

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