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

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Abstract

Chickpea (*Cicer arietinum* L.) an important food legume, ranks third in the world. In Pakistan yield of chickpea is low due to the prevalence of wilt and blight diseases - the two destructive diseases. The control measures available are not feasible and economical, except to exploit host plant resistance mechanism to identify the sources of resistance in existing chickpea germplasm. Fifty four advance chickpea genotypes were screened in blight screening nursery and wilt sick plot. Out of total 54 genotypes 23 were resistant and 16 were moderately resistant to *Ascochyta* blight disease. Among 23 resistant genotypes; K0058-09, K0062-09, K0066-09, D095-09, K07A005, BK05A015 and BK04A013 had disease rating mean of 3. The results of early wilt showed 19 genotypes as highly resistant and 15 as resistant. The genotypes K0070-09, BKK17106, CH 65/02 and BK04A013 were highly susceptible to wilt during early pathogen infection at seedling stage while the genotypes K0063-09, BKK17106 & BK04A013 were susceptible during late season. Resistance sources identified could be exploited directly and also may be transferred through hybridization to high yielding disease susceptible genotypes.

Key words: Ascochyta blight, Cicer arietinum, Disease resistance and Fusarium wilt.

Introduction

Chickpea (Cicer arietinum L.) is an important food legume that ranks third in the world (Hirich et al., 2014; Bokhari et al., 2011 & Sarwar et al., 2012). It is a rich source of good quality protein with ability to sustain soil fertility when included in different cropping systems. In Pakistan it covers an area of 9.85 million hectares with an average annual production of 6.73 million tons (Anon., 2012) and is planted mostly under rainfed conditions on marginal lands. The yield of chickpea on these lands is already low but the situation is further aggravated due the prevalence of blight and wilt diseases caused by Ascochyta rabiei and Fusarium oxysporum f. sp. ciceris (Foc), respectively (Sarwar et al., 2012). Ascochyta blight is an important foliar disease of chickpea worldwide that causes grain yield losses up to 100% (Pande et al., 2005) and is the most important yield-limiting factor in Australia and Canada, potentially affecting 95% area (Gan et al., 2006). The spread of the disease is more with cool (15-25°C) and humid weather (>150 mm rainfall) that prevails during the crop season (Pande et al., 2005). Pathogen causing wilt disease is soil born, can affect all stages of plant growth and development but higher incidence at flowering and podding stage (Maitlo et al., 2014). The severity of the disease is maximum under high temperature and drought conditions. Annually 10-90% losses occur due to chickpea wilt disease (Sharma & Muehlbauer, 2007), whereas in Pakistan average yield losses are 10-50% in dry areas (Khan et al., 2004; Naqvi et al., 2014). The control measures for wilt disease are; either to use suitable fungicide for seed dressing which is not feasible in the long run (Chaudhry et al., 2006) due to higher cost or through management i.e., early or late sowing.

There is no proper method of controlling blight disease available yet as spray of fungicide is impossible in the weather conditions favorable for the development of disease. The best option available for integrated management strategy to control these diseases is to exploit host plant resistance mechanism to identify the sources of resistance in existing chickpea germplasm (Duzdemir *et al.*, 2014). But the problem is that the resistance mechanism is not stable due to the introduction of new pathotypes/isolates. Therefore, continuous efforts are required for identification of the new sources. The resistance against *Ascochyta* blight disease is due to either a single dominant gene or recessive gene (Reddy & Singh, 1993) and wilt resistance is race specific governed by major genes.

Keeping in view the objective of the study, advance chickpea genotypes were screened to identify the new sources of resistance against *Ascochyta* blight and *Fusarium* wilt under field conditions.

Materials and Methods

Advance chickpea genotypes (37 Kabuli and 17 Desi) obtained from Nuclear Institute for Agriculture and Biology (NIAB) Faisalabad; Pulses Research Institute (PRI), Faisalabad; and Arid Zone Research Institute (AZRI), Bhakkar were screened against blight and wilt diseases under field conditions in experimental area of Plant Breeding and Genetics Division NIAB, Faisalabad. The experimental design was randomized complete block design (RCBD).

