International Journal of Plant & Soil Science

Volume 35, Issue 19, Page 698-710, 2023; Article no.IJPSS.105143
ISSN: 2320-7035

Screening Chickpea Genotypes against Fusarium Wilt Disease under Controlled Conditions

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

Article Information

DOI: 10.9734/IJPSS/2023/v35i193601

Open Peer Review History:
This journal follows the Advanced Open Peer Review policy. Identity of the Reviewers, Editor(s) and additional Reviewers, peer review comments, different versions of the manuscript, comments of the editors, etc are available here: https://www.sdiarticle5.com/review-history/105143

Original Research Article

Received: 18/06/2023
Accepted: 22/08/2023
Published: 25/08/2023

ABSTRACT

Fusarium wilt caused by *Fusarium oxysporum* f. sp. *Ciceris* is one of the economical important vascular root diseases affecting chickpea which can cause up to 90% yield loss during crop growth stages. In the present investigation, 71 chickpea genotypes including two controls *viz.,* JG315

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(highly resistant) and JG 62 (highly susceptible) were screened by artificial inoculation of pathogen causing Fusarium wilt under controlled conditions in poly house using Completely Randomized Design with two replications during Rabi 2022 with intention to identify potentially wilt resistant genotype (s). Disease incidence was evaluated across distinct developmental phases, specifically the seedling and reproductive stages, employing the metric of percent disease incidence. At the seedling stage, out of the 71 entries, 24 genotypes displayed resistance to the disease, 38 genotypes exhibited moderate resistance, five genotypes found to be moderately susceptible, three susceptible, and only one genotype showed high susceptibility. Upon reaching the reproductive stage, the disease reactions changed drastically as only one genotype was found resistant, 14 genotypes moderate resistance, 17 moderately susceptible, 25 susceptible and 14 highly susceptible.

Keywords: Fusarium wilt; chickpea; resistant; susceptible; genotypes; screening.

1. INTRODUCTION

Chickpea, also known as gram, Bengal gram, Egyptian pea, garbanzo or garbanzo bean, is a self-pollinated, annual diploid (2n = 2x = 16) species [1] with a genome size of 738 Mb [2]. Its seeds are super-nutrient foods providing rich content of protein and certain dietary minerals such as calcium, iron and phosphorus [3-4]. It helps to increase soil fertility by biological nitrogen fixation [5-7]. Chickpea is a vital rabi pulse on the Indian subcontinent with a worldwide production of 15.87 million tones, contributing significantly to the global pulse economy. Currently, 15.004 mha of area are used to cultivate chickpea, with a productivity of 1,057.8 kghah^{-1} and a production of 15.87 mt per year worldwide. As estimated 73.78 % (10.943mha) of the world's total chickpea area and 73.45% (11.91m tones) come from India [8].

Numerous biotic and abiotic factors contribute to the reduced productivity of chickpea [9-18]. A comprehensive survey conducted in 1995 across 55 countries revealed the presence of 172 pathogens causing various diseases in chickpea. These included 67 fungi, 3 bacteria, 22 viruses and phytoplasma, and 80 nematodes [19]. Among these, *Fusarium oxysporum* f. sp. *ciceris,* the causal agent of chickpea wilt, stands out as a significant concern for legume pathologists and breeders because of its detrimental impact on chickpea production [10]. The pathogen is known to persist in the soil for extended periods of up to six years, even in the absence of its host, making it both seed and soil-borne [20]. The primary mode of infection occurs through chlamydospores or mycelia. Interestingly, the fungus can thrive in the roots and stem, even in seemingly healthy plants growing alongside diseased ones that harbor a substantial amount of the pathogen.

Continuous and exclusive reliance on systemic fungicides for disease control has proven ineffective in achieving complete eradication of the wilt disease from infected areas, even with the development of wilt-resistant pathotypes [21,10]. To address this limitation, the development of resistant chickpea cultivars has emerged as a sustainable alternative approach for disease management [15]. Consequently, the current focus lies on creating wilt-resistant cultivars, conserving genetic diversity, and screening genotypes against specific pathotypes, which are crucial steps towards sustainable farming practices [22].The substantial dependence on intensive fungicide usage as a major agricultural management practice has confirmed inadequacy in reducing the severity of diseases [23-32], including *Fusarium* wilt [10, 20]. Consequently, exploring host plant resistance has been pursued in the past as an economically viable management strategy for this disease [33]. However, widespread deployment of resistant varieties has been hindered by undesirable agronomic traits associated with wild donor parents of chickpea, as well as the high degree of pathogenic variability observed among the population of *Fusarium oxysporum* f. sp. *ciceris* [34].

