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EVALUATION OF CHICKPEA GERMPLASM FOR WILT RESISTANCE

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Abstract

Fusarium wilt caused by *Fusarium oxysporum* f.sp. *ciceri* is a devastating disease of chickpea in Pakistan. In the present study one hundred and fifty eight genotypes of recent origin were evaluated under artificial disease condition to identify genetic sources of resistance against this disease. The experiment was planted in an augmented design with single replication. Disease observations were recorded at seedling and reproductive stages. There was a considerable variation between genotypes with respect to their disease reaction at both stages of evaluation. At seedling stage the disease incidence ranged from 0% to 57.2% and at reproductive stage it varied from 0% to 100%. At seedling stage, 107 genotypes exhibited resistant response, 29 were tolerant and 22 were susceptible. On the contrary, only 3 genotypes with disease incidence 0%, 6.7% and 8.3% were resistant, 4 with disease incidence of 18.2 to 20% were tolerant and 151with disease incidence of 25% to 100% were susceptible at reproductive stage. Three genotypes, F98-75C, F98-181C and F98-193C were consistently resistant at both stages. Three genotypes (CM32/91 X PAIDAR 91, F98-166c, and F98-28C) were resistant at seedling stage and tolerant at reproductive stage whereas a single genotype (ICC 11514 x ICC422 X C44) was consistently tolerant. These genotypes may be exploited in breeding programs to pyramid resistant genes.

Introduction

Chickpea is the most important pulse crop of Pakistan. The 5 years average data shows that it is annually cultivated on 1074 thousands hectares with 615 kg/hectare yield and 660.7 thousand tones production (Anon., 2000). The productivity of chickpea in Pakistan is below world average, and has been uncertain, erratic and low amounting to only about 10% of the world’s produce (Auckland & Van-der-Maesan, 1980). One of the factors responsible for low yield is the occurrence of diseases particularly the wilt caused by *Fusarium oxysporum* Schlecht. Emend Snyd. & Hans. f.sp. *ciceri* Padwick. It is a serious disease of chickpea in India. Iran, Pakistan, Nepal, Burma, Spain and Tunisia and has also been reported from Bangladesh, Ethiopia, Malawi, Mexico, Peru, Syria, the USA (Nene *et al*., 1984). The yield losses due to this disease may vary from 10-90% (Jimenez-Diaz *et al.*, 1989, Ratnaparkhe *et al*., 1998). According to an estimate the annual loss of US $ 1 million may be caused by this disease in Pakistan (Sattar *et al*., 1953). Wilt has reduced the share of chickpea from 50% in 1950s to 10% in 1990s on irrigated lands in Pakistan (Hanif *et al*., 1999). An annual yield loss of 12-15% in chickpea, caused by wilts and root rot, in Spain was estimated by Trapero-Casas & Limenez-Diaz (1985). The production of chickpea in California declined largely because of chickpea wilt (Buddenhagen *et al*., 1988). At ICRISAT, it was found that early wilting causes more loss than late wilting and the seeds harvested from late wilted plants were less heavy and dull than that from healthy plants (Haware & Nene, 1980). At least 7 races of fungus causing wilt disease have been reported (Haware & Nene, 1982b; Philips, 1988; Jimenez-Diaz *et al*., 1989). However, no information on existence of races in Pakistan is available despite the variation in isolates collected from different sites (Iftikhar *et al*., 2002).

Chemical control of wilt is not much effective and economical because the pathogen is soil as well as seed-borne in nature and is difficult to eradicate. Fungal chlamydospores survive in soil up to 6 years even in the absence of the host plants (Haware *et al*., 1996). The use of resistant cultivars is the best and the cheapest method to minimize losses caused by wilt. There is no reliable information in the literature on resistant sources against Pakistani isolates of fungus causing wilt in chickpea. The present study was therefore, undertaken to evaluate the newly developed genotypes of chickpea for resistance against local isolates of wilt fungus.

