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Screening for Resistant Sources in Chickpea Accessions against Fusarium Wilt

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Abstract: ***One hundred one genotypes of chickpea were screened for resistance to fusarium wilt disease caused by Fusarium oxysporum f. sp. Ciceri at Student's Instructional Farm of Narendra Deva University of Agriculture and Technology, Narendra Nagar, Kumarganj, Faizabad (U.P.) during Rabi, 2014-15. It was observed that 57 lines were resistant, 28 were tolerant while 16 were susceptible to the wilt disease at seedling stage. Whereas, 31 genotypes were resistant, 26 were tolerant and 44 were susceptible at reproductive stage. On an average basis 56.44% disease resistance was recorded at early stage and 30.69% at reproductive stage, whereas 15.84% disease incidence was observed at seedling stage and 43.56% at reproductive stage. The disease incidence of tolerant genotypes was screened at seedling stage of 27.72% and 25.74% at reproductive stage.***

Keywords: *Chickpea; Fusarium oxysporum; resistance.*

1. Introduction

Chickpea (*Cicer arietinum* L.) is the most important pulse crop of India. It is an important source of human food and animal feed that also helps in the management of soil fertility particularly in dry lands. It can be a promising alternative crop for rotation with barley, peas and wheat in dry land areas. Chickpea is also known as King of pulses. In India, total pulses are grown on an area of 23.47 m ha with production of 18.34 m t and productivity of 751 kg/ha in 2012-13. Chickpea (*Cicer arietinum* L.) is the premier pulse crop of India covering 9.51 million hectares area and production contributing 8.83 million tones with the productivity of 929 kg/ha . The area, production and productivity of Utter Pradesh has been possessed 604.00 thousand ha, 732.00 thousand tones, 1212 kg/ha respectively in year 2012-13 (Anonymous, 2013).

Chickpea wilt (*Fusarium oxysporum* f. sp. *Ciceri*) is very common seed/ soil born disease causing 10-12% annual loss in India. It is typical vascular disease causing xylem necrosis the disease is systemic in nature and plants may be infected at any stage. The fungus can survive in soil up to six years even in absence of host. It is widely spread disease covering all major chickpea growing states. Wilt pathogen can destroy the crop completely or cause significant annual yield losses; however, its prevalence is less common where cold temperature persists for longer period. Since spores of the fungus are found in soil. Presence of sufficient soil moisture and 20-30^{0}C temperature can cause fast spread of disease. In case, fungus is present in soil and infected seeds have been sown, then crop has to face severe damage. Early wilting causes more loss than late wilting. The most practical cheapest, economical and ideal way of managing chickpea wilt, is the use of resistant cultivars. **(Nene & Haware, 1980 and Iqbal *et al.*, 2005).** Present study was undertaken to evaluate the genotypes of chickpea for resistance against wilt fungus in order to identify new genetic sources of resistance.

2. Material and Methods

One hundred one genotypes procured from Pulse section; Department of Genetics and Plant Breeding N.D.U.A.&T. Kumarganj Faizabad (UP) were screened for their level of resistance/susceptibility against *Fusarium* wilt under field condition at Student's Instructional Farm. Each genotype was planted in augmented block design with two replications with susceptible check **JG 62** which, repeatedly planted after every two test entries and the experimental plot was also surrounded by two rows of JG 62 to ensure uniform spread of the disease. Plot size was kept 4 m whereas; row to row and plant to plant distances were maintained at 30 cm and 10 cm, respectively. Data on the number of wilted plants in each pot for each test line 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 killing of the susceptible check had occurred. The second stage data on wilted plants were recorded at the initiation of physiological maturity. At reproductive stage data on wilted plants of test entries were recorded at 100% killing of the susceptible check. The level of resistance and susceptibility of each test line was determined by using 1-9 rating scale given by **(Iqbal *et al.*, 2005)**; where 1=highly resistant (0-10% plants wilted), 3=resistant (11-20% plants mortality), 5=moderately resistance (21-30% mortality), 7=susceptible (31-50% mortality) and 9= highly susceptible (more than 50% mortality).

The wilt incidence per cent of each test entry was calculated by the following formula:

$$\text{Wilt incidence (\%)} = \frac{\text{Number of plants wilted}}{\text{Total number of plants}} \times 100$$

3. Results and Discussion

The disease incidence of 101 chickpea genotypes was recorded at seedling and reproductive stage (Table 1). The

