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RESEARCH ARTICLE

Identification of resistant sources of castor against Fusarium wilt disease

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Abstract Castor is an important oilseed crop grown in India. However, its cultivation is seriously affected by several diseases, e.g. wilt disease caused by *Fusarium oxysporum* f. sp. *ricini* being economically relevant. Host plant resistance is the best option for the management of this disease. A total of 524 castor genotypes were screened against wilt disease under sick plot conditions for five years from 2013–2014 to 2017–2018. The wilt sick plot was maintained with a inoculum load of 2×10^3 CFU/gm of soil and the genotypes were screened alongside resistant (48–1) and susceptible (JI-35) cultivars. It was observed that 109 castor genotypes were susceptible to wilt with more than 50% wilt incidence whereas, 32 genotypes; i.e., DCS-86, DCS-118, DCS-108, DCS-105, DCS-107, DPC-17, DPC-18, DPC-21, DPC-23, DPC-24, DPC-25, M-571, DPC-28, PMC-9, PMC-11, PMC-14, PMC-15, PMC-16, PMC-17, PMC-24, PMC-38, PMC-55, PMC-60, PVT- 11–3, PVT-11–18, PVT-11–17, PVT-11–21, PVT-12–4, PVT-12–6, PVT-12–72 and PVT-11–26 recorded < 20% wilt incidence during the consecutive years of testing. Three wilt-resistant parental lines of castor IPC-21/DPC-21(INGR No.21107), M-571 (INGR No. 21230), and ICS-200 (INGR No. 21157) were identified and registered by the Plant Germplasm Registration Committee. Among the screened parental lines, PMC-14 showed a resistant reaction of 10–16.7% to wilt for three years and was used as a parent ICS-164 (PMC-14) in developing wilt-resistant hybrid ICH-66, which was released for cultivation in five states of India, namely, Andhra Pradesh, Telangana, Tamil Nadu, Karnataka and Odisha. The resistant lines identified in this study could be used for breeding wilt-resistant castor hybrids.

Keywords Advanced breeding material · Castor wilt · *Fusarium oxysporum* f. sp. *ricini* · Host plant resistance · Parental lines · Wilt sick plot

Introduction

India is the major producer of castor in the world followed by Mozambique, China, Brazil, and Myanmar. Castor crop (*Ricinus communis* L.) has good industrial and medicinal value and the importance of the crop lies in its unique seed oil that is composed of greater than 80% ricinoleic acid, an unusual, monounsaturated, 18-carbon fatty acid, and has many desirable industrial properties. Castor oil finds its use in the manufacture of industrial products like nylon fibers, jet engine lubricants, dyes, hydraulic fluids, detergents, ointments, cosmetics, greases, paints, soaps, perfumes, varnishes, etc., (Dange 2003). Castor is now emerging as a commercial crop with immense

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export potential, capable of securing valuable foreign exchange. In 2021–2022, India exported 689,656 MT of castor oil, which was worth 75260 million as per data available with the solvent extractors association of India. India recorded castor yield of 2288 kg/ha during 2021–2022 and the total production was 179.5 million tons in 2021–2022 according to the solvent extractors association of India (Tilhantec 2022).

Total area under castor seed cultivation in India for the year 2022–2023 was estimated to be 0.918 million hectares as per governments estimates against 0.8–1.1 million hectares in 2021–2022, which has increased by 13% compared to the previous year. After revision of Gujarat and Rajasthan yield estimate, India's average castor seed productivity for 2022–2023 has been revised to 2048 kg/ha against a second estimate of 2074 kg/ha and first estimate of 2129 kg/ha. Total castor seed production in India has been revised to 1.881 million tons for 2022–2023 against the 1st estimate of 1.963 million tons and second estimate of 1.906 million tons. The production for 2023 has been predicted to be higher by 11% when compared with production estimate of 1.694 million tons for 2022 (Anonymous 2023; Castor crop survey 2022–2023, SEA, INDIA).

Fusarium oxysporum f. sp. *ricini* causes wilt disease in all stages of castor crop growth from seeding to fully mature stage, leading to significant losses. The symptoms appear as yellowing of seedlings and sickly appearance with marginal necrosis of the leaves. The cross-sections of the affected roots show the fungus in vascular tissues (Nanda and Prasad 1974). The young seedling of the two–three leaf stage exhibits discoloration of hypocotyl and a decrease of turgidity along with color change. The plants infected during flowering, spike formation, and development stages show sickly appearance, yellowing, and marginal necrosis of leaves that later advances to interveinal areas and covers the leaves completely. The leaves subsequently shrivel, leading to shedding of leaves from the lower parts of the plant, leaving only a few top leaves intact. This is then followed by an irreversible wilting of the plants. The infected plants did not bear healthy capsules, subsequently leading to reduced yields (Moshkin 1986). In Russia the extent of castor wilt disease incidence was up to 80% (Moshkin 1986). The yield loss depends on the stage at which wilt affects the crop, the loss could be nearly 77% at the stage of flowering, nearly 63% at 90 days, and 39% at the subsequent stages of secondary branch formation as reported by Pushpavathi et al. (1998). Losses of yield were observed in most of the cultivated castor hybrids in Gujarat and it was very high with nearly 85% in north Gujarat areas (Dange 2003). Fusarium wilt appears as patches in field conditions at different stages of castor crop. Lakshminarayana and Raoof (2006) reported a 10-40% reduction in yield, 8–14% reduction in seed weight and 1–2% less in seed oil content.

The disease reaction changes with the variation in environmental temperature. Navas-Cortes et al. (2000) reported that in chickpea genotypes the wilt disease was observed with fungal inoculum of 6–8 × 10^3 CFU/g of soil and within the temperature range of 10–30 °C. Models assessing the combined effect of temperature and inoculum density on wilt-disease reaction have been developed. Ben-Yephet and Shtienberg 1997 reported that the wilt disease in carnation genotypes caused by *F. oxysporum* f. sp. *dianthi* differed with changes in temperature i.e. at 22 °C the genotypes differentiated into highly resistant, moderately resistant, and susceptible diseased genotypes but at 26 °C most of the genotypes showed a susceptible reaction.

Identification of disease-resistant sources is a good management option for abatement castor wilt disease. The resistant sources were obtained by screening several genotypes under sick plot conditions. As wilt is a vascular disease caused by a soil-borne fungus, chemical and physical control methods are not very effective. Due to the systemic nature of the pathogen and the difficulties in controlling the pathogen after the onset of infection, development of castor genotypes with inherent resistance to wilt is the only viable option to manage the disease problem (Dange et al. 2006; Desai et al. 2003). The ability of plants to resist disease onset was tested under sick pot conditions wherein, 88 germplasm lines grown in pots were screened against *F. oxysporum* f.sp. *ricini* by artificial inoculation method (Prasad and Bhatnagar (1978).

