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Genetic analysis and mapping of ascochyta blight resistance in kabuli chickpea (*Cicer arietinum*)

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ABSTRACT

Ascochyta blight is a fungal disease of chickpea (*Cicer arietinum* L.) caused by *Ascochyta rabiei*, resulting in up to 100% crop yield loss under favourable conditions. The experiment was conducted during 2018–19 and 2019–20 at Punjab Agricultural University, Ludhiana, Punjab aimed to transfer ascochyta blight resistance, a commercially important susceptible kabuli cultivar L 552 was crossed with an exotic resistant kabuli line FLIP 05-43. The F2 and F2:3 populations developed from L 552 × FLIP05-43 cross were screened for ascochyta blight resistance using cut twig method at Punjab Agricultural University, Ludhiana. Genetic studies in these populations revealed that resistance to the disease was controlled by single recessive gene designated as *arr6*. The genotyping of F2 population was carried out using 46 polymorphic SSR markers. The linkage analysis mapped 31 markers into eight linkage groups with a total map length of 377.14 cM. The gene *arr6* was located on LG 4 at a distance of 8.6 and 16.1 cM from markers CGMM072 and NCPGR247, respectively. Thus, the present study identifies genomic location of the gene conditioning resistance to ascochyta blight. The detected region will further be fine mapped to follow marker assisted breeding for ascochyta blight resistance. To our best knowledge, this is the first report of mapping *arr6* gene using kabuli line FLIP05-43.

Keywords: Chickpea (*Cicer arietinum* L.), Disease resistance, Intra-specific population, Molecular mapping, SSR markers

The cultivated chickpea (*Cicer arietinum* L. Fabaceae) is a self-pollinated diploid crop (2n = 16) with a genome size of 738.09 Mb (Varshney *et al.* 2013). It is grown in 57 countries, with India, Australia, Myanmar, Turkey, Ethiopia, and Russia as key producers (Merga and Haji 2019). Chickpea seeds are rich in protein (22%), minerals, fiber and β-carotene (Jukanti *et al.* 2012). The cultivated chickpea is classified into desi and kabuli. The kabuli chickpeas have large, light colored seeds with a smooth, thin coat and 100-seed weight of 26 g. These fetch upto 3 times higher prices than desi chickpeas that have smaller, angular, dark brown seeds with a thick, rough coat and 100-seed weight of 21 g (Khan *et al.* 1995, Purushothaman *et al.* 2014).

Fungal pathogen *Ascochyta rabiei* causes ascochyta blight (AB), a major biotic stress in chickpea. AB affects both vegetative and reproductive stages, potentially causing 100% yield loss under cool, wet conditions (Choudhary *et al.* 2022). Development of resistant/tolerant chickpea cultivars is considered the most effective approach to manage AB (Sharma and Ghosh 2016). Breeding efforts focus on incorporating AB resistance (ABR) in kabuli cultivars from resistant kabuli germplasm to maintain seed quality traits (Vir *et al.* 1975, Reddy and Singh 1984, Kaur *et al.* 2012). As compared to conventional breeding, genomics-assisted breeding approaches can help develop desirable cultivars quickly (Stephens *et al.* 2013). Till date, only a few intraspecific maps have been documented in chickpea based on populations segregating for ABR (Flandez-Galvez *et al.* 2002, Flandez-Galvez *et al.* 2003a; b, Udupa and Baum 2003, Cho *et al.* 2004, Stephens *et al.* 2013), which has limited identification of gene(s)/QTL governing ABR in pure *C. arietinum* genetic background.

To incorporate ABR into a commercial kabuli cultivar (L552), we developed an intraspecific cross between L552 and FLIP 05-43, a kabuli germplasm line from International Center for Agricultural Research in the Dry Areas (ICARDA), Syria possessing stable resistance to AB. The study reports genetics of ABR derived from FLIP 05-43 and the genomic location of ABR gene through construction of genetic linkage map using F2 mapping population and

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SSR markers. The identified flanking markers will be useful in marker-assisted breeding programs for chickpea improvement.

MATERIALS AND METHODS

Development of populations: The experiment was conducted during 2018–19 and 2019–20 at Punjab Agricultural University, Ludhiana, Punjab. F1 plants developed by crossing a kabuli cultivar L 552 as female parent (susceptible to *A. rabiei* and originating in India) with a kabuli line FLIP 05-43 as male parent (resistant to *A. rabiei* and originating in Syria) were used to generate F2 population. The population was grown in rows at the experimental field area of Punjab Agricultural University, Ludhiana, Punjab, India during 2018–19. In the subsequent year (2019–20), 15 plants of each F2 derived F2:3 families were grown in the field area in one row of 2 m length with row to row spacing of 40 cm.

