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PHYTOPATHOLOGICAL EFFECT AND DISEASE TRANSMISSION OF *FUSARIUM OXYSPORUM* AND *RHIZOCTONIA SOLANI*

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Abstract : Studies on phytopathological effects and disease transmission of *Fusarium oxysporum* and *Rhizoctonia solani* were carried out using seeds with natural field infection. Symptoms were observed on root, stem, leaves and pots of these plants. A close examination of infected parts under steriobinocular microscope revealed mycelium and abundant microsclerotia in the peripheral region of the lesions. Microsclerotia were also observed on root, stem surface and inner surface of splitted stem, and on pod surface of mature plants. Rarely these were recorded on root nodules as well. Sequential observations were made to study the effects of pathogen on seed germination, seedling survival, mortality, symptomatology, disease transmission and recovery of pathogen. Seed categorized as asymptomatic and symptomatic (weakly and heavily infected) were used separately for each experiment. Two seed samples ac. nos. 3527, 3529(*F.oxysporum)* and 3530, 3538(*R.solani)* naturally infected were used for the study.

Keywords: Fusarium oxysporum, RHIZOCTONIA SOLANI, lentil, DISEASE TRANSMISSION, PHYTOPATHOLOGICAL EFFECT

I. INTRODUCTION

INTRODUCTION

Lentil (Lens culinaris L.) is the second most important cool-season legume crop in India (Ram and Punia, 2018). It covers an area of 1.51 million ha with a production of 1.56 million tons and productivity of 1,032 kg ha−1 (Directorate of Economics and Statistics, 2020). In India, the pulse cultivation occupies nearly about 24.70 million hectare of land every year and the annual production touches over 14.50 million tones and productivity are 587 kg/ha.

Lentil is currently an important pulse crop grown widely throughout the Indian subcontinent, Middle East, Northern Africa, Southern Europe, North and South America, Australia and west Asia (Ford and Taylor, 2003; Erskine, 1997).

Lentil are among ancient plants known to be cultivated by man carbonized lentil found in Neolithic villages in the Middle East have been dated as being between 8 and 9 thousand years old. After initial cultivation of the crop in the Middle East. Lentil use began to spred around the Mediterranean.By 2200B.C lentil begain to appear in Egyptian tombs (Pooja, 2005).

Fusarium wilt of lentil, caused by *Fusarium oxysporum* schlecht, emendd. Synder and Hansan f. sp. lentis Vasudeva and srinivasan (fol) is a devastating disease in most countries where lentils grown. Fol has great variability (Allen D.J. et. al., 1998).

The pathogen persists in the soil as chlamydospores that can remain viable for several seasons (Erskine and Bayaa, 1996), and is capable of colonizing crop residue and roots of most crops grown in rotation with lentil.

It can enter the host through root tips, primarily in the area of elongation, a process which is aided by wounding (Bhalla et al., 1992).

Seeds of healthy and infected lentils from different plots were collected and both produced healthy plants. It is concluded that lentil wilt caused by *F. oxysporum f.sp. lentis* is not seedborne (Pundir et al.,1991).

Initial disease symptoms included yellowing of leaves, root and collar rot and stunting. As the disease progressed, some affected plants wilted and died. The causal organism was identified as *Rhizoctonia solani* (*Thanatephorus cucumeris*) based on morphological and cultural observations and pathogenicity test. This is thought to be the first report of *R. solani* causing root and collar rot of lentil in Italy (Tosi et al., 2002).

Surveys by Saskatchewan (Morrall et al. 1972; McKenzie and Morrall 1973) and southern Alberta (Swanson et al. 1984) indicated that root diseases of lentil were of minor importance.

MATERIALS AND METHODS

The study was carried out by applying following methods (i) Petriplate method (ii) Water agar seedling symptom test and (iii) Pot experiment. Data on seed germination, disease symptom, rotting, survival and mortality of seedling or plants were recorded.

Petriplate Method

Two replicates of 100 seeds per category per sample pretreated with aqueous solution of sodium hypochlorite with 0.2% chlorine were grown on moistened blotters. The seeds were incubated at 26 ± 2°C under 12h of alternating cycles of light and darkness for 7 days. Observations were made at 24h interval up to 8th day.

Water Agar Seedling Symptom Test

Fifty Seeds per category per sample treated with 2% available chlorine were sown in sterilized water agar medium in test tubes (1 seed/test tube) under aseptic conditions, incubated at 26 ± 2°C for 12h of alternating cycles of artificial light and darkness. Observations were taken daily up to 15 days.

Pot Experiment

Sample ac.nos.3527, 3529 for *Fusarium oxysporum* and ac. nos.3530, 3538 for *Rhizoctonia solani* was used for the experiment. Two replicates of 100 seeds per category per sample were sown in pots (10 seeds/pot), containing sterilized soil in the month of July. The seeds used were treated with 2% aqueous chlorine solution for 5 minutes. Data was recorded on seedling emergence, seedling survival, mortality and transmission of disease regularly at weekly intervals up to maturity of the plant (nearly 120 days).

