CHICKPEA BLIGHT: FORMER EFFORTS ON PATHOGENICITY, RESISTANT GERMPLASM AND DISEASE MANAGEMENT

Muhammad Tariq Mahmood[1], Mushtaq Ahmad[1], Imtiaz Ali[2]

[1]Gram Breeding Research Station, Kallurkot, Pakistan
[2]Regional Agricultural Research Institute, Bahawalpur, Pakistan

KEYWORDS: Ascochyta Blight, Chickpea, Pathogen, Disease Management

ABSTRACT

Ascochyta blight (AB) is a devastating and widespread fungal disease of chickpea caused by *Ascochyta rabiei* L. AB-prone environments are characterized by prolonged cool, cloudy and moist climatic conditions during the crop season. Former reports are evident that AB epidemics caused partial to complete yield losses of the chickpea crop depending upon severity of infections. The pathogen generally survives between seasons through infected crop debris and infected seeds. Exploitation of resistant genotypes, extensive tillage, manipulation of sowing dates, destruction of crop residues, seed treatment with fungicide, rotation of non-host crops and foliar fungicide applications are helpful in disease management. The previous research findings put emphasis on further exploration of the genetics, ecology, variability and the host-pathogen interaction to devise more effective disease management strategies. Through this review we have attempted to summarize former efforts related to pathogen, its biology, genetic variability, influential factors, resistant sources and disease management option with an emphasis to future prospects of AB.

INTRODUCTION

Chickpea (Cicer areitinum L.) is the third most important food legume pulse crop across the world. It is primary source of high quality protein, carbohydrates and minerals in human food throughout the chickpea growing regions around the globe (Shah, Imran, Atta, Shafiq, Aslam & Hussain, 2015; Megersa, Losenge & Chris, 2017; Iqbal, Zafar, Ashraf & Hassan, 2018; Rubiales, Fondevilla, Chen & Davidson, 2018, Mohammdi, 2019). The present status of chickpea production depicts a gloomy picture with an erratic harvest caused by certain biotic and the abiotic stresses (Upadhyaya, Dwivedi, Gowda & Singh, 2007; Asnake, 2016; Pandey, Irulappan, Bagavathiannan & Kumar, 2017, Aslam, Jiang, Zafar, Usama & Haroon, 2018). Among the biotic stresses *Ascochyta Blight* (AB) is the most disastrous fungal disease of the chickpea caused by *Ascochyta Rabiei*. AB has been reported in almost all chickpea cultivating regions across the world and is deemed to be most devastating biotic factor resulting in significant loss of yield and degradation of seed quality (Singh & Sharma 1998; Bhardwaj, Sandhu, Kaur, Gaur & Varshney, 2010; Ghosh, Sharma, Telangre & Pande, 2013; Chen, 2016; Megersa et al., 2017; Pandey et al., 2017; Khan, Arshad, Zeeshan, Ali, Nawaz & Fayyaz, 2018).

The Ascochyta Blight was first ever reported in Attock region (India, now in Punjab, Pakistan) in 1911 (Butler, 1918; Shah et al., 2015; Iqbal et al., 2018). Formerly, AB was reported by Morall and Mckenzie (1974) around Saskatoon region of America. Now this disease prevails in more than 40 chickpea growing countries around the world (Nene, 1982; Bhardwaj et al., 2010; Sharma & Ghosh, 2016). *Ascochyta blight* was first named and identified by Labrousse in 1930 (Benzohra, Bendahmane, Labdi & Benkada, 2013; Khan et al., 2018). The disease primarily spreads through seed and plant residues, however it is accelerated by wind and rain splashes. Disease prevalence and severity is variable in response to environmental conditions. The disease attack usually becomes more devastating and epidemic in cool, cloudy and the humid environmental conditions

(>150mm rainfall and 15-25 ^{o}C temperature) (Nene, 1982). Disease infections can appear on all the aerial parts of chickpea plant including leaves, pods, stem and branches developing necrotic lesions on leaves, abortion of pods and breakage of the stem and branches leading to death of the plants (Labrousse, 1931; Nene, 1984; Li, Rodda, Aftab, Redden, Hobson, Rosewarne, Materne, Kaur & Slater, 2015).