Screening of populations using cut twig method: The spores of virulent isolate 8 of race 6 (3968) of *A. rabiei* prevalent in Punjab region (Singh 1990) were collected from infected chickpea plants and used for preparing inoculums at pathology laboratory as described by Lekhi *et al.* 2022. The inoculums containing approximately 5×10^4 spores/ml were used for screening of F2 and F2:3 populations (derived from L552 FLIP 05-43 cross) for AB resistance or susceptibility during two successive years using cut twig method (Singh and Sharma 1998). The tender shoot twigs (15 cm long) of parents and individual F2, F2:3 plants were cut with the help of a scissor, dipped in water and transferred to seedling trays containing field soil. The trays were then kept in the experimental field area. The twigs were artificially inoculated by spraying inoculum of virulent isolate of *A. rabiei* in the evening using a knapsack sprayer. The congenial conditions (relative humidity above 85% and temperature around 25°C) for disease development were created according to Lekhi *et al.* 2022.

Readings for disease incidence on a single twig basis were taken on 15th day after inoculation, using a 1–9 scale, where: 1, No symptoms; 1.1–3.0, Lesions on <10 % twig area with no stem girdling; 3.1–5.0, Lesions on up to 25% twig area and stem girdling on <10% twig area; 5.1–7.0, Lesions on most part of the twigs and stem girdling on 50% twig area; 7.1–9.0, Profuse lesions on twigs and stem girdling on >50% twig area (Nene *et al*. 1981, Singh and Sharma 1998). The F2 twigs with an average disease score of ≤5 were classified as resistant and >5 as susceptible. The F2:3 families where twigs had a mean disease score of ≤3 were considered as homozygous resistant, >5 as homozygous susceptible, and the families in which twigs were exhibiting both resistant, susceptible phenotypes were regarded as segregating. The experiments were performed twice to confirm the results. The data were analyzed and the segregation ratios were tested against expected Mendelian segregation ratios using chi-square test to know the number and nature of gene(s) controlling resistance to AB.

Genotyping of F2 mapping population using SSR markers: Young twigs (3–4 in number) were collected from each parent (L 552, FLIP 05-43) and F2 plant, and used for DNA extraction using CTAB method (Sika *et al.* 2015). Parental polymorphism was surveyed using a total of 300 SSR markers (spanning all linkage groups of chickpea) selected from linkage maps of Winter *et al.* (1999), Lichtenzveig *et al.* (2005), Nayak *et al.* (2010), Bharadwaj *et al.* (2011) and Thudi *et al.* (2011). PCR mixture (10 µl) contained 20 ng genomic DNA (3 µl), 5 µM of each primer (0.6 µl), 1 mM dNTP mix (2 µl), 25 mM $MgCl_2$ (0.6 µl), 5 × PCR buffer (2 µl), 5 units GoTaq DNA polymerase (1 µl) [Promega, USA] and nuclease-free water (0.2 µl). The reaction mixtures were placed in an Eppendorf master cycler programmed for an initial denaturation at 94°C for 5 min, followed by 35 cycles of denaturation at 94°C for 1 min, annealing at 55–60°C for 2 min, extension at 72°C for 2 min, concluded by a final extension at 72°C for 7 min. The amplicons were resolved on 6% (w/v) polyacrylamide gel electrophoresis (PAGE), visualized under UV gel documentation system and photographed. The polymorphic markers were applied on DNA of F2 plants, and scored on basis of differential separation of amplicons. The allele from L 552 was scored as 'A', from FLIP 05-43 as 'B', heterozygous plants containing both alleles were marked as 'H' and missing data as 'M'.

Table 1 Reaction of F2 and F2:3 populations derived from L 552 × FLIP 05-43 to ascochyta blight under controlled field conditions at Punjab Agricultural University, Ludhiana

Pathotype	Generation	Total number of plants	No. of resistant plants (R)	No. of susceptible plants (S)		Genetic ratio	Calculated χ^2 value
Virulent isolate 8 of race 6 (3968) of *A. rabiei*	F2	119	33	86		1R: 2.6S	0.46
	Generation	Total number of families	No. of homozygous resistant families (HR)	No. of segregating families (Seg)	No. of homozygous susceptible families (HS)	Genetic ratio	Calculated χ^2 value
	F2:3	119	31	58	30	1HR: 1.9Seg: 1HS	0.096

χ^2, Table value (5% level of significance) @1 d.f. = 3.841, and @2 d.f. = 5.991; R, Resistant; S, Susceptible; HR, Homozygous resistant; HS, Homozygous susceptible.

