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Exploitable variation for fusarium wilt resistance in advanced safflower lines

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Abstract

Fusarium wilt caused by *Fusarium oxysporum* f. sp. *carthami* is a significant constraint to safflower (*Carthamus tinctorius* L.) production in India. Identifying resistant sources is the most economical and sustainable approach for managing soil and seed-borne diseases. The present investigation was conducted during Rabi 2022-23 at the safflower Fusarium wilt-sick plot of the Agricultural Research Station, Tandur, Telangana, to evaluate safflower breeding lines for wilt resistance. Ninety-one entries, comprising 88 advanced breeding lines and three checks, were screened. Wilt incidence was recorded at 15-day intervals from 30 to 120 days after sowing and genotypes were classified based on standard disease rating scales. Analysis of variance revealed highly significant treatment effects, indicating considerable genetic variability for wilt resistance. Wilt incidence in breeding lines ranged from 0 to 100 per cent. Five genotypes were immune, nine resistant, and eighteen moderately resistant, whereas most genotypes exhibited susceptible or highly susceptible reactions. The susceptible checks recorded high wilt incidence, confirming uniform disease pressure. The immune and resistant entries identified in this study can serve as valuable sources for breeding Fusarium wilt-resistant safflower cultivars.

Keywords: Safflower, fusarium wilt, disease resistance, screening, sick plot

Introduction

Safflower (*Carthamus tinctorius* L.) is a vital rabi oilseed crop cultivated predominantly under rainfed conditions in the semi-arid regions of India. Owing to its adaptability to marginal environments, safflower plays a crucial role in sustaining oilseed production in dryland agriculture. However, its productivity is severely constrained by several biotic stresses, among which Fusarium wilt is the most destructive (Mayee and Dattar, 1986; Kolte, 2014) [7, 6]. *Fusarium oxysporum* f. sp. *carthami* Klisiewicz and Houston, a soil-borne fungal pathogen, causes Fusarium wilt of safflower. The disease affects the crop at all growth stages, resulting in characteristic symptoms such as leaf yellowing, vascular discolouration, wilting, and eventual plant death (Weiss, 1983) [15]. In severe cases, the disease can cause yield losses of up to 80% or more, particularly in poorly drained soils (Sastry and Ramachandram, 2003) [13]. In India, Fusarium wilt is widely distributed and poses a significant production constraint in safflower-growing states, including Telangana, Maharashtra, and Andhra Pradesh. The disease was first reported in India by Singh *et al.* in 1975 [14].

The pathogen is both soil- and seed-borne in nature. It survives in the soil for prolonged periods through the formation of chlamydospores and also persists as mycelium and spores on infected seeds and seed coats. Seed transmission of the pathogen has been reported to range from 10 to 40%, facilitating its spread across seasons and locations (Chakrabarti, 1980) [3]. Continuous cultivation of wilt-susceptible traditional varieties has further aggravated the problem, leading to increased disease incidence and yield losses as high as 93% under severe conditions (Sastry *et al.*, 1993) [13].

Management of Fusarium wilt through chemical means has mainly proven ineffective and economically impractical due to the soil-borne nature and long-term survival of the pathogen (Kolte, 2014) [6]. Therefore, breeding for host plant resistance is considered the most economical, environmentally safe, and sustainable approach for managing the disease (Mayee

and Dattar, 1986; Agrios, 2005) [7, 1]. Although a few germplasm lines and cultivars with partial or complete resistance have been identified globally, the availability of stable resistance sources remains limited. Moreover, the continuous evolution and genetic variability of the pathogen necessitate ongoing efforts to identify new and durable sources of resistance.

In view of the economic importance of safflower and the persistent threat posed by *Fusarium* wilt, the present study was undertaken to identify safflower breeding lines possessing resistance to *Fusarium oxysporum* f. sp. *carthami*, which can be effectively utilized in resistance breeding programmes.

Materials and Methods

The experiment was conducted during *Rabi* 2022-23 at the *Fusarium* wilt-affected plot of the Agricultural Research Station (ARS), Tandur, Telangana, which has a well-established history of severe and uniform wilt incidence, ensuring consistent disease pressure with a pathogen Foc population of 3.5×10^3 cfu/g. A total of 91 genotypes, comprising 88 advanced breeding lines and three checks, were evaluated. The checks included two susceptible checks, NIRA and PBNS-12, and one resistant checks, TSF-1. The trial was laid out in an Augmented Block Design (ABD) with six blocks, following the procedure described by Federer (1956) [5]. The test entries were unreplicated, while the checks were replicated across all blocks. The entries were sown at a spacing of 45×15 cm, and all recommended agronomic practices were followed uniformly, except for plant protection measures against wilt.

