Pak. J. Bot., 46(4): 1479-1483, 2014.

SOURCES OF RESISTANCE IN CHICKPEA (*CICER ARIETINUM* L.) LAND RACES AGAINST *ASCOCHYTA RABIEI* CAUSAL AGENT OF ASCOCHYTA BLIGHT DISEASE

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Abstract

Ascochyta blight disease, caused by the fungus *Ascochyta rabiei*, is a major yield limiting factor of chickpea in Turkey and around the world. This study was conducted to identify sources of genetic resistance against chickpea blight caused by *Ascochyta rabiei*. For this purpose, 68 chickpea land races of different origins were evaluated in both field and growth chamber conditions during 2008-2009 growing seassons. Two standard cultivars were used as a reference, Inci (resistant) and Canitez (susceptible). Disease severity scoring was conducted on a 1–9 rating scale 21 days after inoculation in growth chamber test and at flowering and pot filling stages in field tests. Analysis of variance (ANOVA) test showed a significant difference among the chickpea landraces in ascochyta blight resistance at $p<0.05$. None of the chickpea land races was highly resistant to the pathogen in growth chamber and field conditions. Only two landraces (10A and 28B) were moderately resistant to the disease. Some of the landraces resulted in a particular plant to exhibit no disease symptoms, indicating that the variation within chickpea land races was high. Therefore, seeds of this plant were harvested separately and preserved for further evaluations.

Introduction

Chickpea (*Cicer arietinum* L.) is an important legume crop grown under a wide range of ecological conditions in the world with 11.081.938 ha sowing area and 9.774.082 ton annual production. Turkey is the third major chickpea producer in the world after India and Pakistan with an annual production of 562.564 tones/year (Anon., 2009). There are several fungal diseases of chickpea, causing economical yield loss, including Ascochyta blight disease (*Ascochyta rabiei* (pass.) Lab.) (Acikgoz & Demir, 1984; Reddy & Singh, 1984; Singh & Reddy, 1991; Singh & Reddy 1993). Ascochyta blight disease of chickpea causes about 20% to 100% yield loss annually and may cause total failure to the crop under epidemic conditions (Reddy & Singh, 1990; Jimenez-Diaz *et al*., 1993). The pathogen fungus attacks all aerial parts of the plant, causing necrotic lesions. Lesions on leaves and pods are circular while they are elongate on petioles and stems. When the lesions encircle stems and petioles, they usually break (Nene & Reddy, 1987). Management of chickpea blight disease rely on the application of foliar and seed dressing fungicides (Rauf *et al*., 1996; Pande *et al*., 2006) and on the use of disease free seeds and field sanitation. Under favorable conditions for disease development and spread, these practices cannot be sufficient to get effective disease control. Also most of the chickpea growers can not afford the cost of chemical control in Turkey. Under these conditions the cheapest and most effective control strategy against blight disease is use of resistant or tolerant cultivars. Since blight resistance levels of current cultivars are not high (Chongo & Gossen, 2001; Pande *et al*., 2006), identification of resistance sources and use of these sources for developing resistant cultivars is an important component of integrated control programs of the disease. Numerous studies have been conducted to determine resistant line or cultivar against *Ascochyta rabiei* (Reddy & Singh, 1984; Dolar, 1995; Haware *et al*., 1995; Singh & Reddy, 1996; Toker & Canci, 2003; Sagir *et al*., 2004). Iqbal *et al*., (2002) reported that seven out of 356 chickpea genotypes found resistant against *Ascochyta rabiei* in greenhouse conditions, but none of these genotypes were highly resistant. Similarly, Toker & Canci, 2003) reported that only 5 genotypes out of 41 (FLIP 95 - 53C, FLIP 95 - 68C, FLIP 97 - 74C, FLIP 95 - 53C, and FLIP 98 - 177C) were resistant to Ascochyta Blight in field conditions since the pathogen fungus develop new pathotypes which overcome the host plant resistance (Akem, 1999; Pande *et al*., 2006). Therefore research is needed for identification of resistant sources and developing resistant or tolerant chickpea cultivars against new patotypes. This study was carried out to screen landraces of chickpeas to identify the new sources of resistance against *Ascochyta rabiei* and to develop blight resistant chickpea cultivars.

