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Madhu Saatu
Department of Genetics and Plant
Breeding, Faculty of Agriculture,
SKUAST-K, Srinagar, Jammu and
Kashmir, India

Hima Kumar Anakali
Department of Genetics and Plant
Breeding, College of Agriculture,
PJTSAU, Rajendranagar,
Hyderabad, Telangana, India

Shaik Faheem Akhtar
Department of Genetics and Plant
Breeding, Faculty of Agriculture,
SKUAST-K, Srinagar, Jammu and
Kashmir, India

Corresponding Author:
Madhu Saatu
Department of Genetics and Plant
Breeding, Faculty of Agriculture,
SKUAST-K, Srinagar, Jammu and
Kashmir, India

Screening of chickpea germplasm for *Ascochyta* blight (*Ascochyta rabiei*) under Himalayan Region

Madhu Saatu, Hima Kumar Anakali and Shaik Faheem Akhtar

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Abstract

Ascochyta blight of chickpea, caused by *Ascochyta rabiei*, is most prevalent in regions with cool and humid climatic conditions. The fungus exhibits significant variation in pathogenicity. The disease manifests as small, circular, brown-black lesions resembling a bull's eye. Chickpea (*Cicer arietinum* L.), one of the most widely cultivated legumes globally, plays a vital role in the economies of several countries and serves as a rich source of nutrients. However, its yield can be significantly affected by *Ascochyta* blight. The available resistant sources are insufficient, as resistance in existing chickpea varieties frequently breaks down due to the rapid evolution of new pathogen pathotypes. Therefore, it is essential to continuously identify new sources of resistance. The present study was conducted to identify sources of resistance to *Ascochyta* blight among 124 chickpea genotypes at Regional Research Station, Faculty of Agriculture, Wadura, Sopore, SKUAST-Kashmir during rabi season of 2019-2020. The screening results categorized the genotypes into three groups: susceptible, tolerant, and moderately resistant. Based on percentage of disease incidence and severity, among 124 genotypes, 16 chickpea lines were identified as moderately resistant, 88 were classified as tolerant, and 20 were found to be susceptible. Further research is recommended on the inheritance of resistance, identification of existing physiological races using Marker-Assisted Selection to assess their aggressiveness, and rigorous screening of genotypes against *Ascochyta* blight.

Keywords: Chickpea, *Ascochyta* blight, disease incidence, severity, resistant, marker-assisted selection

Introduction

Chickpea (*Cicer arietinum* L.) is an annual legume belonging to the Fabaceae family and is primarily cultivated as a rabi season crop. Based on seed colour and geographic distribution, chickpea biotypes are classified into two distinct groups as Kabuli and Desi. In India and Pakistan, the predominant type is Desi, whereas Kabuli chickpeas are mainly cultivated in Turkey (Macar *et al.*, 2017) [8]. Desi cultivars, which originated in India, are characterized by small, wrinkled, and dark coloured seeds, while Kabuli cultivars, originating from the Mediterranean and the Middle East, have large, smooth coated seeds with white to cream coloured (Purushothaman *et al.*, 2014) [17]. Chickpea is considered unique due to its high protein content, which accounts for nearly 40% of its weight (Merga and Haji, 2019) [11]. Its seeds are widely used for both food and animal feed, valued not only for their excellent nutritional profile but also for their rich bioactive compounds, including phenolics and flavonoids (Magalhaes *et al.*, 2017) [9]. As an annual grain legume, chickpea is significant not only for its high nutritional value but also for its role in improving soil fertility by fixing atmospheric nitrogen (Dutta *et al.*, 2022) [4].