Materials and Methods

One hundred and fifty eight genotypes of diverse origin, obtained from various sources constituted the experimental material of this study. These genotypes were planted in a wilt sick plot at Nuclear Institute for Agriculture and Biology (NIAB), Faisalabad. A mixture of various isolates of wilt fungus was used to develop this wilt sick plot. The experiment was planted on 14th of October 2001 in an augmented design with a single replication. Each genotype was planted in a 4m long single row plot. Row to row and plant-to-plant distance were respectively maintained at 30cm and 10cm. A highly wilt susceptible genotype, AUG424, was repeatedly planted after every two test entries. The disease data were recorded at two stages of plant growth i.e. at seedling stage and at reproductive stage (near physiological maturity). The data on wilted plants of test entries at seedling stage were recorded when 100% killing of the susceptible check had occurred. The second stage data on wilted plants were recorded at the initiation of physiological maturity. The wilt incidence of each entry was calculated by the following formula:

$$\text{Wilt incidence} = \frac{\text{No. of plants wilted}}{\text{Total number of plants}} \times 100$$

The level of resistance and susceptibility of each test entry was determined by using the disease rating scale of Iqbal *et al*., (1993) where genotypes with 0-10% disease were rated as resistant, with 11-20% disease as tolerant and with above 20% disease as susceptible.

Results

The disease incidence of 158 entries at seedling and reproductive stage is presented in Table 1. There was a significant variation between genotypes for their disease reaction. Disease incidence at seedling stage varied from 0% to 66% whereas at reproductive stage it ranged from 6.7% to 93.3%. On average basis, 8.82% disease incidence was recorded at early stage and 58.73% at late stage (Fig. 1 & 2). There was no relationship between disease incidence at early and late stage (Fig. 3). The categorization of germplasm showed that at seedling stage 107 genotypes were resistant, 29 were tolerant and 22 were susceptible. On the other hand, at late stage 3 genotypes were resistant, 4 were tolerant and 151 were susceptible (Table 2). The disease incidence at physiological maturity stage increased invariably in all the genotypes as compared to that at seedling stage except for two genotypes in which it remained stable at 7.1 and 8.3 (Table 2). The disease incidence of tolerant genotypes (Table 3) showed that increase in their disease rating ranged from 0% (at seedling stage) to 20% (at reproductive stage).

Table 1. Disease rating of various chickpea genotypes to wilt disease at two stages.

S. No	Entries	Disease rating at seedling stage		Disease rating at flowering stage	
1.	(NEC-138-2 X E100YM) X C44	00.0	R	46.7	S
2.	(1CC11514 X 1CC422) X C44	13.3	T	20.0	T
3.	(C44 X E100YM) X NIFA-88	00.0	R	28.6	S
4.	(C44X1CC7770) X Parbat	06.7	R	46.7	S
5.	(CM72 X ICC11514) X ILC482	06.7	R	40.0	S
6.	(CM72 X ILC3279-195) X C44	13.3	T	67.7	S
7.	42-R 11042	00.0	R	42.9	S
8.	8612 X CM88	00.0	R	75.0	S
9.	89021 X (E100YM X ILC482)	00.0	R	78.6	S
10.	89021XPB 91	14.2	T	42.9	S
11.	91A039	00.0	R	30.8	S
12.	A16 X 1CC 13497	7.10	T	57.1	S
13.	AZRI-BK-NO.5	20.0	T	33.3	S
14.	Bittle-98	21.4	S	64.2	S
15.	C44 X (1CC3856 X E100YM)	00.0	R	60.0	S
16.	C44 X (C44 X 1CC7770)	00.0	R	73.3	S
17.	C44 X (ILC3856 X E100YM/90)	00.0	R	80.0	S
18.	C44 X 1CC7770	06.7	R	60.0	S
19.	C44 X C44 X 1CC7770	20.0	T	53.3	S
20.	C44 X E100YM X Paidar-91	00.0	R	66.7	S
21.	C44 X ILC3856 X E100YM/90	07.7	R	84.6	S
22.	C89/90 X PB91	00.0	R	58.3	S
23.	CM 32-1/90 X Paidar-91	00.0	R	20.0	T
24.	CM 88 X (PK51814 X NEC138-2)	13.3	T	80.0	S
25.	CM 89/90 X PB 91	00.0	R	25.0	S
26.	CM 89-1/90 X PB91	00.0	R	40.0	S
27.	CM32-1/90 X Paidar 91	00.0	R	93.3	S
28.	CM72 X ICC11514 X ILC482	28.6	S	64.2	S
29.	CM72 X ICC7770	00.0	R	28.6	S
30.	CM87-1190 X PB91	07.1	R	50.0	S
31.	CMC 0211S	07.1	R	50.0	S
32.	CMC 129	00.0	R	80.0	S
33.	CMC 132T	21.4	S	50.0	S
34.	CMC 150M	13.3	T	66.7	S
35.	CMC 186M	06.7	R	46.7	S
36.	CMC 191S	28.6	S	50.0	S
37.	CMC 201S	00.0	R	73.3	S
38.	CMC 228S	00.0	R	80.0	S
39.	CMC 2305	23.1	S	61.5	S
40.	CMC 44 X 1CC14734	00.0	R	67.7	S
41.	CMC 44S	00.0	R	64.2	S

Table 1. (Cont'd.).