Anjani et al. (2004) reported several germplasm accessions and breeding lines that were resistant to vascular wilt of castor. Castor germplasm lines and promising entries were screened under glasshouse conditions by root dip inoculation technique against wilt disease (Raoof and Rao 1996). Shaw et al. (2016) conducted an experiment in the glass house and practiced four different inoculation methods namely seed

soaking in the filtrate, soil drenching with inoculum, root dip inoculation, and sick pot method for screening castor genotypes against wilt disease. The results revealed that the sick pot method was more prominent and accurate to evaluate the disease while ensuring a uniform spread of the pathogen.

Over the years, screening of numerous test lines in sick plots and sick pot conditions has also been practiced in several crops like chickpea, tomato, etc. The use of varietal resistance, seed treatment, and crop rotation are the best practices to manage this disease (Prasad et al. 2019). Screening of resistant genotypes would reduce pathogen spread in the field and therefore contribute to effective integrated disease management (Desai and Dange 2003). A systematic programme on the development of wilt-resistant parents was strengthened using the standard screening procedures and identification of wilt-resistant sources. The development of wilt-resistant genotypes requires the identification of dependable sources of resistance by screening large diverse germplasm collections and understanding the mode of inheritance of resistance. Hence, several different parental and advanced breeding lines of castor were screened under wilt sick plot conditions at Indian Institute of Oilseeds Research—Hyderabad from 2013–2014 to 2017–2018 to identify the sources resistant to wilt disease.

Materials and methods

Screening of castor genotypes against *F. oxysporum* f. sp. *ricini* under sick plot conditions

The trials were conducted to identify castor genotypes that were resistant to fusarium wilt. The field trials were conducted under sick plot conditions maintained at ICAR-Indian Institute of Oilseeds Research (IIOR), Rajendranagar, Hyderabad (17° 250 2700 N & 78° 350 400 E) during the rainy season for five years i.e. 2013–2014, 2014–2015, 2015–2016, 2016–2017 and 2017–2018 under fusarium wilt sick plot conditions. Sowing of entries was conducted in the first fortnight of July every year. The castor parental lines and advanced breeding material were selected based on diverse pedigree and desired agro-morphological features like plant height, bloom (waxiness on plant parts), branching, spike characters and duration from castor breeding programme. A total of 524 castor genotypes were evaluated for resistance to wilt disease. This included 157 genotypes in 2013–2014, 79 genotypes in 2014–2015, 76 genotypes in 2015–2016, 100 genotypes in 2016–2017 and 112 genotypes in 2017–2018. The augmented block design was followed with hundreds of genotypes evaluated in the same field experiment being compared susceptible and resistant checks as control treatments. Each test entry was sown in 6 m long rows with a spacing of 60 cm × 45 cm. The susceptible check (JI-35) and resistant check (48–1) were sown at regular intervals after every five rows of test entries to determine the spread of inoculum uniformly across the sick plot and three replications of each entry (40 plants) were maintained (Raoof 2006; Prasad et al. 2019).

Preparation of inoculum

Fusarium oxysporum f. sp. *ricini,* the causal agent of wilt disease was isolated from the wilt infected root samples of castor on a PDA medium, and the culture was purified by single spore isolation method and maintained at 25 ± 2 °C for seven days. Discs of approximately 5 mm size were cut from seven day old culture of *F. oxysporum* f. sp. *ricini*. Five–ten discs of the pathogen were inoculated on boiled and autoclaved sorghum grains in an autoclave bag for mass multiplication of the pathogen and were incubated for 14 days until the entire bag was filled with fungal mycelium. The sorghum bags were kept at a temperature of 25 ± 2 °C for incubation. The sorghum bags were shaken thoroughly on alternate days to ensure complete, and uniform growth of the culture.

The permanent wilt sick plot at Hyderabad was developed by growing highly susceptible cultivars, Aruna/VP1/JI-35/Kranti, along with in situ incorporation of wilt infected plant debris of susceptible cultivars along with incorporation of pathogen inoculum before sowing to screen for different castor genotypes resistant to wilt disease. The pathogen was mass multiplied on sorghum grain medium and applied to the sick plot during ploughing and again at the near seedling stage of the crop, which was 20 days after sowing. The inoculum load was maintained at 2×10^3 CFU/g of soil (Shaw et al. 2016).

The inoculum load of *F. oxysporum* f. sp. *ricini* in soil was tested before and after sowing and also at the end of the trial. The standard soil dilution method was

followed for the isolation of colonies as per Waksman (1922). Fusarium-specific medium (FSM) (2 g sodium nitrate, 1 g potassium hydrogen phosphate, 0.5 g magnesium sulphate, 0.5 g potassium chloride, 0.01 g ferrous sulphate, 30 g sucrose, 2 g yeast, 20 g agar, 0.05 g penta chloro nitro benzene, 0.025 g malachite green and 1.4 g streptomycin per 1 L distilled water) was used to count the isolated colonies of *F. oxysporum* f. sp. *ricini* present in the soil samples collected from the sick plot. Streptomycin solution (1%) was added to the medium to prevent bacterial contamination. After inoculation, the plates were incubated for 4–7 days at 25 ± 2 °C. The colonies were counted at the 5th day and fungal colonies were spotted on medium easily as they developed surface colonies that spread well at the tested dilutions.

Fungal genomic DNA was extracted using the method described by Lee and Taylor 1990. PCR was performed using the ITS specific primers namely: ITS1 (50-TCC GTA GGT GAA CCT GCG G-30) and ITS4 (50-TCC TCC GCT TAT TGA TAT GC-30) according to Lee and Taylor (1990). PCR products were purified and sequencing was performed by Eurofins MWG Operon (Ebersberg, Germany), using the ITS1 and ITS4 primers. The obtained sequences were used to perform a BLAST search in the NCBI database (https://blast.ncbi.nlm.nih.gov/Blast.cgi) using the blastn function to identify the isolated fungi on the genus level and the sequence of *Fusarium oxysporum* f. sp. *ricini* was submitted to NCBI with accession no. MW063666.

Statistical analysis

The seeds of different parental lines and advanced breeding material to be tested were sown in the wilt sick plot. The recommended dose of fertilizers and level of irrigation were provided and pest (other than wilt) control measures were taken as required. The data on the initial plant stand was noted 20 days after sowing and the number of wilted plants was recorded at 30 days intervals up to 150 days after sowing. The number of wilted plants was recorded at different intervals and at each interval, the newly wilted plants were counted without including the previously infected ones; finally, the wilted plants counted at each interval were cumulated to calculate the wilt incidence of each genotype. Percentage wilt incidence was calculated from the formula [(number of wilted plants/total number of plants observed) × 100]. Based on the wilt incidence (%), the standard scale was followed as given by Mayee and Datar (1986) and Anjani et al. (2014). Castor lines showing 0–10% wilt were recorded as highly resistant, 11 to 20% wilt as resistant, 21–40% as moderately resistant; 41–50% as moderately susceptible; 51–75% as susceptible, and > 75% wilt as highly susceptible. The data of wilt incidence at 150 days after sowing were investigated by the analysis of variance (ANOVA) and CD (0.05) values were calculated for each year. INDOSTAT statistical software, Indostat services, Hyderabad, India (www.indostat.org) was used for statistical analysis of the data.

