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CHICKPEA ADVANCED LINES SCREENING FOR SOURCES OF RESISTANCE AGAINST TWO MAJOR DISEASES OF CHICKPEA"WILT AND BLIGHT"

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A B S T R A C T

Chickpea (*Cicer arietinum* L.) is a major food legume which ranks third in the world. Wilt and blight, two damaging diseases of chickpea, are prevalent in Pakistan, and this has resulted in a low output of the crop. Except for exploiting the host plant's resistance mechanism, the available control techniques are neither practicable nor cost-effective. To determine the sources of resistance in chickpea germplasm that is currently available.278chickpea genotypes (168 Desi and 110 Kabuli) originated from different springs were evaluated for disease resistance against *Ascochyta rabiei* in poly house and 102 different genotypes were screened for *Fusarium oxysporum* resistant sources in the sick field at Arid Zone Research Institute Bhakkar. Experiments were laid out following augmented design without replications during 2019-20 and highly susceptible check (AUG-424) was replicated as indicator for disease advent. The disease frequency was assessed twice at different growth stages and genotypes were categorized as per ICARDA rating scale (1-9). *Ascochyta* blight incidences revealed that39 lines displayed resistant (R) reaction and 12 had moderately resistant reaction (MR) in desi. However, 38 were found resistant and 26 genotypes were recorded as moderately resistant in kabuli chickpea, respectively. Similarly, 102 genotypes were sown in sick plot for screening against *F. oxysporum*, out of which only10 entries showed resistant and 21 moderately resistant reaction. The collected information was the most valuable to be used in breeding program for exploiting the genetic resistance and its direct use in severely blight and wilt hit areas may be preferred on the basis of resistance type.

Keywords: Chickpea, genotypes, susceptibility, resistance, *Ascochyta rabiei, Fusarium oxysporum* Pakistan.

INTRODUCTION

Chickpea (*Cicer arietinum* L.) is a less labor-intensive crop and its production demands low external inputs as compared to cereals (Bekele *et al*., 2007). The chickpea is a third-ranked edible legume (Bokhari *et al*., 2011; Sarwar *et al*., 2012; Hirich *et al*., 2014). It is grown by resource-poor farmers in the world's arid and semi-arid regions, particularly in Pakistan (Varshney *et al*., 2014; Maqbool *et al*., 2017). In varied cropping systems, it can retain soil fertility as well as being a rich source of high-quality protein (Malik *et al*., 2011). It is an important source of protein for human food and animal feed (Millan *et al*., 2006; MoARD, 2008). It supplies protein to the poor and thus known as poor man's meat. It covers 940 thousand hectares in Pakistan and produces 545 thousand tons annually (Economic Survey of Pakistan, 2019-20). It is primarily cultivated on marginal soils that are rainfed. It's already difficult to grow chickpeas in poor soils because of the prevalence of diseases such *Ascochyta rabiei* blight and wilt produced by *Fusarium oxysporum*, but the situation is made even more difficult because of the presence of these illnesses (Sarwar *et al*., 2012). A major worldwide foliar disease of chickpea known as ascochyta blight results in up to a 100%

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reduction in grain output (Pande *et al*., 2005; Atta *et al*., 2006) and is one of the major chickpea yields limiting factor in Pakistan and responsible for its yield gap (Shahbaz *et al*., 2014). Both *Ascochyta* blight and *Fusarium* wilt can wipe out an entire crop (Shivalinga *et al*., 2018) or cause noteworthy annual yield losses (Gan *et al*., 2006). The spread of *Ascochyta* blight is more with cool (15-25^{o}C) and humid weather (>150 mm rainfall) that prevails during the crop season (Pande *et al*., 2005). *Fusarium* wilt adversely affects chickpea plant health (Asrat and Tolesa, 2020) and is wide spread in dry and warm areas (Asrat and Tolesa, 2018) perpetrating accountable quantitative and qualitative losses (Khilare *et al.*, 2009; Thaware *et al*., 2016).Wilt disease causing pathogen is a soil borne, causes problems for plants throughout their life cycle, but the prevalence is highest during the flowering and pod development stage (Maitlo *et al*., 2014). High temperatures and drought were found to exacerbate the severity of disease outbreaks. Chickpea wilt disease can cause annual losses of 10 to 90 percent (Sharma and Muehlbauer, 2007), whereas average yield losses in Pakistan are 10 to 50 percent in dry locations (Khan *et al*., 2004; Naqvi *et al*., 2013).

Chickpea production losses owing to Ascochyta blight and Fusarium wilt varies from 10 to 15 percent globally, and in severe situations, the infection can entirely devastate the crop in some areas (Navas-Cortes *et al*., 2000; Sharma *et al*., 2005, Jimenez-diaz and Jimenez-gasco, 2011). *Fusarium* wilt is prevalent in almost all chickpea-growing areas of the world, and its incidence varied from 14 to 32% in Thal region of Pakistan (Islam *et al*., 2011). Early wilt disease incidence was reported to cause 77-94% yield loss (Haware and Nene, 1980).Till the eighties, the disease incidence was recorded in about twenty-six countries of the world (Nene, 1980). Now it is assumed that disease exists in more than 40 countries (Bhardwaj *et al*., 2010; Sharma and Ghosh, 2016).Different management methods for Fusarium wilt of chickpea recognized by Merkuz and Getachew, (2012) and reported that raised bed preparation, tolerant variety and optimum time of planting prevented the wilt incidence and reduce mortality of wilt (Agrios, 2005; Iqbal *et al*., 2005; Ahmad *et al*., 2012) while Chaudhry *et al*. (2006) concluded that for seed dressing, fungicides were ineffective because of their high cost and short-term efficacy. Landa *et al*. (2004) reported that integrated management of Fusarium wilt of chickpea with sowing date, host resistance and biological control and concluded sowing date has the greatest effect on incidence of *Fusarium* wilt and yield of chickpea.

Disease suppression can be achieved by the use of host-plant resistance mechanisms, as well as the identification of sources of resistance in existing germplasm (Bakhsh *et al*., 2007; Duzdemir *et al*., 2014; Tariq *et al*., 2015). When it comes to disease management, adopting resistant crop varieties is the most effective technique (Karimi *et al*., 2012).