Screening against blight

Inoculum preparation

Isolation: Samples of chickpea plants infected with *Ascochyta* blight were collected from areas with severe disease incidence. Pods, stems and leaflets with blight lesions were separated and sterilized in 5% sodium hypochlorite for 1 minute and dried on sterilized filter paper. The material was plated on 2% water agar and incubated at $20^{\circ}C\pm2$ with 12 h light/dark cycle for 5-7

days for fungal growth. Fungal colonies growing from the plant material were sub-cultured on chickpea seed meal agar which consists of hot water extract obtained by boiling 60 g chickpea seed for 30 minutes. Sucrose and agar 120 g each were added to the extract and made volume up to 1 liter. Incubation on this media for 1-2 weeks resulted in the development of colonies of the fungus with pycnidia (Alam & Strange., 1987).

Multiplication: Chickpea seeds were softened by boiling for 15-30 minutes in water drained and autoclaved for 30 minutes at 121°C in a conical flask. Spore suspension was prepared from a slant of the fungus growing on chickpea seed agar by the addition of sterilized distilled water and agitation with a sterile loop. The concentration of the spore suspension was determined with a haemocytometer and adjusted to 10^6 spores/ml by adding water. Spore suspension in adjusted volume was added to wet the seeds and flask was shaken to ensure distribution of the inoculum. After incubating for 7-10 days at 20° C abundant pycnidia were present on the seed. Agitation with sterile distilled water resulted in spore suspension. The suspension was filtered through muslin cloth (Alam & Strange., 1987)

Chickpea cultivation and inoculation: Fifty four genotypes were sown during last week of October in blight screening nursery at NIAB, Faisalabad during 2010-11.Ten seeds for each genotype were planted in 1.5 meter single row in three replicates keeping 15cm plant to plant and 30cm row to row distance. K-850 (highly susceptible to *A. rabiei*) was planted after each two genotypes as check. A sprinkle system was developed to create artificial humidity for disease spread. All genotypes were inoculated equally by spraying fungal suspension during initial flowering and pod filling stages as described by Singh & Reddy (1993), Muehlbauer *et al.* (1998) and Toker *et al.* (1999) to ensure good disease development. Disease assessment was carried out using 1-9 disease severity (DS) rating scale as suggested by Toker *et al.* (1999).

where,

- 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).

Means of control and test genotypes were compared using *t*-test (Shah *et al.*, 2005). The average blight score for each genotype was taken as a round figure (through rounding of the data).

Screening against wilt: The same material was tested for response against *Fusarium oxysporum* in wilt sick plot during the year 2010-11. Ten seeds of each genotype were

planted in 1.5 meter row in three replicates with inter and intra row spacing of 15cm and 30cm, respectively. A highly susceptible variety Aug-424 (susceptible to wilt and tolerant to blight) was planted after each two test genotypes for comparison. Data were recorded on the number of wilted plants in every row to calculate wilt incidence using the following formula suggested by Shah *et al.* (2009);

Wilt incidence (%) = $\frac{\text{No of wilted plants}}{\text{Total number of plants germinated}} \times 100$

Using the above formula early, late and combined wilt incidence (percentage) was calculated to see the response of the genotypes under high inoculum pressure. Early and late wilt data were recorded at last week of December and first week of March, respectively. Level of resistance/susceptibility of each test line was determined by using rating scale suggested by Iqbal *et al.* (2005).

where genotypes with wilt percentage 0-10% = highly resistant; 11-20% = resistant; 21-30% = moderately resistant; 31-50% = susceptible; and 51-100% = highly susceptible.