These lesions gradually increase in size and cover the whole surface of leaves, stems and pods. Initially round to elongate shaped lesions with dark margins arise that are clearly visible on leaves and pods. These lesions penetrate inside the pods resulting in shriveled fruit bodies and yield loss (Nene, 1982). In severe infections, the disease may cause the complete death of plants and 100 % yield loss (Labrousse, 1930; Sahi, Burhan, Iqbal & Sarwar, 2012; Islam, Qasim, Noman, Idrees & Wang, 2017). In this connection, despite the global recognition of disastrous potential of *Ascochyta blight* in chickpea production, a very little headway in pathogenic explorations and management has been made yet. This review aims to summarize all the valuable scientific information about symptoms, lifecycle of the pathogen, factors affecting disease incidence, variability, resistant genetic resources and ascochyta blight management in chickpea.

REVIEW OF LITERATURE

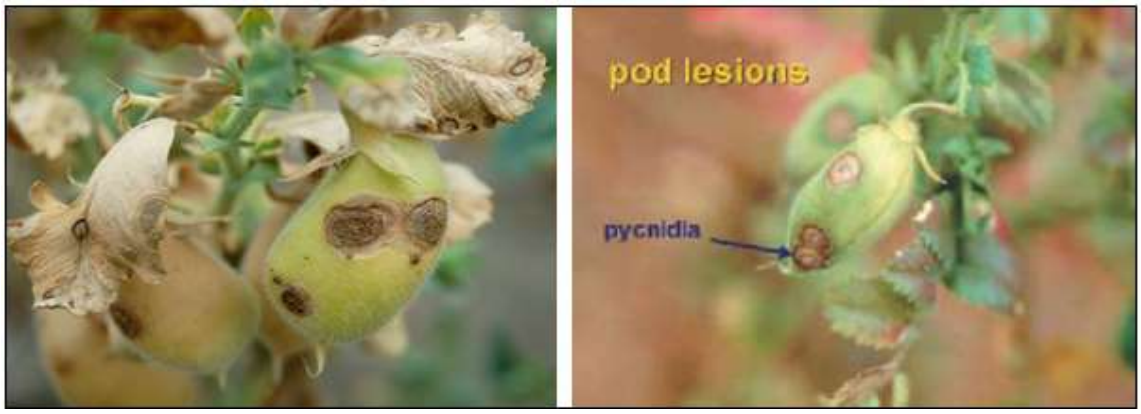
The major parts of the available literature and former efforts regarding the disease symptoms, life cycle of the pathogen, pathogen variability, factors affecting the disease incidence, the resistant genetic resources and the disease management were explored to generate valuable information about the disease.

Disease Symptoms

The symptoms of AB can be observed on all the aerial parts of chickpea plant. Disease symptoms are primarily visible on leaves, pods, stem and branches. AB symptoms are usually prominent during flowering to podding stage. Infections may be seed-borne, water-borne and air-borne. Seed-borne infections form brown lesions at stem basis of young seedlings. These lesions gradually increase in the size, cause girdle in stem and eventually death of plants (Nene, 1982). Lesions appearing on pods and leaves are brown circular in shape spots having a grey center containing pycnidia (Figure 1 & 2) while the lesions appearing on stem and branches are elongated. Conidia could be water-borne which are spread to cause infection in all aerial parts of parts including petioles, leaves, stem branches and pods which lead to quick death of plants. The lesions developing on stem and branches vary in ranges which subsequently girdle the infected parts of plants (Figure 1). The regions over these girdled parts collapse and detached from the rest of plant. Infected pods usually fail to develop seed or develop discolored and shriveled seed due to infection of seed testa and cotyledon (Nene, 1982; Li et al., 2015; Baite, Dubey & Singh, 2016; Islam et al., 2017).