S. No	Entries	Disease rating at seedling stage		Disease rating at flowering stage	
42.	CMC 55S	00.0	R	53.3	S
43.	CMC 71M	00.0	R	64.3	S
44.	CMC 71S	00.0	R	60.0	S
45.	CMC 71T	07.1	R	57.1	S
46.	CMC 85M	00.0	R	93.3	S
47.	CMC 86M	00.0	R	80.0	S
48.	CMC 87	00.0	R	73.3	S
49.	CMC102S	00.0	R	33.3	S
50.	CMC114S	00.0	R	35.7	S
51.	CMC204S	00.0	R	66.7	S
52.	CMC32M	13.3	T	46.7	S
53.	CMC70T	06.7	R	60.0	S
54.	CMC71S	00.0	R	33.3	S
55.	CMC94M	07.7	R	46.2	S
56.	CMNK 287-3K	00.0	R	93.3	S
57.	E101 X PB 91	26.7	S	40.0	S
58.	FLIP 82-150C	66.2	S	33.3	S
59.	FLIP 97-11C	28.6	S	64.2	S
60.	FLIP 97-121C	00.0	R	93.3	S
61.	FLIP 97-135C	00.0	R	100	S
62.	FLIP 97-159C	06.7	R	93.3	S
63.	FLIP 97-168C	06.7	R	80.0	S
64.	FLIP 97-168C	00.0	R	73.3	S
65.	FLIP 97-217C	00.0	R	100	S
66.	FLIP 98-166C	06.7	R	20.0	T
67.	FLIP 98-174C	16.7	T	83.3	S
68.	FLIP 98-175C	40.0	S	06.7	R
69.	FLIP 98-181C	00.0	R	06.7	R
70.	FLIP 98-185C	28.6	S	71.4	S
71.	FLIP 98-193C	08.3	R	08.3	R
72.	FLIP 98-198C	28.6	S	71.4	S
73.	FLIP 98-20C	20.0	T	80.0	S
74.	FLIP 98-222C	50.0	S	50.0	S
75.	FLIP 98-28C	00.0	R	18.2	T
76.	FLIP 98-75C	07.1	R	07.1	R
77.	FLIP 98-79C	00.0	R	85.7	S
78.	FLIP 98-80C	18.2	T	81.8	S
79.	ICI4641 X CMC-14	00.0	R	26.7	S
80.	L8612 X PK51949	07.1	R	35.7	S
81.	L89120 X PK51929	00.0	R	26.7	S

Table 1. (Cont'd.).

S. No	Entries	Disease rating at seedling stage		Disease rating at flowering stage	
82.	NCS 950018	00.0	R	80.0	S
83.	NCS 950115	00.0	R	67.7	S
84.	NCS 950145	00.0	R	46.7	S
85.	NCS 950176	06.7	R	73.3	S
86.	NCS 950201	00.0	R	67.7	S
87.	NCS 950204	00.0	R	53.3	S
88.	NCS 950212	00.0	R	60.0	S
89.	NCS 9901	06.7	R	80.0	S
90.	NCS 9909	00.0	R	100	S
91.	NCS 9910	00.0	R	26.7	S
92.	NCS 9912	06.7	R	73.3	S
93.	NCS 9916	21.4	S	78.6	S
94.	NCS 9917	06.7	R	60.0	S
95.	NCS 9918	14.2	T	85.7	S
96.	NCS 9919	21.4	S	71.4	S
97.	NCS 9921	00.0	R	93.3	S
98.	NCS 9922	33.3	S	60.0	S
99.	NCS 9923	00.0	R	61.5	S
100.	NCS 9927	20.0	T	67.7	S
101.	NCS950021	00.0	R	40.0	S
102.	NCS950048	07.1	R	50.0	S
103.	NCS950079	00.0	R	85.7	S
104.	NCS950145	00.0	R	26.7	S
105.	NCS950185	07.1	R	71.4	S
106.	NCS950189	00.0	R	71.4	S
107.	NCS950195	06.7	R	33.3	S
108.	NCS950204	06.7	R	60.0	S
109.	NCS950208	00.0	R	21.4	S
110.	NCS950209	00.0	R	46.7	S
111.	NCS950219	14.2	T	35.7	S
112.	NCS950220	06.7	R	26.7	S
113.	NCS950222	06.7	R	26.7	S
114.	NCS950225	00.0	R	60.0	S
115.	NCS950235	00.0	R	83.3	S
116.	NCS950257	00.0	R	40.0	S
117.	NCS950258	13.3	T	46.7	S
118.	NCS950259	06.7	R	46.7	S
119.	NCS950264	13.3	T	33.3	S
120.	NCS95038	07.1	R	42.9	S
121.	NCS95079	06.7	R	40.0	S

