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Management of collar rot of chickpea through host plant resistance and fungicides

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Abstract
Chickpea is one of the vital source of protein required for humans it has various health benefits. Two hundred and six entries were screened under field conditions and promising entries under greenhouse conditions (Artificial inoculation) against collar rot. And seven fungicides were used as seed dressing to manage the collar rot disease of chickpea. Among the Kanpur *desi* entries 73 entries were found resistant, 25 entries moderately resistant, 10 moderately susceptible and five (BGD 1073, RKG 201-95, NDG 11-5 and JG 32 and GL 29389) as susceptible and none was highly susceptible and in *kabuli* type 42 entries were resistant, 11 were moderately resistant, five were moderately susceptible (IPCK 06-143, GNG 2104 (AVT 2), JGK-1, CSJK 27 and SKUA-C-23311) and three were susceptible (HK 09-219, JGK 18 and CSJK 70). Twenty one entries from ICRISAT were found resistant, eight entries were moderately resistant and one each was moderately susceptible, susceptible and highly susceptible. Amongst the fungicides used for seed treatment Vitavax power was found to be most effective with 9% infection of collar rot, next good was Hexaconazole, with 38% infection.

Keywords: Chickpea, genotypes, screening, artificial inoculation, field, fungicides

Introduction

Chickpea (*Cicer arietinum* L.) is a legume also known as *garbanzo beans*, are one of the oldest cultivated crop for consumption in the world, is a vital source of protein augmented human food and animal feed, mainly for the low-income population of Southeast Asia (Suzuki & Konno, 1982). It offers a range of health benefits, Chickpeas help to increase digestion, keep blood sugar levels stable, increase protection against disease and more. Chickpeas provides protein, vitamins and minerals, hence included in many healing diets. Being a sub-tropical and drought resistant crop, it grows well in cooler and dry climates. Among different factors causative towards its low production, natural constraints, chiefly diseases are the most significant. Due to these ailments, there is a very low yield in India as compared to potential yield of commercial chickpea cultivars (Ilyas *et al.*, 2007) [6]. Collar rot caused by *Sclerotium rolfsii* Sacc. is one of the fungal disease affecting this crop and is reported almost all over the world wherever chickpea is grown (Nene *et al.*, 1984) [11].

Chickpea is not only good for human health but also for soil health. It meets 80% of its nitrogen (N) requirement from symbiotic rhizobial interactions, which enables the crop to fix up to 140kg N ha^{-1} from atmosphere (Saraf *et al.*, 1998) [16]. It leaves substantial amount of residual nitrogen behind for subsequent crops and adds much needed organic matter to maintain and improve soil health, long-term fertility and sustainability of the ecosystems and is a boon to the resource-poor marginal farmers in the tropics.

This soil-borne pathogen causes rot of collar region on a wide range of plant species belonging to families Compositae and Leguminosae whereas members of Graminae are less susceptible to this disease (Mahen *et al.*, 1995) [9]. The most common hosts are legumes, crucifers and cucurbits. This disease according to Gurha and Dubey (1982) [4] is a serious threat, which under conducive conditions causes 55-95% mortality of the crop at seedling stage. The genetic resistance and seed treatment with fungicides is regarded, as the cost-effective disease management practice for such a devastating soil-borne pathogen.

Therefore, the present study was carried to screen the chickpea entries against *S. rolfsii* for the identification of resistant sources in field as well as in green house conditions, and protection of crop from the collar rot in seedling stage by treating chickpea seeds with fungicides.

Materials and Methods

1. Field screening in sick plot

Kabuli and Desi, chickpea entries provided by IIPR, Kanpur from ICRISAT Patancheru, Hyderabad, 206 entries were screened under field conditions against collar rot at Zonal agricultural research station (ZARS), University of agricultural sciences, G.K.V.K., Bangalore, during *rabi* season 2013. The field experimentations were laid out in a randomized block design with two replications. Every line was sown in five-meter row length. Subsequently after every five test entries, one row of susceptible check L-550 was sown. Observations on per cent collar rot incidence were documented at 10 days interval.