Chickpea is the most widely produced food legume in South Asia and the third largest globally. In 2020, it was cultivated across 14.84 million hectares, yielding 15.08 million tonnes, with an average productivity of 1.01 tonnes per hectare. It is grown worldwide under diverse environmental conditions. Asia leads in chickpea production, contributing 86% of the global output, followed by Africa (4.70%), the America (4.60%), Europe (1.90%), and Oceania (1.80%). Although chickpea is cultivated across all continents, a few countries dominate its production. In 2020, the major producers included India (73.46%), Pakistan (3.30%), Russia (1.93%), the United States (1.28%), Myanmar (3.19%), Australia (1.86%), Canada (1.42%),

Turkey (4.18%), Mexico (0.83%), and Ethiopia (3.03%). Together, these countries accounted for 94% of the global chickpea production. India is the world's largest producer, contributing 73% (11.08 million tonnes) of the total chickpea output (FAOSTAT, 2022) [5].

Ascochyta blight of chickpea caused by *Ascochyta rabiei* (teleomorph: *Didymella rabiei*) (Singh and Reddy, 1996) [19], is the most severe foliar disease affecting chickpea. It leads to a significant reduction in both yield and crop quality. The disease thrives in regions with cool temperatures (15-25°C) and humid conditions, particularly when seasonal rainfall exceeds 150 mm (Pande *et al.*, 2005) [16]. Symptoms include small, circular, brown-black spots (pycnidia) that develop at the center of lesions. These lesions are arranged in concentric circles, resembling a bull's-eye pattern. Under favourable conditions, it can cause crop losses ranging from 50% to 70%, and in severe cases, it may lead to total crop failure. Environmental conditions significantly affect the severity and prevalence of the disease in the natural field condition (NFC) and artificial epidemic field condition (AEC). In natural conditions, no intervention is made on the spread and severity of the disease, except for the presence of sensitive varieties in the trial. Under natural and artificial field epidemic conditions, there is a strong genotype × environment (G×E) interaction, which causes the disease state to alter significantly from year to year depending on the presence of the pathogen in the environment (McDonald and Linde, 2002) [10]. As the plant matures, transitioning from the vegetative to the reproductive stage, its resistance to the disease gradually declines.

Severe outbreaks of the disease occurred between 1981 and 1983, leading to the near eradication of chickpea in the northern regions of the country. As a consequence of the frequent epidemics, several prevalent landraces are threatened from the cultivation. Moreover, the *A. rabiei* keep evolving and so it breaks down the host resistance systems in newly bred chickpea varieties. Therefore, identifying new sources of resistance is crucial to sustaining chickpea cultivation and production. Owing to the economic importance of the disease, the present study was aimed to evaluate the chickpea germplasm against this disease to identify novel sources and understand the level of Ascochyta

blight resistance available in chickpea germplasm.

Materials and Methods

The field experiment entitled, “Screening of Chickpea Germplasm for Ascochyta Blight (*Ascochyta rabiei*) Under Himalayan Region” was conducted at Regional Research Station, Faculty of Agriculture, Wadura, Sopore, SKUAST-K during rabi season of 2019-20. The experimental site was located at 34.35 latitude & 74.40 longitude and about 1589m above mean sea level. It comprised of 124 genotypes along with 4 checks ILC 482, ILC263, ILC533 and Shalimar chickpea-1 (Table 2). The trial was carried out by using an Augmented block design, whereas checks were repeated and randomized within each block. All the genotypes, except for Shalimar chickpea-1 were obtained from International Centre for Agriculture Research in the Dry Areas (ICARDA), Syria. Shalimar chickpea-1 is a locally adopted variety of Jammu and Kashmir. Each genotype was sown in a plot of 2m length, with a row spacing of 30 cm and 10 cm spacing between plants within rows. The chickpea lines were evaluated by recording incidence and severity of disease. The genotypes were scored as per disease rating modified scale as shown in table 1 (Reddy and Singh, 1984) [18].

$$\text{Incidence(\%)} = \frac{\text{No. of infected plants}}{\text{Total no. plants observed}} \times 100$$

$$\text{Disease severity(\%)} = \frac{\sum \text{All disease ratings}}{\sum \text{Total ratings} \times \text{maximum grade}} \times 100$$