For the isolation and recovery of pathogen, diseased and healthy looking seedlings were carefully uprooted washed in running water and cut into small pieces. After surface sterilization with 2% chlorine solution, different parts were cut longitudinally and one half was incubated on blotter while the other halves were cleared by boiling in aqueous solution of 10% KOH. The plant parts were washed thoroughly in tap water, stained with cotton blue and mounted in PVA. After harvesting (120 days) the seeds were collected from infected pods and sown in sterilized petriplates containing three well moistened blotters, for the incidence of pathogen. The root system was washed with water thoroughly and examined by naked eye as well as under stereo binocular microscope for any abnormality and disorder.

RESULTS AND DISCUSSION

The performance of *F.oxysporum* infected seed samples of lentil and the disease transmission from seed to seedling and plant in naturally field infected were studied by petriplate method, water agar seedling symptom test and pot experiments.

Sequential observations were made to study the effects of pathogen on seed germination, seedling survival, mortality, symptomatology, disease transmission and recovery of pathogen. Seed categorized as asymptomatic and symptomatic (weakly and heavily infected) were used separately for each experiment. Two seed samples ac. nos. 3527, 3529(*F.oxysporum)* and 3530, 3538(*R.solani)* naturally infected were used for the study.

I. *F. oxysporum*

Petriplate Method (Table- 1; Fig.-1A-D)

Germination started after 24 h of sowing and 85% and 89% (3527,3529) on 8th day in asymptomatic seeds, whereas in weakly, moderately, and heavily symptomatic seeds, it was 13,20; 9,17 and 3,14 percent in both samples respectively. The ungerminated asymptomatic as well as symptomatic seeds showed pure growth of *Fusarium oxysporum* and revealed sees rot.

The initial disease symptoms appeared as pale yellow discolouration on the hypocotyle region on 3rd-4th day in asymptomatic and 2nd to 3rd day in symptomatic seeds. These symptoms developed rapidly in the upper region as compared to the lower radicular region. Microconidia formation was observed on the hypocotyle region and cotyledonary leaves showed yellowing leading to their fall. In some seedlings, fungal growth was observed on seedcoat attached to the cotyledons for a long period, from where it spreads to the hypocotyle region, whole shoot turned yellow to brown, pulpy and showed wilting and finally these rotted on 5th to 7th day in asymptomatic seeds and 3-5th day in symptomatic seeds of both the seed samples. Similar symptoms were also observed on roots. The intensity of symptoms increased gradually and the number of infected seedlings was 4, 5% (asymptomatic seeds) and 3-9%; 12-13% (symptomatic seeds) on 8th day in the two samples (Fig. -7).

On 14th day, most of the infected seedlings wilted (3-9%; 10-12%) in symptomatic seeds but in asymptomatic seeds only 5, 7% seedlings showed wilting.

Water agar seedling symptom test (Table-2; Fig.-3A-E)

Germination started after 24h of sowing and it was maximum on 3rd day in asymptomatic seeds 80% and 90% in ac.no. 3527 and 3529 respectively. In symptomatic seeds on 2nd day, the germination was 14, 10, and 10% in sample ac.no. 3527 wheras, in ac.no. 3529 on 3rd day it was 18, 18 and 10% in weakly, moderatory and heavily infected seeds respectively.Diseased symptoms were similar to that observed in petriplate test. On 8th day, infected seedlings were 10 and 8% in the population from asymptomatic seeds and 10-14% in that of symptomatic seeds of both samples. The wilting occurred little early and the total loss was much in higher in seedling from symptomatic seeds (94-100%) than in those of asymptomatic ones (30, 16%) (Fig.-8).

Pot experiment (Fig.-5A-F&Fig.-11)

The growing of categorized seeds (healthy, weakly and heavily infected) in pots showed that seedling emergence was affected due to infection. Germination started after 5th day of sowing and it was 80%, 50%, 30% and 85%, 55%, 35% in asymptomatic, weakly and heavily infected seeds of two ac. nos. 3527, 3529 samples respectively. Survivability of seedling was also affected due to infection of pathogen. The seedling/plants obtained from

asymptomatic seeds were more vigorous showing low intensity of the symptom than weakly and heavily infected plants. Pre-and post-emergence mortality was 5%, 4% in asymptomatic seeds, 10%, 8% and 15%, 17% mortality in weakly and heavily infected seeds respectively (Fig.-11).