Table 1. (Cont'd.).

S. No	Entries	Disease rating at seedling stage		Disease rating at flowering stage	
122.	NCS96001	00.0	R	26.7	S
123.	NCS9903	06.7	R	66.7	S
124.	NCS9904	00.0	R	60.0	S
125.	NCS9905	13.3	T	60.0	S
126.	NCS9906	00.0	R	71.4	S
127.	NCS9907	00.0	R	73.3	S
128.	NCS9908	06.7	R	66.7	S
129.	NCS9913	00.0	R	60.0	S
130.	NCS9914	00.0	R	46.7	S
131.	NCS9928	08.3	R	83.3	S
132.	NCS994	00.0	R	40.0	S
133.	NIFA 88 X (PK51814 X NEC138-2)	07.7	R	76.9	S
134.	NOOR-91	20.0	T	80.0	S
135.	PAIDAR 91 X 1CC11514 X ILC3279	13.3	T	67.7	S
136.	Paidar 91 X HI 11287	00.0	R	73.3	S
137.	Paidar-91 X CM3279	00.0	R	21.4	S
138.	PB 91 X 1CC13508	00.0	R	67.6	S
139.	PB91X (1CC11514 X ILC3279)	13.3	T	46.7	S
140.	PB91XParbat	00.0	R	40.0	S
141.	PRSI 830 X (C44 X E100YM/45) Parbat	06.7	R	86.7	S
142.	SEL 96 TH 11507	57.2	S	42.9	S
143.	SEL96 TH 11488	46.7	S	53.3	S
144.	X 98 T 82	00.0	R	66.7	S
145.	X 98 T 91	23.1	S	76.9	S
146.	X 98 TH 10	25.0	S	75.0	S
147.	X 98 TH 102	15.4	T	76.9	S
148.	X 98 TH 109	28.6	S	71.4	S
149.	X 98 TH 37	14.2	T	85.7	S
150.	X 98 TH 52	14.2	T	78.6	S
151.	X 98 TH 59	15.4	T	76.9	S
152.	X 98 TH 60	40.0	S	53.3	S
153.	X 98 TH 61	15.4	T	76.9	S
154.	X 98 TH 62	20.0	R	73.3	S
155.	X 98 TH 68	13.3	T	80.0	S
156.	X 98 TH 71	30.8	S	61.5	S
157.	X 98 TH 80	16.7	T	83.3	S
158.	X 98 TH 99	13.3	T	80.0	S