Figure 1 Ascochyta Blight Symptoms on Chickpea Leaves and Braches

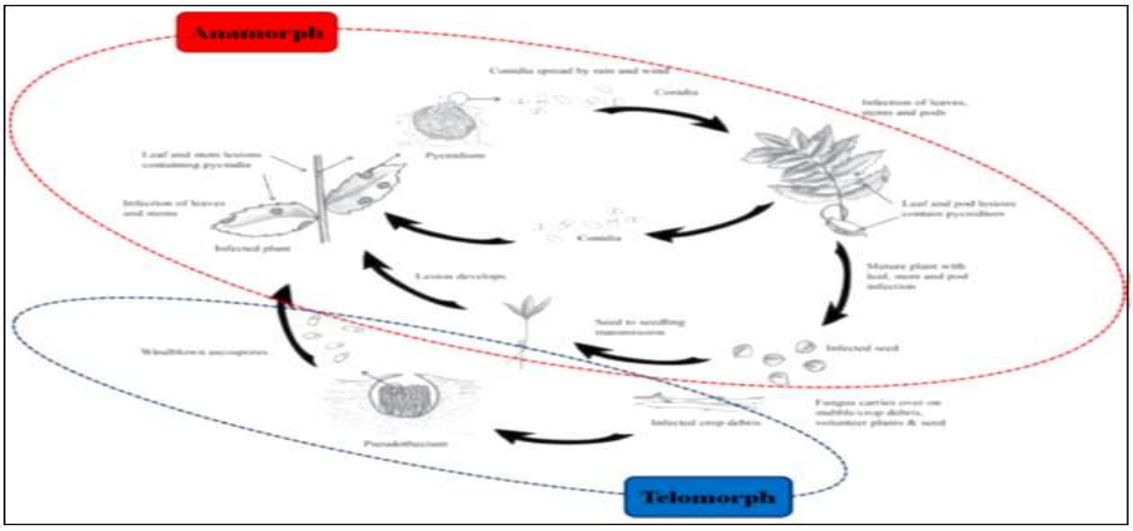
Figure 2 Lesions and Development of Pycnidia on Chickpea Pods



Life Cycle of Pathogen

The causal agent, *A. rabiei* exists in both anamorph and teleomorph stages (Figure 3). In anamorph stage *A. rabiei* forms pear shaped fruit bodies called pycnidia. Unicellar and bicellular pycnidiospores or conidia are contained in a pycnidium. These pycnidiospores are oval to oblong measuring 6-23 µm (Nene, 1982; Casas, Cortes & Diaz, 1996). Fungus can grow on the range of nutrient medium producing the cream color mycelium having pycnidia immersed inside. Teleomorph, *Didymella rabiei* (Kovacheski) var. Arx (Syn. *Mycosphaerella rabiei* Kovacheski), belonging to heterothallic ascomycete developing dark brown pseudopodia measuring 120-270 µm in diameter. The Teleomorph requires combination of two specific mating types (MAT1-1 and MAT 1-2) for their successful sexual reproduction (Punithalingam & Holliday, 1972; Kaiser & Okhovat, 1996; Casas et al., 1996; Armstrong, Chongo, Gossen, Duczek, 2001; Barve et al., 2003). *A. rabiei* involves several asexual reproductions during its parasitic phase whereas, undergoes a single sexual reproduction in a season. The fungus penetrates inside within 24 hours of its adhesion to host and this process requires seven days to accomplish (Kovachevski, 1936; Pandey et al., 1987; Illarslan & Dolar, 2002; Islam et al., 2017).

Figure 3 Life Cycle: Slight Modification of Previous Illustration by Kelly Flower



Pathogen Variability

The scientific information regarding *Ascochyta rabiei* illustrate a high extent of genetic variability in host-pathogen interaction. *A. rabiei* has different physiological races that have become challenge for host resistance breeding programs (Sharma & Ghosh, 2016; Iqbal et al., 2018). Pathogenicity, growth and colony attributes of various isolates from

Italy, India, USA, Pakistan, Australia, Canada and Syria were recorded and were found varying in infecting processes (Pandey et al., 1987; Armstrong et al., 2001). The genes involved in infection initiation or symptom induction may also be variable in expression and infection generation among different isolates (Hanselle et al., 2001). Teleomorph (*D. rabiei*) present in life cycle of *A. rabiei* contribute to variability by generating new arrangement of virulent genes which produce new pathotypes (Baite et al., 2016). In *A. rabiei* the number of new pathotypes have been reported and are being screened at ICARDA and through screening low levels of resistance in available germplasm have been observed so for. Due to the genetic diversity of pathogen management of disease has become more complicated (Nourollahi et al., 2010). The existence of such pathogen variability allows pathotypes to become more virulent by developing resistance against fungicides (Imtiaz et al., 2011; Baite et al., 2016; Iqbal et al., 2018).