Table 1: Disease rating (scale) for screening of chickpea accessions

Rating	Percentage of damage	Reaction description
1	0	Immune, no infection
2	1-5	Highly resistant
3	6-10	Resistant
4	11-15	Moderately resistant
5	16-40	Tolerant
6	41-50	Moderately susceptible
7	51-75	Susceptible
8	>75	Highly susceptible

Table 2: List of chickpea genotypes used in the experiment

S. No	Genotype	S. No	Genotype	S. No	Genotype	S. No	Genotype
1	FLIP12-21C	32	FLIP12-71C	63	FLIP12-297C	94	FLIP12-43C
2	FLIP12-26C	33	FLIP12-39C	64	FLIP12-123C	95	FLIP12-38C
3	FLIP12-339C	34	FLIP12-285C	65	FLIP12-329C	96	FLIP12-301C
4	FLIP12-115C	35	FLIP12-58C	66	FLIP12-220C	97	FLIP12-318C
5	FLIP12-49C	36	FLIP12-250C	67	FLIP12-338C	98	FLIP12-175C
6	FLIP12-12C	37	FLIP12-59C	68	FLIP12-65C	99	FLIP12-313C
7	FLIP12-157C	38	FLIP12-62C	69	FLIP12-336C	100	FLIP12-303C
8	FLIP12-27C	39	FLIP12-41C	70	FLIP12-229C	101	FLIP12-213C
9	FLIP12-95C	40	FLIP12-218C	71	FLIP12-267C	102	FLIP12-284C
10	FLIP12-74C	41	FLIP12-225C	72	FLIP12-237C	103	FLIP12-249C
11	FLIP12-120C	42	FLIP12-116C	73	FLIP12-292C	104	FLIP12-293C
12	FLIP12-81C	43	FLIP12-270C	74	FLIP12-111C	105	FLIP12-211C
13	FLIP12-106C	44	FLIP12-230C	75	FLIP12-141C	106	FLIP12-235C
14	FLIP12-254C	45	FLIP12-226C	76	FLIP12-228C	107	FLIP12-212C
15	FLIP12-70C	46	FLIP12-291C	77	FLIP12-234C	108	FLIP12-232C
16	FLIP12-156C	47	FLIP12-149C	78	FLIP12-340C	109	FLIP12-233C
17	FLIP12-272C	48	FLIP12-227C	79	FLIP12-82C	110	FLIP12-126C
18	FLIP12-56C	49	FLIP12-134C	80	FLIP12-283C	111	FLIP12-306C
19	FLIP12-251C	50	FLIP12-266C	81	FLIP12-236C	112	FLIP12-222C
20	FLIP12-200C	51	FLIP12-239C	82	FLIP12-194C	113	FLIP12-314C
21	FLIP12-117C	52	FLIP12-84C	83	FLIP12-302C	114	FLIP12-286C
22	FLIP12-326C	53	FLIP12-199C	84	FLIP12-214C	115	FLIP12-54C

23	FLIP12-29C	54	FLIP12-238C	85	FLIP12-295C	116	FLIP12-224C
24	FLIP12-22C	55	FLIP12-243C	86	FLIP12-312C	117	FLIP12-92C
25	FLIP12-76C	56	FLIP12-307C	87	FLIP12-221C	118	FLIP12-45C
26	FLIP12-130C	57	FLIP12-244C	88	FLIP12-242C	119	FLIP12-223C
27	FLIP12-158C	58	FLIP12-216C	89	FLIP12-46C	120	FLIP12-345C
28	FLIP12-103C	59	FLIP12-337C	90	FLIP12-231C	121	ILC482
29	FLIP12-181C	60	FLIP12-247C	91	FLIP12-323C	122	ILC263
30	FLIP12-321C	61	FLIP12-274C	92	FLIP12-98C	123	ILC533
31	FLIP12-273C	62	FLIP12-143C	93	FLIP12-294C	124	shalimar chickpea-1

