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RESEARCH **P**APER

Identification of potential sources of resistance for wilt caused by *Fusarium oxysporum* f.sp. *ricini* in castor (*Ricinus communis* L.)

P. BINDU PRIYA[1], M.V. NAGESH KUMAR[2] **AND** V.GOURI SHANKAR[3]

Regional Agricultural Research Station (Pr. JTSAU), Palem, MAHABUBNAGAR (TELANGANA) INDIA
Email : bindupriya.gpb@gmail.com

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An experiment was carried out in wilt sick plot of Regional Agricultural Research Station, Palem to screen diverse germplasm accessions for evaluation of resistance to Fusarium wilt, a devastating disease in castor caused by *Fusarium oxysporum* f.p. *ricini* with an objective to identify new potential sources of wilt resistance. Observations were recorded as per cent disease infection in each entry periodically at thirty days interval upto 150 days after sowing. 'Kranthi' and 'Haritha' varieties were used as susceptible and resistant checks, respectively sown after every five test entries. Out of two hundred germplasm accessions screened, twenty nine accessions were found to be *Fusarium* wilt resistant with less than 20 per cent wilt incidence. Fifty accessions were screened for confirmation of wilt resistance out of which twelve accessions revealed resistant reaction. Further forty advanced lines were screened among which nineteen entries were found to be resistant. The identified resistant cultures serve as potential sources for developing diverse wilt resistant varieties/hybrids in castor.

Key words : Castor, Fusarium wilt, Sick plot, Germplasm screening

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INTRODUCTION

Castor (*Ricinus communis* L.) is an important non-edible oilseed crop particularly grown in arid and semi-arid regions. Castor oil enjoys tremendous demand globally in view of its numerous industrial uses. Wilt incited by *Fusarium oxysporum* f.sp. *ricini* is one of the major diseases of castor causing up to 80 per cent yield losses. Nanda and Prasad first reported castor wilt in India from Udaipur (Rajasthan) in 1974 (Nanda and Prasad, 1974). Wilt is a seed and soil borne disease that appears in patches where chemical control is not much effective and sanitary measures are not practically applicable implying the importance of host plant resistance. Although varieties and hybrids developed during the recent past are wilt resistant, they incline to become susceptible over a period of time. GCH-4, a renowned wilt resistant castor hybrid eventually turned out to become wilt susceptible (Patel *et al.*,1991). Similarly, Anjani *et al.* (2004) reported that the wilt resistant variety DCS-9 showed upto 60 per cent wilt incidence indicating gradual breakdown of resistance. This may be due to the continuously evolving new Fusarium pathotypes which necessitate the identification of newer potential sources for wilt resistance. Germplasm is the basic gene pool to search for useful genes and genotypes needed for achieving desirable genetic improvement (Anjani, 2012) and screening the available germplasm for desirable traits is

a key step in resistance breeding. All the popular wilt resistant castor varieties/ hybrids available today are more or less derived from the same pistillate lines and male combiners resulting in vertical resistance. Further, presence of genetic variation in different races of the wilt pathogen *Fusarium oxysporum* f.sp. *ricini* isolated from different regions has been reported (Prasad *et al*., 2008) which stresses the need for gene pyramiding. New resistant cultivars with a broader genetic base that perform well across different locations and ecosystems are to be developed in castor for achieving durable resistance.

The present study was taken up to evaluate various castor germplasm accessions for identification of novel Fusarium wilt resistant genes which can be introgressed in the otherwise well-performing castor varieties/hybrids or can be used in the development of broad genetic based wilt resistant parental lines. Incorporation of new resistant genes into the promising cultivars through different breeding and biotechnological methods was also reported by Shankar *et al.* (2010).