Results and Discussion

Screening against blight: All tested genotypes differed significantly for their response to Ascochyta blight disease. Susceptible check (K-850) showed severe symptoms of disease on all parts of the plant with disease severity rating mean of 8.7 (highly susceptible). Out of the total 54 genotypes tested 23 were resistant, 16 were moderately resistant, 8 were tolerant, 5 were moderately susceptible, 1 was susceptible and one was highly susceptible to Ascochyta blight disease (Table 2). Among 37 Kabuli and 17 Desi, 12 Kabuli and 11 Desi genotypes were resistant. This reveals that the Desi germplasm is a good source of resistance against Ascochyta rabiei than Kabuli chickpea. The resistance of chickpea against Ascochyta blight disease is due to either a single dominant gene or recessive gene (Reddy & Singh, 1993). Ascochyta blight has a wide range of resistance from different sources having different genes of resistance (Collard et al., 2003). Different genes conferring different levels of resistance could be introduced into commercial cultivars through gene pyramiding to facilitate increased level and durability of resistance in the commercial cultivars (Tekeoglu et al., 2000).

Among 23 genotypes observed to be resistant, 16 genotypes were resistant with disease severity rating mean values of 2.7 or 3.3 and 7 genotypes Viz. K0058-09, K0062-09, K0066-09, D095-09, K07A005, BK05A015 and BK04A013 were resistant with severity rating mean of 3 (Table 1). So, these all are the best sources of resistance against the pathogen. The genotypes; CH82/02, CH 38/03, CH 47/04, FG0902 & FG0901, BKK02182 and BKK02209 showed varying degrees of susceptibility with no utility in the breeding programme. Similar kind of study was reported by Hassan *et al.* (2012). Many others also reported the sources of resistance under field conditions (Alam *et al.*, 2003; Iqbal *et al.*, 2004; Chaudhry *et al.*, 2005 & Bashir *et al.*, 2006).

Sr. No.	Genotype	Туре	Severity mean ± SE	Class	Sr. No.	Genotype	Туре	Severity mean ± SE	Class
1.	K009-09	Κ	4.3±0.9**	MR	28.	BKK02231	Κ	4.7±1.2*	Т
2.	K0010-09	Κ	3.3±0.9***	R	29.	BKK07124	Κ	3.7±0.9***	MR
3.	K0021-09	Κ	3.3±0.9***	R	30.	BKK07151	Κ	2.7±0.7***	R
4.	K0025-09	Κ	3.3±0.9***	R	31.	BKK02182	Κ	7.0 ± 0.6^{ns}	S
5.	K0026-09	Κ	4.3±0.9**	MR	32.	CH 65/02	Κ	5.3±0.7*	Т
6.	K0030-09	Κ	3.3±0.9***	R	33.	CH82/02	Κ	$6.0\pm0.6*$	MS
7.	K0031-09	Κ	5.3±0.7*	Т	34.	CH 38/03	Κ	6.3±0.7*	MS
8.	K0034-09	Κ	4.3±0.3***	MR	35.	CH 47/04	Κ	$6.0\pm0.6*$	MS
9.	K0035-09	Κ	4.3±0.9**	MR	36.	FG0901	Κ	5.7±0.9*	MS
10.	K0039-09	Κ	5.3±0.9*	Т	37.	FG0902	Κ	$6.0\pm0.6*$	MS
11.	K0051-09	Κ	3.3±0.3***	R	38.	D075-09	D	4.3±0.9*	MR
12.	K0054-09	Κ	3.3±0.9***	R	39.	D080-09	D	3.3±0.9***	R
13.	K0057-09	Κ	3.3±0.7***	R	40.	D084-09	D	3.3±0.9***	R
14.	K0058-09	Κ	3.0±0.6***	R	41.	D085-09	D	5.0±0.6***	Т
15.	K0062-09	Κ	3.0±0.6***	R	42.	D089-09	D	3.3±0.9***	R
16.	K0063-09	Κ	4.0±1.0*	MR	43.	D090-09	D	3.3±0.9***	R
17.	K0065-09	Κ	4.0±0.6***	MR	44.	D094-09	D	3.3±0.9***	R
18.	K0066-09	Κ	3.0±0.6***	R	45.	D095-09	D	3.0±1.0***	R
19.	K0068-09	Κ	4.0±1.0*	MR	46.	D096-09	D	4.0±1.2*	MR
20.	K0069-09	Κ	5.3±0.7*	Т	47.	D098-09	D	4.0±0.6***	MR
21.	K0070-09	Κ	4.3±1.5*	MR	48.	D0100-09	D	4.0±1.2*	MR
22.	BKK17115	Κ	3.7±0.9***	MR	49.	BK07A005	D	3.0±0.6***	R
23.	BKK17106	Κ	5.0±0.6***	Т	50.	BK96A2055	D	3.3±0.7***	R
24.	BKK17124	Κ	3.3±0.9***	R	51.	BK05A015	D	3.0±0.6***	R
25.	BKK02174	Κ	5.0±0.6***	Т	52.	BK04A013	D	3.0±0.6***	R
26.	BKK02209	Κ	7.7 ± 0.9^{ns}	HS	53.	FG-0904	D	3.7±0.9***	MR
27.	BKK02213	Κ	3.7±0.9***	MR	54.	FG-0908	D	3.3±0.3***	R
						K-850		8.7±0.3	HS