Results and Discussion

The present study aimed to evaluate the performance of 124 chickpea genotypes, including four standard check varieties, for resistance to *Ascochyta* blight (AB) under natural field conditions. Given the widespread susceptibility of currently cultivated commercial varieties to AB, it is imperative to identify resistant genotypes for effective disease management and breeding interventions. Therefore, evaluating a diverse range of genotypes was essential to identify those with better resistance to *Ascochyta* blight. Previous studies have established various reliable methodologies for screening chickpea genotypes for AB resistance under both field and controlled environments (Nene 1982; Pande *et al.*, 2010; Nasir *et al.*, 2000; Du *et al.*, 2012) [14, 15, 3]. Considering the rapid and aggressive spread of AB, so it would not be advisable to carry out assessments using artificial inoculations in the field. For this reason, the study was done under natural field conditions, to avoid generating new infection sources in the chickpea producing fields.

Resistance to *Ascochyta* blight in chickpea is governed by a complex interplay of anatomical, biochemical, physiological, and genetic factors (Pande *et al.*, 2005) [16]. In terms of host defence, metabolic activities that prevent pathogen invasion play a crucial role in resistance. Key components of this defense include the induction and accumulation of hydrolytic enzymes such as chitinases and β -1,3-glucanases (Boller, 1985) [2]. Notably, Nehra *et al.* (1994) [13] reported significantly higher chitinase activity in the leaves and pods of resistant chickpea cultivars infected with *Ascochyta rabiei*, compared to susceptible genotypes, highlighting the enzyme's role in mediating resistance. Besides Histological investigations following *Ascochyta rabiei* infection have demonstrated that resistant chickpea genotypes exhibit restricted lesion development, fewer petioles were affected and stems showing only localized brown spots, in contrast to the extensive tissue damage and stem girdling observed in susceptible cultivars (Hohl *et al.*, 1990) [6]. In the present study, out of 124 chickpea genotypes evaluated under natural field conditions, sixteen genotypes (26C, 157C, 27C, 95C, 254C, 200C, 117C, 29C, 130C, 321C, 273C, 39C, 291C, 338C, 237C, 301C) exhibited moderate resistance to *Ascochyta* blight. A majority 88 genotypes were classified as tolerant, including ILC482, ILC533, 21C, 339C, 115C, 49C, 120C, and others. In contrast,

twenty genotypes, including ILC263, 74C, 270C, 149C, and Shalimar Chickpea-1, were found to be susceptible to the disease. Notably, none of the genotypes were categorized as immune, highly resistant, resistant, or moderately susceptible (Table 3). These findings are consistent with those reported by Aydogan (2024) [1].

The wide range of variations among the tested genotypes and across the experimental station could be depends on differences in the amount of inoculums in the soil, extent of rain fall distribution, duration of leaf wetness, variations in the genetic diversity within population of *Ascochyta rabiei* and aggressiveness of pathotype difference. Although current literature provides limited and inconclusive insights into the precise genetic architecture underlying resistance, the variability in host resistance is likely influenced by the number and nature of resistance genes, the genetic variability of the pathogen, and prevailing environmental conditions. These factors collectively contribute to the differential expression of resistance observed in the field. Therefore, it is yet to be resolved by conducting more experiments on mode of inheritance resistance using molecular techniques for introgression with other high yielding genotypes. Nevertheless, the presence of moderately resistant genotypes in our collection suggested that those genotypes could be regarded as potential genetic resources for developing *Ascochyta* blight resistant varieties.