RESEARCH METHODOLOGY

Field screening embraces the advantage of ability to grow large populations, evaluate plants under natural conditions and record disease progress throughout the entire life cycle of the plant (Kumar and Srivastava, 2013). A wilt sick plot (WSP) was developed and maintained at Regional Agricultural Research Station, Palem by growing wilt susceptible varieties and incorporating wilt debris in the soil (Nene and Kannaiyan, 1982). Isolate of the local virulent wilt pathogen Palem-1 obtained by single spore isolation technique from infected castor roots was used for maintaining the inoculum load in WSP. The isolate was grown on potato dextrose agar (PDA) medium and fungal discs were inoculated on autoclaved and semi-cooked sorghum seed for fifteen days under sterilized conditions (Reddy *et al*., 2011). The culture thus, multiplied on sorghum seed was added to WSP regularly and uniformly to maintain the inoculum load. Care was taken to regularly irrigate the WSP for active multiplication of the fungal mycelium in soil (Anjani *et al*., 2014 and Pande *et al.*, 2012). Two hundred germplasm accessions obtained from Indian Institute of Oilseeds Research (IIOR), Hyderabad were evaluated in the WSP during *Kharif* 2011-12. Further, fifty germplasm accessions obtained from IIOR were re-evaluated in WSP for confirmation of resistance to Fusarium wilt. Resistant (Haritha) and susceptible (Kranthi) checks were grown along with the test material after every five rows. The germplasm accessions were sown in augmented Block Design with a spacing of 30x45 cm and row length of 6 m comprising 20 plants per row. Forty lines of advanced breeding material (including checks) were also evaluated in the WSP under 'All India Co-ordinated Research Project for Oilseeds' (AICORPO) trials sown in Randomized Block Design with three replications. Pathogen culture Palem-1 was regularly added to the WSP and uniformity of the inoculum was confirmed by testing randomly collected soil samples. Initial and final inoculum load was estimated before sowing and after harvest of the crop (Sharma and Singh, 1973). Inoculum load at the start of the experiment was found to be 1.64 x 10^3 colony forming units (CFU)/g soil which was subsequently increased and maintained at 2.0 x 10^3 CFU/g soil. Normal management practices were followed to control other diseases and insect pests in the crop. Observations were recorded for per cent wilt incidence at an interval of 30 days from the first appearance of wilt upto crop maturity (150 days). The resistance of the test material was reported only when it was free from any wilt symptoms upto 150 days after sowing, while the susceptible variety showed wilting and died subsequently. Per cent disease infection (PDI) was calculated at 150 days after sowing using the formula (Ahmad *et al.*, 2010; Ahammed and Reddy, 2009):

$$\text{Wilt incidence (PDI)} = \frac{\text{Number of plants infected}}{\text{Total number of plnats in each genotype}} \text{x100}$$

Considerable variability was observed among the genotypes for severity of the disease. On the basis of reaction to the disease (PDI), the genotypes were grouped into following categories based on the scale devised by Mayee and Datar (1986) :

Wilt incidence (PDI)	Category
0.0	Highly resistant
0.1-20.0	Resistant
20.1-40.0	Moderately resistant
40.1-50.0	Moderately susceptible
50.1-75.0	Susceptible
>75.0	Highly susceptible

RESEARCH FINDINGS AND ANALYSIS

Wide variability was observed among the accessions screened for Fusarium wilt resistance apart from other important agro-economic traits. Out of two hundred germplasm accessions evaluated twenty nine accessions were found to be resistant with less than 20 per cent wilt incidence (0.1 – 20.0 PDI) upto 150 days after sowing. Twenty two entries showed moderately resistant disease reaction (20.1 to 40.0 PDI), thirty accessions exhibited moderately susceptible reaction (40.1to 50.0 PDI), ninety two germplasm accessions were recorded as susceptible (50.0 to 75.0 PDI) while twenty seven of them were found to be highly susceptible to Fusarium wilt with more than 75 PDI (Table 1). Susceptible check Kranthi recorded 91.4 to 95.0 PDI indicating good spread of the disease in WSP while resistant check Haritha exhibited

Table 1 : Screening of germplasm for resistance to wilt

Disease reaction	Range of PDI (%)	Germplasm accessions
Highly resistant	0.0	Nil
Resistant	0.1 – 20.0	RG-815,844,1146,1221,1577,1697,1766,2100,2720,2759, 2924, 3225, 3242, 3253, 3292, 3296, 3336, 3338, 3352,3352, 3359, 3361, 3378, 3383, 3386, 1714, 2093, 2145, 2161 and 2254
Moderately resistant	20.1 – 40.0	RG-1180,1305, 1357, 1621, 1718, 1788, 2082, 2098, 2121, 2155, 2173, 2184, 2860, 3312, 3315, 3330, 3332, 3368, 3378, 3375 and 3390
Moderately susceptible	40.1 – 50.0	RG-133, 1582, 1612, 1685, 1709, 1904, 1999, 2076, 2077, 2081, 2111, 2112, 2116, 2137, 2140, 2148, 2191, 2253, 2614, 2697, 2761, 2773, 2870, 3314,3328, 3357, 3371, 3380 and 3382
Susceptible	50.1 – 75.0	RG-62, 190, 1103, 1125, 1142,1313, 1340, 1353, 1414, 1545, 1548, 1696, 1721, 1772, 1937, 2005, 2035, 2062, 2071, 2072, 2073, 2075, 2097, 2099, 2102, 2110, 219, 2129, 2131, 2132, 2138, 2142, 2150, 2166, 2171, 2240, 2242, 2243, 2250, 2334, 2422, 2451, 2486, 2681, 2685, 2769, 2775, 2776, 2804, 2822, 2833, 3061, 3102, 3116, 3177, 3187, 3188, 3206, 3218, 3223, 3233, 3262, 3297, 3298, 3304, 3307, 3309, 3311, 3320, 3322, 3325, 3329, 3331, 3334, 3335, 3339, 3340, 3342, 3344, 3346, 3347, 3348, 3350, 3351, 3352, 3355, 3364, 3366, 3367, 3370, 3372, 3377 and 3387
Highly susceptible	>75.0	RG-755, 1139, 1148, 1413, 1523, 1526, 1546, 1916, 1986, 2011, 2064, 2080, 2091, 2118, 2378, 2783, 3240, 3251, 3294, 3302, 3313, 3337, 3341, 3343, 3345, 3349 and 3363

