Evaluation of chickpea germplasm against collar rot disease caused by *Phytophthora megasperma*

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Abstract

One hundred and sixteen chickpea (*Cicer arietinum* L.) varieties/elite lines were evaluated under field conditions during the years 2009 and 2010 to identify source of genetic resistance against collar rot disease caused by *Phytophthora megasperma*. The fungus was isolated from diseased chickpea plants, purified and maintained on pimaricin + ampicillin + rifampicin + pentachloronitribenzene (PARP) medium and multiplied on chickpea seeds. Amongst 116 germplasm/lines, 33 genotypes displayed resistant, 33 moderately resistant, 38 moderately susceptible and 12 susceptible reaction. The resistant sources found in this study can further be exploited in breeding program for the development of disease resistant commercial cultivars against *P. megasperma*.

Key Words: Chickpea, collar rot, genotypes, *Phytophthora megasperma*.

Introduction

Chickpea is an important legume crop of Pakistan. It is cultivated on 2636.42 thousand acres with an annual production of 561.50 thousand tones in Pakistan. Punjab alone contributes 488 thousand tones from 2388.57 thousand acres (Agricultural Statistics of Pakistan, 2009-2010). Chickpea production is always uncertain and has low yield as compared to its potential yield in the country. Among other biotic and abiotic factors responsible for low yield, different diseases play a very key role in this context.

Phytophthora collar rot is an important soil and water-borne disease caused by P. megasperma in irrigated areas. Disease mostly appears in the early growth stage of the crop i.e. before pod formation. Damage is huge in periods with above average rainfall. Development of the disease depends upon both, the pathogen in the soil, and a period of inundation. Only a single saturating rain event is needed for infection. Losses in a Phytophthora infested paddock may be minor if soil saturation does not occur (Ryley et al., 2001). Pathogen survives in soil mainly as thick-walled oospores for at least 10 years. Under favourable conditions, oospores germinate and produce lemon-shaped sporangia. Inside these sporangia, zoospores develop and are released into the soil and surface water, from where they are carried by moving water and swim towards the roots and collar portions of host plants. Zoospores germinate to produce hyphae that invade the roots. Zoospores themselves are only capable of swimming for a few millimetres, while long distance dispersal of *P. megasperma* is due to movement of soil by farm equipments and water infested with oospores, sporangia, zoospores and/or chlamydospores on assent of floods and irrigation or by machinery (Kevin *et al.*, 2011). Collar rot *p*athogen when colonize the roots cause collapse of whole vascular system with death of the plants. If same crop is cultivated every year in the same field the losses increase to many folds.

Chemical control of soil-borne diseases is very costly, uneconomical and somewhat impracticable. The ideal and the most economical way of managing this disease would be the use of host resistance. Work in exploring the resistant source or cultivar seems scanty which calls for screening of all available genetic stock of chickpea for recognition/identification of resistant genotype against the pathogen. The present study was therefore carried out to screen available chickpea genotypes against Phytophthora collar rot.

Materials and Methods

Available genetic material was evaluated for its reaction against the disease by planting it in the soil made sick with abundant culture of the pathogen. The trial was conducted at Plant Pathology Research Institute, Faisalabad area. Chickpea genetic material (lines/cultivars/elite lines) received from Pulses Research Institute, Faisalabad were included in the screening trial. Trial was performed under augmented design. After every two test lines, check variety paidar-91 was sown.

Chickpea seeds were soaked in water for 24 hours then were semi-cooked, surface dried and were put into polypropylene bags at 200 g per bags of 20×30 cm². The bags' open ends were transformed into necks with the help of hard plastic rings and were sealed with cotton plugs.

These bags were autoclaved for 20 minutes. These sterilized bags were then inoculated with fresh culture bits of P. megasperma grown on PARP mediaand were incubated at $20 \pm 1^{\circ}C$ under alternate light and darkness condition for 15 days. The gram seeds fully impregnated with the growth of P. megasperma were thoroughly and vigorously mixed before spreading into field. Seeds of all the varieties/lines were sown in $3.04 \times 0.45 \text{ m}^2$ plot. Second inoculation was done by applying fresh culture suspension of the pathogen three weeks after sowing. The culture suspension was made by blending four dishes (90-mm) fully covered with mycelial growth of the pathogen in 1 liter of water. Suspension (200 mL) was applied to each entry followed by irrigation. Data on percent mortality of the plants was taken and analyzed following the 0-9 scale (Mayee and Datar, 1986).