Factors Affecting the Disease Incidence

Occurrence, spread and severity of disease in nature are primarily controlled by different environmental factors. The favorable environmental conditions have weighty effects on initiation and spread of the disease infections. The climatic factors like relative humidity, temperature, wetness duration and windiness have critical effects on the prevalence and spread of disease (Casas & Kaiser, 1992; Pande et al., 2017; Megersa et al., 2017). Disease attack usually becomes destructive when the temperature remains about 20 ^{o} C showing positive correlation to disease establishment (Casas et al., 1996). The Fungal growth and development of fruit bodies occurs rapidly at 20^{o} C (Casas & Kaiser, 1992). The long cool and moist spells are considered the most encouraging for conidia oozing out and the rain splashes disperse them to surrounding plant populations (Armstrong et al., 2001). The disease becomes epidemic in the cool and humid environment (Sharma & Ghosh, 2016). Subsequent wetness, strong wind and rain splash accelerate dispersal of conidia from infected plant parts to healthy populations (Pande et al., 2005). Relatively low humidity has been found more critical factor in limiting the disease incidence rather than the temperature (Nene, 1982). Heat treatment (55-60 ^{o} C) is also responding factor helping in pathogen suppression (Tripathi *et al.*, 1987; Sharma & Ghosh, 2016).

Genetic Resources of AB Resistance

Exploitation of resistant genetic resources is most successful way to minimize the yield losses occurring through incidence of chickpea blight. A number of screening methods were employed by different researcher for identification of resistant genetic resources. For field screening involving natural environmental conditions was practiced by Pandey et al. (2005). While, Chen, Coyne Peever and Muehlbauer (2004) performed evaluation of the genotypes under controlled conditions of temperature and relative humidity by applying artificial mist of the irrigation foggers. Similarly, Chen and Muehlbauer (2003) invented a new technique for the exploration of resistant sources and named mini dome technique in USA which was found successful for screening of AB resistant germplasm (Pandey et al., 2005; Islam et al., 2017).

Most of researchers have emphasized the use of screening method adopted by ICARDA which involves inoculation of the nurseries with disease debris and the artificial spore suspension (Megersa et al., 2017; Islam et al., 2017; Iqbal et al., 2018; Rubiales et al., 2018). Once disease is established, two methods are used to measure the severity of the disease. First method involves 1-9 scale disease rating as proposed by Reddy and Singh (1984) and documented in table 1. Scale 1-9 involves rating of genotypes by calculation of the percentage of infected plants and on the basis of percentage respective infection rank is marked to genotypes. Germplasm is screened out by providing favorable conditions for disease incidence and availability of inoculums, genotypes ranking from 1-3 (0-10% infections) are classified as resistant to AB.

Table 1 Ascochyta Blight, Disease Rating Scale

S.No.	Infected area (%)	Scale	Host reaction
1	0	1	With no infection
2	1-5	2	Highly Resistant
3	6-10	3	Resistant
4	11-15	4	Moderately Resistant
5	16-40	5	Tolerant
6	41-50	6	Moderately Susceptible
7	51-75	7	Moderately susceptible to Susceptible
8	76-100	8	Susceptible
9	Up to 100		Highly susceptible

A similar scale for screening of chickpea genotypes against disease was later proposed by Manjunatha and Saifulla (2013) which was also found helpful for evaluation of resistant, moderately resistant, tolerant and susceptible genotypes. The second screening method has found objective oriented which involves determination of percentage of infected leaves of all plants (Kanouni et al., 2010). For screening of large scale experimentation these techniques are extensively being utilized by several researchers in India, Australia, Syria, USA and Pakistan (Islam et al., 2017). Screening of a large number of chickpea genotypes for genetic resistance against AB was focused in early 1980s. 1258 Desi and 174 Kabuli genotypes were evaluated by Verma *et al.*, 1981. Singh *et al.*, 1981 put 3200 Kabuli types under screening tests. Several other researchers screened thousands of chickpea genotypes and reported resistant genetic resources. The list of these resistant genotypes is shown in table 2.