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results showed that out of 101 accessions, none was free from infection thus on the basis of disease incidence these chickpea lines were grouped in three categories. It was observed that 57 lines were resistant, 28 were tolerant while 16 were susceptible to the wilt disease at seedling stage. Whereas, 31 genotypes were resistant, 26 were tolerant and 44 were susceptible at reproductive stage. The disease incidence at physiological maturity stage increased invariably in all the genotypes as compared to that at seedling stage. On an average basis 56.44% disease resistance was recorded at early stage and 30.69% at reproductive stage, whereas 15.84% disease incidence was observed at seedling stage and 43.56% at reproductive stage. The disease incidence of tolerant genotypes were screened at seedling stage was 27.72% and at reproductive stage it was 25.74%. The results showed that chickpea accessions had significant genetic variation between genotypes for their disease reaction at two stages i.e., at seedling stage and reproductive stage. Our study revealed that at seedling stage majority of the genotypes were resistant whereas, at reproductive stage majority of the genotypes appeared to be susceptible. Variation in wilt resistance at two stages was also reported by **(Ahmad Ansar *et al*., 2010 and Chaudhry *et al.*, 2006)**. Most of the genotypes that showed resistant response at seedling stage appeared to be susceptible at physiological maturity stage. Although little information on the mechanism of resistance is available, a detailed research based on this material is needed to throw light on it. Fifty seven accessions showing resistance reaction at seedling stage and thirty one at reproductive stage may be utilized in breeding programme to develop resistant/tolerant varieties against fusarium wilt disease. Development of disease is slow in resistant lines and fast in susceptible lines. As the resistant lines at reproductive stage also became susceptible thus field screening at reproductive stage seems to be more reliable.

4. Acknowledgment

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Table No.1: Reaction of 101 chickpea accessions against wilt disease during *Rabi*, 2014-15.

Disease reaction	No. of Entries		Accession	
	Seedling stage	Reproductive stage	Seedling stage	Reproductive stage
Immune (No disease)	0	0	Nil	
Resistant (1-3)	57	31	Avrodhi, DCP 92-3, H 82-2, HK 94-134, ICCV 2, JG 11, JG 315, JG 322, KWR 108, SAKI 9516, Vijay, BG 1053, BG 1088, BG 209, GCP 101, GCP 105, GNG 663, GNG 146, JAKI 92-18, JG 1, JG 16, NDG 54, NDG 93-1, Pant G 186, RSG 888, Udai, Vishal, NDG 3-32-1, KGD 1209, JGK 1, IPC 2004-52, IPC 2008-36, IPCK 2008-36, IPCK 2005-23, IPCK 2005-62, BG 3012, BG 2030, CSJ 592, JSC 35, IPC 2005-74, IPC 2005-59, GNG 1958, GNG 1888, HK 06-152, GL 26054, GJG 0714, BGM 547, BGM 570, CSJK 18, BG 3004, BGD 1053, BGD 1066, GNG 1999, H 06-63, JG 74, ICCV 10, BG 3002	Avrodhi, DCP 92-3, H 82-2, HK 94-134, ICCV 2, JG 11, JG 315, SAKI 9516, Vijay, GCP 101, GNG 663, GNG 146, JAKI 9218, JG 16, NDG 54, RSG 888, Vishal, NDG 3-32-1, KGD 1209, JGK 1, IPC 2004-52, IPCK 2008-36, CSJ 592, JSC 35, IPC 2005-74, GNG 1888, CSJK 18, BGD 1053, BGD 1066, GNG 1999, JG 74

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Tolerant (4-5)	28	26	BG 2053, BG 3003, KGD 1249, NDG 30, NDG 31, NDG 5-21, Rajas, NDG 97-1, IPCK 2006-56, IPCK 441, ICCV 37, HK 06-153, HK 06-163, H 0449, GNG 1996, GNG 1947, GL 21107, CSJ 610, CSG 8962, CSJK 24, GJG 0809, GJG 0814, BGD 1057, BGD 1060, GNG 0703, HC 3, H 07-157, IPC 2006-19	BGM 547, H 06-63, JG 1, KWR 108, CSG 8962, IPCK 2005-23, IPC 2005-59, BGM 570, GL 26054, GJG 0714, NDG 93-1, BG 2030, IPC 2008-36, ICCV 10, HK 06-152, BG 3002, Pant G 186, BG 1088, BG 209, BG 2053, H 0449, CSJK 24, HK 06-153, BGD 1057, GJG 0809, NDG 31
Susceptible (6-9)	16	44	**JG 62**, K 850, BG 1003, GCP 107, BG256, C 235, BG 267, BG 329, Viswas, BG 362, BG 372, GNG 469, JG 218, L 550, RSG 44, Vikas	**JG 62**, JG 322, BG 3004, Udai, GCP 105, IPCK 2005-62, BG 1053, GNG 1958, BG 3012, BG 3003, KGD 1249, NDG 30, NDG 5-21, Rajas, NDG 97-1, IPCK 2006-56, IPCK 441, ICCV 37, HK 06-163, GNG 1996, GNG 1947, GL 21107, CSJ 610, GJG 0814, BGD 1060, GNG 0703, HC 3, H 07-157, IPC 2006-19, K 850, BG 1003, GCP 107, BG256, C 235, BG 267, BG 329, Viswas, BG 362, BG 372, GNG 469, JG 218, L 550, RSG 44, Vikas
Highly susceptible	0	0	Nil	

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