Wilt observations were recorded starting from 30 days after sowing (DAS) and subsequently at 15-day intervals, with observations taken at 30, 45, 60, 75, 90, 105, and a final observation at 120 DAS. The per cent incidence of *Fusarium* wilt for each entry was calculated at each observation by recording the number of wilted plants and expressing it as a proportion of the total plant population using the formula: percentage wilt incidence (%) = (number of wilted plants / total plant population) $\times 100$. The final disease reaction of each entry was determined based on the cumulative wilt incidence recorded up to 120 DAS.

The per cent wilt incidence data were subjected to angular (arcsine) transformation prior to statistical analysis to stabilize error variance. Entries were classified into different reaction categories based on the *Fusarium* wilt disease rating scale of the All India Coordinated Research Project (AICRP) on Safflower as immune (0% wilt), highly resistant (<1%), resistant (1-10%), moderately resistant (11-20%), susceptible (21-50%), and highly susceptible (>51%). Analysis of variance was conducted according to the procedures for the Augmented Block Design, with separate evaluations of block and treatment effects before and after block adjustment. Critical difference (CD) values were used for comparison of adjusted treatment means.

Results and Discussion

The present study evaluated safflower breeding material for resistance to *Fusarium* wilt under field conditions, aiming to identify stable and reliable sources of resistance. The analysis of variance showed that block effects were highly significant when treatments were ignored, indicating the presence of environmental variation across the experimental field (Table 1). However, after block adjustment, these effects became non-significant (Table 2). Highly significant treatment effects were observed in both unadjusted (Table 1) and block-adjusted analyses (Table 2), clearly indicating substantial genetic variability among the safflower genotypes for *Fusarium* wilt

incidence. The replicated checks differed significantly and provided a reliable estimate of experimental error, thereby enhancing confidence in the experiment's precision. The significant contrast between checks and test entries further emphasized the clear distinction between susceptible and resistant checks and the evaluated breeding lines.

Wilt incidence among the genotypes ranged from complete absence of symptoms to 100 per cent plant mortality, as presented in Table 3, reflecting a wide spectrum of disease responses and confirming strong and uniform disease pressure in the sick plot. Based on the wilt reaction scale [immune (0%), resistant (1-10%), moderately resistant (11-20%), susceptible (21-50%), and highly susceptible (>51%)], six genotypes were classified as immune, nine as resistant, and eighteen as moderately resistant (Table 3). In contrast, thirty-one genotypes were susceptible and twenty-five were highly susceptible. The predominance of susceptible and highly susceptible reactions highlights the persistent threat posed by *Fusarium* wilt in safflower cultivation and the limited availability of resistance in existing breeding material.

The susceptible checks, NIRA and PBNS-12, recorded mean wilt incidences of 100% and 72.73%, respectively (Table 3), confirming their effectiveness as standard susceptible controls and validating the severity of the disease pressure. The present study clearly demonstrates the existence of variability for *Fusarium* wilt resistance within safflower germplasm. Earlier studies have reported resistant sources, including safflower lines 86-93-36A, 237550, VI-92-4-2, and II-13-2A (Sastry & Chattopadhyay, 2003) [13, 4], and line 96-508-2-90 (Anjani *et al.*, 2005) [2]. Similar differential responses among safflower genotypes have also been documented by Murumkar *et al.* (2013) [9], Reddy *et al.* (2017) [12], and Rajendraprasad *et al.* (2021) [11], indicating consistency with the present findings.

Recent screening studies have demonstrated the potential for identifying new sources of resistance to *Fusarium* wilt in safflower. Moka *et al.*, (2023) [8] reported that the majority of multiparent cross-derived breeding lines exhibited immunity to wilt, while Prabhavathi *et al.*, (2025) [10] identified the elite safflower line DSAF as resistant. The immune and resistant entries identified in the present investigation further strengthen this evidence and highlight the importance of systematic evaluation of breeding material under wilt sick plot conditions for identifying reliable sources of resistance.

The highly significant genotypic differences observed for *Fusarium* wilt incidence clearly indicate the presence of exploitable genetic variability in the evaluated breeding material. Similar conclusions were drawn by Mayee and Dattar (1986) [7] and Chattopadhyay *et al.* (2011) [4], who emphasized host plant resistance as the most practical, economical, and environmentally safe approach for wilt management. The immune and resistant entries identified in this study can serve as valuable donor parents in resistance breeding programmes. In contrast, moderately resistant genotypes may contribute to the development of cultivars with more durable resistance across diverse agro-climatic conditions.