Per cent disease incidence was calculated by using following formula

$$\text{Per cent disease incidence} = \frac{\text{Number of plants infected}}{\text{Total number of plants}} \times 100$$

Based on PDI, the entries were categorized into different disease reactions using AICRP Chickpea scale.

Reaction	Collar rot incidence (%)
Resistant	0 – 10
Moderately resistant	11 – 20
Moderately susceptible	21 – 30
Susceptible	31-50
Highly susceptible	51-100

2. Screening in pots

Sterilized soil, sand and FYM were mixed in 1:1:0.5 proportion (w/w basis) and filled in sterile earthen pots. Four per cent mass culture of *S. rolfsii* cultured on sorghum seeds was added to soil in pots and mixed properly. For confirmation of promising entries which showed resistant reaction in field conditions, eight entries were selected *viz*., Vishal, BG-256, HIR-55, BBG-1, HIR-60, BBG-2, KAK-2, and HIR-70 were sown in the pots along with Annigeri-1 as susceptible check.

3. Evaluation of fungicides in pots

Healthy viable seeds of chickpea variety Annigeri-1 were treated with different fungicides at recommended concentrations.

The slurry/dry forms of fungicide *viz.* Thiram + carboxin (0.2%), chlorothalonil (0.2%), Carbendazim (0.1%), Hexaconazole, (0.1%) propiconazole (0.1%), difenconazole (0.1%) and thiophanate methyl (0.2%) were used for seed dressing (Table 1) and left for twelve hours. After 12 h the seeds were sown in the pots containing soil to which 4% of *S. rolfsii* inoculum (sclerotia) was added. For check treatment, seeds were moistened with the equal quantity of water. The moisture content in the soil was maintained at field capacity by adding required amount of water daily and the observations on germination and disease incidence were recorded at regular intervals.

Table 1: List of fungicides used for seed treatment

Sl. No.	Common name	Chemical name	Trade name
1	Carbendazim	Methyl -2-benzimidazole carbomate	Bavistin 50% WP
2	Hexaconazole	(RS)-2-(2,4-dichlorophenyl)-1-(1H-1, 2, 4-triazol-1yl) hexane -2-01	Contaf 5 EC
3	Thiophanate methyl	1,2-bis(3-methoxy carbonyl -2-thiouredo) benzene	Topsin M 70 WP
4	Difenconazole	1-2-(2- chloro-4-(4-chlorophenoxy) phenyl1)-4-methyl-1, 3-dioxolan-2 ylmethyl)-1H-1, 2, 4-triazole	Score 25 EC
5	Propiconazole	1-2-(2- chloro-4-(4-dichlorophenoxy))-4-propyl -1, 3-dioxolan-2 yl methyl)- 1H-1, 2, 4-triazole	Tilt 25 EC
6	Chlorothalonil	2,4,5,6-tetrachloroisophthalonitrile	Kavach
7	Carboxin 37.5 + Thiram 37.5	5,6 dihydro 2 methyl 1, 4 oxathin 3 carboxaniline + Tetramethylthiuram disulphide	Vitavax power