0 No symptom of disease Immune/highly resistant 1-10% plants affected Resistant 1 11-20% plants affected Moderately Resistant 3 5 21-50% plants affected Moderately Susceptible 51-70% plants affected 7 Susceptible 9 71% and above Highly susceptible

Results and Discussion

Data presented in the Table 1 shows that all the tested genotypes, varied greatly for their response against the disease. None of the variety behaved as immune/highly resistant. Thirty three varieties/lines found resistant these were: 88189, 89015, 89027, 89062, 90036, 90216, 90270,90275, 90277, 90279, 90280, 91013,91016, 91053, 91066, 91082, 91095, 91099, 91116, 91132, 91137, 94183, 94184, 94201, 94202, 94204, 94227, 96114, CM-72, C 727, C44, Noor 91 and ICC-82436.

Thirty three varieties/lines exhibited moderately resistant response. These varieties/lines were: 89008, 89011, 89021, 89089, 89117, 90064, 90065, 90224, 90253, 90304, 90305, 90313, 90387, 91040, 91048, 91050, 91054, 91055, 91063, 91080, 91103, 91175, 93081, 94192, 94198, 94205, 94206, 94218, 94222, 94224, 94249, 94259, and C-41. Thirty eight varieties/lines i.e. 86037, 86134, 86205, 87145, 87192, 88022, 89007, 89023, 89120, 89144, 90015, 90026, 90056, 90062, 90147, 90241, 90273, 90315, 90395, 90406, 91001, 91003, 91004, 91005, 91107, 91047, 91060, 91065, 91102, 91123, 91124, 91125, 93127, GG 688, Aug 1434, CAM 68, P-235 and C-87 responded as moderately susceptible. Remaining twelve varieties/lines exhibited susceptible response i.e. 86120, 86135, 89033, 90101, 90222, 90248, 90386, 91006, 96315, Pb-91, GL-769, ICC-5127. The resistant genotypes can further be exploited in breeding program for the development of disease resistant commercial cultivar by determining their genetics.

The frequency of highly resistant lines was generally low. This shows a high level of aggressiveness of the pathogen or relatively narrow diversification of genetic material under study. These findings are in conformity with the findings of other scientists in the world. Brinsmead et al. (1985) screened 200 chickpea exhibited resistance to P. megasperma f. sp. medicaginis in two trials on land known to be naturally infested with the pathogen. Several of theses lines were shown to have significantly superior field resistance compared with the commercial cultivars. Sugha et al. (1991) evaluated 210 chickpea lines/cultivars from different sources and none of these was resistant or even moderately resistant. Hussain et al. (2005) screened 57 cultivars and found only one genotype as highly resistant. In Pakistan, this is the first report about evaluation of chickpea germplasm against P. megasperma causing collar rot.

Conclusion

Collar rot at seedling stage causes a high level of infection, therefore, a large number of germplasm lines can be screened at seedling stage under green-house conditions saving much time and labour.



Fig. 1: Frequency (%) of different varieties of chickpea against collar rot disease caused by *P. megasperma*.

Mortality	Reaction	Varieties/Lines	No. of varieties/Lines
(%)			
1-10	Resistant	88189, 89015, 89027, 89062, 90036, 90216,	33
		90270,90275, 90277, 90279, 90280,	
		91013,91016, 91053, 91066, 91082, 91095,	
		91099, 91116, 91132, 91137, 94183, 94184,	
		94201, 94202, 94204, 94227, 96114, CM-72,	
		C 727, C44, Noor 91 and ICC-82436.	
11-20	Moderately Resistant	89008, 89011, 89021, 89089, 89117, 90064,	33
	2	90065, 90224, 90253, 90304, 90305, 90313,	
		90387, 91040, 91048, 91050, 91054, 91055,	
		91063, 91080, 91103, 91175, 93081, 94192,	
		94198, 94205, 94206, 94218, 94222, 94224,	
		94249, 94259, and C-41.	
21-50	Moderately Susceptible	86037, 86134, 86205, 87145, 87192, 88022,	38
		89007, 89023, 89120, 89144, 90015, 90026,	
		90056, 90062, 90147, 90241, 90273, 90315,	
		90395, 90406, 91001, 91003, 91004, 91005,	
		91107, 91047, 91060, 91065, 91102, 91123,	
		91124, 91125, 93127, GG 688, Aug 1434,	
		CAM 68, P-235, C-87.	
51-70	Susceptible	86120, 86135, 89033, 90101, 90222, 90248,	12
	-	90386, 91006, 96315, Pb-91, GL-769, ICC-	
		5127.	
71 above	Highly Susceptible		-
		Total	116

Table 1: Level of resistance/susceptibility of chickpea germplasm accessions against collar rot caused by *Phytophthora megasperma*.

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