By focusing on genetic resistance, breeders aim to develop cultivars that can better withstand Fusarium wilt and reduce the reliance on chemical interventions. Sustainable management practices, coupled with the deployment of resistant varieties, hold promise for achieving effective disease control and enhancing chickpea productivity in the long-term [35,10]. Considering these challenges, the present study was conducted to identify wilt disease-resistant genotype (s) of chickpea under controlled polyhouse condition.

2. MATERIALS AND METHODS

2.1 Experimental Materials

The experimental material consists of 71 chickpea genotypes including two checks JG315 (highly resistant) and JG 62 (highly susceptible) acquired from RAK College of Agriculture Sehore, RVSKVV, Gwalior, M.P., India and College of Agriculture, JNKVV, Jabalpur, M.P., India. These genotypes were screened for host plant resistance against Fusarium wilt in pot in poly house situated at Biotechnology Centre, RVSKVV, Gwalior, M.P., India during Rabi 2022. Each genotypes contains 10 plants in each pot. Completely Randomized Design with two replications was adopted to analyze data.

2.2 Isolation, Purification and Identification of *Fusarium oxysporum* f. sp. *Ciceris*

2.2.1 Isolation of pathogen

The pathogen *Fusarium oxysporum* f. sp. *Ciceris* was isolated from infected chickpea plants by tissue segment method [36]. Plants exhibiting wilt symptoms were collected from the field-grown plants and brought to the laboratory. The diseased samples were prudently placed in labeled polythene bags and subjected to microscopic examination and tissue isolation. Upon arrival at the laboratory, the samples were washed with running tap water to remove soil particles. Subsequently, small tissue bits, approximately 5 mm in size, were excised from the root portions showing characteristic diseased symptoms, such as browning of vascular tissue, ensuring both healthy and diseased portions were included. To prevent contamination, the tissue bits were surface sterilized using 1% sodium hypochlorite solution for 40-60 seconds and rinsed twice with sterilized double distilled water to remove any traces of sodium hypochlorite. These surface sterilized tissue pieces were then placed on sterilized tissue paper and allowed to air dry for two minutes. Afterward, four tissue bits were transferred onto petri plates containing Potato Dextrose Agar (PDA) in a sterile environment. The plates were incubated at a controlled temperature of 26±2°C for 3 to 4 days until early fungal mycelial growth became visible.

2.2.2 Purification and identification of wilt pathogen

Pure culture was identified as *Fusarium oxysporum* f. sp. Ciceri based on morphological characters as reported by Booth [37-38]. A spore suspension of the isolated pathogen, *Fusarium oxysporum* f. sp. Ciceris, was meticulously prepared by dissolving spores in sterile distilled water. One milliliter of the spore suspension was evenly spread across two percent agar plates and allowing excess suspension to drain off. Subsequently, the plates were placed in an incubator at a temperature of 28±2°C, and under microscopic observation, the germination of spores was tracked. The emergence of hyphae from a single spore was carefully identified and marked on the reverse side of the Petri plates using a marker. For further multiplication, the tip of the hypha was excised and then transferred onto Potato Dextrose Agar (PDA) plates. These plates were incubated at a temperature of 28±2°C for a span of 10 days. Following incubation, the resulting pure culture of the fungus was transferred to slants. To ascertain the identity of the purified isolates of Fusarium, a comprehensive assessment of their cultural and morphological traits was conducted. Factors such as colony color, mycelial growth, pigmentation, and sporulation were examined in accordance with the guidelines provided in Booth's monographs on Fusarium [39]. Detailed observation of conidia morphology was carried out utilizing low-power magnification (40X) on a stereo binocular microscope, with all pertinent data accurately documented.

Ultimately, based on the distinctive combination of cultural and morphological characteristics delineated above, the isolates of Fusarium were conclusively identified and classified. To confirm its pathogenicity, disease development was demonstrated by inoculating susceptible plants with the isolated pathogen. For long-term preservation, the pathogen was sub-cultured monthly and stored at 4°C in a refrigerator.