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

R-resistant, MR-moderately resistant, T-tolerant, S-susceptible, HS-highly susceptible

 $\ast, \ast\ast$ and $\ast\ast\ast$ indicate Significance at 0.05, 0.01 and 0.001 probability levels.

SE= standard error

Table 2. Grouping of chickpea advance genotypes against Ascochyta blight.

Class	Genotypic frequency	Name of genotypes
Ι	-	-
HR	-	-
R	23	K0010-09, K0021-09, K0025-09, K0030-09, K0051-09, K0054-09, K0057-09, K0058-09, K0062-09, K0066-09, BKK17124, BKK07151, D080-09, D084-09, D089-09, D090-09, D094-09, D095-09, BK07A005, BK96A2055, BK05A015, BK04A013 & FG-0908
MR	16	K009-09, K0026-09, K0034-09, K0035-09, K0063-09, K0065-09, K0068-09, K0070-09, BKK17115, BKK02213, BKK07124, D075-09, FG-0904, D098-09, D0100-09 & D096-09
Т	8	K0031-09, K0039-09, K0069-09, BKK17106, BKK02174, BKK02231, CH 65/02 & D085-09
MS	5	CH82/02, CH 38/03, CH 47/04, FG0902 & FG0901
S	1	BKK02182
HS	1	BKK02209