Materials and Methods

Plant materials: In the present study, 68 chickpea landraces collected from 38 different locations of Tokat province, and one susceptible and one tolerant cultivar were used (Table 1).

Pathogen isolation and inoculum preparation: The *Ascochyta rabiei* isolates were isolated from diseased plant parts collected from 38 chickpea fields located at different sites of Tokat province. For isolation of the fungus from diseased plant parts, stems or pods showing typical ascochyta blight symptoms were cut into 1cm or 0.5×1.0 cm^2 segments, were surface-disinfested with 2% sodium hypochlorite for 3 minute, rinsed three times with sterile distilled water, and then blotted dry on sterile paper towels. Stem or pod pieces were placed in 2% water agar containing 50 mg/l streptomycine sulfate and incubated at 22 ± 2 °C for 48 – 72 h. Pure single spore (pycnidiospore) isolates of the fungus were obtained and maintained either

in potato dextrose agar (PDA) or chickpea seed meal dextrose agar (CSMDA) containing 40 g chickpea seed meal; 20g dextrose, 20g agar, and 1 litre water at 4°C until use (Chen *et al*., 2004). Based on a preliminary pathogenicity tests results, the most aggressive isolate (AR - 8) (results not shown) was used as an inoculum throughout the growth chamber experiments.

The fungal isolate (AR - 8) was grown in Petri plates at 22 ± 2°C, on PDA medium. After sporulation 15 day-old cultures, the plates were soaked in 10 ml of sterile distilled water and spores were disloged with a sterile glass rod. Conidial concentrations were determined with a haemocytometer and adjusted to 2×10^5 conidiospores mL^{-1} before inoculation.

Table 1. Survey areas and their distances from the center of Tokat.

Survey areas	Round-trip (Km)
Tokat-Erbaa (Karaağaç, Tanoba)	220
Tokat-Erbaa (Demirtaş, Endikpınarı)	210
Tokat-Zile (Ali Bağı, Belpınarı, Elmacık)	224
Tokat Zile (Hacılar, Karayün, Ütük)	150
Tokat-Zile (Çamdere, Yaylakent, Gölcük)	196
Tokat –Sulusaray (Balıkkaya, Buğdaylı)	184
Tokat –Sulusaray (Dutluca, Tekkeyeni)	184
Tokat –Artova (Aşağı Güçlü, Yukarı Güçlü, Taşpınar)	90
Tokat –Artova (Gür ardıç, Ağamusa)	120
Toplam	**1578**

Growth chamber evaluation: Seeds of 68 chickpea land races were planted in 7.5 × 15.0 cm plastic pots containing sterile peatmoss and commercial cultivars, Canitez (susceptible) and Inci (moderately resistant) were used as control for comparison and spread of the disease. Two weeks old chickpea seedlings were sprayed with spore suspension of isolate AR - 8 (2×10^5) until runoff using hand sprayer and immediately covered with translucent plastic bag to produce uniformly high relative humidity for 24 h to facilitate infection (Ilyas & Khan 1986). Plants were then placed in a growth chamber (Rektor Makina Istanbul, Turkey) that was set at 12 h day (20°C) and 12 h night (16°C) at 95% relative humidity. Disease monitoring was conducted twenty - one days after inoculation and cultivars were assessed using 1 - 9 rating scale as described by Reddy and Singh, 1984) as follows: 1 = No infection; 2 = Highly resistant (1 – 5% of plant blighted); 3 = Resistant (6 – 10%); 4 = Moderately resistant (11 – 15%); 5 = Intermediate (16 – 40%); 6 = Moderately susceptible (41 – 50%); 7 = Susceptible (51 – 75%); 8 = Highly susceptible (76 – 100%); 9 = Plant killed. These scores were converted to disease severity (DS) value (Xi *et al*. 1990) by Eq. (1);

$$DS = \frac{\sum nc}{Nxc_m} x100 \qquad (1)$$

where, n is the number of plan in each category, *c* is the value for the category, N is the total number of plants and c_m is the maximum number for categories. Land races were considered resistant if the disease severity was lower than 50% and those with disease severity of 50 - 100% were considered susceptible.