2.2.3 Screening of genotypes under controlled conditions

For screening, plastic pots filled with sterilized (autoclaved) soil were used under controlled poly house conditions (Fig. 1). To maintain control and ensure reliability of the results, a set of highly resistant JG315 and susceptible genotypes JG62 were included in the experiment and repeated after every five entries. For inoculation, spore suspension was prepared from 15- days-old culture of *F. oxyporum* f. sp. *ciceri* multiplied on Potato Dextrose (PD) using sterile distilled water and then strained through muslin cloth. The spore concentration was adjusted to $1x10^6$ conidia ml^-

[1] distilled water using hemocytometer. The sterilized (autoclaved) soil was inoculated with *F. oxyporum* f. sp. *ciceri* spore suspension before sowing the chickpea seeds. Ten seeds of each genotype were sterilized using 1% sodium hypochlorite solution for two minutes and then washed with double distilled water before being sown in individual pots. The plants were sprayed with freshly prepared spore suspension using an atomizer [40].

2.3 Data Collection

The data on disease incidence was recorded at 30 (seedling) and 45 (reproductive) days of sowing. Data at seedling stage was recorded when killing of susceptible check had occurred and second stage data at the initiation of physiological activity.

2.4 Disease Assessment

To quantify the disease incidence, the percentage of wilted plants was calculated using the following formula as described by Shanmugam et al. [41].

$$Disease\ incidence\ (\%) = \frac{Numbers\ of\ plants\ exhibiting\ wilt\ symptoms}{Total\ numbers\ of\ plants\ evaluated} \times 100$$

Table 1. Disease categorization rating scale (1-9) against fusarium wilt [30]

Grade	% wilt incidence	Disease reaction
1	0-10	Resistant(R)
3	11-20	Moderately Resistant (MR)
5	21-30	Moderately Susceptible (MS)
7	31-50	Susceptible(S)
9	>50	Highly Susceptible (HS)

Based on the disease incidence, genotypes were categorized as resistant, moderately resistant, moderately susceptible, susceptible and highly susceptible.

2.5 Statistical Analysis

Test entries were arranged in a complete block design with two replications. The variances (ó), averages and standard deviation (SD) of various repetitions were calculated and analyzed by the software OPSTAT [42].

3. RESULTS AND DISCUSSION

3.1 Wilt Incidence at Seedling Stage

The results demonstrated a diverse range of wilt incidence, spanning from 0% to 90.83% (Table 2). Based on their response to the pathogen, the genotypes were classified into five categories *viz*., resistant, moderately resistant, moderately susceptible, susceptible and highly Susceptible (Table 3; Fig. 2). Out of the 71 genotypes, 24 were considered resistant as showing minimal to no symptoms of wilt incidence at seedling stage. Moderately resistant genotypes, totaling 38 entries, exhibited relatively lower but discernible levels of wilt symptoms. On the other hand, five genotypes displayed moderate susceptibility to Fusarium wilt, signifying a moderate degree of disease progression. While three entries, were considered as susceptible owing their notable wilt symptoms. However, genotype JG62 (check) was identified as highly susceptible, succumbing to severe wilt infection. Remarkably, a previous study conducted by Yadav and Kumar [43] also investigated the resistance of chickpea genotypes against Fusarium wilt. Our current findings complement and extend upon their research, providing valuable insights into the disease reaction of diverse chickpea genotypes and their potential resistance to Fusarium wilt. Such knowledge is critical for developing effective disease management strategies and breeding programmes to enhance chickpea resistance against this devastating pathogen. Our research aligns with previous studies investigating the response of chickpea genotypes to Fusarium wilt disease. Bajwa et al. [44] evaluated 32 genotypes and found only one resistant, while the remaining 31 were found to be susceptible at the seedling stage.

3.2 Wilt Incidence at Reproductive Stage

At this critical growth phase, wilt incidence arrayed between 7.41% to 100%, providing a wide spectrum of disease reactions. Based on their responses to the pathogen, the genotypes were categorized into five groups: resistant, moderately resistant, moderately susceptible, susceptible and highly susceptible (Table 2).