Results and discussion

1. Field screening

Results are presented in Table 2 to 4 (Fig 1). One hundred and seventy four entries (113 desi and 61 *Kabuli*) from IIPR Kanpur and 32 entries from ICRISAT were screened for collar rot between the 113 desi entries 73 entries *viz.*,, NDG 11-24, PG 0100, H 09-53, CSJ 692, GNG 2127, H 09-65, RKG 207-61, RKG 206-31, CSJ 515 (AVT-2), RSG 888, H 08-71, Vijay, JG 25, GJG 0904, RSG 931, CSJ 730, NDG 11-41, GNG 2146, IPC 2009-21, GJG 1012, GL 28186, JG 33, CSJ 741, Phule G 0752, BG 3035, H 09-54, RSG 888, GJG 1003, IPC 2008-76, GNG 2145, H 09-23, NBeG 47-1IPC 06-126, AKG 1108, JG 29, GJG 1010, BG 3033, DKG 972, JG 30, DKG 1030, H 09-90, CSJ 697, BGD 1071, GJG 0921, IPC 06-77, H 08-75, BG 3032, Phule G 0204-16, JG 14, NDG 11-21, GNG 1581, GJG 0809, PBG 5, GNG 469, GL 27104, GNG 1958, Phule G 0204-4, GCP 101, GJG 0906, RSG 888, GJG 0907, JAKI 9218, PG 0120, CSJ 563, BGD 1074, H 09-96, Phule G 0302-10, IPC 08-57, Phule G 08108GL 29390, IPC 07-13, Vijay and RKG 160 showed resistant reactions with 0-10 per cent disease incidence, whereas 25 entries *viz.*, IPC 07-09, CSJ 739, BG 3036, GPF 2, Vishal, NBeG 165, NDG 11-12, RKG 202-22, CSJ 724, GNG 2124, GNG 2171, GJG 1001, GL 28295, GL 28297, BG 3037, RSG 963, JG 31, NBeG 3, GNG 2144, PG 099, GNG 1995 (R), GJG 0814, GJG 0922, RKG 11-301 and Phule G 0305-3 showed moderately resistant reactions with 11-20 per cent disease, 10 entries *viz.*, NDG 11-11, BG 3038, GJG 1004, BGD 1075, GJG 1013, BG 3034, CSJ 513, IPC 08-69, JG 34 and RSG 931showed moderately susceptible reaction with 21-30 per cent disease, 5 entries *viz.*, BGD 1073, RKG 201-95, NDG 11-5, JG 32 and GL 29389 showed susceptible reaction with 30-50 per cent disease and none of the entries showed highly susceptible reactions (Table 2). These results are in agreement with Vannia Rajan *et al.* (2012) [21] they recorded 11 genotypes tolerant, 26 were resistant, 31 were moderately resistant, against collar rot of chickpea disease, similarly, Saifulla M. *et al.* (2011) [10] reported 67 chickpea genotypes as resistant to collar rot disease.

Amongst the 61 *Kabuli* entries screened for collar rot 42 entries *viz.*, BG 3040, NDGK 11-32, HK 2, GNG 2182, NBeG 72, BGD 1076, GNG 2196, Phule G 10406, GLK 27211, BGD 1077, JGK 13, GLK 28127 (AVT 2), IPCK 08-120, JGK 17, BG 3028, CSJK 69, BGD 1079, CSJK 54, BG 3026, BDNGK 798, CSJK 74, Phule G 09316, JGK 2, BG 3025, JGK 20NBeG 119, IPCK 2008-108, Phule G 10404, JGK 21, BG 3041, Kripa, HK 09-201, IPCK 2008-109, BG 3042, PKV 4, HK 09-206, JGK 22, BGD 1078, HK 04, BG 3039, BDNG 799 and Phule G 10306 showed resistant reaction with 0-10 per cent disease incidence; 11 entries *viz.*, HK 06-171, NDGK 11-31, IPCK 2009-164, IPCK 2009-47, HK 08-231, GLK 28331, JGK 19, BG 3022, Kripa, CSJK 72 and HK 09-202 showed moderately resistance reaction with 11-20 per cent disease incidence; 5 entries *viz.*, IPCK 06-143, GNG 2104 (AVT 2), JGK-1, CSJK 27 and SKUA-C-23311 showed moderately susceptible reaction with 21-30 per cent disease incidence; 3 entries *viz.*, HK 09-219, JGK 18 and CSJK 70 showed susceptible reaction with 31-50 per cent disease incidence and none of the entry was highly susceptible (Table 3). Likewise Om Gupta and Anita Babbar (2006) recorded HK 00297 and PG 97-313 (Kabuli) genotypes resistance to collar rot disease