Molecular mapping of ascochyta blight resistance: For each segregating marker, a χ^2 goodness of fit analysis was performed using MAPMAKER software (Lander *et al.* 1987) to evaluate deviation from expected segregation ratio 1:2:1. The markers were included on the map only if threshold LOD score of 3.0 and recombination fraction of 0.3 were obtained. The distances between markers were acquired from recombination frequencies using Kosambi (1944) mapping function of MAPMAKER and Map Chart program version 2.1 (Voorrips 2002).

RESULTS AND DISCUSSION

Screening of F2 and F2:3 populations for genetic analysis of ABR: L 552 and FLIP 05-43 plants were grown in experimental field area and screened under artificial epiphytotic conditions with virulent isolate of *A. rabiei*. With a disease score of 9.0, L 552 was observed to be highly susceptible to AB (Fig. 1a), and FLIP 05-43 with a score of 1 was highly resistant to AB (Fig. 1b). Further, the evaluation of parents, F2, F2:3 populations using cut twig method revealed susceptibility of L 552 to the disease with a mean disease score of 9.0, and resistance of FLIP 05-43 with a score of 1.0. The distinct reaction of parents implied that the disease development in the field was appropriate for evaluating the populations. Out of a total of 119 F2 plants tested, 33 were classified as resistant (R), and 86 were categorized as susceptible (S) [Table 1, Fig. 1c]. Likewise, in the subsequent year, amongst 119 F2:3 families tested, 31 were categorized as homozygous resistant (HR), 30 as homozygous susceptible (HS) and 58 as segregating (Seg). The segregation of F2 plants into resistant/susceptible phenotypes upon inoculation with *A. rabiei* fitted into 1R:3S ratio with $\chi 2$ value of 0.46, suggesting that resistance against the pathogen was controlled by a single recessive gene and designated as *arr6*. Henceforth, segregation of F2:3 families into resistance/susceptible phenotypes exhibited a perfect fit into 1 HR:2 Seg:1 HS ratio with χ^2 value of 0.096, confirming that resistance was controlled by a monogenic recessive gene. The phenotypic screening was carried out using cut twig method as it is fast, reproducible and specifically useful for screening segregating chickpea breeding material (Pande *et al*. 2011). Here, we foremost reported that AB resistance transferred from exotic kabuli line FLIP 05-43 was controlled by a single novel recessive gene. Whilst the disease resistance in kabuli genotypes ILC 72, ILC 183, ILC 200 and ILC 4935 has been reported to be conferred by a single dominant gene (Singh and Reddy 1983), and by three major recessive genes in FLIP 84-92C (Tekeoglu *et al*. 2000).

Molecular mapping of arr6 gene: The limited genetic diversity within cultivated gene pool is the reason for availability of a few intraspecific linkage maps in chickpea (Thudi *et al*. 2011). The markers detected using interspecific mapping populations have a limited chance of transfer to cultivated chickpea breeding programs as these pertain to domestication-related traits that are unlikely to be present in cultivated chickpea gene pool (Stephens *et al*. 2013). In this regard, construction of an intraspecific genetic linkage map from cross between cultivated chickpea genotypes would be more helpful for accelerated gene transfer into commercial genotypes through marker assisted selection (Flandez-Galvez *et al*. 2003a, Millan *et al*. 2003). The parental genotypes were screened using 300 SSR markers as the SSR marker system is co-dominant, reproducible and abundant (Stephens *et al*. 2013), 46 markers (15.33 %) were observed to be polymorphic, mainly attributed to low level of polymorphism between parents. 31 markers (67.39% of polymorphic markers), viz. CaM2049, CGMM011, CGMM025, CGMM028, CGMM029, CGMM066, CGMM067, CGMM072, CGMM074, CGMM0257, GA16, GA17, GA20, GA26, GA117, GAA41, GAA50, GAA51, GAA117, H1116, ICCM0257, NCPGR247, SCY117, TA25, TA44, TA96, TA103, TA104, TR40, TR43 and TR59 showed Mendelian segregation ratio of 1:2:1. The remaining 15 markers (32.61% of polymorphic markers), namely CGMM012, CGMM023, CGMM024, CGMM046, CGMM064, GA46, H5G01, TA28, TA43, TA72, TA76s, TA140, TA200, TS81 and TS82 exhibited distorted ratios. The banding patterns of SSR markers in F2 population are shown in Fig. 2a, 2b. Udupa and Baum (2003) reported a low SSR marker distortion of 3.85% in kabuli×kabuli derived mapping population. Flandez-Galvez (2003a) and Bharadwaj *et al*. (2011) documented 26.8, 28% marker

Fig. 1 (a) Susceptibility of L 552 to ascochyta blight, (b) Resistance of FLIP 05-43 to AB under artificial epiphytotic field conditions, (c) Disease incidence on twigs of F2 population and (d) Disease incidence on F2:3 twigs on 15th day after inoculation.