II. Rhizoctonia solani

Patriplate Method (Table-3; Fig.-2A-C)

Germination of seeds began after 24 h of incubation which increased rapidly for the next 24 h and was maximum 75 and 92 percent in asymptomatic seeds, whereas, germination was very poor 10-14, 8-10 and 2-6 percent in weakly, moderately and heavily symptomatic seeds (Seeds with microsclerotia) of sample ac. nos. 3530 and 3538 respectively on 3rd day of sowing. Ungerminated seeds were mostly covered with pure growth of *Rhizoctonia solani* rarely with bacterial ooze causing pre-emergence damping off and seed rot. Seed rot was low 26 and 4% in asymptomatic seeds and was very high 90-96 and 88-95% in symptomatic seeds. The initial disease symptoms appeared as brown streaks on root-shoot transition zone on 2nd or 4th day of sowing which progressed upward to hypocotyl and downwards to radicular region. In some seedlings when the seed coat with microsclerotia remained attached to the cotyledons for a long period, infection spread to cotyledons and turn to other parts of the seedlings forming necrotic brown areas on the cotyledons and turn to other parts of the seedlings forming necrotic brown areas on the cotyledons and pale to brown streaks on the hypocotyl region. The streaks coalesced and whole shoot turned brown, pulpy and rotted on the 3rd to 7th day in symptomatic seeds. Similar symptoms were also observed on roots. The intensity of symptoms and number of symptomatic seedlings increased gradually and was 26 and 5 in asymptomatic seed on 8th day, whereas, in symptomatic seeds 10, 8, 2 and 14, 10, 6 seedlings showed symptoms in weakly, moderately and heavily infected seeds of sample ac. nos. 3530 and 3538 respectively. Microsclerotial formation was observed on all the parts of infected seedlings from asymptomatic as well as symptomatic seeds. The seedling mortality was 10 and 8 in asymptomatic seeds and 2-6 and 6-14 % in different categories of symptomatic seeds respectively. Total pre- and post-germination losses were very high in symptomatic seeds e.g. 90-97 and 96-98 % as compared to 36 and 12 in asymptomatic seeds of the three samples ac. nos. 3530 and 3538 respectively. In control experiment, 98% germination was recorded (Fig.-9).

Water agar Seedling Symptom Test (Table-4, Fig.-4A-D)

Seed germination started after 24 h of incubation and increased for next 24 h. It was 94 and 92% in asymptomatic seeds and 4-18 and 6-26% in symptomatic seeds respectively on 14th day of sowing. Ungerminated seeds showed heavy growth of *R.solani*. The first symptoms appeared between 4th-7th days in asymptomatic seeds, whereas in symptomatic seeds it appeared during 2nd-5th day. Disease symptoms were similar to that observed in petriplate test. On 8th day, infected seedlings were 2-6% in asymptomatic seeds and 8, 4, 0 and 8, 8, 2 per cent in weakly, moderately and heavily infected seeds of sample ac. nos. 3530 and 3538 respectively. Total loss on 14th day in the two samples was 18, 8% in asymptomatic seeds. In symptomatic seeds it was 86-100% and 86-94% in weakly, moderately and heavily infected seeds of the three samples respectively. In control experiment, 97% germination was observed (Fig.-10).

Pot Experiment (Histogram Figs.-6 A-G&12)

Seed germination started on 5th day of sowing and was 91%, 85% in asymptomatic seeds and 54%, 41% and 35%, 25% in symptomatic category of sample ac. nos. 3530, 3538 respectively.

Mortality and total loss varied from 2 to 25%, 3 to 10% and 7 to 75%, 11 to 85% in asymptomatic and symptomatic (weakly and heavily) categories of both the samples respectively.

Seeds with and without microsclerotia of infected seed samples and uninfected seeds (Control) were used. On 4th day of sowing the germination was 98% in control, whereas in seeds with microsclerotia it ranged from 8-20 % in the two samples. The ungerminated seeds were covered with *R.solani*. All the seedlings obtained from symptomatic seeds developed symptoms as brown streaks and discoloured patches on root-shoot transition zone after 6th day of sowing. Subsequently symptoms appeared on cotyledons as pale yellow to brown or black, circular or oval, concentric spots. The spots enlarged, became irregular and necrotic causing shot holes. All such seedlings finally i.e. by 14th day collapsed in sample ac.no. 3530 whereas 4 and 6 seedlings out of those which developed symptoms survived in ac.nos. 3530 and 3538 respectively. Later on, in the 3rd week of sowing these also rotted.

In asymptomatic seeds of the sample ac.nos. 3530 and 3538, the maximum germination recorded was 52 and 88 % respectively on 8th day of sowing. In sample ac. no. 3530 out of 52 seedlings 14 had developed symptoms of *R.solani* on this day whereas there were no diseased seedlings in those of other one sample. Gradually more seedlings in ac. no. 3530 and some seedlings in other one sample acquired symptoms and the number of seedlings that developed symptoms became 16 and 20 by 60th day of sowing. Some of the seedlings which developed heavy symptoms rotted and collapsed. These numbers was 12 and 10 in ac. no. 3530 and 3538 respectively. The surviving infected plants flowered and fruited but the incidence of flowering and fruiting was rather poor. The lessions on 50 days old infected plants also developed microsclerotia. At maturity in harvested plants, sclerotia were observed on the inside of the longitudinally splitted stems and on roots. Leaves developed sclerotia only on dry necrotic areas. Rarely sclerotia were also observed on root nodules.

