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Management of Dry Root Rot in Chickpea (*Cicer arietinum* L.) Caused By *Macrophomina phaseolina* by Utilizing Host Plant Resistance, Fungicides and Bioagents

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ABSTRACT

Background: For the management of soil borne disease like dry root rot of chickpea caused by *Macrophomina phaseolina*, by using fungicides alone is not feasible due to environmental and health hazards. Hence integrated management of the disease by using resistant varieties, fungicides and bio-control agents is the best alternative. So the present study was aimed to identify resistant varieties, best fungicide and bioagent for management of dry root rot in chickpea.

Methods: Two hundred and twelve genotypes were screened using blotter paper technique for identifying resistant genotypes for dry root rot. The experiment on management of dry root rot was conducted during *Kharif* and *Rabi* of 2013-14 using a susceptible chickpea variety JG-11 with 14 treatments including control with 3 replications.

Result: Of two hundred and twelve chickpea genotypes screened for host plant resistance against *Macrophomina phaseolina* by blotter paper technique only one genotype *i.e*. PBG-5 showed moderately resistant reaction. Among fourteen treatments including fungicides and bioagents imposed for the management of dry root rot, seed treatment with tebuconazole @ 2 g/kg recorded lowest per cent disease incidence of 9.43, with a highest yield of 722.81 kg/ha compared to untreated control which recorded the highest per cent disease incidence (40.10) with a lowest mean yield of 362.02 kg/ha.

Key words: Blotter paper technique, Chickpea, Dry root rot, *Macrophomina phaseolina*, Management.

INTRODUCTION

Chickpea (*Cicer arietinum* L.) is the oldest and third most important pulse crop after dry beans (*Phaseolus vulgaris* L.) and dry peas (*Pisum sativum* L.). It is believed to be originated in south-eastern Turkey (Ladizinsky, 1975). The genus *Cicer*, includes nine annuals and 34 perennial herbs (Van Der Maesen, 1972). In India, chickpea ranks second in area and third in production, perhaps is the largest producer of chickpea in the world covering 80 per cent area and 85 per cent of total production with a productivity of 844 kg/ha (www.iipr.res.in).

Among several constrains that lead to low productivity of chickpea, biotic and abiotic factors are very important. The chickpea is suffering from 172 pathogens including 67 fungi, 22 viruses, 3 bacteria, 80 nematodes and mycoplasma from all over the world (Nene *et al*., 1996). Among all the pathogens, only a few causes massive damage to the crop. Some of the diseases in order of their importance are wilt (*Fusarium oxysporum* f. sp. *ciceri*), wet root rot (*Rhizoctonia solani*), dry root rot (*Macrophomina phaseolina*), Ascochyta blight (*Ascocthya rabiei*) and collar rot (*Sclerotium rolfsii*). Among them, dry root rot caused by *M. phaseolina* is becoming serious threat to chickpea production, it causing 10-35 per cent loss in yields in most of the chickpea growing regions of India (Mahendra Pal, 1998).

Macrophomina phaseolina is primarily seed and soil-borne fungal pathogen having wide host range. In chickpea, infected seeds and microsclerotia surviving in the soil are the major source of primary inoculum. For soil borne diseases, use of fungicides alone is not feasible due to environmental hazards involved. Hence integrated management of the disease by utilizing host plant resistance, fungicides and bio-control agents is the best alternative (Ramarethinum *et al*., 2001). Investigations were made in the present study to identify resistant varieties, best fungicide and bioagent for management of dry root rot in chickpea.

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MATERIALS AND METHODS

Two hundred and twelve genotypes consisting of one hundred and ten desi, sixty one Kabuli types supplied by IIPR Kanpur, ten genotypes of local germplasm and advanced breeding material entries from AICRP on chickpea, GKVK, UAS, Bangalore and thirty one chickpea

entries supplied by ICRISAT Patancheru, Hyderabad were screened using blotter paper technique (Nene *et al*., 1981) for identifying resistant genotypes for dry root rot.

Blotter paper technique

The mycelial discs of *Macrophomina phaseolina* culture KAMP-1 (seven days old) was inoculated to 500 ml conical flasks consisting sterilized 250 ml of Potato Dextrose Broth (PDB) and incubated for seven days. The mycelial mat from the conical flasks was removed and macerated in a warring blender along with 100 ml of sterilized distilled water for a minute. The inoculum was later collected in a beaker. In the mean time, the chickpea seedlings were raised in plastic pots containing sterilized sand mixed soil. One week old seedlings were uprooted and the roots were immersed in sterile water in order to remove the adhered soil particles. The seedlings were then immersed completely in the inoculum in a beaker for a minute. The seedlings particularly the root portion were then placed side by side on a blotter paper (45 cm x 25 cm with one fold) in such a way that only the cotyledons and roots are covered and the green tops of seedlings remained outside and then blotter paper was folded. The folded blotter papers as a heap of ten were then placed in trays and kept in an incubator at 35°C for eight days provided with 12- h artificial light, 80 per cent RH and the blotters were moistened with sterile water every day. One of these ten blotters had the seedlings of susceptible check (L-550). At the end of the incubation period (8 days), the seedlings were examined for the extent of root damage, and scored for the disease on a 1-9 rating scale (Nene *et al*. 1981). Ten seedlings of each entry were considered as one replication, Based on the disease score the genotypes were grouped into different disease reaction categories as mentioned below.