Overall, the elimination of block effects after adjustment, together with the clear differentiation of genotypes based on disease response, highlights the robustness of the experimental design and screening methodology. The strong and consistent reaction of susceptible checks further confirms the uniformity and severity of disease pressure, ensuring that the resistance identified in this study is reliable and meaningful for future safflower improvement programmes.

Table 1: Analysis of variance for Fusarium wilt incidence (%) under Augmented Block Design

Source of variation	df	Sum of squares	Mean squares	F value	Significance
Blocks (ignoring treatments)	5	9,833.49	1,966.70	38.84	***
Treatments (eliminating blocks)	87	54,275.86	623.86	12.32	***
Checks	2	19,942.30	9,971.15	196.92	***
Varieties + checks vs varieties	85	34,333.57	403.92	7.98	***
Error	10	506.36	50.64		
Total	102	64,615.72			

***= Significance at P= 0.001 probability levels.

Table 2: Block-adjusted analysis of variance for Fusarium wilt incidence (%)

Source of variation	df	Sum of squares	Mean squares	F value	Significance
Blocks (eliminating treatments)	5	101.15	20.23	0.4	NS
Treatments (ignoring blocks)	87	64,008.21	735.73	14.53	***
Checks	2	19,942.30	9,971.15	196.92	***
Varieties	84	42,192.28	502.29	9.92	***
Checks vs varieties (C vs V)	1	1,873.64	1,873.64	37	***
Error	10	506.36	50.64		
Total	102	64,615.72			

***= Significance at P= 0.001 probability levels.

Table 4: Reaction of safflower advanced breeding material against Fusarium wilt at ARS, Tandur sick plot during *Rabi* 2022-23

S. No.	Entry	Wilt incidence (%)	Wilt reaction	S.No	Entry	Wilt incidence (%)	Wilt reaction
1	NIRA (SC)	100 (90.01)	HS	47	TSF-841	100 (90.01)	HS
2	PBNS-12 (SC)	72.73 (58.52)	HS	48	TSF-842	96.67 (79.49)	HS
3	TSF-1 (RC)	0 (0.00)	Immune	49	TSF-843	97.67 (81.23)	HS
4	TSF-790	44.00 (41.56)	S	50	TSF-844	91.67 (73.23)	HS
5	TSF-791	36.00 (36.87)	S	51	TSF-845	96.15 (78.70)	HS
6	TSF-792	20.69 (27.06)	S	52	TSF-846	100 (90.01)	HS
7	TSF-793	6.45 (14.72)	R	53	TSF-847	18.18 (25.24)	MR
8	TSF-794	0.00 (0.00)	Immune	54	TSF-848	23.08 (28.71)	S
9	TSF-795	29.17 (32.69)	S	55	TSF-849	90.48 (72.03)	HS
10	TSF-796	26.09 (30.72)	S	56	TSF-850	53.85 (47.21)	HS
11	TSF-798	9.09 (17.55)	R	57	TSF-851	29.63 (32.98)	S
12	TSF-799	29.63 (32.98)	S	58	TSF-852	56.00 (48.45)	HS
13	TSF-801	20.00 (26.57)	MR	59	TSF-853	48.15 (43.94)	S
14	TSF-803	24.00 (29.34)	S	60	TSF-854	64.29 (53.30)	HS
15	TSF-804	20.83 (27.16)	S	61	TSF-855	34.78 (36.14)	S
16	TSF-805	8.00 (16.43)	R	62	TSF-856	0.00 (0.00)	Immune
17	TSF-806	11.11 (19.47)	MR	63	TSF-857	26.47 (30.97)	S
18	TSF-807	19.23 (26.01)	MR	64	TSF-858	21.05 (27.31)	S
19	TSF-808	0.00 (0.00)	Immune	65	TSF-859	33.33 (35.27)	S
20	TSF-809	11.54 (19.86)	MR	66	TSF-860	20.83 (27.16)	S
21	TSF-810	3.03 (10.03)	R	67	TSF-861	17.39 (24.65)	MR
22	TSF-811	25.81 (30.53)	S	68	TSF-862	100 (90.01)	HS
23	TSF-812	11.43 (19.76)	MR	69	TSF-863	20.00 (26.57)	MR
24	TSF-814	16.13 (23.68)	MR	70	TSF-864	33.33 (35.27)	S
25	TSF-816	6.45 (14.72)	R	71	TSF-865	44.44 (41.81)	S
26	TSF-817	15.15 (22.91)	MR	72	TSF-866	83.33 (65.91)	HS
27	TSF-818	23.33 (28.89)	S	73	TSF-867	46.15 (42.80)	S
28	TSF-819	10.34 (18.76)	MR	74	TSF-868	51.85 (46.06)	HS
29	TSF-820	11.11 (19.47)	MR	75	TSF-869	57.69 (49.43)	HS
30	TSF-821	32.35 (34.67)	S	76	TSF-870	43.48 (41.26)	S
31	TSF-822	12.90 (21.05)	MR	77	TSF-871	80.00 (63.44)	HS
32	TSF-823	13.33 (21.42)	MR	78	TSF-872	94.12 (75.97)	HS
33	TSF-824	7.14 (15.50)	R	79	TSF-873	76.47 (60.99)	HS
34	TSF-825	11.54 (19.86)	MR	80	TSF-874	33.33 (35.27)	S
35	TSF-826	12.90 (21.05)	MR	81	TSF-875	70.37 (57.03)	HS
36	TSF-827	9.68 (18.13)	R	82	TSF-876	40.00 (39.23)	S
37	TSF-828	26.67 (31.09)	S	83	TSF-877	35.71 (36.70)	S
38	TSF-829	0.00 (0.00)	Immune	84	TSF-878	57.14 (49.11)	HS
39	TSF-830	34.48 (35.96)	S	85	TSF-879	62.50 (52.24)	HS
40	TSF-831	44.44 (41.81)	S	86	TSF-880	75.00 (60.00)	HS
41	TSF-832	76.00 (60.67)	HS	87	TSF-881	44.44 (41.81)	S
42	TSF-833	100 (90.01)	HS	88	TSF-882	94.74 (76.74)	HS
43	TSF-835	43.75 (41.41)	S	89	TSF-447	0.00 (0.00)	Immune
44	TSF-837	12.12 (20.38)	MR	90	TSF-446	5.60 (13.69)	R
45	TSF-838	10.00 (18.44)	R	91	TSF-443	12.50 (20.71)	MR
46	TSF-840	90.48 (72.03)	HS				