Among the 32 entries screened for collar rot from ICRISAT, 21 entries *viz.*, ICC 11322, ICC 4951, JG62, ICCV 04514, ICCV 07107, ICCV 07111ICCV 07309, ICCV 08113, ICCV 08124, ICCV 08125, ICCV 93706, ICCV 07304, ICCV 07305, ICCV 08117, ICCV 08120, ICCV 08123, ICCV 08315, ICCV 08321, ICCV 08323, ICCV 96854 and ICCV 98505 showed resistant reaction; eight entries *viz.*, ICCV 08116, ICCV 08310, ICCV 07105, ICCV 07118, ICCV 07306, ICCV 073111, ICCV 08319 and ICC 5003 were moderately resistant reaction; one line ICCV 08305 was moderately susceptible the other line ICCV 08311 showed susceptible reaction and another line ICCV 08317 showed highly susceptible reaction (Table 4). Similar results were also recorded by Om Gupta and Anita Babbar (2006) [12] they found the genotypes H 99-264, PG 9425-5 and PG 9425-9 as resistant to collar rot disease.

Fig 1: Field view of screening plot (right) and effect of seed treatment (left).

Legend

T1: Carbendazim (0.1%)
T2: Propiconazole (0.1%)
T4: Difenconazole (0.1%)
T3: Chlorothalonil (0.2%)
T5: Thiophanate methyl (0.2%)
T6: Hexaconazole (0.1%)
T7: Vitavax power (0.2%)

Table 2: Grouping of Kanpur desi type chickpea genotypes based on their resistance reaction to collar rot under field condition

Sl. No.	Disease Reaction	Genotypes	No. of genotypes
1	Resistant (0-10 per cent disease incidence)	GNG 1581, GJG 0809, PBG 5, GNG 469, GL 27104, GNG 1958, Phule G 0204-4, GCP 101, GJG 0906, RSG 888, GJG 0907, JAKI 9218, PG 0120, CSJ 563, BGD 1074, H 09-96, Phule G 0302-10, IPC 08-57, Phule G 08108, IPC 06-126, AKG 1108, JG 29, GJG 1010, BG 3033, DKG 972, JG 30, DKG 1030, H 09-90, CSJ 697, BGD 1071, GJG 0921, IPC 06-77, H 08-75, BG 3032, Phule G 0204-16, JG 14, NDG 11-21, GL 29390, IPC 07-13, NDG 11-24, PG 0100, H 09-53, CSJ 692, GNG 2127, H 09-65, RKG 207-61, RKG 206-31, CSJ 515 (AVT-2), RSG 888, H 08-71, Vijay, JG 25, GJG 0904, RSG 931, CSJ 730, NDG 11-41, GNG 2146, IPC 2009-21, GJG 1012, GL 28186, JG 33, CSJ 741, Phule G 0752, H 09-54, RSG 888, GJG 1003, IPC 2008-76, GNG 2145, BG 3035, H 09-23, NBeG 47-1, Vijay and RKG 160	73
2	Moderately resistant (11-20 per cent disease incidence)	GPF 2, Vishal, NBeG 165, NDG 11-12, RKG 202-22, CSJ 724, GNG 2124, GNG 2171, GJG 1001, GL 28295, GL 28297, BG 3037, RSG 963, JG 31, NBeG 3, GNG 2144, PG 099, GNG 1995 (R), GJG 0814, IPC 07-09, GJG 0922, CSJ 739, BG 3036, RKG 11-301 and Phule G 0305-3	25
3	Moderately susceptible (21-30 per cent disease incidence)	NDG 11-11, BG 3034, CSJ 513, IPC 08-69, BG 3038, GJG 1004, BGD 1075, GJG 1013, JG 34 and RSG 931.	10
4	Susceptible (31-50 per cent disease incidence)	BGD 1073, RKG 201-95, NDG 11-5 and JG 32, GL 29389.	5
5	Highly susceptible (>50 per cent disease incidence)	-	0

Table 3: Grouping of Kanpur kabuli type chickpea genotypes based on their resistance reaction to collar rot under field condition