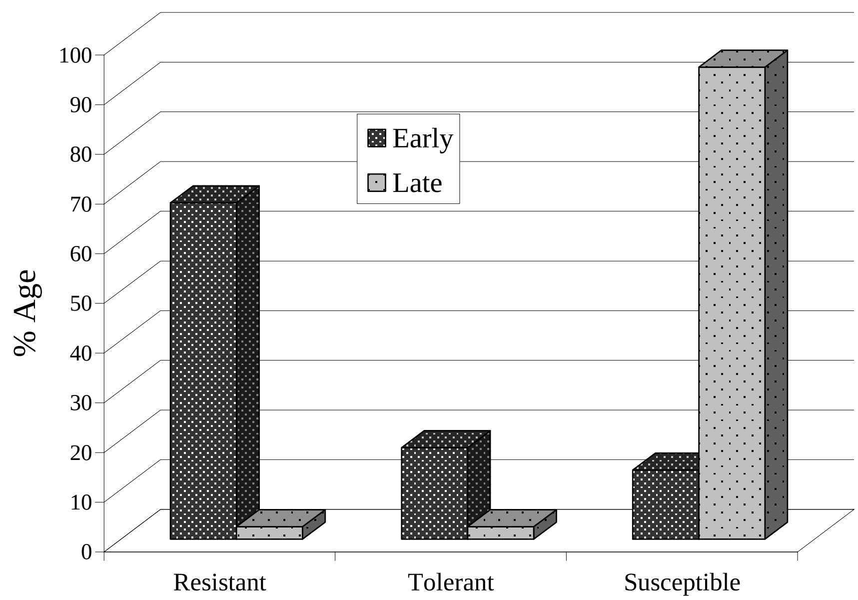

Fig. 1. Response of chickpea genotypes to wilt at seedling (early) and reproductive (late) stage.

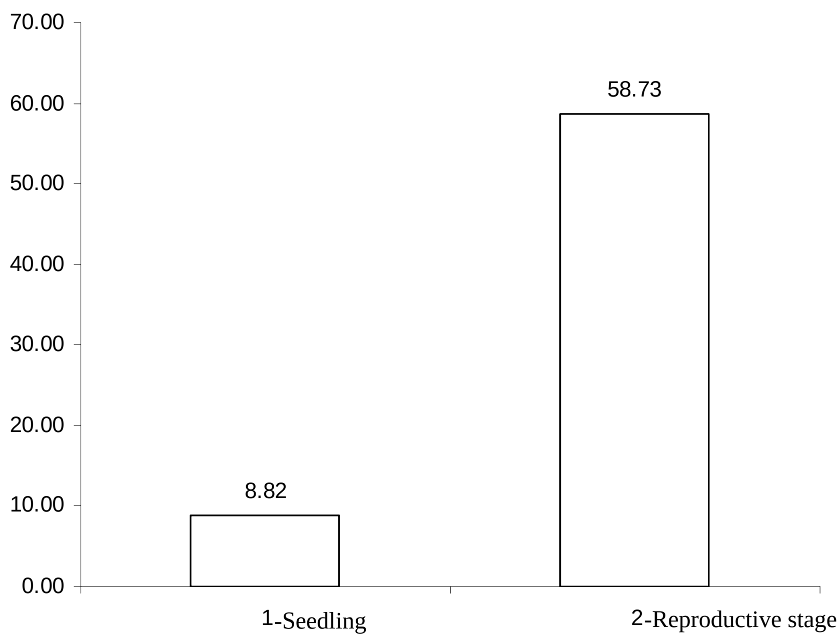

Fig 2. Disease incidences of chickpea wilt at two stages.

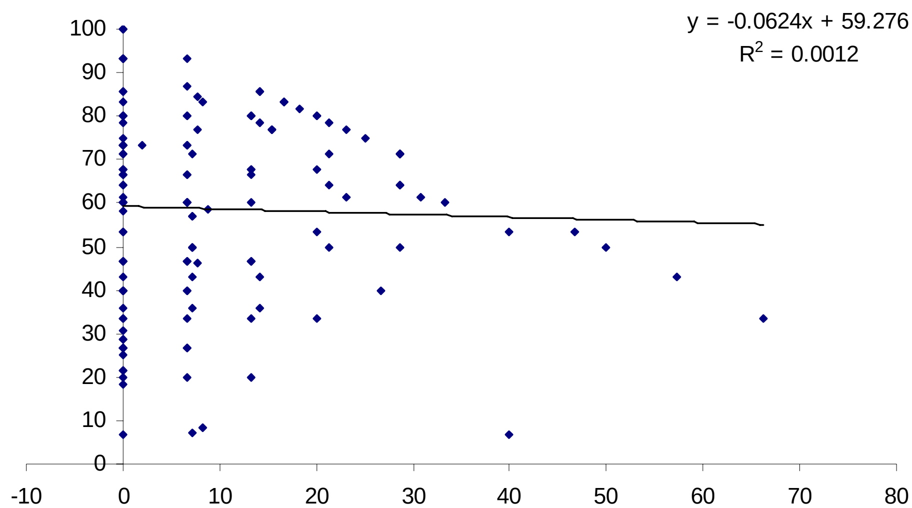

Fig. 3. Relationship between wilt disease severities at two stages.

Table 2. Classification of chickpea genotypes with respect to their wilt response at seedling and reproductive stage.