The pods harvested from the infected plants had brown patches and streaks on surface. The seeds from these pods carried (7-11 %) infection of *R.solani* which was expressed on incubation.

100% infection of *R.solani* was recorded from the infected seedlings and plant parts viz. leaf, stem, root and pod. In pod the infection was observed in pericarp, and seeds from these pods yields the fungus. The wholemount cleared preparation and thin sections of seedling and plant parts revealed presence of thick, septate, knotty, brown mycelium in epidermis, cortex, pith and vascular elements of stem.

Declaration

The author declare that they have no competing interests.

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REFERENCES

Allen, D.J. and J.M. Lenne, 1998. The pathology of food and pasture legumes. CAB International, Wallingford, United Kingdom, pp: 750.

Bhalla MK, Nozzolillo C, Schneider E (1992). Observation on the responses of lentil root cells to hypha of *Fusarium oxysporum*. *J. Phytopathol* **135**: 335-341.

Directorate of Economics Statistics (2020). Agricultural Statistics at a Glance 2019 Government of India Ministry of Agriculture and Farmers Welfare Department of Agriculture, Cooperation and Farmers Welfare Directorate of Economics and Statistics. Available online at: http://eands.dacnet.nic.in

Erskine, W.; Bayaa, B. and Saxena, M.C. (1996). Registration of ILL 5588. Lentil germplasm resistant to vascular wilt and ascochyta blight. Crop Sciense. **36(4)**: 1080, ICARDA, Aleppo, Syria.

Erskine W (1997). Lessons for breeders from landraces of lentil. Euphytica **93:** 107–112.

Ford RR, Taylor PWJ (2003). Construction of an intraspecific linkage map of lentil (*Lens culinaris ssp. Culinaris*). Theor. Appl.Genet. **107**: 910-916.

Morrall, R. A. A., McKenzie, D. L., Duczek, L. J. and Verma, P. R. 1972. A qualitative survey of diseases of some specialty crops in Saskatchewan in 1970 and 1971: sunflower, safflower, buckwheat, lentil, mustards and field pea. Can. Plant Dis. Surv. **52:** 143–148.

McKenzie, D. L. and Morrall, R. A. A. 1973. Diseases of three specialty legume crops in Saskatchewan in 1972: field pea, lentil, and fababean. Can. Plant Dis. Surv. **53**: 187–190.

Pooja, 2005. Legumes. Economic botany. Discovery Publishing House New Delhi. 271-272 pp.

Pundir, C.S.; Om Singh and Verma, H.C. 1991. Effects of Fusarium wilt on free amino acids in the progency of heating and infected lentil. *Indian Phytopathology.* **43(4)**: 580-582.

Ram, B., and Punia, S. S. (2018). Effect of seed priming and foliar urea spray on yield and economics in lentil (Lens culinaris) under rainfed condition. Int. J. Agric. Sci. 10, 5801–5803. Available online at: http://www.bioinfopublication.org/jouarchive.php?opt=&jouid=BPJ0000217 (accessed March 30, 2021).

Swanson, T. A., Howard, R. J., Flores, G. H. A. and Sumar, S. P. 1984. Incidence of root rot in pulse crops in southern Alberta, 1978–1983. Can. Plant Dis. Surv. **64**: 39–41.

Singh Anita,2022. Bio-Efficacy of Trichoderma Species Against Lentil Wilt Pathogen. International Journal of Creative Research Thoughts (IJCRT) .**10:** b740-b743.

Toshi L, Natalimi G and Cappelli C, 2002. First report of *Rhizoctonia solani* on lentil in Italy. Annalidella-Facolta-di- Agraria, - Universita – degli-studi-di perugia. **54**:201-203.

TABLE: 1 PHYTOPATHOLOGICAL EFFECTS OF NATURAL INFECTION OF FUSARIUM OXYSPORUM DURING GERMINATION DETERMINED IN SBM (100 SEEDS/CATAEGORY/SAMPLE)