Table 2. List of Chickpea Genotypes Reported Resistant against Ascochyta Blight

SN	AB Resistant Genotypes Reported	References
1	ICC4324, ICC3996, ICC 4475, ICC6988, ICC6981, ILC2467	Verma et al., 1981
2	CM72 and CM86	Haq et al., 1981
3	ICC 4200, ICC4248, ICC5124, ICC3634, ICC6981, ILC196, ILC3346, ILC3956, ILC4421	Redy and singh ,1984
4	ILC3856, ILC3279, ILC 191, ILC72	Singh et al., 1984
5	ICC2160, ICC1257, ICC1069	Kalia, 1984
6	ILC3956	Redy and kabbabeh ,1985
7	NEC2451,P919, PB82-1, BRG8, P1252-1, EC26446	Tiwari and Pandey, 1986
8	ILC183 and ILC82-11	Kinaki and Dalkiran, 1987
9	ILC-236, ILC-484 and ILC- 484	Redy and singh ,1990
10	ILC-3864, ILC-3870 and ILC-4221	Pal and Singh, 1990
11	ILC 6482, ILC5925, ILC 5586 ,ILC-482 and ILC-3279	Reddy and Singh, 1993
12	ICC-4475, FLIP 90-95C, ICC-12004, ICC-13508, ICC-13269 and ICC-13555	Iqbal et al., 1994
13	CM72 and ILC191	Sarwar et al., 1996
14	FLIP97-227C, FLIP97-132C, C FLIP98-224 FLIP95-68C, FLIP95, FLIP94-90C,	Iqbal et al., 2002
15	F16-90 C,NCS950038, ,NCS950088 CMC228S,SEL96TH11488,FLIP-75C	Hussain et al., 2002
16	FLIP 95-68C, FLIP 95-53C, FLIP 97-74C FLIP 95-53C and FLIP 98-177C	Toker and Seyin, 2003
17	CC106199, CM1966193, CMC77S, CM843198, CM1441198, CM1223198, CC104199,	Alam et al., 2003
18	PI 559361, PI 559363 and W6 22589	Chen et al., 2004

19	ATC-46934, ATC-46892 and ATC-46935	Nguyen et al., 2005
20	Punjab-91, Bital-98, Punjab-2000, Balkassar-2000 and Vanhar	Chaudry et al., 2005
21	NCS , AZRI 7130 and AZRI 17115	Malik et al.,2006
22	03159, 93A-086, 93A-111 ,03039, 03041, 03053, 03115,	Ilyas et al., 2007
23	Himachal Channa 1, Himachal Channa 2, GPF 2, HPG-17, PBG-1 and PBG-2	Basandrai et al., 2009
24	FLIP 98-133C and FLIP 98-136C	Chandirasek et al., 2009
25	Vinhar, Bittle-98, 06025 ,06056, , 06031, , 06027, 06026, 06035, 06040, 06041	Ghazanfar et al., 2010
26	54247, 53651, 53045, 53217, 53218, 53323and 53398	Iqbal et al., 2010
27	FLIP 97-121C	Kaur et al., 2012
28	04A09, 06A083 and 07A006	Sahi et al., 2012
29	FLIP 4107, FLIP 1025 and FLIP 10511	Benzohra et al., 2013
30	Thal-2006, Dasht and Vanher-2000	Rehman et al.,2013
31	ICC7052, ICC4463, ICC4363, ICC2884, ICC7150, ICC15294 and ICC11627	Ghosh et al., 2013
32	K-60013, K-96022 K-98008, D-97092, K-96001, Punjab-2008, D-91055, D90272, D-96050	Ahmad et al., 2013
33	Thal-2006, 5CC-109 and 06001	Rashid et al., 2014
34	ILC72, ILC182, ILC187, ILC200 and ILC202	Benzohra et al., 2015
35	ILC 8068, ICC 4475, ILC 200, ILC 7374 and ILC 7795.	Labdi et al.,2015
36	K0010/09, K0021/09, K0025/09, K0030/09, K0051/09, K0054/09, K0057/09, FG-0908	Shah et al., 2015
37	Genesis 425, CICA1007 and CICA0912	Moore et al., 2016
38	ICC15978, ICC 3996 and ICC 76	Baite et al., 2016
39	ICCV-96836 and Arerti	Zewdie & Tadesse, 2018
40	Punjab 2008, Bittal 2016 and D.09027	Iqbal et al., 2018