At the reproductive stage, genotype JG315 (check) demonstrated highest resistance, showing minimum wilt symptoms despite the pathogen's presence. Fourteen genotypes including ICCV201207, SAGL22-118, SAGL-152238, SAGL-153226, SAGL-152223, SAGL-

152234, SAGL-162376, RVSSG84, RVSSG92, JG130, JAKI 9218, JG6, ICC4958 and RVSSG52 displayed moderate resistance, exhibiting relatively lower wilt incidences compared to susceptible counterparts. On the other hand, seventeen genotypes demonstrated moderate susceptibility to Fusarium wilt, showing an apparent yet manageable level of disease progression. A total of 25 genotypes were found susceptible, indicating a noteworthy susceptibility to the pathogen. While 14 genotypes were considered highly susceptible as displaying severe wilt symptoms, (Table 3; Fig. 2). These genotypes succumbed to considerable wilt incidence and thus require attention in breeding and disease management strategies. Mirzapour et al. [45] assessed 18 genotypes/cultivars, noting disease incidence ranging from 0% to 46.6% at the seedling stage and up to 100% at the reproductive stage.

Table 2. Disease scoring/indexing of chickpea genotypes against *Fusarium* wilt under controlled condition

S. No.	Genotypes	Mean SS (%)	Reaction (SS)	Mean RS (%)	Reaction (RS)
1	ICCV 201211	11.26	MR	33.56	S
2	ICCV 201210	8.39	R	26.66	MS
3	ICCV 201109	8.01	R	52.27	HS
4	ICCV 20116	10.47	MR	54.16	HS
5	ICCV 201115	13.80	MR	32.05	S
6	ICCV 201214	11.02	MR	48.70	S
7	ICCV 201112	7.17	R	54.16	HS
8	ICCV 201205	13.57	MR	40.06	S
9	ICCV 201104	13.80	MR	48.07	S
10	ICCV 201206	11.26	MR	33.56	S
11	ICCV 20117	14.83	MR	48.10	S
12	ICCV 201207	7.17	R	14.16	MR
13	Pant Gram 5	8.012	R	26.13	MS
14	H12-55	15.38	MR	45.83	S
15	RVG 202	11.02	MR	24.35	MS
16	SAGL 22-110	11.66	MR	34.28	S
17	SAGL 22-116	10.47	MR	38.69	S
18	SAGL 22-117	6.66	R	28.57	MS
19	SAGL 22-118	6.90	R	19.25	MR
20	SAGL 22-119	11.68	MR	45.83	S
21	SAGL 22-120	7.73	R	33.56	S
22	SAGL 22-121	10.47	MR	46.42	S
23	SAGL 22-122	10.47	MR	23.21	MS
24	SAGL 22-123	14.35	MR	54.19	HS
25	SAGL 22-124	34.35	S	67.13	H.S
26	SAGL- 152327	7.41	R	24.03	MS
27	SAGL- 152324	7.87	R	34.28	S
28	SAGL- 152237	20.19	MS	20.20	MS
29	SAGL- 152278	24.83	MS	43.56	S
30	SAGL- 152250	28.20	MS	53.07	HS
31	SAGL- 152330	11.85	MR	27.27	MS
32	SAGL- 152238	15.10	MR	17.69	MR
33	SAGL- 152405	10.47	MR	23.21	M.S
34	SAGL- 152339	10.71	MR	32.05	S
35	SAGL- 152344	7.41	R	24.03	MS
36	SAGL- 162299	31.90	S	51.92	HS
37	SAGL- 162387	17.14	MR	33.33	S
38	SAGL- 152227	14.35	MR	41.95	S
39	SAGL- 162381	7.17	R	23.21	MS
40	SAGL- 162364	10.98	MR	41.66	S
41	SAGL- 152356	12.42	MR	36.50	S
42	SAGL- 152337	12.17	MR	55	HS

