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An *in vitro* Test for Evaluating the Efficacy of Mycoparasites on the Sclerotial Germination of Ergot (*Claviceps fusiformis* Lov.) of Pearl millet

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Ergot of pearl millet (*Pennisetum americanum* (L.) Leeke) caused by *Claviceps fusiformis* Loveless is causing severe losses in several high yielding varieties and hybrids of pearl millet in India. In addition to directly reducing grain yield, the admixture of sclerotia with grain poses a health hazard to consumers because of a toxic alkaloid. The methods of management of the disease include removal of sclerotia from the seed prior to planting by floatation in 10% salt solution, post harvest deep ploughing to bury sclerotia and adjustment of sowing time to avoid conditions conducive to infection. However, these methods do not give the desired level of disease control (Thakur *et al.*, 1985). Since the sclerotia constitute the primary source of inoculum, their destruction will reduce the perpetuation of the disease. Since the fungicides are not effective in this direction, antagonistic fungi like *Gliocladium virens* Miller and Foster, *Trichoderma viride* Pers and *T. harzianum* Rifai were tested to find out whether the germination of sclerotia can be delayed or inhibited by them. Kulkarni and Moniz (1974) reported that *Cerebella andropogonis* inhibited the sclerotial development of ergot of bajra. In this pathogen, Tripathi *et al.* (1981) reported that *Fusarium semibucinum* Fuckel and *Dactylium fusarioides majus* Wollen W., parasitized honeydew and sclerotial stages.

Mature sclerotia of uniform size (4.5 x 2 mm) were collected and washed with sterile water and 25 sclerotia were partially buried in sterilized sand in 9 cm Petri dish. The Petri dishes were kept at $25 \pm 2^{\circ}\text{C}$ under 12 h

darkness, alternated with light (Prakash *et al.*, 1981). The sand was kept moist by pouring sterile water at regular intervals. Two ml of the spore suspension ($10^5/\text{ml}$) of the antagonistic organisms i.e. *T. viride*, *T. harzianum* and *G. virens* was added to each Petri dish. Sterilized water was added to control plates. The treatments were replicated six times. The number of germinating sclerotia was recorded at weekly intervals for four months.

The earliest germination of sclerotia was observed after eight weeks. The control plates recorded 58.6 per cent germination (Table 1). All the three antagonistic organisms significantly reduced the germination of sclerotia and were on par with each other. The antagonistic organisms not only reduced the germination per cent but also delayed the germination of sclerotia. In the control plates, the sclerotia germinated after 9 weeks whereas none of the sclerotia in antagonist - treated plates germinated at that time. In antagonist treated plates germination took place after 12 weeks. Even after 15 weeks, the germination per cent in antagonist-treated plates was 3.3, 4.0 and 3.3 for *T. viride*, *T. harzianum* and *G. virens* respectively.

Tripathi *et al.* (1981) reported that size of sclerotia of *C. fusiformis* was reduced considerably and germinability was inhibited by 50% when they were parasitized by *F. sambucinum* and *Dactylium fusarioides*. Muller *et al.* (1985) reported that treatment of sclerotia of *Sclerotinia sclerotiorum* with *G. virens* inhibited carpogenic germination of the pathogen.

Table 1. Effect of antagonists on the germination of ergot sclerotia

Treatment	No. of sclerotia germinated (out of 150) weeks after			Per cent germination
	9	12	15	
<i>T. viride</i>	0.0	1.0	5.0	3.3 ^a
<i>T. harzianum</i>	0.0	1.0	6.0	4.0 ^a
<i>G. virens</i>	0.0	1.0	5.0	3.3 ^a
Control	66.0	82.0	88.0	58.6 ^b

In a column, means followed by similar letters are not different statistically ($P = 0.05$) by DMRT

In Maryland, USA, 100 and 1000 macro conidia of *Sporidesmium sclerotivorum* Uecker/g soil introduced in the plough layer reduced the number of sclerotia of *S.sclerotiorum* (Lib.) de Bary by 75 and 94 per cent respectively (Ayers and Adams, 1979). The results clearly indicate the potential of antagonistic fungi in reducing the inoculum load of the pathogen.

Key words : Ergot, mycoparasites, biological control

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