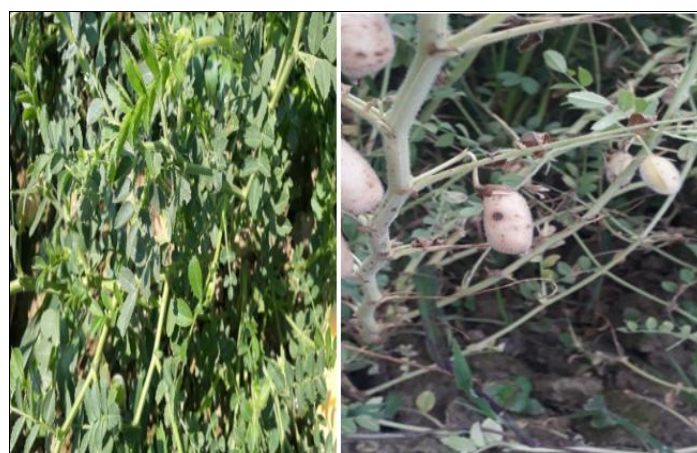


Fig 1: *Ascochyta* blight symptoms on leaf and pod

Table 3: Classification of chickpea (*Cicer arietinum* L.) genotypes into different groups based on resistance reaction (FLIP12-)

S. No	Disease Reaction	No. of Genotypes	Genotypes
1	Moderately resistant	16	FLIP12-26C, 157C, 27C, 95C, 254C, 200C, 117C, 29C, 130C, 321C, 273C, 39C, 291C, 338C, 237C, 301C.
2	Tolerant	88	ILC482, ILC533, 21C, 339C, 115C, 49C, 120C, 81C, 106C, 70C, 156C, 272C, 251C, 326C, 22C, 76C, 158C, 103C, 181C, 71C, 285C, 58C, 250C, 59C, 62C, 41C, 218C, 225C, 230C, 226C, 227C, 134C, 266C, 239C, 84C, 199C, 238C, 307C, 216C, 337C, 247C, 143C, 297C, 123C, 329C, 220C, 65C, 336C, 229C, 267C, 141C, 228C, 340C, 82C, 283C, 236C, 194C, 302C, 214C, 295C, 221C, 242C, 46C, 231C, 323C, 294C, 43C, 38C, 318C, 175C, 313C, 303C, 213C, 249C, 293C, 211C, 212C, 232C, 233C, 306C, 222C, 314C, 286C, 54C, 224C, 45C, 223C, 345C.
3	Susceptible	20	ILC263, 74C, 270C, 149C, 244C, 243C, 312C, 284C, 126C, 92C, Shalimar Chickpea- 1, 12C, 56C, 116C, 243C, 274C, 292C, 111C, 98C, 235C.

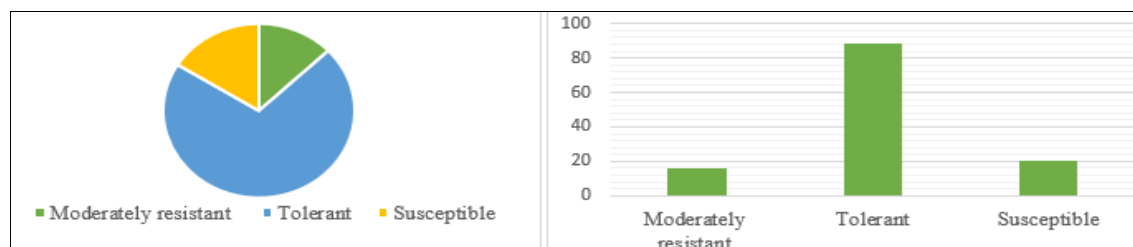


Fig 2: Distribution of Chickpea Genotypes based on Ascochyta Blight Reaction

Conclusion

The findings of this study revealed that the majority of chickpea genotypes ranged from tolerant to susceptible in their response to Ascochyta blight, with only a limited number exhibiting moderate resistance. These moderately resistant genotypes have been incorporated into ongoing breeding programs aimed at developing cultivars with enhanced resistance to Ascochyta blight. Utilizing these resistant sources in commercial breeding can help to enhance seed yield by minimizing crop damage caused by the disease. Due to the frequent resistance break down because of the rapid evolution of new pathogen pathotypes, therefore, continuous efforts are required to identify stable resistance sources and development of Ascochyta blight resistance varieties for sustainable production.