Field evaluation: Field studies were conducted in the experimental fields at the Gaziosmanpasa University Agricultural Faculty, Tokat-Turkey, during 2007–2008 and 2008–2009. The soil of field was sandy clay loam with a pH of 7.5, moisture content of 12.4%, organic matter of 18.9 mg g^{-1}; exchangeable K of 287 kg ha^{-1}, available sodium of 34.0 mg g^{-1}, and available P_2O_5 of 20.6 kg ha^{-1}. The landraces and two commercial cultivars (Canitez and Inci) were sown with a 40 cm row to row and 10 cm plant to plant distance, in a randomized block design, with three replication. Each treatment consisted 1 row with 3-m length and 30 seeds in per row. The seeds were sown by hand. A susceptible control, Canitez, was repeated every 5 rows. Di-ammonium phosphate (18 kg N ha^{-1} and 20 kg P ha^{-1}) was applied as at the time of seed bed preparation. Weeds were removed by hand during the growing period as needed. The plots were inoculated by spraying conidial inoculum of isolate AR - 8 (2×10^5), with pressure sprayer at the time of flowering. Ascochyta blight - infected debris, collected from different locations, was also broadcast in each plot along with spray inoculation to achieve uniform development of the disease and to prevent disease escape.

Disease scoring was recorded on the basis of 10 randomly selected plants in each row twice during the growing season (flowering and pod filling stages) using the 1 – 9 rating scale (Reddy & Singh, 1984) and DS value was calculated as mentioned above.

Statistical analysis: The data were analysed using Analysis of Variance (ANOVA) test. The means of treatments were grouped on the basis of Duncan's multiple range test at the 0.05 probability level. The software SAS was used to conduct all the statistical analysis.

Results

Growth chamber evaluation: The disease severity (DS) of 68 chickpea landraces was recorded at seedling stage in growth chamber. Results of analysis variance (ANOVA) showed a significant differences ($p<0.05$) [a4] among the chickpea landraces in Ascochyta blight resistance. According to disease severity these chickpea landraces were grouped in two categories (Table 2). Sixty six landraces were susceptible to the ascochyta blight disease,

while two of them (10A and 28B) were resistant to the disease at seedling stage in the growth chamber. None of the 68 landraces was highly resistant (Table 2). On the other hand, cultivar Canıtez (Susceptible control) showed susceptible reaction to the pathogen isolates with a 88.89% of DS value and cultivar Inci (resistant control) showed resistant reaction with a DS value of 35.18%. Disease severity values ranged from 88.89 to 52.59% among the susceptible landraces (Table 2). None of the genotypes was highly resistant, and this indicated presence of conducive environmental conditions for disease during screening.

Field evaluation: Disease development varied slightly between two years but the differences were not significant. Overall disease severity followed similar trends in both years for the land races and two cultivars. Therefore the data were combined. Analysis variance (ANOVA) test showed a significant differences among the chickpea landraces in ascochyta blight resistance at ($p<0.05$). Only two landraces (10A and 28B) showed disease severity of 39.26% (less than 50%) they were resistant over 2 years under field conditions. Cultivar Canıtez, susceptible to ascochyta blight, showed a disease severity of 91.85% over 2 years. On the other hand, cultivar Inci exhibited resistant reaction with a disease severity of 45.19% (Table 3). Disease severity values ranged from 86.89 to 50.18% among the susceptible landraces. Based on the field experiment results none of the landraces were also highly resistant to the disease.

Table 2. Disease reaction of chickpea land races, collected from Tokat province, to *Ascochyta rabiei In vitro* conditions.