SI. No.	Disease Reaction	Genotypes	No. of genotypes
1	Resistant (0-10 per cent disease incidence)	CSJK 54, BG 3026, BDNGK 798, CSJK 74, Phule G 09316, JGK 2, BG 3025, JGK 20, BG 3040, NDGK 11-32, HK 2, GNG 2182, NBeG 72, BGD 1076, GNG 2196, Phule G 10406, GLK 27211, BGD 1077, JGK 13, GLK 28127 (AVT 2), IPCK 08-120, JGK 17, BG 3028, CSJK 69, BGD 1079, NBeG 119, IPCK 2008-108, Phule G 10404, JGK 21, BG 3041, Kripa, HK 09-201, IPCK 2008-109, BG 3042, PKV 4, HK 09-206, JGK 22, BGD 1078, HK 04, BG 3039, BDNG 799 and Phule G 10306	42
2	Moderately resistant (11-20 per cent	HK 08-231, HK 06-171, NDGK 11-31, IPCK 2009-164, IPCK 2009-47, GLK 28331,	11

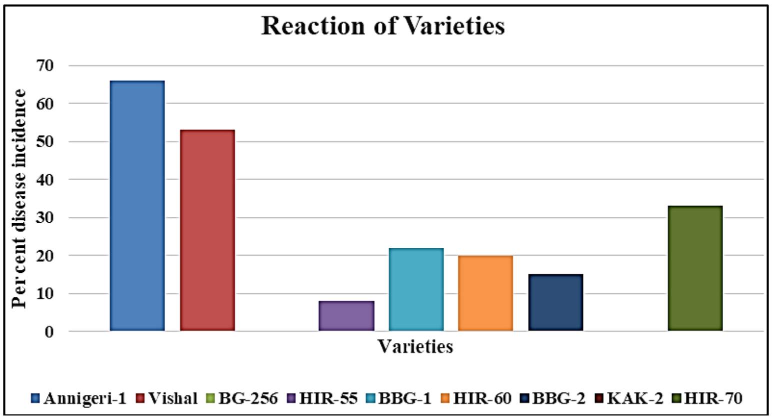
	disease incidence)	JGK 19, BG 3022, Kripa, CSJK 72 and HK 09-202	
3	Moderately susceptible (21-30 per cent disease incidence)	IPCK 06-143, GNG 2104 (AVT 2), JGK-1, CSJK 27 and SKUA-C-23311	5
4	Susceptible (31-50 per cent disease incidence)	HK 09-219, JGK 18 and CSJK 70.	3
5	Highly susceptible (>50 per cent disease incidence)		0

Table 4: Grouping of ICRISAT chickpea genotypes based on their resistance reaction to collar rot under field condition

Sl. No.	Disease Reaction	Genotypes	No. of genotypes
1	Resistant (0-10 per cent disease incidence)	ICCV 07107, ICCV 07111, ICCV 07304, ICCV 07305, ICCV 08117, ICCV 08120, ICCV 08123, ICCV 08315, ICCV 08321, ICCV 93706, ICCV 11322, ICCV 4951, JG62, ICCV 08323, ICCV 96854 ICCV 98505, ICCV 07309, ICCV 08113, ICCV 08124, ICCV 08125, and ICCV 04514	21
2	Moderately resistant (11-20 per cent disease incidence)	ICCV 07105, ICCV 07118, ICCV 07306, ICCV 073111, ICCV 08116, ICCV 08310, ICCV 08319 and ICCV 5003	8
3	Moderately susceptible (21-30 per cent disease incidence)	ICCV 08305	1
4	Susceptible (31-50 per cent disease incidence)	ICCV 08311	1
5	Highly susceptible (>50 per cent disease incidence)	ICCV 08317	1