Results

Fusarium oxysporum f. sp. *ricini* was isolated from the wilt infected castor root samples, and the isolated cultures were identified based on colony characteristics with the help of monographs of Fusarium and illustrated books (Booth 1971; Summerell and Leslie 2006). The fungal characteristics include white fluffy mycelia when grown on potato dextrose agar medium (PDA). The mycelia have been found to turn pinkish when incubated under fluorescent light. The fungus produces both macro conidia and micro conidia. The microconidia are hyaline, round to ovoid, and can be composed of one or two cells. Higher numbers of micro conidia are observed when compared to macro conidia. The single-celled microconidia measure 6.31 × 3.66 µ in size while the double-celled microconidia measure 15.29 × 3.76 µ. Macroconidia are septate with, 2–6 septa (mostly 3), can be straight, spindle or sickle shaped and measure 17.50 − 70.00 × 3.50 − 5. 25 µ. Both terminal and intercalary chlamydospores appear that measure 8.7 × 4.44 µ.

The inoculum load in the sick plot was tested every year and in a few years, the inoculum load was observed to reach $1.0 - 1.5 \times 10^3$ CFU/g before sowing in the sick plots. However by adding sorghum grown Fusarium inoculum and wilted susceptible plants to the plots the inoculum load of 2×10^3 CFU/g soil could be constantly maintained in the sick plot during all the years throughout the duration of the experiment.

The different genotypes of castor that included parental lines and advanced breeding material were screened against Fusarium wilt disease under wilt sick plot field conditions. The sowing of different genotypes was carried out in July every year. The plants were observed for wilt symptoms at regular intervals from 20 to 150 days after sowing. The symptoms of wilting such as yellowing, thickening, necrosis and wilting of the leaves, drooping of the plant, creation of a black streak from the collar to the growth point, ultimately leading to plant mortality were observed.

Among the 157 genotypes evaluated against wilt disease in 2013–2014, 25 castor genotypes showed susceptible reaction with > 50% wilt incidence. This included seven genotypes Kh12-83–3, Kh12-321–1, Kh12-321–2, Kh12-1369–2, Kh12-1419–2, Kh12-1419–3, Kh12-1460–1, PHT-2013–5 and PHT-11–13-55 that showed > 75% wilt incidence with highly susceptible reaction and 16 genotypes showed > 50% wilt incidence with susceptible reaction (Table 1). Fifty genotypes showed highly resistant reaction with 0–10% wilt incidence while 44 genotypes showed resistant reaction with 11–20% wilt incidence (Table 2). Thirty-one genotypes showed 21–40% wilt incidence with moderately resistant reaction. The susceptible check JI-35 showed 96.4% wilt incidence and the resistant check 48–1 showed 0% wilt incidence. The weather parameters like temperature ranged from 22 °C (min.) to 32.2 °C (max.) in July month and 11 °C (min.) to 28.1 °C (max.) in December month. Relative humidity ranged from 69.6% (I) to 92.1% (II) in July and 51% (I) to 90% (II) in December during the cropping period.

Wilt incidence was not recorded in DCS-108, while a < 10% incidence was observed in DPC-17, and DPC-23. The disease progressed gradually in DCS-86, DCS-107, and DPC-25 but it was < 20% by the end of the season in 2013–2014 (Fig. 1). At 30 days after sowing the wilt incidence was low in all entries, but by 90 days after sowing the disease progressed. The wilt incidence did not increase after 120 days after sowing in most of the promising lines except DPC-25 and M-571 in which disease progression was observed up to 150 days after sowing.

Among the 79 genotypes evaluated against wilt disease in 2014–2015, 17 castor genotypes showed susceptible reaction with > 50% wilt incidence and among them nine castor genotypes PMC-45, PMC-46, PMC-53, PMC-54, PMC-56, PMC-57, PMC-59, PMC-61 and, DCS-113 showed > 75% wilt incidence. Fifteen castor parental lines showed highly resistant reaction with 0–10% wilt incidence whereas 20 genotypes showed resistant reaction with 11–20% wilt incidence (Table 2). Moderately resistant reaction was recorded in 21 castor parental lines. Wilt incidence of 96.8% was recorded in JI-35, the susceptible check, and 5.3% wilt incidence was recorded in 48–1, resistant check. During cropping period the temperature was 23.2 °C (min.) to 35.9 °C (max.) with relative humidity from 64.1% (I) to 83.1% (II) in July and temperature was of 12 °C (min.) to 30.6 °C (max.) with relative humidity of 47% (I) to 89% (II) in December.

Wilt incidence was not recorded in DCS-86 and, DCS-118, while it was < 10% in DCS-108, DCS-107, DPC-21, DPC-23, M-571, PMC-11, PMC-14, and, PMC-55 (Fig. 2). The wilt disease did not increase in all the promising parental, advanced breeding lines upto 120 days after sowing. It increased gradually in advanced breeding lines PMC-60, and PMC-24.

Among 76 genotypes evaluated against wilt disease six castor genotypes showed susceptible reaction

Table 1 Wilt incidence and severity in the different genotypes of castor screened in wilt sick plot from 2013–2014 to 2017–2018

Year	Total cultivars tested	Highly resistant (0–10% wilt)	Resistant (11–20% wilt)	Moderately resistant (21–40% wilt)	Moderately susceptible (41–50% wilt)	Susceptible to highly susceptible (> 50% wilt)	Analysis of variance CD ($P = 0.05$)
2013–2014	157	50	44	31	7	25	18.7
2014–2015	79	15	20	21	6	17	23.6
2015–2016	76	25	24	13	8	6	19.0
2016–2017	100	15	17	23	11	34	23.3
2017–2018	112	17	18	37	13	27	11.8
Total	524	122	123	125	45	109	

Treatments found significant at 1% and 5% level

Table 2 Castor cultivars that showed resistant and highly resistant reaction to wilt disease (2013–2014 to 2017–2018)