AMPLE	ASYMPTOMATIC %								SYMPTOMATIC%																							
									Weakly								Moderately								Heavily							
3527																																
Days	2	3	4	5	6	7	8	14	2	3	4	5	6	7	8	14	2	3	4	5	6	7	8	14	2	3	4	5	6	7	8	14
Germination	45	64	81	85	85	85	85	85	8	10	13	13	13	13	13	13	9	9	9	9	9	9	9	9	3	3	3	3	3	3	3	3
Ung. without pathogen	55	29	9	5	5	5	5	5	92	16	13	13	13	13	13	13	91	9	9	9	9	9	9	9	97	4	4	4	4	4	4	4
Ung. with pathogen	0	7	10	10	10	10	10	10	0	74	74	74	74	74	74	74	0	82	82	82	82	82	82	82	0	93	93	93	93	93	93	93
Seedling symptoms	0	2	4	4	4	2	2	0	0	3	5	5	5	6	5	0	0	4	4	6	7	7	7	0	2	2	1	1	1	1	1	0
Seedling mortality	0	0	0	1	1	3	3	7	0	0	3	3	3	3	4	9	0	0	0	2	2	2	2	9	0	0	1	1	2	2	2	3
Incidence of pathogen	0	9	14	15	15	15	15	17	0	77	82	82	82	83	83	83	0	86	86	90	91	91	91	91	95	95	95	95	96	96	96	96
Normal Seedlings	45	62	77	80	80	80	80	78	8	7	5	5	5	4	4	4	9	5	5	1	0	0	0	0	1	1	1	1	0	0	0	0
3529																																
Germination	55	68	81	89	89	89	89	89	12	19	20	20	20	20	20	20	14	17	17	17	17	17	17	17	14	14	14	14	14	14	14	14
Ung. without pathogen	45	23	10	2	2			2	88	8	7	7	7	7	7	7	86	4	4	3	3	3	3	3	86	3	2	2	2	2	2	2
Ung. with pathogen	0	9	9	9	9	9	9	9	0	73	73	73	73	73	73	73	0	79	79	80	80	80	80	80	0	83	84	84	84	84	84	84
Seedling symptoms	0	0	1	1	3	3	3	0	0	2	5	9	9	8	8	3	0	9	6	8	7	6	6	3	4	4	4	5	5	7	7	2
Seedling mortality	0	0	0	0	0	1	1	5	0	0	0	0	1	2	4	10	0	0	3	4	5	7	7	12	0	2	2	2	3	4	5	12
Incidence of pathogen	0	9	10	10	12	13	13	14	0	75	78	82	83	83	85	86	0	88	88	92	92	93	93	95	4	89	90	91	92	95	96	98
Normal Seedlings	55	68	80	88	86	85	85	84	12	17	15	11	83	10	8	7	14	8	8	5	5	4	4	2	86	8	8	7	6	3	2	0

TABLE: 2 PHYTOPATHOLOGICAL EFFECTS OF NATURAL INFECTION OF FUSARIUM OXYSPORUM DURING GERMINATIODETERMINI IN WATER AGAR SEEDLING SYMPTOM TEST (100 SEEDS/CATAEGORY/SAMPLE)

SAMPLE	ASYMPTOMATIC %								SYMPTOMATIC%																							
									Weakly								Moderately								Heavily							
3527																																
Days	2	3	4	5	6	7	8	14	2	3	4	5	6	7	8	14	2	3	4	5	6	7	8	14	2	3	4	5	6	7	8	14
Germination	62	80	80	80	80	80	80	80	14	16	16	16	16	16	16	16	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10	10
Ung. without pathogen	38	4	4	4	4	4	4	4	86	24	24	24	24	24	24	24	90	20	20	20	20	20	20	20	90	24	24	24	24	24	24	24
Ung. with pathogen	0	16	16	16	16	16	16	16	0	60	60	60	60	60	60	60	0	70	70	70	70	70	70	70	0	66	66	66	66	66	66	66
Seedling symptoms	0	6	6	4	4	4	6	4	0	4	6	6	4	4	4	6	0	6	4	4	4	4	6	2	0	4	4	4	4	4	4	0
Seedling mortality	0	0	0	2	4	4	4	10	0	0	2	4	6	6	6	10	0	0	2	2	4	4	4	8	0	0	2	2	4	6	6	10
Incidence of pathogen	0	22	22	22	24	24	26	30	0	64	68	70	70	70	74	76	0	76	76	76	78	78	80	80	0	70	72	72	74	76	76	76
Normal Seedlings	62	74	74	74	72	72	70	66	14	12	8	6	6	6	6	0	10	4	4	4	2	2	0	0	10	6	4	4	2	0	0	0
3529																																
Germination	84	90	90	90	90	90	90	90	14	18	18	18	18	18	18	18	18	18	18	18	18	18	18	18	8	10	10	10	10	10	10	10
Ung. without pathogen	16	4	4	4	4	4	4	4	86	20	20	20	20	20	20	20	82	12	12	12	12	12	12	12	92	12	12	12	12	12	12	12
Ung. with pathogen	0	6	6	6	6	6	6	6	0	62	62	62	62	62	62	62	0	70	70	70	70	70	70	70	0	78	78	78	78	78	78	78
Seedling symptoms	0	0	0	0	0	4	6	2	0	4	10	10	8	8	6	6	0	6	6	8	6	6	6	6	0	4	4	2	2	4	4	0
Seedling mortality	0	0	0	0	0	0	2	6	0	0	2	4	6	6	8	12	0	0	2	2	4	6	8	12	0	0	2	4	4	4	6	10
Incidence of pathogen	0	6	6	6	6	10	14	14	0	0	76	76	76	76	76	80	0	76	78	80	80	82	84	88	0	82	84	84	84	86	88	88
Normal Seedlings	84	90	90	90	90	86	82	82	14	14	6	4	4	4	4	0	18	12	10	8	8	6	4	0	8	6	4	4	4	2	0	0