2 Screening in pot conditions

Eight entries of chickpea (Table 5 and graph 1) along with one susceptible check Annigeri-1 were used for evaluation against collar rot resistance in pot condition as explained in "Materials and Methods". Among them, two entries *viz.*, BG-256 and KAK-2 were free from infection (0%) whereas HIR-55, BBG-2 HIR-60 BBG-1 HIR-70 showed 8, 15, 20, 22 and 33 per cent infection, respectively. Vishal was most susceptible with 53% infection compared to 60% infection in susceptible check (Annigeri-1). Similarly Abida Akram *et al.* (2008) evaluated the germplasm under greenhouse conditions in Islamabad, to identify sources of genetic resistance against collar rot disease caused by *S. rolfsii*. Out of 98 germplasm accessions only 5 genotypes *viz.*, FLIP 97132C, FLIP 97-85C, FLIP 98-53C, ILC -5263 and NCS 9905 exhibited highly resistant response to disease while 9 genotypes *viz.*, FLIP 96-153C, FLIP 97-129C, FLIP 97-172C, FLIP 97-185C, FLIP 98-227C, FLIP 98-107C, FLIP 98-230C, ILC-182 and NCS 9903 showed resistant reaction. Twenty five genotypes showed moderately resistant to tolerant response while the remaining were susceptible to highly susceptible to this disease.



Graph 1: Reaction of selected varieties in green house (Artificial inoculation)

Different varieties exhibited different kind of reactions against the collar rot disease of chickpea when tested indicate differential interactions in chickpea genotypes. Significant host-pathogen interactions indicated existence of specificity of resistant genes in chickpea pathosystems as hypothesized by Vanderplank (1984) [20].

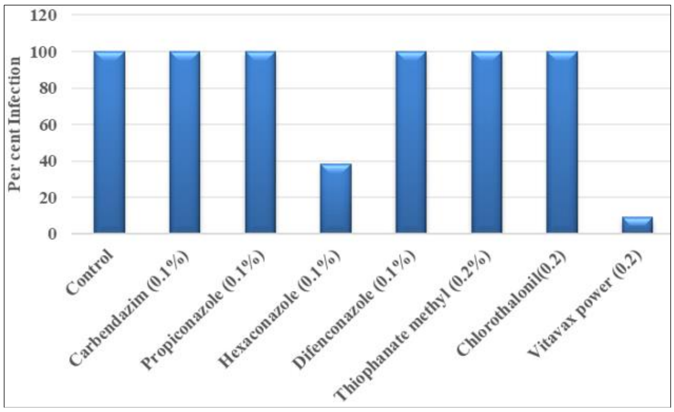
Table 5: Reaction of selected chickpea entries to collar rot resistance under artificial screening

Sl. No.	Variety	PDI	Disease reaction
1	Annigeri-1	66	HS
2	Vishal	53	HS
3	BG-256	0	R
4	HIR-55	8	R
5	BBG-1	22	MS
6	HIR-60	20	MR
7	BBG-2	15	MR
8	KAK-2	0	R
9	HIR-70	33	S

The utilization of resistant varieties is a classical approach to prevent losses caused due to diseases. This approach is novel in the management of diseases as it involves no or less cost of production.

3 Efficacy of fungicides in pot trials

The different fungicides tested among all *viz.*, Carbendazim (0.1%) propiconazole (0.1%), Hexaconazole (0.1%), difenconazole (0.1%), thiophanate methyl (0.2%), chlorothalonil (0.2) and Vitavax power (0.2); Vitavax power was found to be most effective with 9% infection of collar rot. Next good was Hexaconazole, with 38% infection (Table 6 and Fig 2) and other fungicides were least effective with total pre-emergence seedlings death, these observations are correlated with the findings of Prabhu (2003) [14] also recorded cent per cent mycelial suppression of *S. rolfsii* by Carboxin. Thiram was inhibiting the growth of *S. rolfsii*, the causal agent of foot rot of wheat (Harlapur (1988) [5] whereas, Sheoraj Singh *et al.* (2005) [17] observed Mancozeb, Thiram and Carboxin able to inhibit cent per cent control of the *S. rolfsii* causing collar rot in lentil *in vitro* and Vyas and Joshi, 1977 [22]; Siddaramaiah *et al*. 1979 [18]; Kulkarni *et al*. 1986 [7] also recorded effectiveness of Plantvax and Vitavax in inhibiting the vegetative growth of *S. rolfsii* of foot rot of various crop plants.