S. No.	Genotypes	Mean SS (%)	Reaction (SS)	Mean RS (%)	Reaction (RS)
43	SAGL- 153226	18.68	M.R	18.33	MR
44	SAGL- 152336	21.02	MS	64.93	HS
45	SAGL- 152222	29.93	MS	63.33	HS
46	SAGL- 152318	32.69	S	60.98	HS
47	SAGL- 152258	6.90	R	29.67	MS
48	SAGL- 152231	7.5	R	40.58	S
49	SAGL- 152223	10.83	MR	16.78	MR
50	SAGL- 152234	7.5	R	16.23	MR
51	SAGL- 152329	7.17	R	23.21	MS
52	SAGL- 162376	15.47	MR	18.33	MR
53	SAGL- 162377	6.90	R	22.25	MS
54	RVSSG 84	11.26	MR	16.78	MR
55	RVSSG 74	11.66	MR	25.71	MS
56	JG 130	10.98	M.R	16.66	MR
57	RVSSG 83	10.51	MR	24.03	MS
58	JAKI 9218	8.01	R	17.84	MR
59	RVG 204	7.73	R	16.78	MR
60	JG 6	7.14	R	15.38	MR
61	RVSSG 92	10.23	MR	23.07	MS
62	ICC 4958	10.51	MR	19.05	MR
63	RVSSG 71	7.87	R	34.28	S
64	RVSSG 52	6.66	R	14.28	MR
65	RVSSG 68	10	R	22.25	MS
66	SAGL- 161024	14.83	MR	34.84	S
67	SAGL- 163006	18.68	MR	36.66	S
68	SAGL- 161025	14.35	MR	41.95	S
69	SAGL- 163007	19.04	MR	66.81	HS
70	JG 315 (Check)	0	R	7.41	R
71	JG 62 (Check)	90.83	HS	100	HS

Where, SS=Seedling Stage; RS=Reproductive Stage; R=Resistance; MR= Moderate Resistance; MS= Moderate Susceptible; S= Susceptible; HS= Highly Susceptible

Fig. 1. Screening of chickpea genotypes against *fusarium* wilt under controlled condition in polyhouse

In the present investigation 24 chickpea genotypes were found resistant for wilt during seedling stage. Among these lines, only one genotype found to be resistant at reproductive stage. These results are accordance to findings of Iqbal et al. [46] as they also reported the sources of resistance against Fusarium wilt in chickpea germplasm originating from national and international research institutes. They identified 14 chickpea lines to be resistant to wilt at seedling stage but no line found to be resistant at reproductive stage.

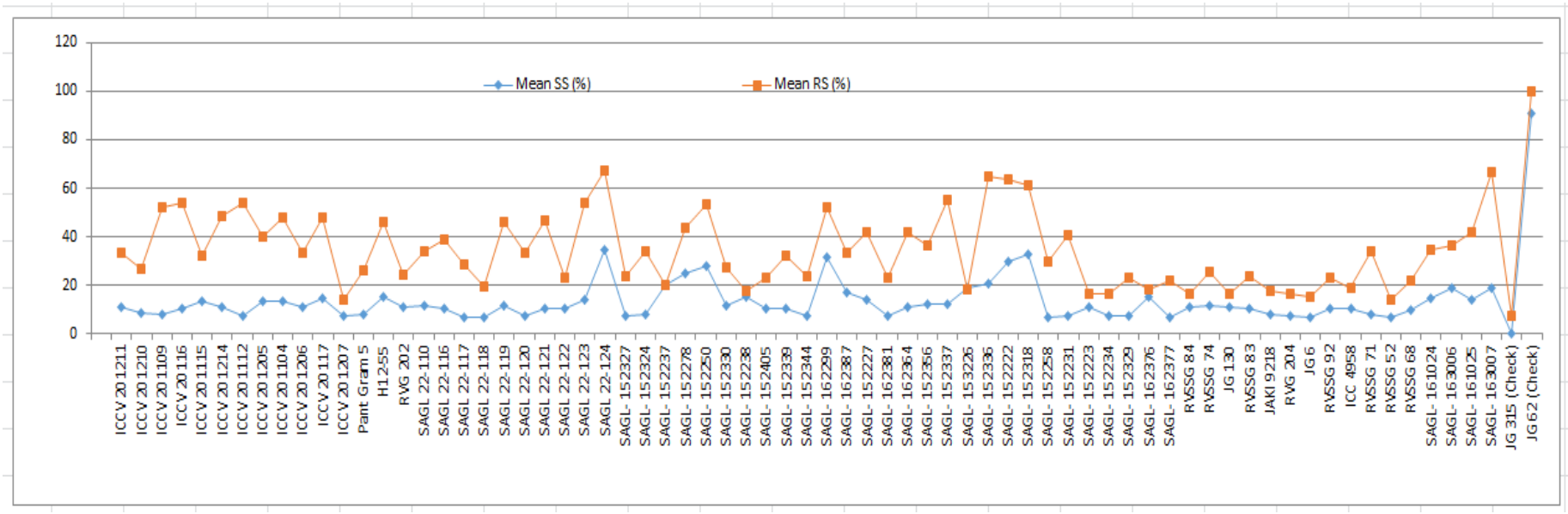

Fig. 2. Disease incidence of chickpea genotypes under artificial inoculation condition against fusarium wilt disease at seedling and reproductive stages