Currently, there is no effective way to manage blight disease since spraying fungicides under disease-friendly conditions is difficult. The greatest approach for integrated disease control is to use host plant resistance mechanisms in current chickpea germplasm (Duzdemir *et al*., 2014). But new pathotypes/isolates keep changing the resistance mechanism. Thus, coordinated efforts are essential to identify genetic origins. After all, development of resistant varieties is the most effective method to manage Ascochyta blight and *Fusarium* wilt to realize chickpea yield stability. Host resistance is the main component of integrated disease management and most efficient, cheapest, environmentally safe and economical way of managing two major diseases of chickpea (Seid and Melkamu, 2006; Asnakech, 2014). Identifying resistant chickpea varieties against Ascochyta blight and Fusarium wilt is an important solution to minimize the yield gap of chickpea production. For this, cultivars that are resistant to pests and diseases are a well-known fact. Natural epidemics or intentional inoculation in the field or in a controlled environment have yielded a number of sources of resistance to Ascochyta blight and Fusarium wilt. Blight and wilt resistant cultivars, on the other hand, have failed, either due to a breakdown in genetics or a shift in the pathogen's virulence (Nene, 1980; Jamil *et al*., 2010). Since diseases are best controlled through host plant resistance, a reliable screening technique is necessary for introducing long-lasting resistance into cultivars. Individual, virulent isolates are far more trustworthy than diseased plant detritus or even an inoculum including a variety of isolates (Ilyas *et al*., 2007). It is difficult to manage the disease because of the complexity of the target pathosystem and the inherent complexities of the management strategy itself. In the present study, chickpea germplasm was tested against an *A. rabiei* highly virulent isolate and the same genotypes were also tested against Fusarium wilt in a wilt sick field containing heavy *F. oxysporum* inoculums (Jiménez-díaz and Jiménez-gasco, 2011).

The results of these investigations were used in the search for new resistant sources of chickpea blight and fusarium wilt in poly houses (screen houses where pathogens are artificially inoculated) and field conditions (naturally and artificially infested fields), respectively. Advance chickpea genotypes, such as the Desi and Kabuli, were screened for new sources of resistance.

MATERIALS AND METHODS

Screening against wilt: A wilt sick plot was equipped with a mixture of isolates representing different chickpea growing areas. Fresh inoculum of *F. oxysporum* was prepared in laboratory and added in sick plot for infection and disease development. A total of 102 chickpea germplasm lines were screened for their response to *Fusarium* wilt disease. Augmented design was used without replication. A highly wilt susceptible genotype, AUG-424 (Atta *et al.,* 2006), was repeatedly planted after every two test entries. Each genotype was laid out in a 2 m row length. Row to row and plant to plant distance was kept as 30 and 10 cm, respectively. Wilt incidence was calculated by counting the number of wilted plants in each row by the formula provided by Shah *et al*. (2009).

$$\text{Disease Incidence (\%)} = \frac{\text{No. of infected plants}}{\text{Total no. of plants}} \times 100$$

In order to determine the reaction of the genotypes to high inoculum pressure, the early, late, and combination wilt incidence percentages were determined using the aforementioned formula. During the fourth week of December and the first week of March, respectively, data on early and late wilt were collected. According to Iqbal *et al*. (2005) utilizing a rating scale devised by them, they determined the amount of resistance/susceptibility of each test line. A wilt reaction percentage of 0-10 percent indicates high resistance; 11-20 percent indicates moderate resistance; 21-30 percent indicates moderate resistance; 31-50 percent indicates susceptible; and 51-100 percent indicates extremely susceptible.

Screening against blight

Preparation of Inoculum

Isolation: Several samples of Ascochyta blight infected chickpea plants were gathered from places where the disease was prevalent and spread rapidly. These infected samples were plated on PDA media for the isolation of fungus. Fifteen days after fungus colonies developed, spores were examined under microscope (CETI Magnum-PH Trinocular. Code MED-2738.0000M). Pods, stem and leaflets with blight lesions were alienated and sterilized in 5 percent sodium hypochloride for 1 minute before being dried on sterilized filter paper to prevent contamination. The sample was plated on 2 percent water agar and cultured at 20°C±2 with a 12 h light/dark cycle for 5-7 days to see if it would grow into a fungus. Plant material-derived fungal colonies were subcultured on chickpea seed meal agar, which is made from hot water extract obtained by boiling 60 g chickpea seeds for 30 minutes. It was necessary to dilute the extract with 120 g of sucrose and 120 g of agar each in order to make it up to 1 L in volume. Following two to three weeks of incubation on this medium, colonies of the fungus containing pycnidia began to grow and spread (Alam and Strange, 1987).

Multiplication: Seeds of chickpea were boiled in water for 15-30 minutes to soften them, draining them, and then autoclaving them for 30 minutes at 121°C in a conical flask. In order to inoculate chickpea seed, spore suspension of required fungus was developed by stirring distilled water on a mushroom. To assess the concentration of the spore suspension, a hemocytometer (Model No. QJ1102) was used, and the concentration was adjusted to 10^6 spores/ml by adding water. The seeds were wetted with spore suspension in an adjusted volume, and the flask was shaken to ensure that the inoculum was distributed evenly across the flask. Pycnidia were plentiful on the seed after it had been incubated for 7-10 days at 20 °C. The use of sterile distilled water to stir the spore suspension produced spore suspension. The suspension was filtered using a cotton cloth to remove any impurities before being used (Alam and Strange, 1987).

Chickpea cultivation and inoculation: During 2019-20, 353 genotypes were sown in a blight screening nursery in a poly house at Arid Zone Research Institute, Bhakkar, Pakistan
(31°38'08.0"N71°07'16.0"E, 31.635555, 71.121099). Fifteen seeds of each genotype were planted in a 2-meter single row at a distance of 15cm between plants and 30cm between rows. As a control, AUG-424 (very vulnerable to *A. rabiei*) was planted after every two genotypes. A sprinkler system was devised to artificially humidify the air to aid in disease transmission. Further to ensure proper disease development, fungal suspension was sprayed during the early blooming and pod filling stage on all genotypes, as described by Singh & Reddy, (1993), Muehlbauer *et al*. (1998); Toker *et al*. (1999); Pande *et al*. (2011) disease scale ranging from 1 to 9 modified from

Jan and Wiese, 1991; Toker *et al*. (1999) was used for following criteria to determine the severity of the disease;

1 = Immune (No symptoms on plants),
2 = Highly Resistant (small tissue depression or spot),
3 = Resistant (elongating spot),
4 = Moderately Resistant (coalescent spot),
5 = Tolerant (stem girdling),
6 = Moderately susceptible (stem breaking),
7 = Susceptible (lesion growth downward from breaking point),
8 = Highly susceptible (whole plant nearly dead) and
9 = Highly susceptible (All plants dead).