Graph 2: Effect of seed treatment of different fungicides against collar rot of chickpea

Similarly Arunasri *et al*. (2011) [2] found Propiconazole was highly effective with cent percent inhibition of pathogen at all the concentration tested followed by captan at 1000 ppm concentration (94.89%). And also by Kulkarni (2007) [8] Propiconazole were found to be most effective at all concentrations and Fouzia Yaqub and Saleem Shahzad (2006) [3] showed that dithane M 45 and sancozeb fungicides at high concentration are effective in reducing the growth of *S. rolfsii*.

Table 6: Effect of seed treatment on collar rot incidence of chickpea

Treatment	Germination percentage	Per cent Infection
Control	0	100*
Carbendazim (0.1%)	0	100*
Propiconazole (0.1%)	0	100*
Hexaconazole (0.1%)	36	38
Difenconazole (0.1%)	3	100*
Thiophanate methyl (0.2%)	0	100*
Chlorothalonil(0.2)	0	100*
Vitavax power (0.2)	64	9

0 indicates 100 per cent pre-emergence death of the seedlings

There are many reports on the uses of mixtures of synthetic fungicides for the control of plant pathogenic fungi. When utilized as combi products, such fungicides enhance the level of inhibition of a pathogen as compared to solo applied at higher rates (Pappas, 1997; Raj and Sharma, 2003). Therefore, combi products of fungicides should be preferred for effective management of plant diseases.

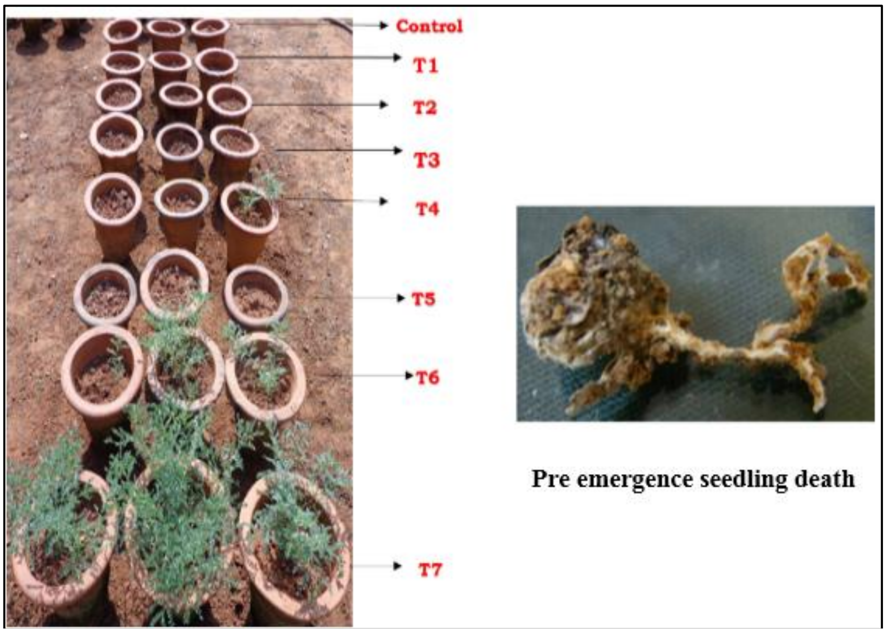


Fig 2: Effect of seed treatment of Annigeri I seeds against collar ro

Legend
T1: Carbendazim (0.1%)
T2: Propiconazole (0.1%)
T3: Chlorothalonil (0.2%)
T4: Difenconazole (0.1%)
T5: Thiophanate methyl (0.2%)
T6: Hexaconazole (0.1%)
T7: Vitavax power (0.2%)

Conclusion
If we use resistant/ moderately resistant varieties along with seed treatment at the time of sowing we can manage this disease to a greater extent by this we can achieve the higher yields which were hindered by collar rot of chickpea.

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