Table 3. Reaction of chickpea genotypes against fusarium wilt under controlled condition

Disease reaction	Number of genotypes		Name of genotypes	
	Seedling stage	Reproductive stage	Seedling stage	Reproductive stage
Resistant	24	1	ICCV 201210, ICCV 201109, ICCV 201112, ICCV 201207, Pant Gram 5, SAGL 22-117, SAGL 22-118, SAGL 22-120, SAGL- 152327, SAGL- 152324, SAGL- 152344, SAGL- 162381, SAGL- 152258, SAGL- 152231, SAGL- 152234, SAGL- 152329, SAGL- 162377, JAKI 9218, RVG 204, JG 6, RVSSG 71, RVSSG 52, RVSSG 68	JG 315

Disease reaction	Number of genotypes		Name of genotypes	
	Seedling stage	Reproductive stage	Seedling stage	Reproductive stage
Moderately Resistant	38	14	ICCV 201211, ICCV 20116, ICCV 201115, ICCV 201214, ICCV 201205, ICCV 201104, ICCV 201206, ICCV 20117, H12-55, RVG 202, SAGL 22-110, SAGL 22-116, SAGL 22-119, SAGL 22-121, SAGL 22-122, SAGL 22-123, SAGL- 152330, SAGL- 152238, SAGL-152405, SAGL- 152339, SAGL- 162387, SAGL- 152227, SAGL- 162364, SAGL-152356 SAGL- 152337, SAGL- 153226, SAGL- 152223, SAGL- 162376, RVSSG 84, RVSSG 74, JG 130, RVSSG 83, RVSSG 92, ICC 4958, SAGL- 161024, SAGL- 163006, SAGL- 161025, SAGL- 163007	ICCV 201207, SAGL 22-118, SAGL- 152238, SAGL- 153226, SAGL- 152223, SAGL-152234, SAGL- 162376, RVSSG 84, RVSSG 92, JG 130, JAKI 9218, JG 6, ICC 4958, RVSSG 52
Moderately Susceptible	5	17	SAGL- 152237, SAGL- 152278, SAGL-152250, SAGL- 152336, SAGL- 152222	ICCV 201210, Pant Gram 5, RVG 202, SAGL 22-11,7 SAGL 22-122, SAGL- 152327, SAGL- 152237, SAGL- 152330, SAGL-152405, SAGL- 152344, SAGL- 162381, SAGL- 152258, SAGL- 152329, SAGL-162377, RVSSG 74, RVSSG 92, RVSSG 68,
Susceptible	3	25	SAGL- 152318, SAGL- 162299, SAGL 22-124	ICCV 201211, ICCV 201115, ICCV 201214, ICCV 201205, ICCV 201104, ICCV 201206, ICCV 20117, H12-55, SAGL 22-110, SAGL 22-116, SAGL 22-119, SAGL 22-120, SAGL 22-121, SAGL- 152324, SAGL- 152278, SAGL- 152339, SAGL- 162387, SAGL-152227, SAGL- 162364, SAGL- 152356, SAGL- 152231, RVSSG 71, SAGL- 161024, SAGL- 163006, SAGL- 161025
Highly susceptible	1	14	JG 62	ICCV 201109, ICCV 20116, ICCV 201112, SAGL 22-123, SAGL 22-124, SAGL- 152250, SAGL- 162299, SAGL- 152337, SAGL-152336, SAGL- 152222, SAGL- 152318, RVSSG 83, SAGL- 163007, JG 62

The evaluation of diverse chickpea genotypes against Fusarium wilt revealed promising results, with many genotypes exhibiting resistance reactions at the seedling stage, while some showed resistance at the reproductive stage. These resistant genotypes hold great potential for utilization in breeding programmes aiming to develop Fusarium wilt-resistant/tolerant varieties. Remarkably, the disease progression was considerably slower in the resistant lines, whereas susceptible lines succumbed swiftly to the pathogen. This stark contrast in disease development underscores the importance of identifying and prioritizing resistant genotypes to combat the detrimental effects of Fusarium wilt effectively.