Management of dry root rot by fungicides and bioagents

The experiment on the management of dry root rot in chickpea was conducted during the *Kharif* and *Rabi* of 2013-14 at ZARS, GKVK, UAS, Bengaluru, Karnataka using a susceptible variety JG-11 with a plot size 3 X 2 sq.m and spacing of 30 x 10 cm in a RCBD design consisting 14 treatments including control and 3 replications. The treatments were -seed treatment with *Trichoderma viride* @ 5 g/ kg of seeds (T1), seed treatment with *T. viride* @ 10 g/ kg of seeds (T2), seed treatment with *Pseudomonas florescence* @ 5 g/ kg of seed (T3), seed treatment with *P. florescence* @ 10 g/ kg of seed (T4), soil application of *T. viride* @ 5 kg in 50 kg of FYM (T5), soil application of *T. viride* @10 kg in 50 kg of FYM (T6), seed treatment with carbendazim @ 2 g + thiram @ 1 g/kg of seed (T7), seed treatment with carbendazim @ 2 g + captan @ 1 g/kg of seeds (T8), seed treatment with carbendazim + mancozeb (Saff) @ 2 g/ kg of seed) (T9), seed treatment with tebuconazole (Raxil) @ 2 g/kg of seeds (T10), seed treatment with tebuconazole (Raxil) @ 2 g + carbendazim 1 g/ kg of seed (T11), seed treatment with carboxin 37.5% + thiram 37.5% (vitavax power) @ 2 g/ kg of seed (T12), seed treatment with carboxin 37.5% + thiram 37.5% (vitavax power) @ 2g + *T. viride* @ 5 g/ kg of seed (T13) and untreated control (T14). The observations on disease incidence and yield parameters were recorded at maturity for each treatment.

Statistical analysis

The experimental data collected were analyzed statistically for its significant or non-significant difference by the normal statistical procedure adopted for completely randomized design and randomized block design and interpretation of data was carried out in accordance with Walter (1997). The level of significance used in 'F' and 'T' test was P=0.05 and P=0.01. Critical differences were calculated wherever 'F' test was significant. The value of per cent disease incidence was subjected to arcsine transformation.

RESULTS AND DISCUSSION

Among two hundred and twelve entries screened in blotter paper method only one entry ie. PBG 5 was found to be moderately resistant, twenty nine entries found moderately susceptible, one hundred and nine entries were found susceptible and seventy three entries were found highly susceptible (Table 2). Earlier workers have also screened chickpea genotypes against dry root rot. Nagamma *et al*. (2015) screened hundred and sixty five chickpea entries and reported none of them to be resistant or moderately resistant. Whereas, Pande *et al*. (2004) screened forty seven chickpea germplasm lines and found three of them to be resistant, 22 moderately resistant, 19 susceptible and three highly susceptible under blotter paper technique. Om *et al*. (2012) screened 170 genotypes for dry root rot during the year 2007-10 under blotter paper method, 68 genotypes exhibited resistant reaction (<10% mortality).

Among the fourteen treatments imposed for the management of dry root rot in chickpea in both *kharif* and *rabi* season of 2013-14. The seed treatment with tebuconazole @ 2 g/kg (T10) recorded the lowest (11.77 and 7.08%) dry root rot incidence with a yield of 666.57 kg/ ha and 779.04 kg/ha, respectively in *kharif* and *rabi* and is at par with T11 [ST with tebuconazole (2 g/kg) + carbendazim

Table 1: Categorization of chickpea genotypes into different disease ratings on 1-9 scale.

Rating	Disease reaction categories	Symptoms
1	Resistant	No infection on roots
3	Moderately resistant	Very few small lesions on roots
5	Moderately susceptible	Lesions on roots clear but small, new roots free from infection
7	Susceptible	Lesions on roots many, new roots generally free from lesions
9	Highly susceptible	Roots infected and completely discolored

Table 2: Disease reaction of chickpea entries for dry root rot under blotter paper method.

