RHIZOCTONIA DISEASES OF LEGUME CROPS AS AFFECTED BY *TRICHODERMA VIRIDE*

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Rhizoctonia disease of legume plants was found to be very prevalent at Ujjain. The disease was most damaging in pre-emergent and post-emergent stages. In the present study Rhizoctonia diseases of two crops, viz., *Phaseolus lunatus* and *Pisum sativum* were taken into consideration for biological control by the use of well-known antagonist *Trichoderma viride*. The attempt was to reduce the entry of *Rhizoctonia solani* to the host roots at the time of infection by previous inoculation of *T. viride* on either seeds or seedling roots. Further, in other series of experiments attempt was also made to infest *T. viride* in soil before inoculating the soil with *R. solani*. The disease was reduced effectively by establishing *T. viride* on seedling roots by different methods. Inoculation of the antagonist on seeds did not give very promising results although the disease was inhibited to certain extent. Considerable reduction in disease was also noted when *Trichoderma* was infested in soil together with amendment prior to the pathogen. The results indicate that Rhizoctonia infection was inhibited in seedling stage by presence of *Trichoderma* in the infection court of the root at the time of infection. *Trichoderma* was detected both in rhizosphere and rhizoplane of the host even after 30–35 days of its inoculation. Further it can also be concluded that the antagonist cannot inhibit disease development once the infection has taken place.

INTRODUCTION

Significant role of *Trichoderma viride*, a well-known soil antagonist, was known from 1936 (Weindling and Fawcett) when Rhizoctonia 'damping off' of citrus seedlings was found to be reduced by increasing the frequency of the antagonist in the root region. Later on, Bliss (1951) has also pointed out that control of *Armillaria mellea* of citrus seedling was solely not due to carbon disulphide fumigation but also because of the destruction of the pathogen by *T. viride* which was also found to be tolerant to fumigation. *Trichoderma* has also been found to be the dominant early recolonizer of soil after fumigation by various chemicals (Evans 1955; Saksena 1960; Moubasher 1963; Warcup 1952) which are generally used to control root-diseases. Further, *Trichoderma* was also reported in the rhizosphere and rhizoplane of moderately resistant varieties (Subba Rao and Bailey 1961; Srivastava and Saksena 1968) which also indicates its significance in disease control. Besides *Trichoderma*, other soil antagonists were also found to be effective in root disease control (Chang and Kommedahl 1968).

Considering the antagonistic activity of *Trichoderma viride* in root-zone and also the reduction of pathogenic activity of *Rhizoctonia solani* in soil by various

amendments (Davey and Papavizas 1960), the present experiment is aimed to inhibit disease from two sides, firstly, suppressing the activity of *R. solani* in soil by various amendments and secondly, by protecting the host from infection by coating the vulnerable host tissue by the antagonist.

In the present study, Rhizoctonia disease of two legume crops, *Phaseolus lunatus* (Lima bean, 'Lobia') and *Pisum sativum* (Pea) were selected to attempt biological control by the use of *Trichoderma viride*. Emphasis is on the establishment of the antagonist on young seedling roots so as to give a chance to the antagonist to invade the root region earlier than the pathogen and then to check the disease induction. This method of control is preferred here because the disease was most damaging in pre-emergent and post-emergent stages and anyhow if the infection is controlled at this stage, the plants remain healthy and the crop is saved from heavy losses.

Materials and Methods

Two pathogenic isolates of *Rhizoctonia solani*, R_1 and R_2, infecting locally grown varieties of *P. lunatus* and *P. sativum* seedlings respectively and two isolates of *Trichoderma viride* T_1 and T_2 were selected for experimental purpose. The pathogen, *R. solani,* was grown in potato dextrose broth for 8–10 days and the mycelial mat was crushed with distilled water in blender. The suspension was first mixed with sand and then with soil at 1 per cent (w/w) of fresh weight of mycelium. This served as the inoculum. Spore suspension of *T. viride* (500000 spores/ml) was added to the soil at 10 ml/100 g of soil or amendments.

Experimental pots were inoculated by amendments either with or without antagonist 28 days before sowing of seeds or planting seedlings. The amendments were added at 10 per cent (w/w) dry weight of the soil. All the experiments were done in triplicates, pots were watered intermittently and controls were also run side by side. The experiments were done in three sets:

I. *Inoculation of* T. viride *on host*

(A) *Seedling roots*—The antagonist was inoculated on roots by two methods:

A_1—Surface-sterilized seeds were allowed to germinate in sterilized sawdust previously inoculated with *T. viride*. Ten to twelve-day-old such seedlings were transferred to the following types of soil.

Type I—Soil infested with *R. solani* only.
II—Soil infested with *R. solani* and wheat straw.
III—Soil infested with *R. solani* and sawdust.

A_2—Roots of 10–12 day-old seedlings were dipped in spore suspension of *T. viride* and transferred to all the three types of soil mentioned above.

(B) *Seeds*—Surface-sterilized seeds were dipped in *T. viride* spore suspension and then sown in all the three types of soil.

II. *Inoculation of* T. viride *in soil*

Trichoderma viride was infested in soil by inoculating it to sterilized amendments (wheat straw and sawdust) which were allowed to grow under aseptic conditions for

two weeks. Such inoculated amendments were added to the soil prior to inoculation of pathogen, seeds were sown to such pots and the plants were sampled for infection rating after 30-35 days.