Table 2 : Screening of germplasm for confirmation of wilt resistance

Disease reaction	Range of PDI (%)	Germplasm accessions
Highly resistant	0.0	Nil
Resistant	0.1 – 20.0	RG-558, 926, 981, 1364, 2087, 2105, 2125, 2126, 3300 and 3406
Moderately resistant	20.1 – 40.0	RG- 1634, 2106, 2130, 2139, 2153, 3224, 3396, 3398, 3410 and 3413
Moderately susceptible	40.1 – 50.0	RG- 735, 1945, 2014, 2876, 3391, 3395, 3397, 3399, 3400, 3411 and 3412
Susceptible	50.1 – 75.0	RG- 565, 574, 988, 1268, 1291, 1354, 1686, 1707, 1746, 2024, 2144, 3223, 3319, 3392 and 3411
Highly susceptible	>75.0	RG- 930 and1558

Table 3 : Screening of advanced material for wilt resistance

Disease reaction	Range of PDI (%)	Advanced material (Including checks)
Highly resistant	0.0	Nil
Resistant	0.1 – 20.0	DCS-9, JC-22, SKI-333, SKI-339, JC-24, 48-1, SHB-918, PCH-254, SHB-891, JHB-981, JHB-985, HSC-5, SHB-874, GCH-7, SHB-872, PCH-111, JI-384, PCH-222, DCS-9
Moderately resistant	20.1 – 40.0	JC-20, JC-26, MCI-11, JHB-977, WESTERN SARPANCH, SHB-890, NBCH-763, PCH-248, SHB-875, SKI-337, DCH-177
Moderately susceptible	40.1 – 50.0	MCI-12, DCH-519, NBCH-66, DCH-177, ANDCI-8
Susceptible	50.1 – 75.0	JC-4, JC-12, SHB-871, RHC-277, HCH-6
Highly susceptible	>75.0	Nil

wilt symptoms ranging from 0.0 to 5.8 PDI. Most of the susceptible entries recorded lesser wilt incidence than Kranthi. Among the fifty accessions screened for confirmation of wilt resistance, twelve accessions were found to be resistant (9.0 to 16.6 PDI), ten entries showed moderately resistant reaction (27.2 to 40.0 PDI), eleven entries exhibited susceptible reaction (42.8 to 50.0 PDI), fifteen of them were observed to be moderately susceptible (52.9 to 66.6 PDI) while two entries were recorded to be highly susceptible (81.8 and 100.0 PDI) (Table 2).Presence of highly resistant to highly susceptible germplasm for wilt in castor was also reported by Anjani (2012).Among the advanced material screened, nineteen entries showed resistance (5.9 to 18.8 PDI), eleven entries were moderately resistant (22.2 to 38.5 PDI), five entries were moderately susceptible (40.7 to 46.9 PDI) while five lines exhibited susceptible disease reaction (51.2 to 66.6 PDI) (Table 3). The resistant entries offer promising potential sources that can be used for future resistance breeding programmes. Similar findings were observed by Anjani *et al*. (2004) ; Anjani (2010) and Anjani and Raoof (2005) who reported several sources of stable resistance to Fusarium wilt identified based on multi-year, multi-location screening in wilt sick plots under high disease pressure. None of the lines were highly resistant indicating the need for further enhancement of wilt resistance through scrupulous screening (both field and greenhouse) of diverse castor genotypes. Similar studies for evaluation of disease resistance were carried out in other crops also where Nene and Kanniyan (1982) screened more than 11,000 entries of pigeonpea for resistance to Fusarium wilt in the wilt plots and selfed the individual plants resistant to *Fusarium udum* to fix resistance in a homozygous condition. Field screening of 106 germplasm accessions of groundnut was carried out for identification of sources of resistance to bacterial wilt by Wang *et al.* (2009). Besides field screening (WSPs), screening under green house conditions (using root dip inoculation technique) can be deployed for identification and confirmation of wilt resistance. Raoof and Rao (1996) screened 160 castor germplasm accessions under greenhouse conditions using root dip inoculation technique and identified forty one wilt resistant accessions.

Research on identification of race specific wilt resistant genes and pyramiding them in a single genotype is required for longer perpetuation of the varieties (Kumar *et al*., 2015). Resistant sources identified in the present study will be further evaluated under greenhouse conditions for confirmation of resistance. Such intensive screening enables to identify valuable sources of resistance that can be used in future breeding programmes in the development of new wilt resistant parental lines and improvisation of existing varieties and hybrids of castor.

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