The t-test was used to compare the means of the control and test genotypes (Shah *et al*., 2005). A round figure was used to represent the average blight score for each genotype (through rounding of the data).

RESULTS AND DISCUSSION

Screening against blight: Managing the genetic resistance in chickpea against *Ascochyta* blight (AB) is a challenge because of high level of diversity in primary gene pool of host, complexity in molecular bases in QTLs and variable pathogen population with continuously emerging new pathotypes (Islam *et al*., 2017). All genotypes studied shown significant variation in their reaction to Ascochyta blight disease. AUG-424which showed susceptible reaction revealed rigorous disease symptoms, with an average disease severity rating of 8.9.Out of 168 (Table 1) genotypes tested (Desi),39 were resistant (R), 12 were moderately resistant (MR), 25 were moderately susceptible (MS), 45 was susceptible (S) and 47 were highly susceptible (HS) to *Ascochyta* blight disease (Table 3). Among 110 Kabuli (Table 2), 38 genotypes were resistant, 20 were moderately resistant, 36 were susceptible and 16 were highly susceptible to Ascochyta blight (Table 4).These findings indicate that the screened germplasm is an excellent resistance source to *A. rabiei.* Chickpea genotypes were resistant to Ascochyta blight disease due to a single dominant gene (Reddy and Singh, 1993). Ascochyta blight possesses a diverse variety of resistance genes derived from many sources (Collard *et al*., 2003). Randhawa *et al.* (2009) studied the role of glandular hairs density, population and size of stomata aperture in chickpea cultivars against Ascochyta blight. It was observed that these characters played significant role in display of in-built resistance. Through gene pyramiding, different genes providing varying degrees of resistance can be inserted into commercial cultivars to ensure lasting resistance in commercial cultivars (Tekeoglu *et al*., 2000). 39 genotypes (Desi) were observed to be resistant with mean disease severity ratings ranging from 2.7 to 3.3 (Table 3) and 38 genotypes (Kabuli) were found to be resistant with mean disease severity ratings ranging from 2.8 to 3.7 (Table 4). These were all the most effective sources of resistance to the disease. Hassan *et al*. (2012) conducted a study of a similar nature. Numerous others detailed the sources of resistance encountered in field situation (Iqbal *et al*., 2004; Chaudhry *et al*., 2005; Bashir *et al*., 2006). Islam *et al*. (2017) revealed that development of AB resistant varieties through incorporation of resistant genes need to be continued to defeat the pathogen and acquiring the desirable results as many genes contribute to plant-pathogen interaction and all of them can increase the resistance responses to Ascochyta blight disease (Andam *et al*., 2020).

Screening against wilt: Chickpea genotypes (Table 5) evaluated for wilt incidence exhibit considerable differences during the crop's early and late seasons. Early wilt percentages were from 7 to 96 percent, late wilt percentages ranged from 6 to 97 percent, and overall wilt percentages ranged from 9.5 to 100 percent (Table 5). In comparison, wilting of plants occurred less frequently throughout the late season than during early spread when favorable circumstances for disease development prevailed. The combined wilt data indicated that ten genotypes were resistant, twenty-one were moderately resistant, three were moderately susceptible, and forty-nine were severely vulnerable (Table 6).Disease infection (wilt) was sustainable and uniform in early, late and combined as inoculum of *F. exospore* was applied in sufficient amount and season was favorable for screening against wilt. The genotypes 09AG006, CH16/06, D08025, TG1410, TG1708, TG1710, TG1712, TG1808, BK-2011and TG1410 showed resistant type reaction in combined to wilt disease and it was suggested that the genotypes may be used in breeding program aimed to develop wilt resistant varieties. In view of combined effect of wilt on genotypes viz., 09AG006, CH16/06, D08025, TG1410, TG1708, TG1710, TG1712, TG1808, Bhakkar-2011, TG1410, Bittle-2016, CH888/06, CM54/05, D088-11, NIAB- 16, TG1401, TG1411, TG1413, TG1613 (B), TG1702, TG1703, TG1704, TG1714, TG1716, TG1718, TG1801, TG1802, TG1812, TG1817 and TG1826 were categorized as resistant to moderately resistant. Resistance is mediated by a genetic mechanism to disease for these genotypes was very stable in the early and late seasons, exhibiting consistency in their reactive response

(Asrat and Tolesa, 2018), i.e., responding similarly to all three wilt categories. The findings corroborated some of Sarwar *et al,* (2012) findings. During screening, it was noted that majority of the entries (70%) displayed moderately susceptible to highly susceptible reaction. This depicts that the most of the genotypes did not have resistance genes. These results are also in accordance with that of Iqbal *et al*. (2010), who studied 145 genotypes against Ascochyta blight and wilt diseases and most of them showed susceptible to highly susceptible reaction. The aggressiveness of the pathogen on both types of chickpea varieties was increased parallel with inoculum concentration and time (Ayana *et al*., 2019).

Table 1.Disease severity (DS) rating means for *Ascochyta* blight (Desi chickpea).