TABLE: 3 PHYTOPATHOLOGICAL EFFECTS OF NATURAL INFECTION OF *RHIZOCTONIA SOLANI* DURING GERMINATION DETERMINED IN SBM (100 SEEDS/CATAEGORY/SAMPLE)

SAMPLE	ASYMPTOMATIC %								SYMPTOMATIC%																							
									Weakly								Moderately								Heavily							
3530																																
Days	2	3	4	5	6	7	8	14	2	3	4	5	6	7	8	14	2	3	4	5	6	7	8	14	2	3	4	5	6	7	8	14
Germination	58	75	75	75	75	75	75	75	6	12	#	12	12	12	10	10	8	8	8	8	8	8	8	8	2	2	2	2	2	2	2	2
Ung. without pathogen	30	15	15	15	15	15	15	15	10	7	7	7	7	5	5	5	10	10	7	7	7	7	7	7	10	7	7	7	7	7	7	7
Ung. with pathogen	7	10	10	10	10	10	10	10	80	86	#	86	86	85	85	85	82	82	83	83	83	83	83	83	88	91	91	91	91	91	91	91
Seedling symptoms	1	3	12	14	16	18	21	15	0	4	2	2	2	2	4	4	2	2	4	5	5	5	5	2	1	1	1	1	1	1	0	0
Seedling mortality	0	0	0	2	2	4	4	10	0	0	1	1	1	1	2	2	0	0	3	3	3	3	3	6	0	2	2	2	3	3	3	3
Incidence of pathogen	10	15	25	28	30	34	36	36	80	90	90	90	90	90	93	93	82	82	89	90	90	90	90	90	87	97	97	97	97	97	97	97
Normal Seedlings	56	70	60	56	54	52	46	46	7	4	4	4	4	4	1	1	6	6	2	2	2	2	2	1	2	0	0	0	0	0	0	0
3538																																
Germination	70	92	92	92	92	92	92	92	14	14	14	14	14	14	14	14	10	10	10	10	10	10	10	10	6	6	6	6	6	6	6	6
Ung. without pathogen	30	4	4	4	4	4	4	4	6	6	3	3	3	3	3	3	4	2	2	2	2	2	2	2	9	4	4	4	4	4	4	4
Ung. with pathogen	0	4	4	4	4	4	4	4	80	80	80	83	83	83	83	83	86	88	88	88	88	88	88	88	85	90	90	90	90	90	90	90
Seedling symptoms	0	4	7	7	7	8	6	0	2	12	12	8	8	8	6	0	0	5	4	4	4	4	5	0	2	4	4	5	6	5	4	0
Seedling mortality	0	0	0	0	0	0	2	8	0	2	2	6	6	6	8	14	0	0	3	5	5	5	5	10	0	0	0	0	0	1	2	6
Incidence of pathogen	70	8	11	11	11	12	12	12	82	94	94	97	97	97	97	97	0	93	95	97	97	97	98	98	87	94	94	95	96	96	96	96
Normal Seedlings	81	88	85	85	85	84	84	84	12	0	0	0	0	0	0	0	10	5	3	1	1	1	0	0	4	2	2	1	0	0	0	0

TABLE: 4 PHYTOPATHOLOGICAL EFFECTS OF NATURAL INFECTION OF *RHIZOCTONIA SOLANI* DURING GERMINATION DETERMINED IN WATER AGAR SEEDLING SYMOTOM TEST (50 SEEDS CATAEGORY/ SAMPLE)