Disease Management

Aschochyta Blight can be effectively managed over cultural practices, use of chemicals and integrated disease management. The cultural practices are adopted to minimize the sources of inoculum. Sowing of diseased free seed, rotation of crops in such a manner that non host crop follow the host crops, elimination of crop residues and deep sowing of crop have been found most effective to minimize disease incidence (Pandey et al., 2005, Mohammdi, 2019). Agronomic practices such as the late sowing of crop, lower seed rate, more plant to plant and row to row distances are also helpful in reduction of disease development. Application of fertilizer with maximum ratios of potassium has also been found effective in retardation of disease (Pandey et al., 2005; Malik et al., 2006). The extensive tillage practices also prevent the production of ascospores and restrain the development of teleomorph stage thus inhibits disease infections (Hanselle et al., 2001). Chemical control of diseases on cereal crops is usually avoided to prevent transmission of their certain negative effects into grains however, in case of severity of diseases use of chemicals have been found much helpful. Several fungicides have been suggested for effective control of AB.

The seed treatment with Calixin-M, thiabendazole, iprodione, thirum and propiconazole have been reported effective by various researchers (Reddy & singh, 1984; Ghazanfar et al., 2010). Application of repeated foliar spray of mancozeb, captan, bordeaux mixture, sulfur, dithianon, cholorthalonil and ferbam have been found effective to successfully

reduce the disease attack (Pandey et al., 2005; Rashid et al., 2014; Rubiales et al., 2018, Mohammdi, 2019). Integrated disease management (IDM) is crucial to take advantage from the genotypes that are not highly resistant to AB. IDM for management of AB has been proposed by various researchers however, practices recommended by Pandey et al., 2005 have been found most effective which include: (i) use of clean seed (pathogen free), (ii) deep ploughing to eradicate the disease debris/ infected plant residues, (iii) proper rotation of crops, (iv) sowing of the resistant varieties, (v) use of the foliar fungicides. Manipulation of sowing dates, destruction of residues of previous crop, seed treatment with fungicide, rotation of non-host crops and foliar fungicide applications have been found most effective to control the disease (Nene & Reddy, 1987; Redy & singh ,1990; Singh & Sharma, 1998; Rubiales et al., 2018).

CONCLUSIONS

The management of AB is vital to acquire stable and increased chickpea yields across the world. Since 1980's, the extra ordinary efforts by the several researchers enabled us to understand pathogen, its life cycle, variability, factors responsible for disease incidence, screening of resistant sources and disease management strategies. The number of the enthusiastic and the practicable researches were conducted involving evaluation of the thousands of advance strains and cultivars. Studies have reported that humid, windy and cool environment favors the prevalence of disease. Various cultural practices and foliar application of fungicides have been found helpful in reduction of disease attack to some extent however, durable management of AB is only possible by exploiting the resistant chickpea genotypes. Further efforts for explorations of the genetics, ecology and host-pathogen interaction of *A. rabiei* are required to develop efficient disease management strategies and to evolve the promising cultivars for AB-prone environments through breeding programs.

REFERENCES

Armstrong, C. L., Chongo, G., Gossen, B. D., Duczek, L. J. (2001). Mating type distribution and incidence of the teleomorph of *Ascochyta* rabiei (Didymella rabiei) in Canada. *Canadian Journal of Plant Pathology*, 23, 110–113.

Aslam, M., Jiang, C., Zafar, U., Usama, M., & Haroon, H. (2018). Functional Genomics Prospective of Chickpea Breeding: Constraints and Future Directions. Modified Conceptual Development Agronomy, 3(4).

Asnake, F. (2016). Unraveling Valuable Traits in Ethiopian Grain Legumes Research Hastens Crop Intensification and Economic Gains: A Review. *Universal Journal of Agricultural Research*, 4, 175-182.

Baite, M. S., Dubey, S. C., & Singh, B. (2016) Morphological variability in the Indian isolates of *Ascochyta rabiei* causing blight in chickpea and evaluation of chickpea cultivars. *Indian Journal of Plant Protection*, 44, 74-82.