Year	Highly resistant (0–10%)	Resistant (11–20%)
2013–2014	Kh12-86–2, Kh 12–91-2, Kh 12–317-2, Kh 12–367-1, Kh 12–1422-1, Kh 12–1498-1, Kh 12–1522-2, Kh 12–1555-1, Kh 12–1841-1, DCS-81, DCS-89, DCS 94, DCS-108, DCS 120, DPC-17, DPC-23, DPC-26, PHT-2013–10, PHT-2013–11, PHT-2013–13, PHT-2013–14,PHT-2013–15, PHT-2013–22, PHT-2013–23, PHT-2013–26, PHT-2013–27, PHT-2013–32, PHT-2013–33,PHT-2013–34, PHT-2013–38, PHT-2013–39, PHT-2013–41, PHT-2013–46, PHT-2013–51, PHT-2013–54, PHT-11–13-56, PHT-11–13-60, PHT-11–13-62, PHT-2013–63, PHT-2013–66, PHT-2013–68, PHT-2013–71, PHT-11–13-72, PHT-11–13-73, PHT-11–13-77, PHT-11–13-78, PHT-11–13-79,PHT-11–13-80, PHT-11–13-81, PHT-13–2	Kh12-77–2, Kh 12–91-3, Kh 12–98-2, Kh 12–111-2, Kh 12–130-3, Kh 12–320-1, Kh 12–339-2, Kh 12–367-4, Kh 12–1373-1, Kh 12–1422-2, DCS-78, DCS 86, DCS 64, DCS-102, DCS 104, DCS 105, DCS 106, DCS 107, DCS 110, DCS 118, M-571, M-574, DPC-24, DPC-25, PHT-2013–2, PHT-2013–3, PHT-2013–8, PHT-2013–9, PHT-2013–18, PHT-2013–21, PHT-2013–29, PHT-2013–30, PHT-2013–36, PHT-2013–40, PHT-2013–42, PHT-2013–43, PHT-2013–44, PHT-2013–47, PHT-2013–48, PHT-2013–49, PHT-2013–61, PHT-11–13-64, PHT-11–13-67, PHT-2013–70
JI-35 (Susceptible check)-96.4%; 48–1 (Resistant check)-0.0%		
2014–2015	PMC-11, PMC-14, PMC-39, PMC-40, PMC-55, DCS-86, DCS-107, DCS-108, DCS-112, DCS-118, DCS-119, Gandhi, DPC-23, DPC-21, M 571	PMC-6, PMC-9, PMC-15, PMC-16, PMC-17, PMC-18, PMC-19, PMC-21, PMC-24, PMC-25, PMC-33, PMC-34, PMC-35, PMC-36, PMC-37, PMC-38, PMC-50, PMC-51, PMC-60, DCS-105
JI-35 (Susceptible check)-96.8%; 48–1 (Resistant check)-5.3%		
2015–2016	PVT-1–12-2, PVT-1–12-3, PVT-1–12-98, PVT-1–12-161, PVT-1–12-167, PVT-11–3, PVT-11–11, PVT-11–17, PVT-11–18, PVT-11–19, PVT-11–21, PVT-11–26, PVT-11–59, PVT-11–61, DPC-20, DPC-21, DPC-25, DPC-29, PMC-65, PMC-67, PMC-78, PMC-10, PMC-13, PMC-66, PMC -77	PVT-1–14-189, PVT-1–12-4, PVT-1–12-6, PVT-1–12-7, PVT-1–12-8, PVT-1–12-9, PVT-1–12-72, PVT-1–12-88, PVT-1–12-90, PVT-1–12-103, PVT-1–12-104, PVT-1–12-160, PVT-11–2, DPC-17, DPC-18, DPC-19, DPC-23, DPC-24, DPC-28, PMC-22, PMC-32, PMC-43, PMC-47, PMC-74
JI-35 (Susceptible check)-95.8%; 48–1 (Resistant check)-2.5%		
2016–2017	PMC-38, PMC-65, PMC-67, PMC-79, DPC-17, DPC-18, DPC-20, DPC-21, DPC-25, DPC-28, PVT-11–3, PVT-11–5, PVT-11–17,PVT-11–18, PVT-11–26	PMC-9, PMC-11, PMC-14, PMC-15, PMC-16, PMC-17, PMC-24, PMC-55, PMC-60, PMC-66, PMC-78, PMC-81, DPC-23, DPC-24, PVT-11–19, PVT-11–21, PVT-11–70
JI-35 (Susceptible check)-100.0%; 48–1 (Resistant check)-0.0%		
2017–2018	PVT-11–3, PVT-11–11, PVT-11–18, PVT-11–59, PVT-12–2, K 16–1520-1, K 16–2018-1, K 16–2048-1, K 16–2058-1, K 16–2164-1, K 16–2193-3, K 16–2205-1, K 16–2206-1, RG-1963, RG 566, P3-207, JI-244	Kh-4–3-44, Kh-3–5-21, Kh-3–5-49, DCS-107, PVT-11–2, PVT-11–17, PVT-11–21, PVT-11–26, PVT-12–3, PVT-12–4, PVT-12–6, PVT-12–72, K 16–1513-1, K 16–2165-2, K 16–2211-1, PMC 14, P2-131, P3-283
JI-35 (Susceptible check)-97.9%; 48–1 (Resistant check)-5.0%		

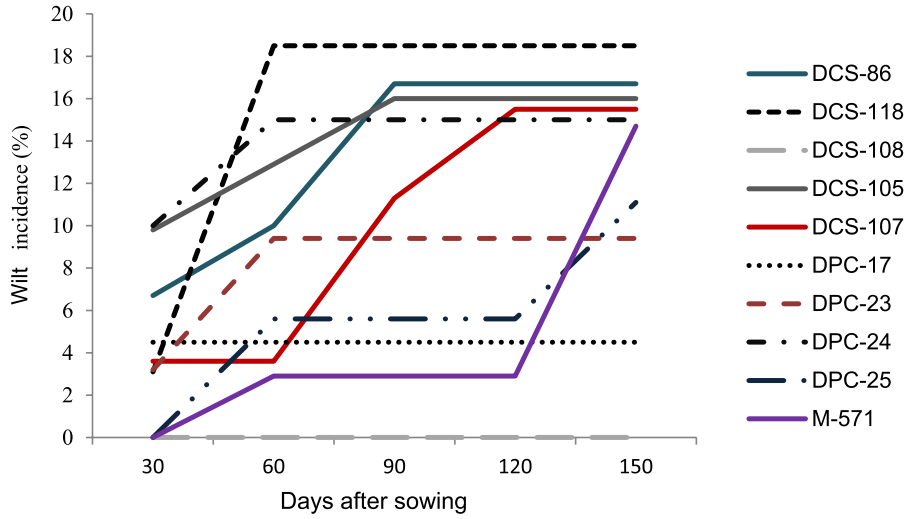

Fig. 1 Progress of wilt disease in promising genotypes of castor during 2013–2014 depicted as percentage wilt incidence recorded periodically from 30 to 150 days after sowing