Sr. #	Genotype	Severity mean ± SE	Class	Sr.#	Genotype	Severity mean ± SE	Class
1	TG1502	4.3±0.9**	MS	85	CH15/12	7.0±0.6ns	S
2	06A056	6.0±0.6*	MS	86	CH-18-D-118	3.0±0.6***	R
3	93A138	7.7±0.9ns	HS	87	GGP-1456	7.0±0.6ns	S
4	GGP1481	3.3±0.3***	HR	88	05A028	6.0±0.6*	MR
5	CH1312	7.7±0.9ns	HS	89	CH-18-D-123	7.0±0.6ns	S
6	GGP1460	4.0±0.6***	MR	90	E-4	6.0±0.6*	MS
7	CH18-D-117	3.3±0.9***	R	91	CH27/12	6.0±0.6*	MS
8	M	7.7±0.9ns	S	92	GGP1484	3.0±0.6***	R
9	TG1505	2.7±0.3***	R	93	G	7.0±0.6ns	S
10	GGP1461	7.7±0.9ns	HS	94	J	5.7±0.9*	MS
11	E-11	6.0±0.6*	MS	95	TG1500	2.8±0.6***	R
12	CH18-D-127	7.0±0.6ns	S	96	K	4.3±0.3***	MR
13	E	7.0±0.6ns	S	97	06A118	3.0±0.6***	R
14	TG1410	2.7±0.9ns	R	98	T	3.0±0.6***	R
15	H	7.7±0.9ns	HS	99	I	3.0±0.6***	R
16	06A117	7.7±0.9ns	HS	100	E-22	7.0±0.6ns	S
17	TG1504	7.7±0.9ns	HS	101	E-9	6.3±0.7*	MS
18	GGP1506	7.7±0.9ns	HS	102	GGP-1425	3.0±0.6***	R
19	P	7.0±0.6ns	S	103	TG1415	7.0±0.6ns	S
20	TG1507	5.7±0.9*	MS	104	06A099	6.3±0.7*	MS
21	TG1503	5.7±0.9*	MS	105	TG1508	7.0±0.6ns	S
22	TG1456	7.7±0.9ns	HS	106	B0097-10	3.0±0.6***	R
23	TG1401	3.3±0.3***	R	107	06A086	7.0±0.6ns	S
24	CH108/12	7.7±0.9ns	HS	108	CH44/12	7.7±0.9ns	HS
25	D	5.7±0.9*	MS	109	TG1424	3.2±0.5***	R
26	06A011	7.8±0.9ns	HS	110	GGP-1451	7.0±0.6ns	S
27	CH18-D-125	3.3±0.3***	R	111	W	3.0±0.6***	R
28	CH18-D-135	7.8±0.9ns	HS	112	TG1312	3.0±0.6***	R
29	GGP1518	3.3±0.3***	R	113	CH12/12	7.7±0.9ns	HS
30	GGP1467	7.0±0.6ns	S	114	TG1501	7.7±0.9ns	HS
31	X	3.3±0.3***	R	115	E-24	6.3±0.7*	MS
32	E-2	3.3±0.3***	R	116	09AG006	7.0±0.6ns	S
33	TG1429	8.7±0.3ns	HS	117	GGP1457	6.3±0.7*	MS
34	GGP1483	4.0±1.2**	MR	118	CH30/12	7.7±0.9ns	HS
35	GGP1516	7.7±0.6***	HS	119	GGP1443	7.0±0.6ns	S
36	E-18	7.0±0.6ns	S	120	CH-18-D-126	7.7±0.9ns	HS
37	CH18-D-121	4.0±1.2**	MR	121	E-1	6.3±0.7*	MS
38	GGP1490	8.7±0.3ns	HS	122	07A007	8.7±0.3ns	HS
39	L	5.7±0.9*	MS	123	CH-18-D-115	6.3±0.7*	MS

40	05A030	3.0±0.6***	R	124	GGP1517	8.7±0.3ns	HS
41	CH14/12	3.0±0.6***	R	125	CH-18-D-130	3.3±0.3***	R
42	GGP-1315	8.7±0.3ns	HS	126	CH19/12	7.7±0.9ns	HS
43	GGP1407	8.7±0.3ns	HS	127	GGP1486	7.0±0.6ns	S
44	NES0613	3.3±0.3***	R	128	E-8	8.7±0.3ns	HS
45	TG1426	2.7±0.6***	R	129	U	3.3±0.3***	R
46	05A056	4.0±1.2*	MR	130	06A126	4.3±0.3***	MR
47	TG1511	5.7±0.9*	MS	131	03A036	6.3±0.7*	MS
48	F	3.3±0.3***	R	132	CH-18-D-124	7.0±0.6ns	S
49	PB2018.500109	7.8±0.9ns	HS	133	R	7.0±0.6ns	S
50	E-23	5.7±0.9*	MS	134	TG1509	7.7±0.9ns	HS
51	06A124	3.0±0.6***	R	135	CH-18-D-134	7.0±0.6ns	S
52	E-16	7.8±0.9ns	HS	136	06A082	7.0±0.6ns	S
53	E-7	3.0±0.6***	R	137	92A260	5.7±0.9*	MS
54	E-15	7.0±0.6ns	S	138	GGP-1429	7.0±0.6ns	S
55	GGP1515	4.3±0.3***	MR	139	GGP1445	7.7±0.9ns	HS
56	CH14/12	7.8±0.9ns	HS	140	GGP1475	7.0±0.6ns	S
57	S	7.9±0.9ns	HS	141	GGP1459	7.0±0.6ns	S
58	E-14	7.0±0.6ns	S	142	CH-18-D-120	5.7±0.9*	MS
59	03A035	4.3±0.3***	MR	143	E-20	5.7±0.9*	MS
60	CH18-D-113	7.0±0.6ns	S	144	TG1480	5.7±0.9*	MS
61	06A055	3.3±0.3***	R	145	C	7.7±0.9ns	HS
62	CH03/12	3.0±0.6***	R	146	CP10070	7.0±0.6ns	S
63	GGP1440	4.3±0.3***	MR	147	CH10/12	7.7±0.9ns	HS
64	D072-11	7.7±0.9ns	HS	148	CH09/12	7.7±0.9ns	HS
65	CH18-D-133	7.0±0.6ns	S	149	06A061	7.0±0.6ns	S
66	E-10	7.0±0.6ns	S	150	05A005	3.0±0.6***	R
67	E-12	7.0±0.6ns	S	151	TG1510	7.0±0.6ns	S
68	CH18-D-132	7.0±0.6ns	S	152	06A089	7.0±0.6ns	S
69	GGP-1482	7.0±0.6ns	S	153	CH23/12	7.7±0.9ns	HS
70	A	7.0±0.6ns	S	154	CH-18-D-119	5.7±0.9*	MS
71	GG1514	3.0±0.6***	R	155	GGP-1424	7.7±0.9ns	HS
72	CH18-D-129	7.0±0.6ns	S	156	GGP-1489	3.3±0.3***	R
73	TG1419	2.9±0.6***	R	157	GGP-1462	7.7±0.9ns	HS
74	GGP1512	7.7±0.9ns	HS	158	06A054	7.0±0.6ns	S
75	CH18-D-128	7.7±0.9ns	HS	159	E-13	7.7±0.9ns	HS
76	06A119	3.0±0.6***	R	160	E-17	3.3±0.3***	R
77	GGP1493	7.0±0.6ns	S	161	92A230	4.3±0.3***	MR
78	GGP1485	3.3±0.3***	R	162	E-9	5.7±0.9*	MS
79	CH-18-D-131	7.7±0.9ns	HS	163	E-15	7.0±0.6ns	S
80	O	7.7±0.9ns	HS	164	CH33/12	7.0±0.6ns	S
81	E-21	4.3±0.3***	MR	165	E-3	7.7±0.9ns	HS
82	E-6	7.7±0.9ns	HS	166	Q	7.0±0.6ns	S
83	GGP-1411	7.7±0.9ns	HS	167	V	7.0±0.6ns	S
84	E-5	3.0±0.6***	R	168	AUG424	8.9±0.3***	HS