SAMPLE	ASYMPTOMATIC %								SYMPTOMATIC%																							
									Weakly								Moderately								Heavily							
3530																																
Days	2	3	4	5	6	7	8	14	2	3	4	5	6	7	8	14	2	3	4	5	6	7	8	14	2	3	4	5	6	7	8	14
Germination	80	94	94	94	94	94	94	94	14	18	18	18	18	18	18	18	10	12	12	12	12	12	12	12	4	4	4	4	4	4	4	4
Ung. without pathogen	20	0	0	0	0	0	0	0	86	6	6	6	6	6	6	6	90	2	2	2	2	2	2	2	96	0	0	0	0	0	0	0
Ung. with pathogen	0	6	6	6	6	6	6	6	0	70	70	70	70	70	70	70	0	86	86	86	86	86	86	86	0	96	96	96	96	96	96	96
Seedling symptoms	0	0	0	0	0	2	2	0	0	0	4	4	2	2	8	2	0	0	0	6	4	6	4	2	0	4	4	0	0	0	0	0
Seedling mortality	0	0	0	0	0	0	0	2	0	0	0	0	4	4	6	14	0	0	0	0	4	4	8	10	0	0	0	4	4	4	4	4
Incidence of pathogen	0	6	6	6	6	8	8	8	0	70	74	74	76	76	84	86	0	86	86	92	94	96	98	98	0	##	##	##	##	##	##	100
Normal Seedlings	80	94	94	94	94	92	92	9	24	24	20	20	18	18	10	8	10	12	12	6	4	2	0	0	96	0	0	0	0	0	0	0
3538																																
Germination	80	92	92	92	92	92	92	92	26	26	26	26	26	26	26	26	10	12	12	12	12	12	12	12	6	6	6	6	6	6	6	6
Ung. without pathogen	20	2	2	2	2	2	2	2	74	4	4	4	4	4	4	4	90	6	6	6	6	6	6	6	94	6	6	6	6	6	6	6
Ung. with pathogen	0	6	6	6	6	6	6	6	0	70	70	70	70	70	70	70	0	72	72	72	72	72	72	72	0	88	88	88	88	88	88	88
Seedling symptoms	0	0	0	0	0	2	6	0	0	0	4	4	2	2	8	2	0	0	8	6	6	10	8	4	0	2	2	2	2	2	2	0
Seedling mortality	0	0	0	0	0	0	0	2	0	0	0	0	4	4	6	14	0	0	8	6	6	10	6	10	0	0	0	4	4	4	4	6
Incidence of pathogen	0	8	8	8	8	8	8	8	0	70	74	74	76	76	84	86	0	78	86	86	86	92	92	92	0	88	88	94	94	94	94	94
Normal Seedlings	80	82	82	82	82	82	82	80	26	26	20	20	18	18	10	8	10	12	6	6	6	0	0	0	6	4	4	0	0	0	0	0

