Screening for chickpea genotypes resistant to Fusarium wiltdisease under controlled conditions

Abstract

Fusariumwiltcaused by *Fusariumoxysporum* f. sp. *ciceris*is one of the economical important vascular root diseases affecting chickpea and can causes up to 90% yield losses depending on unfavorable environmental condition. In the present investigation, 71 chickpea genotypes including two controls *viz.*,JG315 (highly resistant) and JG 62 (highly susceptible)were screened by artificial inoculation of pathogen causingFusariumwilt under controlled conditions in poly housewith intention to identify potentially resistant genotype (s) that could help in mitigating the impact of this destructive disease on chickpea crops. The study assessed the disease incidence at two different stages *i.e.*, seedling and the reproductive stages. At the seedling stage, out of the 71 entries, 24 genotypes displayed resistance to the disease, whilst 38 genotypes exhibited moderate resistance. However, five genotypes were found to be moderately susceptible, three susceptible, and only one genotype showed high susceptibility.Upon reaching the reproductive stage, the disease reactions changed drastically, where only one genotype showed resistance. Among the remaining genotypes, 14 displayed moderate resistance, 17 moderately susceptible, 25 susceptible, and 14 genotypes were exhibited highly susceptible response.The promising genotype (s)may beutilized totransfer of their gene (s) to a commercial cultivar to develop Fusariumwilt resistant cultivar (s) in future.

Key words: Fusarium wilt; Chickpea; Resistant; Susceptible; Genotypes

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]. It has about 34 perennial wild species and 9 annual species. Among nine annual species, *Cicer arietinum* is the only cultivated species worldwide [8]. There are two types of cultivated chickpea *Kabuli* and *Desi*. *Desi* (microsperma) type's plant has anthocyanin pigmentation on stems, pink flowers, and a wild range colored and thick

Comment [E1]: is it for disease development or for growth of the crop

Comment [E2]: 1.Your objective is to identify resistant chickpea genotypes to fusarium wilt disease.

2. Methods of the study not mentioned.

Comment [E3]: Total 71 genotypes. 24 R, 38 mr, 5 ms and 1 hs. What about the other three genotypes? If the two HR and HS checks? Where is the other one?

Comment [E4]: It was one-time study in controlled environment therefore, your recommendation should indicate the need for further study at different times, location and under natural infection condition.

Comment [E5]: Add Screening or evaluation to the key words.

seed coat. Kabuli (macrosperma) type plants are characterized by a lack of anthocyanin pigmentation on their stems, white flowers, and seeds that are white or beige-colored with a distinctive ram's head shape. These seeds have a smooth surface and thin seed coat [9]. Kabuli-type chickpeas hold greater economic significance as they command a higher market price compared to Desi-type chickpeas. Despite this economic importance, most genomic resources developed for chickpeas have focused on the Desi-type [10-12]. These two types exhibit distinct genetic backgrounds, leading to variations in crucial agronomic traits, including disease resistance, cold tolerance, and growth habit [13].

Comment [E6]: In the first paragraph of the introduction it is better to discuss about the production and productivity of the crop in the world or in your country, its economic importance, factors affecting its production with special emphasis on fusarium wilt disease and so on.

Numerous biotic and abiotic factors contribute to the reduced productivity of chickpea [14-21]. 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 [22]. Among these, *Fusariumoxysporum* 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 [15]. 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 [23]. 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[24]. 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 [25].The substantial dependence on intensive fungicide usage as a major agricultural management practice has confirmed inadequacy in reducing the severity of soil-borne diseases[26-35,15], including *Fusarium*wilt [36]. Consequently, exploring host plant resistance has been pursued in the past as an economically viable management strategy for this disease [37]. 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 *Fusariumoxysporum* f. sp. *ciceris*[38].

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 [39].Considering these challenges, the present study was conducted to identify wilt disease-resistant genotype (s) of chickpea under controlled polyhouse condition.

Material and Methods

Experimental material

The experimental material consists of 71 chickpea genotypesincluding 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. Thesegenotypes were screened for host plant resistance against Fusarium wilt in pot in poly housesituated at Biotechnology Centre, RVSKVV, Gwalior, M.P., India. CompletelyRandomizedDesign was adopted to analyze data.

Comment [E7]: 1. How many seedlings were tested for each genotypes?
2. Was there replication?

Isolation, purification and identification of *Fusariumoxysporum* f. sp. *Ciceris*

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 5mm 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 sterilizeddouble 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. The developing mycelia were then transferred to fresh PDA plates and allowed to grow for an additional seven days at the same temperature.

To achieve a pure culture, hyphal tips were isolated from the resulting culture and maintained on PDA slants for future use. The pathogen was identified as *Fusariumoxysporum* f. sp. *ciceri* based on its morphological characteristics. 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.

Comment [E8]: From where did you learn this isolation, purification and identification techniques? You need to add citations and references.