Barve, M. P., Arie, T., Salimath, S., Muehlbauer, F. J., Peever, T. L., (2003). Cloning and characterization of the mating type (MAT) locus from *Ascochyta* rabiei (teleomorph: Didymella rabiei) and a MAT phylogeny of legume-associated *Ascochyta* spp. Fungal Genet. Biol. 2003, 39, 151–167.

Benzohra, I. E., Bendahmane, B. S., Labdi, M., & Benkada, M. Y. (2013). Sources of Resistance in Chickpea Germplasm to Three Pathotypes of *Ascochyta rabiei* (Pass.) Labr. In Algeria. *World Applied Science Journal,* 21 (6), 873-878.

Bhardwaj, R., Sandhu, J. S., Kaur, L., Gupta, S., Gaur, P., & Varshney, R. K. (2010). Genetics of *Ascochyta blight* resistance in chickpea. *Euphytica*, 171, 337–343.

Butler, E. J. (1918). Fungi and disease in plants, Bishen Singh and Mahendra Pal Singh, New Conaught Place, Dehradun Periodical Experts, 42-D, Vivek Vihar, Dehli-32. 547 pp. (reprinted 1973).

Casas, A., & Kaiser, W. J. (1992). The Development of *Didymella rabiei*, The teleomorph.

Casas, A., Cortes, J. A., Diaz, R. M. (1996). Airborne ascospores of Didymella rabiei as a major primary inoculum for *Ascochyta blight* epidemics in chickpea crops in southern Spain. *European Journal of Plant Pathology*, 102, 237–245.

Chen, W., & Muehlbauer, F. J. (2003). An improved technique for virulence assay of the *Ascochyta*

Chen, W., Coyne, C. J., Peever, T. L., & Muehlbauer, F. J. (2004). Charactrization of Chickpea differentials for pathogenicity assay of *Ascochyta blight* and identification of chickpea accessions resistant to *Didymella rabiei*. *Plant Pathology*, 40, 401-407.

Ghazanfar, M. U., Sahi, S. T., Javed, N., & Wakil, W. (2010). Response of advanced lines of chickpea against chickpea blight disease. *Pakistan Journal of Botany*, 42, 3423-3430.

Ghosh, R., Sharma, M., Telangre, R., & Pande, S. (2013). Occurrence and distribution of chickpea diseases in central and southern parts of India. *American Journal of Plant Sciences*. 4, 940–944.

Hanselle, T., Ichinoseb, Y., & Barz, W. (2001). Biochemical and molecular biological studies on infection *(Ascochyta rabiei*)- induced thaumatinlike proteins from chickpea plants *(Cicer arietinum* L.), 1096-1107.

Illarslan, H., & Dolar, F. S. (2002). Histological and ultrastractural changes in leaves and stems of the resistant and susceptible chickpea cultivars to *Ascochyta rabiei*. *Journal of Phytopathology*. 150: 340-348.

Imtiaz, M., Abang, M. M., Malhotra, R. S., Ahmed, S., Bayaa, B., Udupa, S. M., Baum, M. (2011). Pathotype IV, a new and highly virulent pathotype of Didymella rabiei, causing *Ascochyta blight* in chickpea in Syria. *Plant Diseases*, 2011, 95, 1192.

Iqbal, J., Zafar, S. A., Ashraf, A., & Hassan, A. (2018). Pathogenic variability in chickpea genotypes against chickpea blight. *Journal of Agriculture & Basic Sciences, 03 (04).*

Islam, W., Qasim, M., Noman, A., Idrees, A., & Wang, L. (2017). Genetic resistance in chickpea against *Ascochyta blight*: Historical efforts and recent accomplishments. *The Journal of Animal & Plant Sciences*, 27(6), 1941-1957.

Kaiser, W. J., & Okhovat, M. (1996). Distribution of *Didymella rabiei*, the teleomorph of *Ascochyta rabiei* in Iran. *Iranian Journal of Plant Pathology*, 32, 158-162.

Kalia, N.B. (1984). Chickpea improvement in Himachal Pardesh. Chickpea newsletter. 11:9.

Kanouni, H., Taleei, A., Peyghambari, S. A., Okhovat, S. M., & Abang, M. (2010). Impact of different pathotypes and spore concentrations on *Ascochyta blight* incidence in two Chickpea (*Cicer arietinum* L.) genotypes. Iranian Journal of Crop Sciences, 12, 56-68.