Rating	Reaction	Entries	No. of entries
1	Resistant	Nil	0
3	Moderately resistant	PBG 5	1
5	Moderately susceptible	GNG 1581, GNG 469, GNG 1958, CSJ 563, RKG 202-22, IPC 06-126, AKG 1108, CSJ 697, Phule G 0204-16, JG 14, IPC 08-69, CSJ 515 (AVT-2), Vijay, JG 25, GJG 0904, CSJ 730, IPC 07-09, GNG 2104 (AVT 2), GNG 2196, Phule G 10406, HK 09-219, BGD 1077, IPCK 2008-108, CSJK 72, Phule G 10404, JGK 21, BG 3041, ICCV 07111 and ICCV 96854.	29
7	Susceptible	GJG 0809, GL 27104, GPF 2, GCP 101, GJG 0906, GJG 0907, NDG 11-11, PG 0120, RKG 201-95, BGD 1074, Phule G 0302-10, GNG 2124, IPC 08-57, GNG 2171, GJG 1001, GL 28297, JG 29, GJG 1010, DKG 972, BG 3034, DKG 1030, NDG 11-5, GJG 0921, IPC 06-77, H 08-75, NDG 11-21, GL 29390, NDG 11-24, PG 0100, CSJ 513, H 09-53, CSJ 692, GJG 1004, JG 31, GNG 2127, GJG 1013, GNG 1995 (R), RKG 207-61, RKG 206-31, GJG 0814, RSG 888, NDG 11-41, CSJ 739, GJG 1012, GL 28186, CSJ 741, H 09-54, RSG 888(2), GJG 1003, JG 34, Phule G 0305-3, H 09-23, NBeG 47-1, RKG 160, CSJK 54, BDNGK 798, CSJK 74, IPCK 06-143, HK 08-231, Phule G 09316, BG 3025, HK 06-171, CSJK 27, NDGK 11-31, JGK 20, BG 3040, NDGK 11-32, GNG 2182, GLK 27211, IPCK 2009-47, GLK 28331, BG 3039, BDNG 799, HK 09-202, Phule G 10306, CSJK 70, JGK 13, GLK 28127 (AVT 2), JGK 17, Kripa (178), CSJK 69, BGD 1079, NBeG 119, Kripa (189), HK 09-201, BG 3042, HK 09-206, BGD 1078, ICCV 07105, ICCV 07107, ICCV 07304, ICCV 07305, ICCV 07306, ICCV 07309, ICCV 073111, ICCV 08113, ICCV 08116, ICCV 08117, ICCV 08120, ICCV 08125, ICCV 08305, ICCV 08315, ICCV 93706, ICC 11322, ICC 4951, ICC 5003, Vishal, KAK 2 and JG-62.	109
9	Highly susceptible	Phule G 0204-4, RSG 888(1) , JAKI 9218, BGD 1073, NBeG 165, NDG 11-12, CSJ 724, H 09-96, PhuleG 08108, GL 28295, BG 3033, JG 30, H 09-90, BGD 1071, BG 3032, GJG 0922, IPC 07-13, BG 3037, BG 3038, RSG 963, NBeG 3, BGD 1075, GNG 2144, H 09-65, PG 099, JG 32, GL 29389, H 08-71, GNG 2146, IPC 2009-21, JG 33, PhuleG 0752, BG 3035, RSG 931, IPC 2008-76, BG 3036, RKG11-301, GNG 2145, BG 3026, JGK 2, JGK-1, HK 2, IPCK 2009-164, NBeG 72, BGD 1076, JGK 19, HK 04, SKUA-C-23311, JGK 18, IPCK 08-120, BG 3022, BG 3028, IPCK 2008-109, PKV 4, JGK 22, ICCV 04514, ICCV 07118, ICCV 08123, ICCV 08124, ICCV 08310, ICCV 08311, ICCV 08317, ICCV 08319, ICCV 08321, ICCV 08323, ICCV 98505, A1, BBG 1, BBG 2, BG 256, HIR 70, HIR 55 and HIR 60.	73
Total No. of genotypes			212

(1 g/kg)] which recorded 15.09 and 15.05 per cent disease incidence with a yield of 564.43 kg/ha and 629.04 respectively in *kharif* and *rabi*. These treatments were found significantly superior over control which recorded highest dry root rot incidence as 41.87 and 38.33 with a lowest yield of 310.55 kg/ha and 413.49kg/ha respectively, in *kharif* and *rabi* (Table 3).

In both the seasons the treatment T10: ST with tebuconazole @ 2 g/kg recorded lowest per cent disease incidence of 9.43, with a highest yield of 722.81 kg/ha, which is at par with T11: ST with tebuconazole 2 g + carbendazim 1 g/kg which recorded 15.07 per cent disease incidence with a yield of 596.74 kg/ha followed by T8: ST with carbendazim 2 g + captan 1 g/kg of seed which recorded 15.21 per cent disease incidence with a mean yield of 519.92 kg/ha. These treatments are significantly superior to control which recorded 40.10 per cent disease incidence with a lowest mean yield of 362.02 kg/ha (Table 3).