III. In a third set of experiments seedlings already infected with *R. solani* were treated with *T. viride* by dipping them in its spore suspension and transferring them to all the three different soils mentioned in the first set of experiments. Further, infected seedlings were also transferred to the soils in which *T. viride* was infested together with the amendments.

Sampling of the seedlings for rhizosphere and rhizoplane were done twice, i.e., after 21 and 35 days of antagonist inoculation in all sets of experiments.

Results and Conclusions

The results of the first and the second set of experiments are given in Tables I and II respectively. The results of the third set were not promising and hence not given here for brevity.

Table I

Effect of Trichoderma viride *on Rhizoctonia disease of* Phaseolus lunatus *and* Pisum sativum *as expressed by infection rating when antagonist was inoculated on host by different methods.*

Name of plant and antagonist	A_1 method			A_2 method			B method		
	Soil type I	Soil type II	Soil type III	Soil type I	Soil type II	Soil type III	Soil type I	Soil type II	Soil type III
1. *Phaseolus lunatus*									
(a) Control—No antagonist	62.4	53.0	50.9	72.4	53.0	50.9	73.7	60.3	61.0
(b) *Trichoderma viride* T_1	32.6	28.3	35.6	38.2	33.6	38.0	56.8	48.2	55.6
(c) *Trichoderma viride* T_2	38.9	29.4	42.8	42.6	42.8	40.2	58.0	49.6	52.0
2. *Pisum sativum*									
(a) Control—No antagonist	57.8	54.3	50.2	57.8	54.3	50.2	65.9	58.9	62.7
(b) *Trichoderma viride* T_1	42.3	39.4	42.4	44.8	34.2	38.0	52.1	50.3	52.5
(c) *Trichoderma viride* T_2	44.5	32.8	43.4	39.5	42.4	31.9	48.9	45.8	48.9

A perusal of Table I shows that in both the crops, *P. lunatus* and *P. sativum*, inoculation of the antagonist on roots of young seedlings inhibited disease to a greater extent than inoculating it on seeds. Although the inoculation of seeds reduced the pre-emergent killing to a greater extent, this method could not reduce the infection very effectively in post-emergent stage because by this time the antagonist might have dislodged or its action might have been reduced by other soil microorganisms, while inoculating antagonist on seedling roots had reduced the post-emergent infection effectively. Comparing the amendments, wheat straw was always found to be

TABLE II

Effect of Trichoderma viride *on Rhizoctonia disease of* P. lunatus *and* P. sativum *as expressed by percentage of infectio nrating when antagonist was infested in soil with amendments*

Name of plants and antagonist	Without amendments			Wheat straw			Sawdust		
	Infection rating	Rhizosphere	Rhizoplane	Infection rating	Rhizosphere	Rhizoplane	Infection rating	Rhizosphere	Rhizoplane
Phaseolus lunatus									
Control—No antagonist	62.4	—	—	55.6	—	—	56.7	—	—
T. viride T_1	61.4	++	—	38.6	+++	—	35.6	++	—
T. viride T_2	64.5	++	—	44.5	+++	+	36.8	++	—
Pisum sativum									
Control—No antagonist	57.8	—	—	54.8	—	—	50.4	—	—
T. viride T_1	56.23	++	—	34.8	++	+	32.8	++	—
T. viride T_2	56.8	++	—	41.2	+++	—	35.6	++	+

superior to sawdust whether present alone in soil or with the antagonist. The antagonist was found to be more effective together with amendments as compared to unamended soil. It was also detected both in rhizosphere and rhizoplane of all types of plants even after 35 days of its inoculation.

In the second set of experiments (Table II) the disease was reduced considerably when antagonist was inoculated in soil together with the amendments prior to the inoculation of pathogen. Here also wheat straw was found to be more effective than sawdust. *Trichoderma* was also reported here from rhizosphere and rhizoplane but not so frequently as in the first set of experiments.

In the third series of experiments the results were not encouraging because here the disease development could not be checked either by inoculating the antagonist on host-tissue or seeds. Inoculation of antagonist to the soil together with amendments was also ineffective. The results of these experiments indicate that if once the infection has taken place disease development cannot be checked.

The results of the present experiment are in accordance with Srivastava and Saksena (1968), Tiwari (1968), and Claudius (1968).

Considering the two isolates of *T. viride*, *T. viride* T_1 was found to be slightly more active in both the crops.

In conclusions derived from the above experiments *T. viride* may be considered strongly effective in reducing Rhizoctonia disease of *P. lunatus* and *P. sativum* when inoculated on either seeds or seedling roots. This antagonistic action may be because *T. viride* would have formed a sort of defence mantle around the vulnerable host tissue and the pathogen has to pass through this mantle before it can invade the host. This disease reduction was further enhanced when the inoculated seeds or seedlings

were transferred to amended soil because in such soils *Rhizoctonia* may also be suppressed by other soil micro-organisms. The reduction in disease by inoculating *T. viride* in soil together with amendments may be due to the suppression of *R. solani* by high frequency of the antagonist in soil. The third set of experiments indicates that the disease development cannot be checked after the infection has taken place. Further it can also be concluded that *T. viride* can inhibit or even check the entry of *R. solani* and thus protects the seedlings but it is not effective in checking disease development.

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