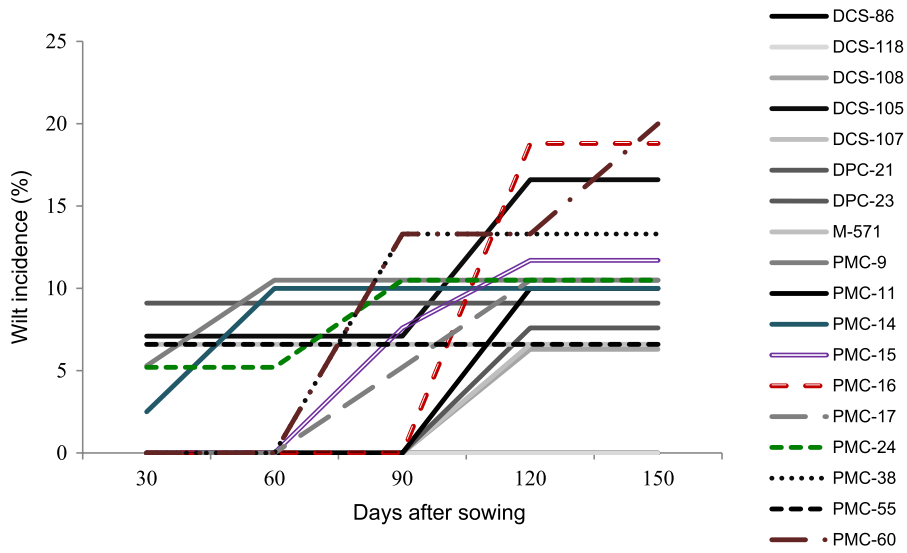

Fig. 2 Progress of wilt disease in promising genotypes of castor during 2014–2015 depicted as percentage wilt incidence recorded periodically from 30 to 150 days after sowing

with > 50% wilt incidence and castor genotypes GP-783 and PMC-80 showed > 75% wilt incidence in 2015–2016 (Table 2). Twenty five castor genotypes showed highly resistant reaction with 0–10% wilt incidence whereas 24 genotypes showed resistant reaction with 11–20% wilt incidence. The wilt incidence was < 10% in DPC-25, PVT-11–8 and PVT-11–17. Wilt disease was not observed in DPC-17, DPC-21, and PVT-11–3. The disease progressed in DPC-18 and PVT-12–6 from 90 days onwards (Fig. 3), while it did not progress much after 120 days from sowing. The weather parameters like temperature ranged from 23.4 °C (min.) to 34.4 °C (max.) and relative humidity from 56.9% (I) to 82.1% (II) during July while temperature of 15.7 °C (min.) and 30.4 °C (max.) with relative humidity ranging from 47% (I) to 92.9% (II) during December in cropping period.

During the 2016–2017 experimentation period, among the 100 genotypes evaluated against wilt disease, 34 castor genotypes showed susceptible reaction with > 50% wilt incidence and of them, 16 castor genotypes showed > 75% wilt incidence. Fifteen parental lines recorded highly resistant reaction with 0–10% wilt incidence whereas 17 genotypes showed resistant reaction with < 20% wilt incidence (Table 1, 2). Moderately resistant reaction with 21–40% wilt incidence was observed in 23 genotypes. The wilt disease was not observed in DPC-25,

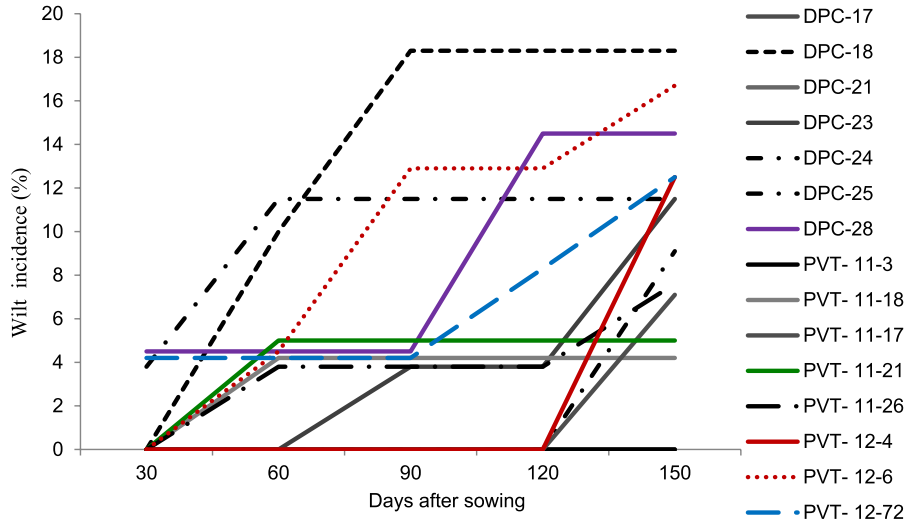

Fig. 3 Progress of wilt disease in promising genotypes of castor during 2015–2016 depicted as percentage wilt incidence recorded periodically from 30 to 150 days after sowing

PVT-11–3 and PVT-11–18 in 2016–2017. The disease progressed in PMC-15, PMC-17, PMC-60, PVT-11–21 and, PVT-11–26, from 90 days onwards and the wilt incidence was < 10% in DPC-17, DPC-18, DPC-38, PVT-11–17 and, PVT-11–26 (Fig. 4). During the cropping season, the temperature ranged from 24.0 °C (minimum) to 32.2 °C (maximum) in July and 14.0 °C (minimum) to 29.4 °C (maximum) in December while relative humidity ranged from 60.9 (I) to 75.0% (II) and 36.7 (I) to 75.0% (II) respectively.

In 2017–2018, 112 genotypes were evaluated against wilt disease, of these, 27 castor genotypes showed susceptible reaction with > 50% wilt incidence (Table 1). Seventeen genotypes showed highly resistant reaction with 0–10% wilt incidence whereas 18 genotypes showed resistant reaction with < 20% wilt incidence (Table 2). Wilt incidence of 21–40% was observed in 37 genotypes. Low wilt incidence of < 10% was observed in PVT-11–3 and, PVT-11–18 and wilt incidence was low at 30 days after sowing in all entries. The disease did not progress significantly

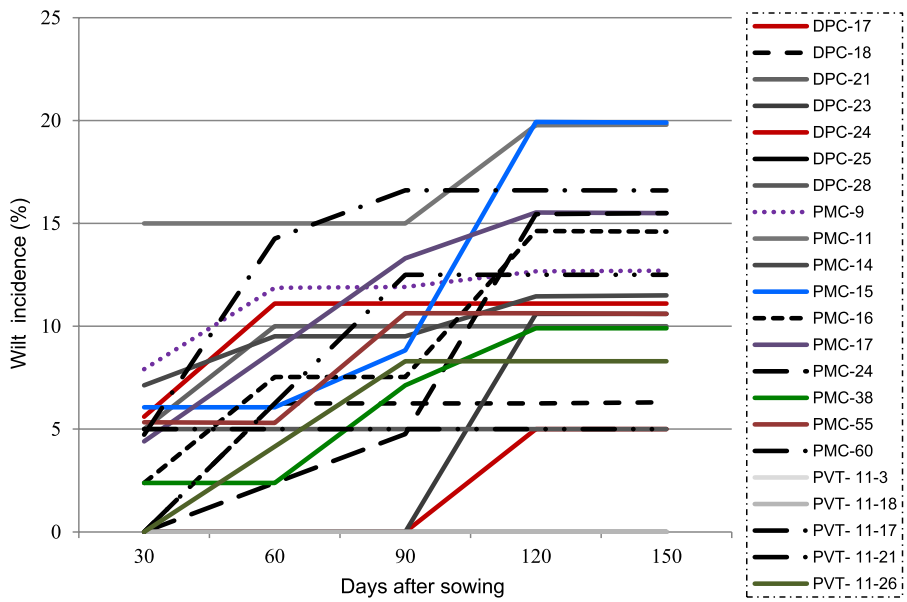

Fig. 4 Progress of wilt disease in promising genotypes of castor during 2016–2017 depicted as percentage wilt incidence recorded periodically from 30 to 150 days after sowing