				Table	3. Resp	onse of chickp	ea adva	nce genotypes	agair	nst <i>fusarium</i> wil	f.				
Genotype	TP	Early wilt % mean ± SE	CLS	Late wilt %	CLS	Combined wilt % age* mean ± SE	CLS	Genotype	TP	Early wilt % Mean ± SE	CLS	Late wilt %	CLS	Combined wilt %age* mean ± SE	CLS
K009-09	К	12.5±1.4***	Я	14.3±0.7***	R	25.0±2.4***	MR	BKK02231	К	25.0±2.2***	MR	***0 + 0	HR	25.0±2.4***	MR
K0010-09	Х	$11.1 \pm 1.0^{***}$	R	$12.5\pm0.6^{***}$	R	22.2±2.0***	MR	BKK07124	Х	22.2±2.6***	MR	***0 + 0	HR	$22.2\pm 2.0^{***}$	MR
K0021-09	К	$10.0\pm0.4^{***}$	HR	$11.1\pm0.1^{***}$	R	$20.0\pm1.4^{***}$	R	BKK07151	К	$0.0\pm0.0^{***}$	HR	$11.1\pm0.6^{***}$	R	$11.1\pm1.1^{***}$	R
K0025-09	К	$0.0\pm0.0^{***}$	HR	$0\pm 0^{***}$	HR	$0.0\pm0.0^{***}$	HR	BKK02182	К	$40.0\pm 2.9^{***}$	\mathbf{s}	$33.3\pm0.6^{***}$	\mathbf{s}	60.0±2.7***	HS
K0026-09	Х	$33.3\pm 2.3^{***}$	S	$16.7\pm0.9^{***}$	R	$44.4{\pm}1.7{***}$	S	CH 65/02	Х	57.1±1.4***	SH	***0 * 0	HR	57.0±3.2***	SH
K0030-09	К	$28.6 \pm 4.1^{***}$	MR	20±0.7***	R	42.9±2.2***	S	CH82/02	К	40.0±2.2***	\mathbf{s}	***0 + 0	HR	$40.0\pm 5.0^{***}$	\mathbf{s}
K0031-09	К	40.0±5.7***	S	$16.7\pm 0.8^{***}$	R	50.0±2.5***	S	CH 38/03	К	$0.0\pm0.0^{***}$	HR	$^{**0}_{0}$	HR	$0.0\pm0.0***$	HR
K0034-09	К	$10.0\pm0.1^{***}$	HR	$11.1\pm0.7^{***}$	R	$20.0\pm1.2^{***}$	R	CH 47/04	К	$0.0\pm0.0^{***}$	HR	$***0 \pm 0$	HR	$0.0\pm0.0***$	HR
K0035-09	К	37.5±2.7***	\mathbf{S}	$40\pm0.9^{***}$	\mathbf{v}	62.5±2.6***	SH	FG0901	К	$0.0\pm0.0^{***}$	HR	***0 + 0	HR	$0.0\pm0.0***$	HR
K0039-09	К	12.5±4.3***	R	$0 \pm 0^{***}$	HR	12.5±1.5***	R	FG0902	К	50±3.3***	\mathbf{S}	$20\pm0.6^{***}$	R	$60.0\pm 6.3*$	SH
K0051-09	K	$28.6 \pm 1.8^{***}$	MR	$^{***}0^{\mp 0}$	HR	28.6±3.8***	MR	D075-09	D	$0.0\pm0.0^{***}$	HR	***0 7 0	HR	$0.0\pm0.0^{***}$	HR
K0054-09	Х	16.7±2.1***	R	$20\pm0.8^{***}$	R	33.3±2.6***	S	D080-09	D	20.0±1.7***	Ч	***0 + 0	HR	20.0±1.2***	R
K0057-09	К	$12.5\pm1.9^{***}$	R	$28.6\pm1.1^{***}$	MR	37.5±1.4***	S	D084-09	D	$11.1 \pm 3.3 * * *$	К	***0 + 0	HR	$11.1\pm 2.2^{***}$	R
K0058-09	К	$10.0\pm1.3^{***}$	HR	$^{***}0^{\mp 0}$	HR	$10.0\pm0.6^{***}$	HR	D085-09	D	$0.0\pm0.0^{***}$	HR	$11.1\pm0.6^{***}$	R	$11.1\pm 1.1^{***}$	R
K0062-09	К	$11.1 \pm 1.3^{***}$	К	$12.5 \pm 1.2^{***}$	R	22.2±2.7***	R	D089-09	D	$0.0\pm0.0^{***}$	HR	***0 + 0	HR	$0.0\pm0.0^{***}$	HR
K0063-09	Х	$11.1 \pm 1.1^{***}$	К	62.5±1.5***	SH	$66.7 \pm 1.7 * * *$	HS	D090-09	D	$11.1 \pm 1.0^{***}$	R	***0=0	HR	$11.1\pm0.6^{***}$	R
K0065-09	К	$0.0\pm0.0^{***}$	HR	$11.1\pm0.5^{***}$	R	$11.1\pm 1.4^{***}$	R	D094-09	D	$0.0\pm0.0^{***}$	HR	***0平0	HR	$0.0\pm0.0^{***}$	HR
K0066-09	К	$11.1\pm0.9^{***}$	К	**0	HR	$11.1 \pm 1.4^{***}$	R	D095-09	D	$11.1\pm0.7^{***}$	R	***0 + 0	HR	$11.1\pm0.4^{***}$	R
K0068-09	К	$0.0\pm0.0^{***}$	HR	$0 \pm 0^{***}$	HR	$0.0\pm0.0^{***}$	HR	D096-09	D	$0.0\pm0.0^{***}$	HR	***0 + 0	HR	$0.0\pm0.0^{***}$	HR
K0069-09	Х	$0.0\pm0.0^{***}$	HR	$11.1 \pm 1.2^{***}$	R	$11.1\pm 1.6^{***}$	R	D098-09	D	$11.1\pm0.6^{***}$	R	12.5±0.3***	R	22.2±4.5***	MR
K0070-09	К	$70.0\pm6.3*$	SH	$33.3\pm0.9*$	S	80.0±2.7***	HS	D0100-09	D	$0.0\pm0.0^{***}$	HR	$20\pm0.8^{***}$	R	$20.0\pm4.4^{***}$	R
BKK17115	К	$50.0\pm 1.8^{***}$	S	50±0.9***	S	$75.0\pm1.4^{***}$	HS	BK07A005	D	$33.3\pm 2.1^{***}$	\mathbf{s}	$50\pm0.6^{***}$	\mathbf{s}	66.7±5.9*	HS
BKK17106	Х	62.5±2.7***	SH	$66.7\pm1.6^{***}$	SH	87.5±1.9***	HS	BK96A2055	D	$0.0\pm0.0^{***}$	HR	***0 + 0	HR	$0.0\pm0.0^{***}$	HR
BKK17124	К	$50.0\pm1.7^{***}$	S	$50\pm0.6^{***}$	S	75.0±3.3***	SH	BK05A015	D	$50.0\pm2.1^{***}$	S	***0=0	HR	$50.0\pm1.8^{***}$	\mathbf{s}
BKK02174	Х	$40.0\pm0.9^{***}$	S	**0	HR	40.0±2.5***	\mathbf{s}	BK04A013	D	62.5±2.3***	SH	100 ± 0 ns	SH	$100.0{\pm}0.0^{\rm ns}$	SH
BKK02209	Х	12.5±2.2***	К	$^{***}0^{\mp 0}$	HR	12.5±1.2***	R	FG-0904	D	12.5±1.2***	К	$14.3\pm0.6^{***}$	К	$25.0\pm 5.1^{***}$	MR
BKK02213	К	50.0±2.5***	S	$^{***}0^{\mp 0}$	HR	$50.0\pm1.2^{***}$	s	FG-0908	D	$0.0\pm0.0***$	HR	***0 + 0	HR	$0.0\pm0.0***$	HR
								AUG-424		90 ,0±0.6	HS	100.0 ± 0.0	HS	100.0 ± 0.0	HS
*Combined w	/ilt % v	vas calculated by	using tot	tal early and late	wilted pl	ants divided by th	ne total g	erminated plants							
Classification	TP-T:	ype, K-Kabuli, C	LS-class.	, HR-highly resis	stant, R-re	esistant, MR-mod	lerately r	esistant, S-susce	ptible,	HS-highly suscep	otible				
*, ** and ***	indica	te Significance at	0.05, 0.0	01 and 0.001 prol	bability h	evels., SE= stand	lard erroi	L							