Screeningofgenotypesundercontrolledconditions

For screening, plastic pots filled with sterilized (autoclaved) soil were used in a poly house. 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. 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.

Comment [E9]: How did you prepare the inoculum (amount of inoculum inoculated on the sterilized soil in the pots)?

Data collection

Comment [E10]: Add data collection sub section, insert some information on how you take samples from the inoculated seedlings, data collecting intervals at each sampling stage (seedling and reproductive).
Eg, days after planting

Disease assessment

Data on wilted plants of chickpea genotypes were recorded at two different stages*viz.*,seedling and reproductive. To quantify the disease incidence, the percentage of wilted plants was calculated using the following formula:

Comment [E11]: How many times? Data collection intervals.

Comment [E12]: Citation and reference needed

$$\text{Diseaseincidence}(\%) = \frac{\text{Numbers ofplantsexhibitingwiltsymptoms}}{\text{Total numbers ofplantsevaluated}} \times 100$$

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

Table1Diseasecategorizationratingscale(1-9) against Fusariumwilt [40]

Grade	**%wiltincidence**	**diseasereaction**
1	0-10	Resistant(R)
3	11-20	ModeratelyResistant (MR)
5	21-30	Moderately Susceptible (MS)
7	31-50	Susceptible(S)
9	>50	Highly Susceptible (HS)

Statistical analysis

Comment [E13]: In this section the statistical analysis toll used, analysis model and the significance test should be mentioned.
Eg. ANOVA or t test or other

Results and Discussion

The disease incidence of 71 chickpea genotypes including two checks*viz*., JG315 and JG 62was recorded at twodifferent stages*i.e.*,seedlingandreproductivestage**.**

Comment [E14]: Already mentioned twice.

Wilt incidence at seedling stage

The results demonstrated a diverse range of wilt incidence, spanning from 0% to 90.83%. Based on their response to the pathogen, the genotypes were classified into five categories*viz*., resistant, moderately resistant, moderately susceptible, susceptibleandhighly Susceptible (Table 2; Table3; Fig. 1). Out of the 71 genotypes, 24 were considered resistant as showing minimal to no symptoms of wilt incidence at seedling stage. Notable resistant genotypes included, ICCV 201210, ICCV 201109,ICCV 201112, ICCV 201207, Pant Gram 5, SAGL 22-117, SAGL 22-118, SAGL22-120, 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 68including check JG315.

Comment [E15]: Put each table or figure in the appropriate place along/matching your results and discussion.

Comment [E16]: You don't have to mention all names of genotypes here; it's already placed in the table. Just focus on main findings.

Moderately resistant genotypes, totaling 38 entries, exhibited relatively lower but discernible levels of wilt symptoms. Examples of such genotypes encompassed ICCV 201211, ICCV 20116, ICCV 201115, ICCV 201214, ICCV 201205, ICCV 201104, ICCV 201206, ICCV20117, 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, RVSSG74, JG 130, RVSSG 83, RVSSG 92, ICC 4958, SAGL- 161024, SAGL-163006,SAGL-161025,and SAGL- 163007.

Comment [E17]: Same here.

On the other hand, five genotypes displayed moderate susceptibility to Fusarium wilt, signifying a moderate degree of disease progression. Among these wereSAGL-152237, SAGL-152278, SAGL-152250, SAGL-152336 and SALG-152222. While three entries, *namely* SAGL-152318, SAGL-162299, and SAGL 22-124, were considered as susceptible owing their notable wilt symptoms. However, genotypeJG62 (check) was identified as highly susceptible, succumbing to severe wilt infection.Remarkably, a previous study conducted by

Yadav and Kumar [41] 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 programs 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*. [42] evaluated 32 genotypes and found only one resistant, while the remaining 31 were found to be susceptible at the seedling stage.

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; Table 3;Fig. 1).

Comment [E18]: Put each table or figure in the appropriate place along/matching your results and discussion.

At the reproductive stage, genotype JG315 (check) demonstrated highest resistance, showing minimum wilt symptoms despite the pathogen's presence. Fourteen genotypes includingICCV201207, SAGL22-118, SAGL-152238, SAGL-153226, SAGL-152223, SAGL- 152234, SAGL-162376, RVSSG84, RVSSG92, JG130, JAKI9218, JG6, ICC4958 and RVSSG52displayed moderate resistance, exhibiting relatively lower wilt incidences compared to susceptible counterparts. On the other hand, seventeen genotypes demonstrated moderate susceptibility to Fusarium wilt, showing anapparent yet manageable level of disease progression. Such genotypes encompassed ICCV 201210, Pant Gram 5, RVG202, SAGL22-117, SAGL22-122, SAGL-152327, SAGL-152237, SAGL-152330, SAGL- 152405, SAGL-152344, SAGL-162381, SAGL-152258, SAGL-152329, SAGL-162377, RVSSG 74, RVSSG 92 and RVSSG 68.