Disease incidence Category	Disease response	No. of genotypes in each category at seedling stage	No. of genotypes in each category at reproductive stage
0-10%	Resistant	107	3
11-20%	Tolerant	29	4
21-30%	Susceptible	11	10
31-40%	Susceptible	5	10
41-50%	Susceptible	1	17
51-60%	Susceptible	1	19
61-70%	Susceptible	0	20
71-80%	Susceptible	0	32
81-90%	Susceptible	0	10
91-100%	Susceptible	0	7
Total genotypes in 21-100% category	**Susceptible**	**22**	**151**

Table 3. Wilt reaction of selected genotypes at two stages.

Genotype	Wilt incidence at seedling stage	Wilt incidence at reproductive stage	Category of wilt reaction at seedling stage	Category of wilt reaction at reproductive stage
ICC11514XICC422XC44	13.3%	20%	Tolerant	Tolerant
CM32/92 X Paidar	0%	20%	Resistant	Tolerant
F98-166C	6.7%	20%	Resistant	Tolerant
F98-28C	0%	18.2%	Resistant	Tolerant
F98-75C	7.1%	7.1%	Resistant	Resistant
F98-181C	0%	6.7%	Resistant	Resistant
F98-193C	8.3%	8.3%	Resistant	Resistant

Discussion

Wilt caused by *Fusarium oxysporum* is a devastating disease of chickpea gaining importance day by day due to prevalence of drought conditions in the country. The chemical control of this disease is expensive and impractical because of the seed as well as soil borne nature of the fungus. Resistant cultivars are the most effective and cheap means of control (Jimenez-Diaz *et al*., 1992). The current study was conducted to identify resistant sources against the prevalent isolates of wilt existing in Pakistan. This study revealed a considerable variation for wilt incidence between the genotypes. Out of 158 genotypes studied, only 7 were either resistant or tolerant. The low number resistant lines may be attributed to the mixture of fungus isolates used for the development of wilt sick bed. The sources of resistance to *Fusarium* wilt in chickpea breeding materials are not uncommon and a number of workers have reported the occurrence of high level of resistance to *Fusarium* wilt (Pathak *et al*., 1982; Zote *et al*., 1983; Ahmad, 1990; Ahmad & Sharma, 1990; Kaushal & Singh, 1990; Reddy *et al*., 1990 & 1991; Iqbal *et al*., 1993; Iftikhar *et al*., 1997; Yu & Su, 1997). Zote *et al*., (1983) studied 42 lines of chickpea for the source of resistance to chickpea wilt in a wilt sick plot infested with *F. oxysporum* f.sp. *ciceri* and reported that none of the 42 lines was highly resistant. However, four developed less than 10% disease and six others developed less than 29% disease. Govil & Rana (1984) evaluated 239 cultivars representing a range of variability among Indian and Iranian germplasm in wilt sick plot for years. None was found to be immune but maximum resistance was shown by Indian cultivars such as P-597, P-621, P-3649, P-4128 and P-4245. Zote *et al*., (1986) reported that only five chickpea lines out of 15 tested for three successive years showed less than 10% wilt incidence. Khalid (1993) evaluated 122 test lines against *Fusarium* wilt under field conditions and found 37 of them to be resistant, while all the remaining test lines exhibited moderate resistance to highly susceptible reaction. Iftikhar *et al*., (1997) screened 31 chickpea germplasm lines received from ICARDA, and found all of them to be highly resistant to wilt disease.