2446

Class	Genotypic frequency	Name of genotypes
HR	19	K0021-09, K0025-09, K0034-09, K0058-09, K0065-09, K0068-09, K0069-09, BKK07151, CH 38/03, CH 47/04, FG0901, D075-09, D085-09, D089-09, D094-09, D096-09, D0100-09, BK96A2055 & FG-0908
R	15	K009-09, K0010-09, K0039-09, K0054-09, K0057-09, K0062-09, K0063-09, K0066-09, BKK02209, D080-09, D084-09, D090-09, D095-09, D098-09 & FG-0904
MR	4	K0030-09, K0051-09, BKK02231 & BKK07124
S	12	K0026-09, K0031-09, K0035-09, BKK17115, BKK17124, BKK02174, BKK02213, BKK02182, CH82/02, FG0902, BK07A005 & BK05A015
HS	4	K0070-09, BKK17106, CH 65/02 & BK04A013

Table 4. Summary of grouping of early response of chickpea advance genotypes against r usu tunt	ance genotypes against <i>Fusarium</i> with	Ivance g	ipea adv	nicki	ot c	y response	c early	grouping of	Summary of	e 4. S	abi	1
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 Class	frequency	Name of genotypes
HR	27	K0025-09, K0039-09, K0051-09, K0058-09, K0066-09, K0068-09, BKK02174, BKK02209, BKK02213, BKK02231, BKK07124, CH 65/02, CH 82/02, CH 38/03, CH 47/04, FG0901, D075-09, D080-09, D084-09, D089-09, D090-09, D094-09, D095-09, D096-09, BK96A2055, BK05A015 & FG-0908
R	17	K009-09, K0010-09, K0021-09, K0026-09, K0030-09, K0031-09, K0034-09, K0054-09, K0062-09, K0065-09, K0069-09, BKK07151, FG0902, D085-09, D098-09, D0100-09 & FG-0904
MR	1	K0057-09
S	6	K0035-09, K0070-09, BKK17115, BKK17124, BKK02182 & BK07A005
HS	3	K0063-09, BKK17106 & BK04A013

Table 5. Summary of grouping of late response of chickpea advance genotypes against Fusarium wilt.