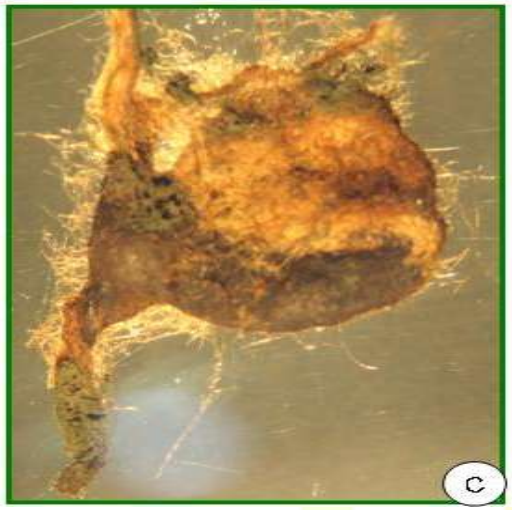

Fig.2

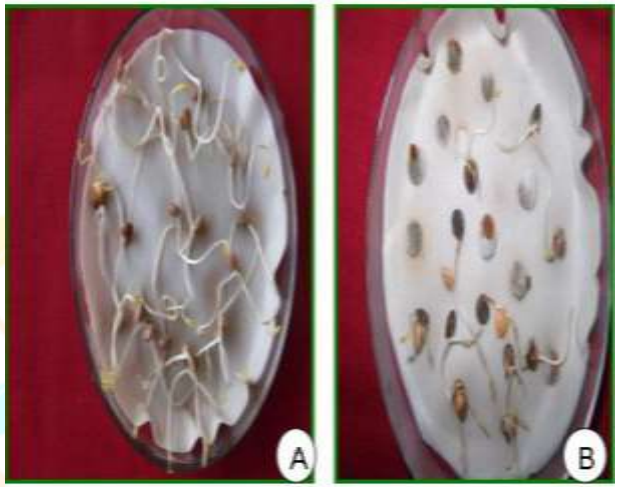

Fig.1

Fig-3

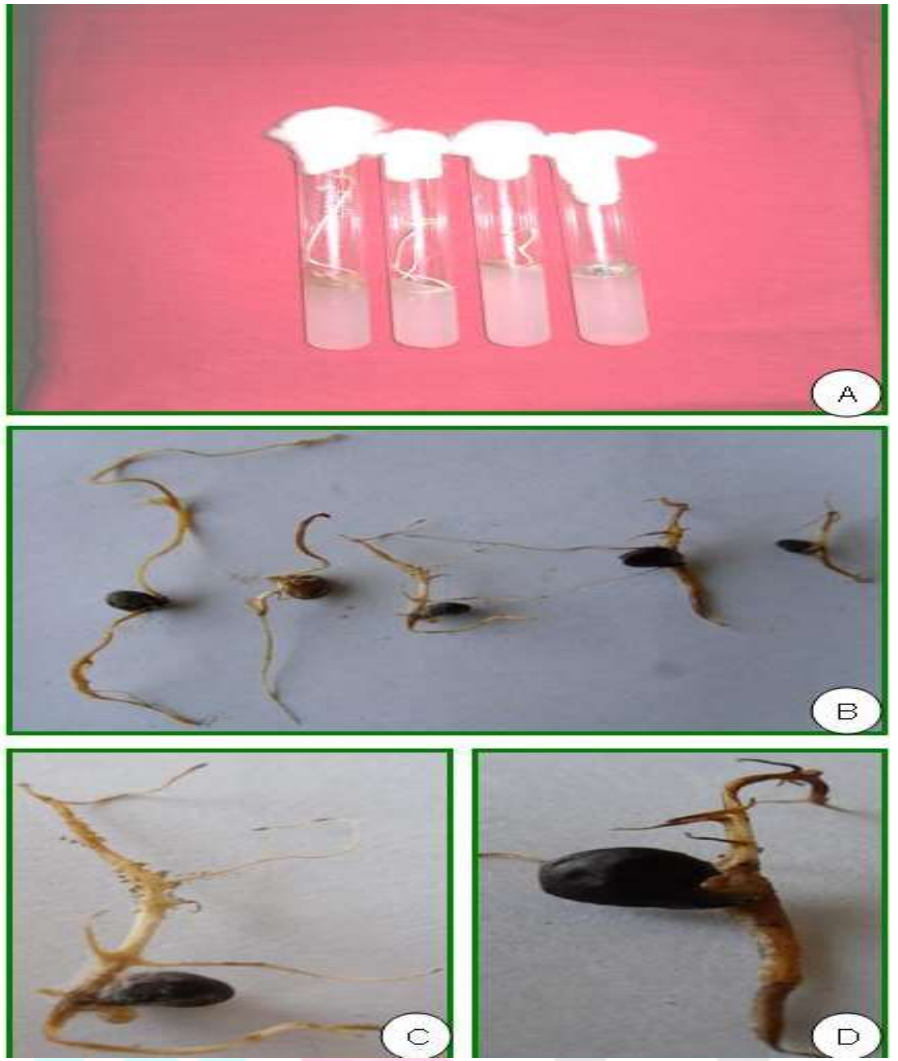

Fig-4

Fig-5

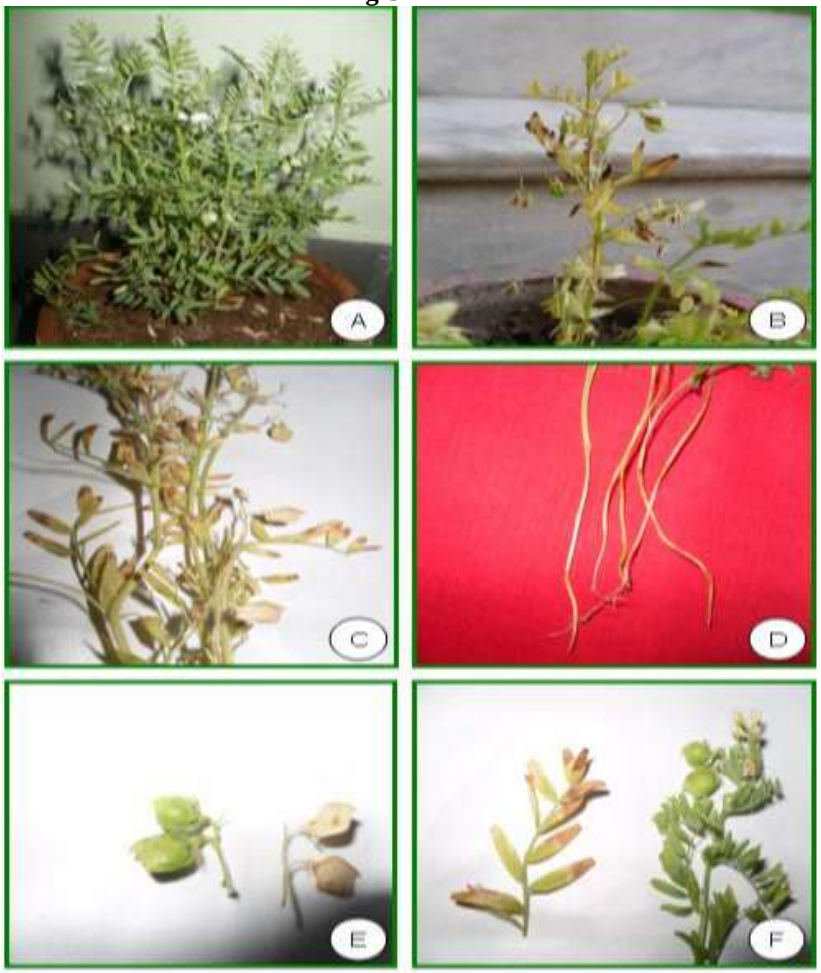

Fig-6