*Figures in Parentheses are angular transformed values

Table 4: Reaction of safflower advanced breeding material against Fusarium wilt at ARS, Tandur sick plot during *Rabi* 2022-23

Disease scale	Wilt incidence (%)	Wilt Reaction	Entries	Number of Entries
0	No wilting symptoms	Immune (I)	TSF-794, TSF-808, TSF-829, TSF-856, TSF-447+ TSF-1(RC)	5
1	<1% plants wilted	Highly Resistant (HR)		0
3	1-10% plants wilted	Resistant (R)	TSF-793, TSF-798, TSF-805, TSF-810, TSF-816, TSF-824, TSF-827, TSF-838, TSF-446	9
5	11-20% plants wilted	Moderately Resistant (MR)	TSF-801, TSF-806, TSF-807, TSF-809, TSF-812, TSF-814, TSF-817, TSF-819, TSF-820, TSF-822, TSF-823, TSF-825, TSF-826, TSF-837, TSF-847, TSF-861, TSF-863, TSF-443	18
7	21-50% plants wilted	Susceptible (S)	TSF-790, TSF-791, TSF-792, TSF-795, TSF-796, TSF-799, TSF-803, TSF-804, TSF-811, TSF-818, TSF-821, TSF-828, TSF-830, TSF-831, TSF-835, TSF-848, TSF-851, TSF-853, TSF-855, TSF-857, TSF-858, TSF-859, TSF-860, TSF-864, TSF-865, TSF-867, TSF-870, TSF-874, TSF-876, TSF-877, TSF-881	31
9	>51% plants wilted	Highly Susceptible (HS)	TSF-832, TSF-833, TSF-840, TSF-841, TSF-842, TSF-843, TSF-844, TSF-845, TSF-846, TSF-849, TSF-850, TSF-852, TSF-854, TSF-862, TSF-866, TSF-868, TSF-869, TSF-871, TSF-872, TSF-873, TSF-875, TSF-878, TSF-879, TSF-880, TSF-882 + NIRA (SC), PBNS-12 (SC)	25
			Total	88

Conclusion

The present study revealed considerable genetic variability among safflower advanced breeding material for resistance to Fusarium wilt. Six entries were identified as immune, while nine and eighteen entries were resistant and moderately resistant, respectively. These promising genotypes can be effectively utilized as donor parents in safflower breeding programmes aimed at developing Fusarium wilt-resistant varieties for the incorporation of resistance into agronomically desirable, high-yielding safflower varieties for rainfed ecosystems.

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