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References

- Aydoğan A. Comparison of different screening methods for the selection of Ascochyta blight disease on chickpea (*Cicer arietinum* L.) genotypes. *Front Plant Sci.* 2024;15:1347884.
- Boller T. Induction of hydrolases as a defense reaction against pathogens. In: *Cellular and molecular biology of plant stress*. 1985. p. 247-62.
- Du W, Zhao X, Raju T, Davies P, Trethowan R. Identification of Ascochyta rabiei disease resistance in chickpea genotypes. *Euphytica*. 2012;186:697-704.
- Dutta A, Trivedi A, Nath CP, Gupta DS, Hazra KK. A comprehensive review on grain legumes as climate-smart crops: challenges and prospects. *Environ Chall.* 2022;7:100479.
- FAO. FAOSTAT statistical database. Rome: Food and Agriculture Organization of the United Nations; 2022. Available from: <https://www.fao.org/faostat/en/#data>
- Hohl B, Pfautsch M, Barz W. Histology of disease development in resistant and susceptible cultivars of chickpea (*Cicer arietinum* L.) inoculated with spores of Ascochyta rabiei. *J Phytopathol.* 1990;129(1):31-45.
- Iqbal SM, Hussain S, Bakhsh A, Bashir M. Sources of resistance in chickpea against Ascochyta blight disease. *Int J Agric Biol.* 2002;4:488-90.
- Macar TK, Macar O, Mart Dİ. Variability in some biochemical and nutritional characteristics in desi and Turkish kabuli chickpea (*Cicer arietinum* L.) types. *Celal Bayar Univ J Sci.* 2017;13(3):677-80.
- Magalhaes SC, Taveira M, Cabrita AR, Fonseca AJ, Valentão P, Andrade PB. European marketable grain legume seeds: Further insight into phenolic compounds profiles. *Food Chem.* 2017;215:177-84.
- McDonald BA, Linde C. Pathogen population genetics, evolutionary potential and durable resistance. *Annu Rev Phytopathol.* 2002;40:349-79.
- Merga B, Haji J. Economic importance of chickpea: Production, value, and world trade. *Cogent Food Agric.* 2019;5(1):1615718.
- Nasir M, Bretag TW, Kaiser WJ, Meredith KA, Brouwer JB. Screening chickpea germplasm for ascochyta blight resistance. *Australas Plant Pathol.* 2000;29(2):102-7.
- Nehra KS, Chugh LK, Dhillon S, Singh R. Induction, purification and characterization of chitinases from chickpea (*Cicer arietinum* L.) leaves and pods infected with Ascochyta rabiei. *J Plant Physiol.* 1994;144(1):7-11.
- Nene YL. A review of Ascochyta blight of chickpea. *Int J Pest Manag.* 1982;28(1):61-70.
- Pande S, Sharma M, Gaur PM, Gowda CLL. Host plant resistance to Ascochyta blight of chickpea. *Inf Bull No. 82.* Patancheru, India: ICRISAT; 2010.
- Pande S, Siddique KHM, Kishore GK, Bayaa B, Gaur PM, Gowda CLL, et al. Ascochyta blight of chickpea (*Cicer arietinum* L.): A review of biology, pathogenicity and disease management. *Aust J Agric Res.* 2005;56:317-32.
- Purushothaman R, Upadhyaya HD, Gaur PM, Gowda CLL, Krishnamurthy L. Kabuli and desi chickpeas differ in their requirement for reproductive duration. *Field Crops Res.* 2014;163:24-31.
- Reddy MV, Singh KB. Evaluation of a world collection of chickpea germplasm accessions for resistance to Ascochyta blight. *Plant Dis.* 1984;68(10):900-1.
- Singh KB, Reddy MV. Improving chickpea yield by incorporating resistance to Ascochyta blight. *Theor Appl Genet.* 1996;92(5):509-15.