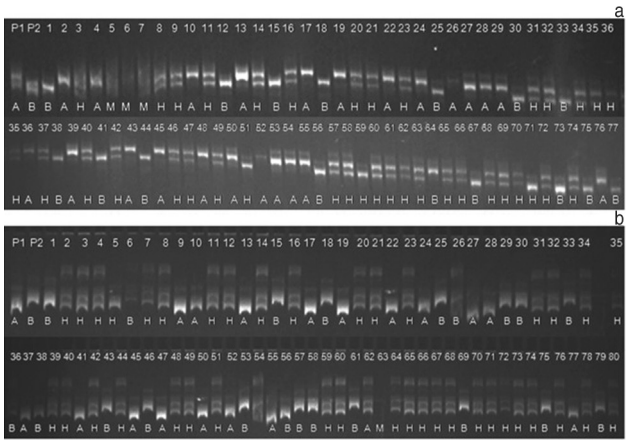

Fig. 2 Banding pattern of SSR marker (a) CGMM072 and (b) NCPGR247 in a representative number of F2 plants derived from L 552 × FLIP 05-43. P1= L 552 (susceptible parent), P2, FLIP 05-43 (resistant parent), A, Allele A (from susceptible parent), B, Allele B (from resistant parent), H, Allele A + Allele B, M, Missing data

distortion in populations derived from desi × desi and desi × kabuli crosses, respectively. Lyttle (1991) stated segregation distortion to be an outcome of selection during gamete formation, fertilization or germination. In an intraspecific chickpea F2 population, the gametophytic factors influenced male or female gametes selectively leading to segregation distortion (Castro *et al*. 2011). Another reason is erroneous PCR resulting from degraded DNA template or primer failure to amplify DNA correctly due to excessive freezing or thawing (John and Jeffery 2015).

A total of eight linkage groups were generated covering a total map length of 377.14 cM with an average distance of 12.57 cM between adjacent markers. The number of markers ranged from 3–8 with a map length ranging from 7.7–40.3 cM. Some skewness towards clustering of markers was observed in linkage groups 5 and 7. Large gaps were obtained on the map due to restricted genomic coverage of markers. The map length was found to be close to other intraspecific chickpea maps i.e. 534.5, 419.0 and 471.1 cM with 8.1, 7.9 and 14.2 cM mean distance between contiguous markers, respectively (Flandez-Galvez 2003a, Udupa and Baum 2003, Bharadwaj *et al*. 2011).

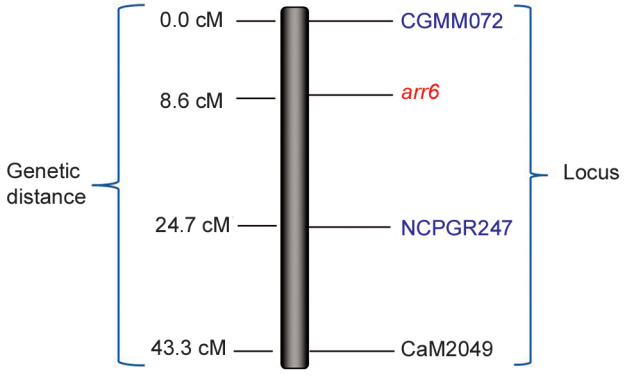

Fig. 3 Mapping of ascochyta blight resistance gene *arr6* on LG 4 using L552 × FLIP 05-43 F2 population.

The analysis of AB and SSR marker data mapped the *arr*6 gene on LG 4 at a distance of 8.6 cM from SSR marker CGMM072 and 16.1 cM from SSR marker NCPGR247 (Fig. 3). A few previous studies mapped AB resistant genes/QTLs on LG 2, 3, 4 and 6 through construction of intraspecific linkage maps obtained from kabuli × kabuli (Udupa and Baum 2003), desi × kabuli (Cho *et al*. 2004, Taran *et al*. 2007), desi × desi and kabuli × desi (Flandez-Galvez *et al*. 2003a, Stephens *et al*. 2013, Garg *et al*. 2018). Furthermore, the resistance in the present population was derived from FLIP 05-43, which is different from the kabuli sources reported by Udupa and Baum (2003), Cho *et al*. 2004 and Taran *et al*. 2007.

The present study identified a novel source for ascochyta blight resistance in kabuli germplasm. The mapping information obtained from segregating populations derived from the novel resistant source would serve as a starting point for fine mapping of the novel *arr6* locus. This would enable identification of co-segregating tightly linked markers that can be used in marker-assisted breeding and genetic enhancement of chickpea germplasm.

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