To ensure the reliability of the breeding programme, field screening at the reproductive stage appears to be a more dependable approach. Despite displaying resistance at the seedling stage, some genotypes transitioned to susceptibility at the reproductive stage. Consequently, evaluating genotypes at the reproductive phase provides critical insights into their long-term resistance potential and aids in selecting more robust and durable resistance traits. Kumar et al. [47] screened 101 genotypes, of which 57 showed resistance, 28 tolerant, and 16 susceptible responses at the seedling stage. At the reproductive stage, 31 genotypes were found resistant, 26 tolerant, and 44 susceptible. Thaware et al. [48] observed varying reactions among 50 chickpea entries against *F. oxysporum* f. sp. ciceris, with six entries being highly resistant, 31 resistant, eight moderately resistant, two moderately susceptible, and three highly susceptible. Patil *et al*. [49] examined seven isolates of *Fusarium oxysporum* f. sp. ciceris in chickpea. Among these isolates, I-19 and I-28 were identified as resistant, while I-20, I-13, and I-1 were classified as moderately resistant. Conversely, I-4 and I-80 were found to be susceptible to the pathogen. Interestingly, our own findings align with these results, as all isolates tested exhibited susceptibility to JG62, reinforcing the observed trends. Mirzapour *et al*. [45] reported that during the seedling stage two genotypes were found highly resistant, 7 genotypes and 3 cultivars resistant, 2 genotypes moderately resistant and 4 cultivars susceptible. whereas, during the reproductive stage under pod culture conditions, 2 genotypes observed resistant, 1 cultivar moderately resistant, 1 cultivar and 2 genotypes susceptible and 5 cultivars and 7 genotypes highly susceptible at reproductive stage under pod culture condition. Yadav *et al*. [50] were conducted pot culture experiments to assess the disease incidence of Fusarium wilt in different genotypes. Among the tested genotypes, DCP92-3, IPC14-28, IPC13-70, and IPC 05-28 demonstrated a resistant reaction, with disease incidences ranging from 0% to 10% under sick pot conditions. On the other hand, genotypes *viz*., IPC10-72, IPC10-217, IPC11-30, IPC12-108, and IPC11-12 exhibited a moderately resistant reaction, with disease incidences ranging from 11% to 20%.

Seedlings are particularly vulnerable to Fusarium wilt due to their underdeveloped root systems and limited ability to defend against pathogens. As plants mature and progress to the reproductive stage, their root systems become more established, which can provide some degree of resistance against initial infections. However, the pathogen may persist in the soil, and when plants allocate more resources to reproduction, their defense mechanisms against Fusarium wilt might be compromised. The wilt can be observed in susceptible genotypes within 25 days after sowing in the field (designated “early wilt”). However, symptoms are usually more visible in the early stages of flowering, 4 to 6 weeks after sowing and can also appear up to podding stage (“late wilt”). Late wilted plants exhibit drooping of the petioles, rachis and leaflets, followed by yellowing and necrosis of foliage. Early wilting causes more loss than late wilting [51, 52].

The consistent findings from these studies emphasize the significance of identifying and utilizing resistant or tolerant chickpea genotype (s) in breeding programmes for developing Fusarium wilt-resistant varieties. Understanding the diverse disease reactions among genotypes at different stages helps in formulating effective disease management strategies and enhancing sustainable chickpea production. The presence of varying resistance levels among different genotypes highlights the potential for selecting promising candidates to breed Fusarium wilt-resistant varieties, thus contributing to improved disease management and sustainable cultivation of chickpea.

4. CONCLUSION

Fusarium wilt remains a highly destructive vascular disease in chickpea. In our current study, we conducted screening of 71 diverse chickpea genotypes against Fusarium wilt using

diseased pots. Among these, the genotypes exhibited resistant and moderate resistance to *F. oxysporum* f. sp. Ciceris hold potential as valuable sources of disease resistance for future chickpea improvement programmes. Moreover, the genotypes showing resistance are well-suited for exploitation in breeding programmes and could be directly sown in wilt-prone regions. Consistently resistant lines may serve as essential disease resistance donors in breeding initiatives. The utilization of these resistant genotypes as donors in breeding programmes warrants further investigation into the mode of inheritance of their disease resistance traits. For comprehensive disease management, continuous mass screening of genotypes under field and pot conditions is recommended, focusing on potential breakdown of resistance sources and phenotyping of major races in major chickpea growing regions. While information on the mechanism of resistance remains limited, in-depth research based on this material is essential to gain insights into the underlying resistance mechanisms.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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The peer review history for this paper can be accessed here:
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