R-resistant, MR-moderately resistant, T-tolerant, S-susceptible, HS-highly susceptible
*, ** and *** indicate Significance at 0.05, 0.01 and 0.001 probability levels.
SE= standard error

Table 2.Disease severity (DS) rating means for *Ascochyta* blight (Kabuli chickpea).

Sr.#	Genotype	Severity mean ± SE	Class	Sr.#	Genotype	Severity mean ± SE	Class
1	TGK1506	4.0±0.6***	MR	56	GGP-31	3.7±0.9***	MR
2	TGK1711	7.1±0.6ns	S	57	CH69/09	4.0±0.6***	MR
3	TGK1502	7.1±0.6ns	S	58	GGP-35	3.7±0.9***	MR
4	TGK1720	7.1±0.6ns	S	59	TGK1507	7.1±0.6ns	S
5	GGP5	7.1±0.6ns	S	60	NOOR-13	3.0±0.7***	R
6	TGK1503	7.0±0.6ns	S	61	TGK1723	7.1±0.6ns	S
7	GGP18	7.0±0.6ns	S	62	TGK1734	7.0±0.6ns	S
8	TGK1712	7.7±0.9ns	HS	63	GGP-15	7.7±0.9ns	HS
9	CH56/12	7.7±0.9ns	HS	64	TGK1501	7.0±0.6ns	S
10	NOOR-2009	7.1±0.6ns	S	65	GGP-22	3.7±0.9***	MR
11	TGK1502	2.8±0.9***	R	66	GGP-16	7.0±0.6ns	S
12	COOP-5-E	7.1±0.6ns	S	67	TGK1749	3.7±0.9***	MR
13	TGK1508	7.1±0.6ns	S	68	COOP-4-BK	7.1±0.6ns	S
14	GGP-7	4.0±0.6***	MR	69	GGP-32	7.1±0.6ns	S
15	GGP-21	7.0±0.6ns	S	70	TGK1740	7.1±0.6ns	S
16	TGK1750	7.0±0.6ns	S	71	GGP-8	7.1±0.6ns	S
17	TGK1727	7.0±0.6ns	S	72	TGK1743	4.0±1.0*	MR
18	TGK1725	7.0±0.6ns	S	73	TGK1752	7.9±0.9ns	HS
19	TGK1714	7.0±0.6ns	S	74	TGK1717	7.1±0.6ns	S
20	NOOR-2013	7.7±0.9ns	HS	75	GGP-12	7.0±0.6ns	S
21	TGK1728	7.1±0.6ns	S	76	CH53/12	7.9±0.9ns	HS
22	GGP-13	7.7±0.9ns	HS	77	GGP-9	4.0±0.6***	MR
23	CH47/12	4.0±1.0*	MR	78	TGK1755	3.3±0.9***	R
24	GGP1755	7.0±0.6ns	S	79	TGK1716	7.1±0.6ns	S
25	TGK1706	2.9±0.9***	R	80	E-5	3.3±0.9***	R
26	GGP-6-F	7.7±0.9ns	HS	81	E-6	3.2±0.7***	R
27	TGK1719	4.0±1.0*	MR	82	E-14	3.1±0.7***	R
28	COOP-3-C	4.0±0.6***	MR	83	E-1	3.0±0.7***	R
29	TGK1733	3.7±0.9***	MR	84	E-4	3.01±0.7***	R
30	NOOR-2009	4.0±0.6***	MR	85	TGK1604	3.0±0.7***	R
31	CH616/10	3.3±0.9***	R	86	E-3	3.3±0.9***	R
32	TGK1619	4.0±1.0*	MR	87	E-13	3.0±0.6***	R
33	GGP-1-K	3.0±0.6***	R	88	TGK1732	3.0±0.6***	R
34	COOP-2-BK	7.0±0.6ns	S	89	E-11	3.3±0.3***	R
35	COOP-1-AK	8.7±0.3 ns	HS	90	GGP-23	3.0±0.6***	R
36	GGP-2-K	4.3±0.3***	MR	91	TGK1754	3.3±0.3***	R
37	GGP-28-K	4.0±1.0*	MR	92	GGP-10	3.3±0.3***	R
38	GGP-17-K	7.0±0.6ns	S	93	E-12	3.3±0.3***	R
39	TGK1702	4.3±0.3***	MR	94	E-8	4.3±0.3***	MR
40	NOOR-13	4.3±0.3***	MR	95	TGK1731	4.3±0.3***	MR
41	GGP-19	7.0±0.6ns	S	96	E-16	3.3±0.3***	R