It has been shown that tebuconazole ((RS)-1-(4-chlorophenyl)-4,4-dimethyl-3-(1H-1,2,4-triazole-1-ylmethyl)-pentan-3-alcohol) was highly effective against *Fusarium graminearum* (Spolti *et al*., 2012). It is a systemic fungicide of the triazole group, and the primary mode of action is the inhibition of ergosterol biosynthesis in fungi (Hewitt, 1998). Even though different triazole fungicides have a similar mechanism of action, they may show marked differences in their activity against different fungal pathogens (Buchenauer, 1987; Scheinpflug and Kuck, 1987). This broad-spectrum, relatively new triazole fungicide is being used for its effectiveness against soil-borne and foliar fungal diseases in nut, fruit, cereal and vegetable crops worldwide (Munoz-Leoz *et al*., 2011).

Results of the present study are in agreement with

Table 3: Management of dry root rot in chickpea by utilizing synthetics and bioagents during the year 2013-14.

Treatments	Dry root rot incidence (%) (2013-14)		Mean dry root rot	Yield (kg/ha.) (2013-14)		Mean Yield
	Kharif	*Rabi*	incidence (%)	*Kharif*	*Rabi*	(kg/ha)
T1: ST* with *T. viridae* @ 5 g/kg of seed	30.34 (33.12)	27.26 (31.17)	28.80 (32.44)^{hi}	368.48	427.38	397.93 ^{ab}
T2: ST with *T. viridae* @ 10 g/kg of seed	28.14 (31.61)	21.08 (27.24)	24.61 (29.67)^{defgh}	437.55	468.49	453.02^{abcd}
T3: ST with *P. fluorescens* @ 5 g/kg of seed	38.03 (37.98)	35.86 (36.72)	36.95 (37.42)^{ih}	345.32	672.38	508.85^{abcd}
T4: ST with *P. fluorescens* @ 10 g/kg of seed	39.57 (38.88)	31.77(34.19)	35.67 (36.63)^{iih}	380.42	555.71	468.06^{abcd}
T5: Application of *T. viridae* @ 5 kg in 50 kg of FYM	28.15(32.03)	24.16 (29.38)	26.16 (30.73)^{efgh}	406.98	452.93	429.96^{abcd}
T6: Application of *T. viridae* @ 10 kg in 50 kg of FYM	26.81 (30.40)	20.27 (26.38)	23.54 (28.96)^{bcdefgh}	360.38	573.49	466.94^{abcd}
T7: ST with carbendazim @ 2 g + thiram @ 1 g/kg of seed	18.32 (24.82)	16.94 (24.17)	17.63 (24.81)^{bc}	552.25	466.26	509.26^{abcd}
T8: ST with carbendazim @ 2 g + captan @ 1 g/kg of seed	21.17(26.58)	9.24 (16.88)	15.21 (22.54)^{b}	453.02	586.82	519.92^{abcd}
T9: ST with carbendazim + mancozeb @ 2 g/kg of seed	27.60 (31.43)	19.83 (26.18)	23.72 (29.06)^{cdefgh}	337.54	474.04	405.79^{abc}
T10: ST with tebuconazole @ 2 g/kg	11.77 (19.84)	7.08 (15.19)	9.43 (17.74)^{a}	666.57	779.04	722.81^{e}
T11: ST with tebuconazole 2 g + carbendazim @ 1 g/kg of seed	15.09 (22.55)	15.05 (22.48)	15.07 (22.83)^{b}	564.43	629.04	596.74^{de}
T12: ST with carboxin 37.5% + thiram 37.5% (Vitavax power) @ 2 g/kg seed	28.52 (32.22)	23.93 (28.59)	26.23 (30.77)^{fgh}	421.98	729.04	575.51^{cde}
T13: ST with TV @ 5 g + carboxin 37.5 % + thiram 37.5% (Vitavax power) @ 2 g/kg seed	36.34 (37.01)	21.40 (27.28)	28.87 (32.30)^{ghi}	417.92	673.49	545.70^{bcd}
T14: Untreated control	41.87 (40.26)	38.33 (37.97)	40.10 (39.27)^{h}	310.55	413.49	362.02^{a}
S.Em±		1.44			80.53	
CD @ 5%		4.41			173.98	
CV		6.89			19.95	

Brenneman *et al*. (1991) who reported the antifungal activity of tebuconazole against *Sclerotium rolfsii* and *Rhizoctonia solani in vitro*. Kanwal *et al*. (2012) evaluated different concentrations *viz*. 35, 70, 105 and 140 ppm of tebuconazole against three problematic soil-borne fungal phytopathogens namely *M. phaseolina, F. oxysporum* f. sp. *lycopersici* and *S. rolfsii* using food poisoning technique, all the concentrations of tebuconazole completely arrested the growth of all the three fungal species.

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