in most of the entries from 120 days after sowing except in DCS-107, PVT-11–17, and PVT-12–72 (Fig. 5). Weather parameters like temperature ranged from 21.5 °C (min.) to 34.5 °C (max.) with relative humidity ranged from 60.9% (I) to 75.0% (II) in July. During December, the temperature varied from 13.5 °C (min.) to 28.5 °C (max.) while relative humidity was between 67.0% (I) and 92.0% (II) in the cropping period of 2017–2018.

A few of the genotypes were tested for two to three years for confirmation of resistance to wilt disease under sick plot conditions. The promising wilt resistant genotypes of castor that showed wilt incidence within 0–20% of disease twice and thrice from 2013 to 2018 are given in Table 3. The castor genotypes i.e. DCS-86, DCS-118, DCS-108, DCS-105, DCS-107, DPC-17, DPC-18, DPC-21, DPC-23, DPC-24, DPC-25, M-571, DPC-28, PMC-9, PMC-11, PMC-14, PMC-15, PMC-16, PMC-17, PMC-24, PMC-38, PMC-55, PMC-60, PVT- 11–3, PVT-11–18, PVT-11–17, PVT-11–21, PVT-12–4, PVT-12–6, PVT-12–72 and PVT-11–26 showed consistent resistant reaction during consecutive years with < 20% wilt incidence (Table 3).

Discussion

Selection of resistant sources by thorough screening of parental lines is common practice for infusing genetic diversity in plant breeding programs. Breeding for wilt resistance is the most cost-effective and eco-friendly disease management method. In the present study, castor genotypes comprising of parental lines and advanced breeding material were screened against wilt disease under sick plot conditions over several years. The wilt sick plot was maintained with a Fusarium inoculum load of 2×10^3 CFU/g soil. The wilt symptoms started appearing as yellowing, necrotic spots in the leaves, and necrosis of seedlings 25 days after sowing in susceptible entries under field conditions. The highly resistant lines did not show any disease symptoms till the end of the experiment. There could be different mechanisms of resistance operating in lines that exhibited varying levels of resistance. The susceptible check, JI-35 (planted across the field) died of wilt disease within 40–50 days of sowing. The resistant check, 48–1 exhibited normal growth with a few wilt symptoms till end of the experiment (150 DAS).

Wilt resistance was tested in approximately 524 castor genotypes comprising of parental lines and advanced breeding material under sick plot conditions for 5 years from 2013–2014 to 2017–2018. Among them, 109 lines showed susceptible to highly susceptible reaction with > 50% wilt incidence and 45 entries recorded moderately susceptible reaction with 41–50% wilt incidence. The wilt incidence of 21–40% was observed in 125 genotypes. 123 genotypes recorded resistant reaction with 11–20% wilt incidence while 122 parental lines showed < 10% wilt incidence with highly resistant reaction over five years of testing. Some of the entries were repeatedly tested for 2–3 consecutive years for confirmation of resistance.

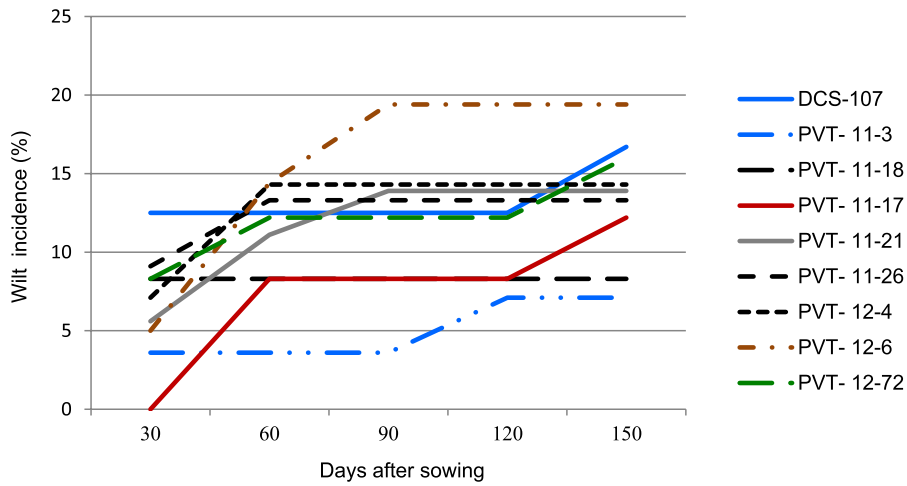

Fig. 5 Progress of wilt disease in promising genotypes of castor during 2017–2018 depicted as percentage wilt incidence recorded periodically from 30–150 days after sowing

Table 3 Promising castor genotypes that showed resistant reaction (< 20% incidence) from 2013–2014 to 2017–2018

Genotype	Year of testing	Wilt incidence (%)	Genotype	Year of testing	Wilt incidence (%)	Genotype	Year of testing	Wilt incidence (%)
DCS-86	2013–2014	16.7	M-571	2013–2014	14.7	PVT-11–18	2015–2016	4.2
	2014–2015	0		2014–2015	6.6		2016–2017	0
DCS-118	2013–2014	18.5	DPC-28	2015–2016	14.5		2017–2018	8.3
	2014–2015	0		2016–2017	5	PVT-11–17	2015–2016	7.1
DCS-108	2013–2014	0	PMC-9	2014–2015	10.5		2016–2017	5
	2014–2015	6.3		2016–2017	12.7		2017–2018	12.2
DCS-105	2013–2014	16	PMC-11	2014–2015	10	PVT-11–21	2015–2016	5
	2014–2015	16.6		2016–2017	19.8		2016–2017	12.5
DCS-107	2013–2014	15.5	PMC-14	2014–2015	10		2017–2018	13.9
	2014–2015	6.6		2016–2017	11.5	PVT-11–26	2015–2016	7.6
	2017–2018	16.7		2017–2018	16.7		2016–2017	8.3
DPC-17	2013–2014	4.5	PMC-15	2014–2015	11.7		2017–2018	13.3
	2015–2016	0		2016–2017	19.9	PVT-12–4	2015–2016	12.5
	2016–2017	5	PMC-16	2014–2015	18.8		2017–2018	14.3
DPC-18	2015–2016	18.3		2016–2017	14.6	PVT-12–6	2015–2016	16.7
	2016–2017	6.3	PMC-17	2014–2015	10.5		2017–2018	19.4
DPC-21	2014–2015	9.1		2016–2017	15.5	PVT-12–72	2015–2016	12.5
	2015–2016	0	PMC-24	2014–2015	10.5		2017–2018	16
	2016–2017	10		2016–2017	15.5	JI-35(Sus. Ch.)	2013–2014	96.4
DPC-23	2013–2014	9.4	PMC-38	2014–2015	13.3		2014–2015	96.8
	2014–2015	7.6		2016–2017	9.9		2015–2016	95.8
	2015–2016	11.5	PMC-55	2014–2015	6.6		2016–2017	100
	2016–2017	10.6		2016–2017	10.6		2017–2018	97.9
DPC-24	2013–2014	15	PMC-60	2014–2015	20	48–1(Res. Ch.)	2013–2014	0
	2015–2016	11.5		2016–2017	16.6		2014–2015	5.3
	2016–2017	11.1	PVT-11–3	2015–2016	0		2015–2016	2.5
DPC-25	2013–2014	11.1		2016–2017	0		2016–2017	0
	2015–2016	9.1		2017–2018	7.1		2017–2018	5
	2016–2017	0						