42	GGP-3	4.0±0.6***	MR	97	TGK1520	3.3±0.3***	R
43	AUG-424	8.8±0.9ns	HS	98	E-10	3.3±0.3***	R
44	TGK1612	7.0±0.6ns	S	99	E-7	3.0±0.6***	R
45	TGK1724	7.9±0.9ns	HS	100	TGK1721	2.8±0.3***	R
46	GGP-4	7.9±0.9ns	HS	101	TGK1704	2.9±0.3***	R
47	TGK1605	7.9±0.9ns	HS	102	CH48/12	3.0±0.6***	R
48	TGK1730	7.9±0.9ns	HS	103	CH57/12	3.0±0.6***	R
49	GGP-1729	3.0±0.6***	R	104	E-2	4.3±0.3***	MR
50	NOOR-09	7.9±0.9ns	HS	105	E-15	4.3±0.3***	MR
51	TGK1726	7.9±0.9ns	HS	106	E-9	3.0±0.6***	R
52	GGP-26	7.0±0.6ns	S	107	GGP-20	3.0±0.6***	R
53	TGK1739	7.0±0.6ns	S	108	TGK1505	3.7±0.9***	MR
54	GGP-36	3.7±0.7***	R	109	TGK1508	7.0±0.6ns	S
55	CH66/10	4.0±0.6***	MR	110	TGK1501	7.0±0.6ns	S

R-resistant, MR-moderately resistant, S-susceptible, HS-highly susceptible *, ** and *** indicate Significance at 0.05, 0.01 and 0.001 probability levels. SE= standard error

Table 3.Grouping of chickpea advance genotypes against *Ascochyta* blight (Desi)

Class	Genotypic frequency	Genotypes
R	39	GGP1481, E-2, NES0613, F, 06A055, TG1424, 05A005, CH18-D-117, TG1505, TG1401, CH18-D-125, GGP1518, X, GGP1516, 05A030, CH14/12, TG1426, 06A124, E-7, CH03/12, GG1514, TG1419, 06A119, GGP1485, E-5, CH-18-D-118, GGP1484, TG1500, 06A118, T, I, GGP-1425, B0097-10, W, TG1312, CH-18-D-130, U, GGP-1489, E-17
MR	12	GGP1460, GGP1483, CH18-D-121, 05A056, GGP1515, 03A035, GGP1440, E-21, 05A028, K, 06A126, 92A230
MS	25	TG1502, 06A056, E-11, TG1507, TG1503, D, L, TG1511, E-23, E-4, CH27/12, J, E-9, 06A099, E-24, GGP1457, E-1, CH-18-D-115, 03A036, 92A260, CH-18-D-120, E-20, TG1480, CH-18-D-119, E-9
S	45	M, CH18-D-127, E, P, GGP1467, E-18, E-15, E-14, CH18-D-113, CH18-D-133, E-10, E-12, CH18-D-132, GGP-1482, A, CH18-D-129, GGP1493, CH15/12, GGP-1456, CH-18-D-123, G, E-22, TG1415, TG1508, 06A086, GGP-1451, 09AG006, GGP1443, GGP1486, CH-18-D-124, R, CH-18-D-134, 06A082, GGP-1429, GGP1475, GGP1459, CP10070, 06A061, TG1510, 06A089, 06A054, E-15, CH33/12, Q, V
HS	47	93A138, CH1312, GGP1461, TG1410, H, 06A117, TG1504, GGP1506, TG1456, CH108/12, 06A011, CH18-D-135, TG1429, GGP1490, GGP-1315, GGP1407, PB2018.500109, E-16, CH14/12, S, D072-11, GGP1512, CH18-D-128, CH-18-D-131, O, E-6, GGP-1411, CH44/12, CH12/12, TG1501, CH30/12, CH-18-D-126, 07A007, GGP1517, CH19/12, E-8, TG1509, GGP1445, C, CH10/12, CH09/12, CH23/12, GGP-1424, GGP-1462, E-13, E-3, AUG-424

Table 4. Chickpea genotypes grouping against Ascochyta blight (Kabuli)

Class	Genotypic frequency	Genotypes
R	38	GGP-36, NOOR-13, E-6, E-14, E-1, E-4, TGK1604, TGK1502, TGK1706, CH616/10, GGP-1-K, GGP-1729, TGK1755, E-5, E-3, E-13, TGK1732, E-11, GGP-23, TGK1754, GGP-10, E-12, E-16, TGK1520, E-10, E-7, TGK1721, TGK1704, CH48/12, CH57/12, E-9, GGP-20, TGK1733, GGP-31, GGP-35, GGP-22, TGK1749, TGK1505
MR	20	TGK1506, GGP-7, CH47/12, TGK1719, COOP-3-C, NOOR-2009, TGK1619, GGP-2-K, GGP-28-K, TGK1702, NOOR-13, GGP-3, CH66/10, CH69/09, TGK1743, GGP-9, E-8, TGK1731, E-2, E-15
S	36	TGK1711, TGK1502, TGK1720, GGP5, TGK1503, GGP18, NOOR-2009, COOP-5-E, TGK1508, GGP-21, TGK1750, TGK1727, TGK1725, TGK1714, TGK1728, GGP1755, COOP-2-B-K, GGP-17-K, GGP-19, TGK1612, GGP-26, TGK1739, TGK1507, TGK1723, TGK1734, TGK1501, GGP-16, COOP-4-B-K, GGP-32, TGK1740, GGP-8, TGK1717, GGP-12, TGK1716, TGK1508, TGK1501,
HS	16	TGK1712, CH56/12, NOOR-2013, GGP-13, GGP-6-F, COOP-1-A-K, AUG-424, TGK1724, GGP-4, TGK1605, TGK1730, NOOR-09, TGK1726, GGP-15, TGK1752, CH53/12