It was obvious from our study that at seedling stage majority of the genotypes were resistant where as at reproductive stage majority of the genotypes appeared to be susceptible. Various workers have already reported variation in response of genotypes at two stages (Nene *et al*, 1981; Haware, 1992). They also reported that some of the sources were resistant against more than one race. However, these workers used different isolates and the genotypes from those used in the current study. On the other hand a high degree of variability has been reported between isolates of same race collected from different areas and between isolates of different races (Sivaramakrishnan *et al*., 2002). Similarly, the isolates from different areas of Pakistan were highly variable with respect to their virulence (Iftikhar *et al*., 2002). The variability in pathogen population in chickpea growing areas of Pakistan pose difficulties in developing stable varieties as they usually succumb to new isolates. Iftikhar *et al*., (1996) showed that resistant cultivars of chickpea did not maintain resistance across locations. The current study was made in a wilt sick plot created by the use of a mixture of isolates representing different chickpea areas. Therefore, the genotypes identified as resistant in this study will maintain their response across the locations. Most of the genotypes that showed resistant response at seedling stage appeared to be susceptible at physiological maturity stage. This phenomenon could be accounted for due to the prevalence of disease for a short period at seedling stage and for a long period at the reproductive stage. Since high temperature plays an important role for wilt development and the high temperature suitable for disease development prevailed for a short period at seedling stage due to onset of winter in December and it prevailed for a long time at reproductive stage due to onset of summer at the time of flower initiation. Therefore, disease prevailed for a longer time at 2nd stage of observation. Consequently, most of the genotypes that were resistant at seedling stage became susceptible at reproductive stage. This means that such genotypes required long wilting time. Therefore, the genotypes used in the present study may be divided into two categories, early wilting genotypes and late wilting genotypes. The resistant genotypes at seedling stage may be planted in those areas where disease prevalence occurs at seedling stage only. Delay in sowing can also help to escape disease in such areas. On the other hand the genotypes that showed resistance or tolerance at both the stages are most suitable for exploitation in breeding programs or for direct sowing in wilt prone areas. As the resistant genotypes expressed resistance against a mixture of isolates, they may posses multiple genes for resistance against this disease. Tullu (1996) reported variation in chickpea for wilting time. He also reported a genotype that was consistently and uniformly resistant. These findings are quite in line with our results obtained from this study. The susceptible genotypes at seedling stage may be categorized as early wilters and susceptible genotypes at reproductive stage may be classified as late wilters. The three genotypes that were consistently resistant at both stages against a mixture of isolates may be exploited in breeding programs aimed at development of wilt resistant varieties.

There was no association between disease severities at two stages (Fig 3). This indicated that different genotypes could be utilized according to prevalence of disease at various growth stages. The resistance of the local germplasm against *Fusarium* wilt can be exploited for the development of commercial cultivars possessing all other desirable agronomic traits.

References

Ahmad, S., S.P.S. Beniwal and N. Tadesse. 1990. Field screening of chickpea for resistance to wilt/root rots in Ethiopia. *International Chickpea Newslet*, 22: 34-36.

Ahmad, Q. and N.R. Sharma. 1990. Sources of resistance to fusarium wilt of chickpea in Bihar. *ICN*, 23: 20.

Anonymous. 1987. ICRISAT Annual Report.

Anonymous. 2000. *Agricultural Statistics of Pakistan*. Ministry of Food. Agriculture and Livestock. Government of Pakistan, Food and Agic. Div. (Planning Unit), Islamabad.

Auckland, A.K. and L.J.G. Van-der-Maesen. 1980. Chickpea-ICRISAT crop Sci. Soc. Of America, 667:249-259. Cf. *Pak. J. Agric. Res*., 2(4): 1981.

Buddenhagen, I.N., F. Workneh and Bosque-Perez. 1988. Chickpea improvement and chickpea diseases in California. *International Chickpea Newsletter*, 19: 9-10.

Govil, J.N. and B.S. Rana. 1994. Stability of host plant resistance to wilt (*Fusarium oxysporum* f.sp. *ciceri*) in chickpea. *International J. Trop. P1. Dis*., 2(1): 55-60.

Halila, H.M., H.E. Gridley and P. Houdiald. 1984. Sources of resistance to *Fusarium* wilt in Kabuli chickpeas. *International Chickpea newsletter*, 10: 13-14.

Haware, M.P. and Y.L. Nene. 1980. Influence of wilt at different stages on the yield loss in chickpea. *Tropical Grain Legume Bulletin* 19: 38-44.

Haware, M.P. and Y.L Nene. 1982. Races of *Fusarium oxysporum* f.sp. *ciceri*. *Plant Disease*, 66: 809-810.

Haware, M.P., Y.L. Nene and M. Natarajan. 1986. Survival of *Fusarium oxysporum* f.sp. *ciceri* in soil in absence of chickpea. In *Management of soil-borne diseases of crop plant*: Proceeding of National Seminar. 8-10 Jan 1986 Coimbatore, India: Tamil Nadu Agricultural University.

Haware, M.P., R.P.S. Pundir and J Narayana Rao. 1992. Screening of world chickpea germplasm for resistance to fussarium wilt. *Field Crop Sci*., 30: 147-154.

Haware, M.P., Y.L. Nene and M. Natarajan. 1996. Survival of *Fusarium oxysporum* f.sp. *ciceri*. *Plant Disease*, 66: 809-810.