Res. Ch. resistant check, *Sus. Ch.* susceptible check

The mechanisms of resistance differ between genotypes. Each cultivar has separate genes responding to pathogen interaction. The pathogen adapts to cope with the host diversity and fluctuating weather and hence resistant genotypes could become susceptible (Prasad et al. 2008). The research on evaluating and identifying resistance sources against Fusarium wilt disease through hybridization techniques has been a continuous process. However, field resistance among available parental lines needs to be assessed for further execution in resistance breeding programs. Findings also showed that as the pathogen switches its strategy of infection, the host tailors its defence strategy to meet the changing situation indicating that a resistant host makes choices that are different from those made by a susceptible host during infection. Most importantly, this defence response was more prompt in the resistant host compared to the susceptible host. The resistance nature of the genotypes was revealed using the same genotypes that showed less wilt incidence (0–20%) for 2–3 years during 5 years.

Research about the activity of defence enzymes viz, superoxide dismutase (SOD), peroxidase (POX), catalase (CAT), ascorbate peroxidase (APX), glutathione reductase (GR), and β-1,3-glucanase revealed that the level of defence-related enzyme

activity of SOD, GR and β-1,3-glucanase was higher in resistant castor cultivars like 48–1. In contrast, the activity of the ascorbate peroxidase enzyme was higher in the susceptible castor cultivar JI-35. The release of these enzymes is related to the expression of resistant mechanisms that restricted the browning of xylem vessels in the resistant cultivar (Bharathi et al. 2019). The restricted growth of mycelium, absence of browning in xylem vessels and increase in activity of defence-related enzymes in resistant cultivar indicates the resistance mechanism in the plant.

The high frequency of resistant lines indicates that genetic resistance for fusarium is widespread in castor genotypes. The highly resistant lines identified here are genetically very diverse and could be exploited as good sources of resistance in the castor breeding programmes. In field conditions, plants grow faster and tolerate a certain level of infection (Eynck et al. 2009). The expression of resistance in the field could also depend on the concentration or rate of production of constitutive antifungal components by the root. Stevenson et al. (1995) found that the kind of plant root exudates (which have antifungal activity) and the rate of exudation differ between susceptible and resistant plants. Among different genotypes tested, some showed high immune reaction with 0% wilt incidence, and others with highly resistant reaction showed wilt incidence with 0–10%.

Cultivating the resistant castor cultivar ensures protection against wilt disease, and saves time, energy, and money spent on other measures of management of disease. Breeding of resistant genotypes is one of the important options to control wilt disease in castor. Among 524 genotypes tested from 2013–2014 to 2017–2018, these 31 genotypes i.e. DCS-86, DCS-118, DCS-108, DCS-105, DCS-107, DPC-17, DPC-18, DPC-21, DPC-23, DPC-24, DPC-25, M-571, DPC-28, PMC-9, PMC-11, PMC-14, PMC-15, PMC-16, PMC-17, PMC-24, PMC-38, PMC-55, PMC-60, PMC-50, PVT- 11–3, PVT-11–18, PVT-11–17, PVT-11–21, PVT-12–4, PVT-12–6, PVT-12–72 and PVT-11–26 showed resistant reaction during consecutive years with 0–20% wilt incidence.

Parental lines DCS-86 and DCS-118 genotypes showed 16.7%, 18.5% wilt incidence in 2013–2014 and 0.0% wilt incidence respectively in 2014–2015. DCS-108 showed 0% wilt incidence in 2013–2014 and was highly resistant with 6.3% wilt incidence in 2014–2015. DCS-105 cultivar showed resistant reaction of 16% and 16.6% wilt incidence respectively in 2013–2014 and 2014–2015. DCS-107 cultivar showed resistant reaction of 15.5%, 6.6% and 16.7% wilt incidence respectively during 2013–2014, 2014–2015 and 2017–2018. DPC-21 cultivar showed highly resistant reaction to wilt disease with 9.1, 0.0, 10% wilt incidence in 2014–2015, 2015–2016 and 2016–2017 respectively. Parental line DPC-17 recorded < 10% wilt incidence in all three years of testing with 4.5%, 0%, 5.0% in 2013–2014, 2015–2016 and 2016–2017 respectively. DPC-18, which was tested consecutively in 2015–2016 and 2016–2017, showed wilt incidence of 18.3% and 6.3% in the respective years.

The parental line DPC-23 showed resistant reaction with 9.4%, 7.6%, 11.5%, and 10.6% wilt incidence in 2013–2014, 2014–2015, 2015–2016 and 2016–2017 respectively. The parental lines DPC-24 showed resistant reaction of 15%, 11.5% and 11.1% wilt incidence. Similarly, DPC-25 also showed resistant reaction with 11.1%, 9.1%, 0% wilt incidence during 2013–2014, 2015–2016, and 2016–2017 respectively. The parental lines PMC-9, PMC-11, PMC-14, PMC-15, PMC-16, PMC-17, PMC-24, PMC-38, PMC-55 and PMC-60 showed resistant reaction with less than 20% wilt incidence in both 2014–2015 and 2016–2017. The wilt incidence in M-571 was 14.7% and 6.6% during 2013–2014 and 2014–2015 respectively while 14.5% and 5.0% wilt incidence was observed during consecutive years of 2015–2016 and 2016–2017.