Table 5. Response of chickpea germplasm against Fusarium wilt

Genotype	TP	Early wilt % mean ± SE	Class	Late wilt %	Class	Combined wilt % mean ± SE	Class
05A030	D	44.3±0.6***	S	40.3±0.6***	S	42.3±0.6***	S
06A124	D	47.2±1.6***	S	45.2±1.6***	S	49.2±1.6***	S
09AG006	D	9.75±1.6***	R	7.75±1.6***	R	10±1.6***	R
AUG424 (Check)	D	95±2.7***	HS	97±2.7***	HS	100±2.9***	HS
AUG424 (Check)	D	96±2.8***	HS	95±2.7***	HS	100±2.7***	HS
Bhakkar-2011	D	25.53±1.5***	MR	23.02±1.5***	MR	24.65±1.5***	MR
Bittle -16	D	27.27±1.5***	MR	25.36±1.5***	MR	24.89±2.5***	MR
BK-2011	D	7.05±1.6***	S	10±1.6***	HR	9±1.6***	HR
CH14/12	D	69.7±2.6***	HS	64.7±2.6***	HS	69.7±2.6***	HS
CH16/06	D	10±0.5***	R	8±0.5***	R	10±0.5***	R
CH18-D-121	D	46.6±1.6***	S	39.6±1.6***	S	45.6±1.6***	S
CH18-D-135	D	66.5±2.6***	HS	62.5±2.2***	HS	68.5±2.2***	HS
CH53/07	D	40±1.6***	S	37±1.6***	S	57±1.6***	HS
CH87/06	D	68±2.6***	HS	60±2.4***	HS	72±2.4***	HS
CH888/06	D	11.75±0.6***	MR	9.75±1.6***	MR	12±1.6***	MR
CM54/05	D	13.75±1.6***	MR	10.75±1.6**	MR	12.75±1.6***	MR
CM770/06	D	46±0.9***	S	41±0.9***	S	48±0.9***	S
D08025	D	7±1.1***	R	6±1.5***	R	10±1.5***	R
D088-11	D	11.5±0.3***	MR	9.5±0.4***	MR	10±0.4***	MR
E-15	D	45.51±1.2***	S	41.51±1.2**	S	47.51±1.6***	S
E-16	D	31.75±0.9***	MS	30.75±0.9**	MS	39.75±0.9***	MS
E-18	D	48.3±1.6***	S	38.3±1.6***	S	41.3±1.6***	S
E-2	D	45±2.6***	S	41±2.1***	S	47±2.1***	S
GGP-1315	D	44.25±1.6***	S	41.25±1.6**	S	48.25±1.6***	S
GGP1467	D	70±2.6***	HS	62±2.1***	HS	69±2.1***	HS
GGP1483	D	47.75±1.6***	S	42.75±1.6**	S	45.75±1.6***	S
GGP1490	D	42.25±2.6***	S	38.25±2.6**	S	45.25±2.6***	S
GGP1516	D	40±1.6***	S	37±1.6***	S	42±1.6***	S
GGP1518	D	62.75±2.6***	HS	61.75±2.9**	HS	69.75±2.9***	HS
K7005	K	40.75±1.8***	S	38.75±2.8**	S	42.75±2.8***	S
NES0613	K	51±2.1***	HS	50±2.1***	HS	55±2.1***	HS
NIAB- 16	D	22.22±1.5***	MR	24.21±1.5***	MR	25.33±1.8***	MR
NIAB-16	D	45.05±1.5***	S	42.5±1.5***	S	48.05±1.02***	S
Punjab-1	K	83.75±3.6***	HS	75.75±2.6**	HS	80.75±2.6***	HS
TG1401	D	16.35±0.9***	MR	10.35±0.9**	MR	12.35±0.9***	MR
TG1402	D	63.25±1.6***	HS	60.25±2.1**	HS	80.25±2.1***	HS
TG1403	D	57.55±2.6***	HS	47.5±2.6***	HS	67.55±2.6***	HS
TG1404	D	43.5±1.6***	S	38.5±1.6***	S	58.5±1.6***	S
TG1405	D	66.5±0.9***	HS	51.5±0.9***	HS	65.5±0.9***	HS
TG1406	D	62.75±0.6***	HS	48.75±0.6**	HS	55.75±0.6***	HS
TG1407	D	45±1.6***	S	40±1.6***	S	60±1.6***	HS
TG1408	D	47.75±2.6***	S	41.75±2.6**	S	55.75±2.6***	HS
TG1409	D	43.25±2.6***	S	38.25±1.6**	S	45.25±1.6***	S
TG1410	D	10.75±0.2***	R	8.75±1.2***	R	9.55±1.2***	R
TG1410	D	8.5±0.1***	HR	7.4±0.2***	HR	9.5±0.2***	HR
TG1411	D	12±0.6***	MR	10±0.6***	MR	11±0.6***	MR
TG1412	D	48±1.6***	S	47±1.9***	S	55±1.9***	HS
TG1413	D	17.5±0.9***	MR	13.5±0.9***	MR	15.5±0.9***	MR
TG1414	D	46.5±0.7***	S	36.5±1.7***	S	45.5±1.7***	S
TG1415	D	40±1.8***	S	30±1.8***	S	45±1.8***	S