Genotype	DS (%)[#]	Genotype	DS (%)	Genotype	DS (%)	Genotype	DS (%)
Canıtez	88.89 **a**[##]	33B	88.89 **a**	18A	77.78 **b-g**	4A	66.67 **h-m**
1B	88.89 **a**	35A	88.89 **a**	12A	77.78 **b-g**	21A	66.30 **i-m**
5A	88.89 **a**	9C	85.19 **ab**	27A	77.78 **b-g**	26A	64.45 **j-n**
9B	88.89 **a**	8B	85.19 **ab**	14A	77.78 **b-g**	1A	64.44 **j-n**
13A	88.89 **a**	21B	84.07 **abc**	34A	77.04 **b-h**	4B	64.44 **j-n**
14B	88.89 **a**	9A	83.70 **abc**	32A	76.55 **b-i**	3B	64.44 **j-n**
15A	88.89 **a**	32B	83.33 **abc**	12B	75.93 **b-i**	29A	62.97 **k-n**
15B	88.89 **a**	2A	83.33 **abc**	8C	74.08 **c-j**	6B	60.74 **l-o**
16A	88.89 **a**	33A	83.19 **abc**	23B	73.40 **c-k**	11C	57.78 **m-p**
16B	88.89 **a**	10B	82.96 **abc**	37B	73.33 **c-k**	7A	55.56 **nop**
17B	88.89 **a**	36A	82.22 **a-d**	17A	71.85 **d-k**	13B	55.56 **nop**
20A	88.89 **a**	33C	80.81 **a-d**	24B	69.63 **e-l**	3A	55.15 **nop**
21C	88.89 **a**	20B	80.37 **a-d**	25A	68.92 **f-l**	8A	52.59 **opq**
23A	88.89 **a**	37A	80.00 **a-e**	6A	68.89 **f-l**	28B	42.96 **qr**
24A	88.89 **a**	19A	78.59 **a-f**	11B	68.89 **f-l**	10A	35.56 **r**
26B	88.89 **a**	22A	78.15 **a-f**	25B	67.41 **g-m**	Inci	35.18 **r**
28A	88.89 **a**	2B	77.78 **b-g**	31A	67.41 **g-m**		
29B	88.89 **a**	5B	77.78 **b-g**	11A	66.67 **h-m**		

[#] DS: Disease severity, [##]Values followed by the same letter within each column do not differ significantly according to Duncan's multible range test at ($p<0.05$)

Table 3. Disease reaction of chickpea land races, collected from Tokat province, to Ascochyta rabiei in field conditions.

Genotype	DS (%)[#]	Genotype	DS (%)	Genotype	DS (%)	Genotype	DS (%)
Canıtez	91.85 **a**[##]	33A	72.26 **d-p**	6A	63.34 **n-x**	17B	59.26 **s-z**
35A	86.89 **ab**	37B	72.04 **d-q**	20B	63.15 **n-x**	24A	58.52 **t-a^{1}**
9C	84.81 **abc**	21C	71.67 **e-r**	4B	62.96 **n-x**	25B	58.15 **t-a^{1}**
33B	82.64 **a-d**	1B	70.00 **f-s**	14B	61.85 **o-y**	32A	57.78 **u-a^{1}**
19A	81.30 **b-e**	16B	69.26 **g-t**	34A	61.79 **o-y**	6B	57.78 **u-a^{1}**
36A	80.74 **b-f**	4A	68.71 **h-u**	3B	61.30 **p-z**	29B	57.47 **u-a^{1}**
37A	80.37 **b-f**	31A	68.52 **h-u**	21B	61.20 **b-i**	5A	55.56 **v-b^{1}**
33C	80.03 **b-g**	11A	68.15 **j-u**	24B	61.11 **p-z**	3A	55.19 **w-b^{1}**
8C	79.26 **b-h**	12A	66.92 **j-u**	12B	61.11 **p-z**	9A	54.45 **x-b^{1}**
15A	78.52 **b-i**	23A	66.70 **j-v**	11B	61.11 **p-z**	17A	53.99 **x-b^{1}**
2A	77.87 **b-i**	16A	66.67 **j-v**	21A	60.93 **q-z**	7A	51.48 **y-b^{1}**
15B	77.04 **b-j**	18A	66.61 **j-v**	29A	60.81 **q-z**	27A	51.39 **y-b^{1}**
2B	75.74 **c-k**	14A	66.39 **j-w**	8B	60.74 **r-z**	32B	50.18 **za^{1}b^{1}**
20A	75.43 **c-l**	9B	65.96 **k-w**	13B	60.74 **r-z**	28B	48.15 **a^{1}-c^{1}**
23B	75.25 **c-l**	1A	65.19 **k-x**	10B	60.56 **r-z**	Inci	45.19 **b^{1}-c^{1}**
13A	74.83 **c-m**	26A	64.45 **l-x**	26B	60.49 **r-z**	10A	39.26 **c^{1}**
25A	73.00 **d-m**	5B	64.08 **m-x**	11C	59.63 **s-z**		
22A	72.48 **d-o**	28A	63.95 **m-x**	8A	59.63 **s-z**		