A total of 25 genotypes were found susceptible, indicating a noteworthy susceptibility to the pathogen. Among these were ICCV201211, ICCV201115,ICCV201214, ICCV 201205, ICCV201104, ICCV201206, ICCV20117, H12-55, SAGL22-110, SAGL22-116, SAGL22-119, SAGL22-120, SAGL22-121, SAGL-152324, SAGL-152278, SAGL- 152339, SAGL-162387, SAGL-152227, SAGL-162364, SAGL-152356, SAGL-152231, RVSSG 71, SAGL-161024, SAGL-163006 and SAGL-161025. While 14genotypes were considered highly susceptible as displaying severe wilt symptoms, including ICCV201109, ICCV 20116,

ICCV201112, SAGL 22-123, SAGL 22-124, SAGL-152250, SAGL- 162299, SAGL-152337, SAGL-152336, SAGL-152222, SAGL-152318, RVSSG83, SAGL-163007 and JG62. These genotypes succumbed to considerable wilt incidence and thus require attention in breeding and disease management strategies.Mirzapour*et al*. [43] 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.

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 programs aiming to develop Fusarium wilt-resistant/tolerant varieties. Notably, 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 program, 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*. [44] 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.Thawar*et al*. [45] 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*. [46] investigated seven isolates and identified I-19 and I-28 as resistant, I-20, I-13, and I-1 as moderately resistant, and I-4 and I-80 as susceptible in chickpea. All isolates exhibited susceptibility to JG62, corroborating our own findings. Zewdie*et al*. [47] conducted a study on Fusarium wilt incidences in desi and kabuli type chickpea genotypes. The results revealed that among the desi genotypes, five lines exhibited resistance, while ten genotypes displayed a moderately resistant reaction to the disease. In the case of kabuli type chickpea, five genotypes were identified as resistant and 14 genotypes showed a moderately resistant reaction. Yadav *et al*.[41]were conducted pot culture experiments to assess the disease incidence of Fusarium wilt in different genotypes. Among

Comment [E19]: What isolates?

Comment [E20]: This experiment was conducted in the field of sick plot. Yours was in controlled house. How do you relate it? Add or find another reference similar with your work.

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%.

The consistent findings from these studies emphasize the significance of identifying and utilizing resistant or tolerant chickpea genotype (s) in breeding programs 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.

Comment [E21]: General comment and suggestion to consider!

1. Compare percentage disease incidence at each growth stages?
2. At which growth stage the disease was found higher? What is/are the reason? Find literature to discuss your findings?
3. Was there any genotype which changes its resistance or susceptibility during the growth stages?

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 the genotypes assessed, 14 exhibited moderate resistance to *F. oxysporum*f. sp. ciceris. These identified moderately resistant genotypes hold potential as valuable sources of disease resistance for future chickpea improvement programs. Moreover, the genotypes showing resistance are well-suited for exploitation in breeding programs 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 programs 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.

Comment [E22]: Has repeated sentences.
It appropriate directly to recommend to be used by breeding programs?
Need major revision.

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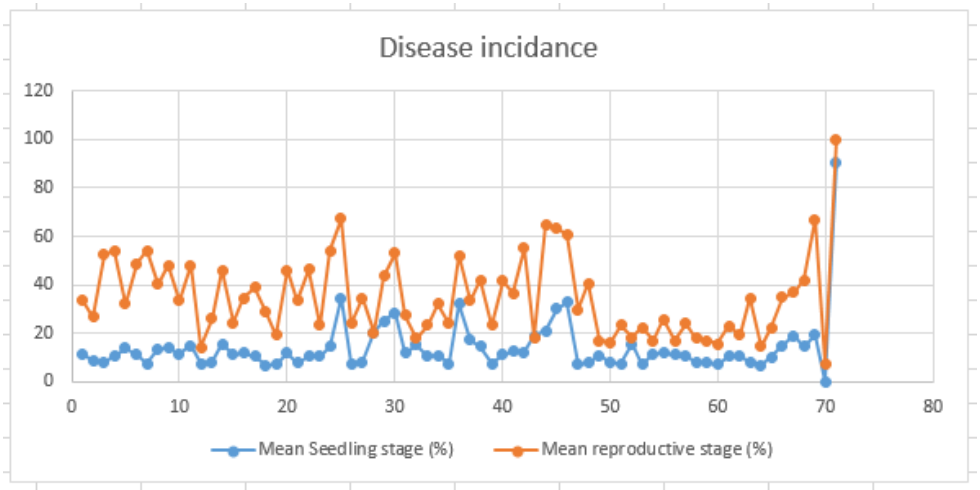

Fig.1 Disease incidence of chickpea genotypes under artificial inoculationcondition forFusariumwilt disease at seedling and reproductive stages

Table 2 Screeningofchickpeagenotypesagainst*Fusarium*wiltundercontrolled 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
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**=Seedlingstage;**RS**=Reproductivestage; **R**=Resistance; **MR**= Moderate resistance; **MS**= Moderate susceptible; **S**= susceptible; **HS**= Highly susceptible

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
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