Table 6. Summary of grouping of combined response of chickpea advance genotypes against Fusarium wilt.

Class	frequency	Name of genotypes
HR	12	K0025-09, K0058-09, K0068-09, CH 38/03, CH 47/04, FG0901, D075-09, D089-09, D094-09, D096-09, BK96A2055 & FG-0908
R	15	K0021-09, K0034-09, K0039-09, K0062-09, K0065-09, K0066-09, K0069-09, BKK02209, BKK07151, D080-09, D084-09, D085-09, D090-09, D095-09 & D0100-09
MR	7	K009-09, K0010-09, K0051-09, BKK02231, BKK07124, D098-09 & FG-0904
S	9	K0026-09, K0030-09, K0031-09, K0054-09, K0057-09, BKK02174, BKK02213, CH82/02 & BK05A015
HS	11	K0035-09, K0063-09, K0070-09, BKK17115, BKK17106, BKK17124, BKK02182, CH 65/02, FG0902, BK07A005 & BK04A013

Screening against wilt: Wilt incidence differ significantly during early and late season of the crop. Early wilt percentage ranged from 10 to 70%, late and combined from 11.1 to 100% (Table 3). Comparatively, wilting of plants during the late season was less than in early spread with prevailing optimum conditions for the development of the pathogen.

Constrais

Early wilt results showed that 19 genotypes were highly resistant, 15 were resistant, 4 were moderately resistant and 12 were susceptible and 4 were highly susceptible (Table 4). However, wilting in late season indicate higher frequency of resistant genotypes (Table 5) due to low pathogen infection. The less wilting during later stages might be due to variable response of some genotypes that were susceptible in early wilting but resistant during late wilting or unfavorable weather conditions may prevail during later stages inhibiting the development of the pathogen. It was observed that the genotypes K0026-09, K0031-09, BKK02174, CH 65/02, CH82/02, FG0902 and BK05A015 were susceptible during early response whereas in late season same genotypes were resistant but susceptible in combined wilt response (Table 3). The genotypes K0070-09, BKK17106, CH 65/02 and BK04A013 were highly susceptible during early pathogen infection at seedling stage while the genotypes K0063-09, BKK17106 & BK04A013 were susceptible during late season. It could be argued that the genotypes prone to early wilt must not be planted in early wilt prone areas. Combined (Total) effect of wilt incidence is given in Table 6. In view of combined wilt genotypes viz., K0058-09, K0025-09, K0068-09, CH 38/03, CH 47/04, FG0901, D075-09, D089-09, D094-09, D096-09, BK96A2055 and FG-0908 had no symptoms of wilting, therefore categorized as highly resistant. The genetic mechanism of disease resistance for these genotypes is quite stable in early and late season i.e., showing same response in all three wilt categories. The results are partially in line as reported earlier by Sarwar et al. (2012).

Conclusion

Chickpea yield per unit area is hampered every year by the spread of these two most destructive diseases. Options available so far are the management or to use cultivars having stable resistance to these diseases. Results from present study revealed that considerable variation was found for resistance against the above mentioned diseases which could be exploited as direct sources or may be transferred through hybridization to high yielding but disease susceptible genotypes. Desi germplasm proved to be better source of resistance compared to the Kabuli material.

Acknowledgements

This study is a part of a research project entitled, 'Improvement of chickpea productivity through the identification of drought tolerant and disease resistant chickpea genotypes for marginal lands' funded by the Punjab Agricultural Research Board, Lahore through a research grant PARB 120. We are thankful to the PARB for providing the financial support.

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(Received for publication 16 October 2014)