PVT-11–3 showed highly resistant reaction of 0%, 7.1% wilt incidence while PVT-11–18 showed 4.2%, 0%, 8.3% wilt incidence during three years of 2015–2016, 2016–2017, and 2017–2018 respectively. The entries PVT-11–17, PVT-11–21, and PVT-11–26 also recorded less than 20% wilt incidence of resistant reaction in 2015–2016, 2016–2017, and 2017–2018. The advanced breeding material of PVT-12–4, PVT-12–6 and PVT-12–72 recorded wilt incidence of 12.5%, 14.3%; 16.7%, respectively in 2015–16 and 19.4% and 12.5%, 16.0% respectively in 2017–2018. These genotypes can be used as resistant sources in the breeding of wilt-resistant hybrids.

Our results can be compared with the results of Khalid (1993) who evaluated 122 test genotypes against wilt disease in chickpea under field conditions and found that 37 lines showed resistant reaction whereas other test lines exhibited mostly susceptible

reaction. Raoof and Rao 1996 reported that out of 160 castor genotypes screened, 42 genotypes were found to be resistant to wilt disease. Our observations are in line with the findings of Pushpavathi et al. (1998) who screened castor genotypes against wilt and showed that cultivar 48–1 had the least wilt incidence (5%) followed by cultivar DCS-9 (13.5%), whereas cultivar Aruna recorded highest wilt incidence (62.5%).

Castor hybrids of susceptible and tolerant parents tended to show disease incidence similar to that exhibited by the susceptible parent, indicating that the susceptible parents potentially have a greater influence on deciding the wilt reaction (Golakia et al. 2005). Similar work done by Chaudhary et al. (2006) reported that among 414 germplasm accessions of chickpea screened against wilt disease in field conditions, 35 lines showed a resistant reaction, 208 lines exhibited an intermediate reaction, 77 lines were susceptible, and 94 lines were highly susceptible. The resistant lines screened were used as resistant sources to chickpea disease. Anjani et al. (2014) identified stable sources of wilt resistance among the global castor germplasm collections available in India. Thirteen accessions viz., RG-43, RG-111, RG-109, RG-297, RG-1608, RG-1624, RG-2758, RG-2787, RG-2800, RG-2818, RG-2822, RG-3016, and RG-3105 consistently showed resistance reaction at both locations in wilt sick plots over years of testing at IIOR, Hyderabad, Telangana, and S.K. Nagar, Gujarat. Both susceptible and resistant checks also confirmed their respective reactions against wilt in wilt sick plots in all the years under study. Shaw et al. (2016) evaluated the castor lines in field conditions of wilt sick plot and reported that advanced breeding lines AP-10, AP-14, AP-27, AP-54, AP-61, AP-85 and AP-121 showed moderate reactions in both sick pot and field screenings. Priya et al. (2016) identified the potential resistance source for Fusarium wilt in wilt sick plots by screening 200 germplasm accessions, 29 accessions found to be resistant to Fusarium wilt with < 20% wilt incidence and 50 accessions were screened for confirmation of wilt resistance under pot culture conditions out of which 12 accessions revealed resistant reaction. Rajput et al. (2023) reported that 36 castor genotypes were wilt resistant and The NJ tree could divide 36 genotypes into three main clusters. ANOVA revealed 15% and 85% variance among and within subpopulations, respectively.

Three parental lines of castor resistant to wilt disease IPC-21/DPC-21(INGR No.21107), M-571 (INGR No. 21230), and ICS-200 (INGR No. 21157) were identified and registered by plant germplasm registration committee (PGRC) of indian council of agricultural research (ICAR), New Delhi based on the results of screening trials conducted during the study period in 2021. IPC-21 / DPC-21 (INGR No.21107) was a pistillate line, with a green stem, double bloom, spiny capsules, normal plant type with elongated internodes, divergent branching, and flat leaves, resistant to wilt, tolerant to leafhopper, and a good combiner for seed yield and yield components. M-571 (INGR No. 21230) with red stem, triple bloom, spiny capsules, loose spike, dwarf plant type with condensed nodes, cup- shaped leaves were resistant to wilt and leafhopper (Lavanya et al. 2023). Castor parental line ICS-200 (INGR No. 21157), a male line was resistant to leafhopper, wilt disease, and thrips (Anonymous 2022).

PMC-14 showed resistant reaction to wilt for three years in 2014–2015 (10.0%), 2016–2017 (11.4%) and 2017–2018 (16.7%) under sick plot conditions. This has been used as one of the parent ICS-164 (PMC-14) in developing the wilt-resistant hybrid ICH-66. ICS-164, a monoecious inbred was developed through the pedigree method of selection involving a 48–1 × RG-1582 cross. This ICH-66 (SKP-84 × PMC-14) hybrid was released for cultivation in Andhra Pradesh, Telangana, Tamil Nadu, Karnataka, and Odisha states of India under rainfed conditions (Prabakaran et al. 2018). This hybrid is a high-yielding hybrid with a potential of 1574 kg/ha and 3375 kg/ha under rainfed and irrigated conditions, respectively and is resistant to wilt and leaf hopper (2015–2016 to 2017–2018). It matures in 94–97 days, earlier than DCH-519 (105–110 days) and GCH-8 (95–105 days) with 46–49% oil content. In the schema for development of the hybrid, the parental lines, and hybrids were screened against wilt under sick plot conditions and found resistant to wilt disease of castor. This response is mandatory for the release of any castor hybrid. Research on the genetics of wilt resistance indicated that for the development of a wilt resistant castor hybrid, both the parents should be resistant to wilt (Desai et al. 2001; Lavanya et al. 2011).

The parental line DCS-94 recorded 10% wilt incidence under wilt sick plot at IIOR, Hyderabad during 2013–2014 and it has been used as the male

parent for the development of hybrid GNCH-1 at Navsari Agricultural University, Navsari, Gujarat (Bhakta et al. 2018; Anonymous 2015). The hybrid GNCH-1 had established its superiority in south & middle Gujarat under AICRP and multi-locations trials in late Kharif/rabi irrigated conditions. The proposed hybrid GNCH-1 high yielding wilt- resistant hybrid is suitable for the late kharif and rabi season of Gujarat, Rajasthan.

Fusarium wilt caused by *F. oxysporum* f. sp. *ricini* is one of the most devastating diseases of castor and causes heavy losses worldwide. The present study screened castor genotypes against wilt disease under wilt sick plot conditions and identified highly resistant and resistant genotypes that are sources of resistance. The information generated through this experiment can be utilized in horizontal resistance breeding programs for the development of resistant hybrids. Among various management practices against Fusarium wilt, breeding for resistant cultivars is an effective, economic, and eco-friendly method to overcome the problem of wilt against castor and this screening method can be incorporated as an effective method and a component for the management of castor wilt disease.

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Author contributions EB, MSLP performed the field experiments, data collection, analysis, preparation of manuscript; CL, MT supplied seeds for screening and supervised the trials. All authors reviewed the manuscript.

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Data availability The data generated during and/or analysed in the current study are available upon request.

Declarations

Conflict of interest The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Ethical approval This article does not contain any research with human participants or animals performed by any of the authors.

Informed consent Informed consent was obtained from all individual participants included in the study.

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