TG1416	D	70±2.8***	HS	60±2.8***	HS	75±2.8***	HS
TG1417	D	77.5±3.6***	HS	65.5±3.1***	HS	75.5±3.1***	HS
TG1418	D	30.75±0.9***	MS	29.75±1.9**	MS	29±1.9***	MS
TG1419	D	50±1.5***	S	48±1.5***	S	52±1.5***	HS
TG1420	D	66.5±2.6***	HS	60.5±2.5***	HS	65.5±2.5***	HS
TG1421	D	48.9±1.9***	S	45.9±1.7***	S	45.9±1.7***	S
TG1423	D	33.5±1.5***	MS	27.5±1.5***	MS	37.5±1.5***	MS
TG1424	D	42.7±1.1***	S	36.7±2.1***	S	47.7±2.1***	S
TG1425	D	40±0.6***	S	34±0.9***	S	42±0.9***	S
TG1426	D	43±2.6***	S	36±2.8***	S	45±2.8***	S
TG1427	D	41.25±2.5***	S	35.25±2.9**	S	47.25±2.9***	S
TG1428	D	45±1.7***	S	41±1.7***	S	48±1.7***	S
TG1429	D	41.25±1.6***	S	37.25±1.8**	S	45.25±1.8***	S
TG1430	D	45±1.5***	S	39±2.5***	S	45±2.5***	S
TG1613(A)	D	52.94±2.2***	HS	48.9±2.2***	S	51.94±2.29***	HS
TG1613(B)	D	25.00±1.2***	MR	23.00±1.2***	MR	23.00±1.2***	MR
TG1617	D	80.76±2.7***	HS	75.76±2.7***	HS	74.76±2.50***	HS
TG1618	D	53.84±1.6***	HS	50.84±1.6***	HS	58.84±1.8***	HS
TG1621	D	84.12±1.3***	HS	74.12±1.3***	HS	80.12±1.3***	HS
TG1622	D	47.05±1.2***	S	45.88±1.2***	S	48.56±1.7***	S
TG1623	D	76.47±1.6***	HS	72.20±1.6***	HS	78.47±1.4***	HS
TG1626	D	73.33±1.5***	HS	68.00±1.5***	HS	77.89±1.11***	HS
TG1702	D	30±1.8***	MR	29.2±1.8***	MR	29.88±1.2***	MR
TG1703	D	25±1.7***	MR	23.01±1.7***	MR	24.23±1.8***	MR
TG1704	D	25±0.6***	MR	22.21±0.6***	MR	26.01±0.54***	MR
TG1707	D	31.25±1.4***	S	32.0±1.4***	S	36.25±1.2***	S
TG1708	D	20±0.2***	R	17.0±0.2***	R	20.56±0.4***	R
TG1710	D	13.63±1.0***	R	11.25±1.0***	R	12.88±1.0***	R
TG1711	D	38.07±1.6***	S	35.21±1.6***	S	38.98±1.7***	S
TG1712	D	20±1.1***	R	18.90±1.1***	R	19.35±1.5***	R
TG1713	D	33.33±1.2***	S	30.32±1.2***	S	32.35±1.2***	S
TG1714	D	25±1.6***	MR	22.03±1.6***	MR	26.02±1.6***	MR
TG1715	D	37.05±1.9***	S	34.20±1.9***	S	38.99.05±1.9***	S
TG1716	D	29.41±1.7***	MR	25.41±1.7***	MR	29.41±1.7***	MR
TG1717	D	29.88±1.5***	S	27.99±1.5***	S	29.88±1.8***	S
TG1718	D	31.25±1.3***	MR	29.99±1.3***	MR	28.25±1.5***	MR
TG1801	D	27.27±1.5***	MR	23.66±1.5***	MR	25.27±1.5***	MR
TG1802	D	26.26±1.7***	MR	22.01±1.7***	MR	25.26±1.89***	MR
X	D	43.5±1.6***	S	39.5±2.6***	S	45.5±2.6***	S
TG1805	D	31.25±2.0***	S	29.32±2.0***	S	29.25±2.01***	S
TG1806	D	40±2.5***	S	41.22±2.5***	S	45±2.8***	S
TG1808	D	15.78±1.7***	R	12.44±1.7***	R	14.78±1.02***	R
TG1812	D	23.52±1.5***	MR	22.14±1.5***	MR	25.52±1.6***	MR
TG1813	D	35.29±1.4***	S	29.99±1.4***	S	36.32±1.8***	S
TG1814	D	40±1.5***	S	38.99±1.5***	S	40.98±2.5***	S
TG1815	D	42.10±1.8***	S	39.87±1.8***	S	47.10±2.8***	S
TG1817	D	30±1.5***	MR	28.31±1.5***	MR	30.99±1.05***	MR
TG1818	D	43.75±1.5***	S	37.98±1.5***	S	45.75±1.6***	S
TG1820	D	66.66±2.5***	HS	59.23±2.5***	HS	68.66±2.8***	HS
TG1825	D	50±1.5***	S	48.70±1.5***	S	52±1.9***	S
TG1826	D	23.07±1.8***	MR	20.98±1.8***	MR	25.07±2.8***	MR
TG1829	D	71.42±2.5***	HS	65.02±2.5***	HS	72.42±1.5***	HS

Table 6. The summarized combined reaction of chickpea genotypes to Fusarium wilt

Class	Genotypic Frequency	Name of genotypes
R	10	09AG006, CH16/06, D08025, TG1410, TG1708, TG1710, TG1712, TG1808, BK-2011, TG1410
MR	21	Bittle-2016, CH888/06, CM54/05, D088-11, NIAB- 16, TG1401, TG1411, TG1413, TG1613 (B), TG1702, TG1703, TG1704, TG1714, TG1716, TG1718, TG1801, TG1802, Bhakkar-2011, TG1812, TG1817, TG1826.
MS	3	E-16, TG1418, TG1423
S	39	05A030, 06A124, CH18-D-121, CM770/06, E-15, E-18, E-2, GGP-1315, GGP1483, GGP1490, GGP1516, K7005, NIAB-16, TG1404, TG1409, TG1414, TG1415, TG1421, TG1424, TG1425, TG1426, TG1427, TG1428, TG1429, TG1430, TG1622, TG1707, TG1711, TG1713, TG1715, TG1717, X, TG1805, TG1806, TG1813, TG1814, TG1815, TG1818, TG1825.
HS	29	AUG424 (Check), AUG424 (Check), CH14/12, CH18-D-135, CH53/07, CH87/06, GGP1467, GGP1518, NES0613, Punjab-1, TG1402, TG1403, TG1405, TG1406, TG1407, TG1408, TG1412, TG1416, TG1417, TG1419, TG1420, TG1613(A), TG1617, TG1618, TG1621, TG1623. TG1626, TG1820, TG1829

CONCLUSION AND RECOMMENDATION

Evaluation of genetic variation in chickpea GenBank is essential for effective selection in genetic improvement for diseases resistance, agronomic and yield traits. Due to the outcome of new virulent strains, there is tireless need to develop resistant cultivars using different breeding techniques against virulent strains and to create variability to obtain sustainable yield. The present study was conducted in field and in poly house to identify the resistant Desi and Kabuli chickpea genotypes against two major destructive pathogens, *A. rabiei* and *F. oxysporum*. Ascochyta blight screening was conducted in control conditions in tunnel while chickpea genetic material was also screened against *Fusarium* wilt in sick plot in open field. Each year, the expansion of these two most devastating diseases had a significant impact on chickpea production per unit area. Currently available options include disease management or the use of genotypes that are resistant to these diseases. The present study discovered significant variation in resistance to the aforementioned diseases, which may be exploited directly or induced via hybridization in high yielding but disease susceptible genotypes. Thus, the resistant lines will be used as disease resistance source donors in breeding programs. Although little information on the disease resistance mechanism was available, however, detailed research by making use of this material is suggested for future effective genetic disease management.

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Muneer Abbas	:	Help in data collection and analysis
Muhammad F. Hassan		Help in write up of manuscript.
Aftab A. Khan	:	Collection of references
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