[#]DS: Disease severity, [##] Values followed by the same letter within each column do not differ significantly according to Duncan's multible range test at ($p<0.05$)

Discussion

The results from growth chamber and field experiments revealed that the chickpea land races were different in resistance due to their high genetic variation. Two land races occurred resistant, and 66 susceptible in growth chamber and field experiments. Similar studies have been conducted by others (Katiyar & Sood 1985; Bashir *et al*., 1985; Guar & Singh 1987; Del-Serrenone *et al*., 1987; Iqbal *et al*., 1989; Reddy & Singh, 1990; Ilyas *et al*., 1991; Dolar & Gurcan 1992; Singh & Reddy 1993). These researchers have studied sources of resistance to Ascochyta blight and reported that some chickpea genotypes were resistant and others were moderately resistant to the disease, where as none of the genotypes was highly resistant. Toker & Canci, 2003) studied the reaction of 41 chickpea lines from ICARDA against *Ascochyta rabiei* in field conditions in Antalya-Turkey and found seven of them were resistant while all the remaining genotypes exhibited moderate resistance to highly susceptible reaction. Similarly, Iqbal *et al*., (2002) evaluated 356 genotypes and observed that none of the 356 genotypes was highly resistant, whereas seven genotypes (FLIP94 - 90C, FLIP95 - 68C, FLIP95 - 47C, FLIP97 - 132C, FLIP97 - 227C, FLIP98 - 224C and FLIP98 - 231C) were resistant and 75 were moderately resistant. Pande *et al*., 2006) evaluated 148 wild accessions from seven *Cicer* spp. *viz., Cicer bijugum*, *Cicer cuneatum*, *Cicerechinospermum*, *Cicer judaicum*, *Cicer pinnatifidum*, *Cicer reticulatum* and *Cicer yamashitae* for resistance to *Ascochyta rabiei* and found that five accessions of *Cicer judaicum* exhibited resistant reaction to the pathogen under greenhouse conditions. Of the remaining lines, 55 accessions were moderately resistant, 61 were susceptible, and 27 were highly susceptible to *Ascochyta rabiei.*

Present study was carried out both in vitro and in vivo conditions, the landraces identified as resistant in vitro maintained their response in vivo. However, disease severity values of most of the susceptible landraces were slightly higher in growth chamber than that in the field experiment, and this was attributed to better environmental conditions for disease development in growth chamber such as high relative humidity (90% RH) that favors the development of disease. Even though none of the land races tested were found highly resistant to *Ascochyta rabiei* in field conditions, some of the land races resulted in a particular plant to exhibit no disease symptoms. These plants were harvested separately and the seeds were preserved for use in a later study to to evaluate their resistance in growth chamber and field conditions.

Consequently, it is evident from data that landraces (10A and 28B) were resistant compared to other tested land races, suggesting that these resistant land races may be evaluated as a source of resistance against ascochyta blight of chickpea. Further research will be conducted to test their resistance in field and growth chamber.

Conclusion

Our study indicated that sufficient resistance to *A. rabiei* exists in chickpea landraces evaluated for resistance. These landraces can be used to build multi-gene resistance in breeding programs, thus improving the levels of disease resistance.

Acknowledgments

The authors acknowledged the financial support from the Gaziosmanpasa University Scientific Research Foundation Commission.

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(Received for publication 8 Janu 2013)