Iftikhar, K., T. Khalid and M.B. Ilyas. 1997. Field screening of chickpea germplasm for the sources of resistance against *Fusarium* wilt. *Pak. J. Phytopathol*. 9(1): 31-33.

Iftikhar, A.K., A. Jabbar and S.S. Alam.2002. Genetic variation among *Fusarium oxysporum* F.sp. *ciceri* isolates in Pakistan as determined by biological pathotyping and vegetative compatibility. *Pak. J. Bot*., 34(4): 433-440.

Infantina, A., A. Porta-Pugalia and K.B. Singh. 1996. Screening wild Cicer species for resistance to *Fusarium* wilt. *Plant Dis*., 80: 42-44.

Iqbal, M.J., K. Iftikhar and M.B. Ilyas. 1993. Evaluation of chickpea germplasm for resistance against wilt disease (*Fusarium oxysporum*). *J. Agric. Res*., 31(4): 449-453.

Jimenez-Diaz, R.M., A Trapero-Casas and Cabrera de la J. Colina. 1989. Races of *Fusarium oxysporum* f.sp. *ciceri* infecting chickpeas in southern Spain. 515-520. In: *Vascular wilt diseases of plants* (Eds.): E.C. Tjamos and C. Beckman.. NATO ASI Series, vol. H28. Berlin, Federal Republic of Germany: North Atlantic Treaty Organization.

Kaushal, R.P. and B.M. Singh. 1990. Pot screening of chickpea germplasm against wilt. *ICN*, 23: 20.

Khalid, T. 1993. *Screening of chickpea germplasm and fungitoxicants against chickpea wilt disease*. M.Sc. Thesis, Deptt. P1. Pathol. Univ. Agric. Faisalabad, Pakistan.

Nash, S.M. and W.C. Snyder. 1962. Quantitative estimation by plate counts of propagules of the bean root rot *Fusarium* in field soils. *Phytopathology*, 52: 567-572.

Nene, Y.L., M.V. Reddy and R.P.S. Pundir 1981. *Sources of resistance to selected chickpea diseases*. ICRISAT Pulses pathology progress report No. 15, ICRISAT, Patancheru.

Nene, Y.L., V.K. Sheila and S.B. Sharma. 1984. *A world list of chickpea (Cicer arietinum* L.) and *pigeonpea (Cajanus cajan* L.) *pathogens*. ICRISAT Pulses Pathology progress report 32. 19 p.

Pathak, M.M., J.S. Sindhu, K.P. Singh and S.B. Lal Srivasta. 1982. Avarodhi, a wilt resistant variety of chickpea. *ICN*, 6: 9.

Phillips, J.C. 1988. A distinct race of chickpea wilt in California. *International Chickpea Newsletter*, 18: 19-21.

Reddy, M.V, T.N. Raju and Y.L. Nene. 1991. Evaluation of chickpea lines in all India coordinator varietal trials for wilt and root rots resistance. *ICN*, 23: 22.

Reddy, M.V, T.N. Raju and P.O.P. Pundi. 1990. Additional sources of resistance to wilt and root rots in chickpea. *ICN*, 12: 36.

Sattar, A., A.G. Arif and M. Mohyuddin. 1953. Effect of soil temperature and moisture on the incidence of gram wilt. *Pak. J. Sci. Res*., 5: 16-21.

Trapero-Casas, A. and R.M. Jimenez-Diaz. 1985. Fungal wilt and root rot diseases of chickpea in southern Spain. *Phytopathology*, 75: 1146 -1151.

Tullu, A. 1996. *Genetics of fusarium wilt resistance in chickpea*. Ph.D. dissertation. Crop and Soil Science Department, Washington Stat University, Pullman, WA, USA.

Yu, K.H. and T. Su. 1997. Pot screening of chickpea germplasm lines against wilt. *Internat. Chickpea. Newslett*., 4: 19-20.

Zote, K.K., P.V. Khalikar and B.P. Dandnaik. 1983. Source of resistance to chickpea wilt. *Internat. Chickpea Newslett*. No. 8(23): Cf: Rev. P1. Path. 63(5): 20-95, 1984.

Zote, K.K., P.V. Khalikar and B.P. Dandnaik. 1986. Reactions of chickpea varieties to *Fusarium* wilt. *Ind. J. Mycol. & P1. Pathol*., 16(1): 80-81

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