Khan, M. I., Arshad, W., Zeeshan, M., Ali, S., Nawaz, A., & Fayyaz, M. (2018). Screening of chickpea kabuli (Cicer arietinum L.) germplasm against *Ascochyta blight* (*Ascochyta* rabiei). *Journal Biology & Environmental Sciences*, 12(2): 128-132.

Kovachevski, I. C. (1936). The blight of chickpea (*Cicer aritinum*). *Mycosphaerella rabiei* f.sp. *Cicri*. Ministry of Agriculture and National Domains, Plant Protection Institute, Sofia, Bulgaria.

Labrousse, F. (1930). Anthracnose of the chickpea (Cicer arietinum). Revue de Patholgie Patholgie Vegetale ed d'Entomologie Agricole de France. 27, 174–177.

Labrousse, F. (1931). Anthracnose of the chickpea. Revue de Pathologie Vegetale et d'Enotmologie Agricole de France. 28, 226–231.

Li, H., Rodda, M., Gnanasambandam, A., Aftab, M., Redden, R., Hobson, K., Rosewarne, G., Materne, M., Kaur, S., & Slater, A. T. (2015). Breeding for biotic stress resistance in chickpea: Progress and prospects. *Euphytica*. 204, 257–288.

Malik, S. R., Iqbal, S. M., & Haqqani, A. M. (2006). Resistance screening to *Ascochyta blight* disease of chickpea in Pakistan. *Chickpea and pigeon pea newsletter*, 13, 30-31.

Manjunatha, H., & Saifulla, M. (2013). Screening of desi and kabuli chickpea (*Cicer arietinum* L.) entries against alternaria blight. *Indian Journal of Scientific Research & Technology,* 1(3), 66-68.

Megersa, T., Losenge, T., & Chris, O. (2017). The Survey of Chickpea (Cicer arietinum L) *Ascochyta blight* (*Ascochyta* rabiei Pass.) Disease Status in Production Regions of Ethiopia. *Plant*, 5 (1), 23-30.

Mohammdi, W. A. D. (2019). "Management of Ascochyta Blight in Chickpea". *Acta Scientific Agriculture*, 3 (3), 105-111.

Moore K., Hobson K., Dron S., Harden S., Bithell P., Sambasivam R., et al. (2016). Available at https://grdc.com.au/Research-and-Development/GRDC-Update-Papers/2016/03/Chickpea-Ascochyta-latest-research-on-variability-and-implications-for-management

Morrall, R., & Mackenzie, D. L. (1974). A note on the inadvertent introduction of *Ascochyta rabiei* to North America, A destructive chickpea disease. *Plant Disease Reporter*, 58(4), 342-345.

Nene, Y. L. (1982). A review of the *Ascochyta blight* of chickpea. Trop. Pest Manag. 28: 61–70.

Nene, Y. L. (1984). A review of *Ascochyta blight* of chickpea (*Cicer arietinum* L.). In: *Ascochyta blight* and winter sowing of chickpeas, Sexena, M.C. and K.B. Singh (Eds.). Martinus Nijhoff/DR W. Junk Publishers, The Netherlands. 17-33.

Nourollahi, K., Javannikhah, M., Naghavi, M. R., Lichtenzveig, J. L., Okhovat, S. M., Oliver, R. P., & Ellwood, S. R. (2010). Genetic diversity and population structure of *Ascochyta rabiei* from the western Iranian Ilam and Kermanshah provinces using MAT and SSR markers. *Mycological Progress*, 10 (1), 1-7.

Pandey, B. K., Singh, U. S., & Chaube, H. S. (1987). Mode of infection of *Ascochyta blight* as caused by *Ascochyta rabiei*. *Journal of Phytopathology*. 119, 88-93.

Pandey, P., Irulappan, V., Bagavathiannan, M., & Kumar, M. (2017). Impact of combined abiotic and biotic stresses on plant growth and avenues for crop improvement by exploiting physio-morphological traits. Front. *Plant Sciences*, 8, 537.

Punithalingam, E., & Holliday, P. (1972). *Ascochyta rabiei*. In descriptions of pathogenic fungi and bacteria; commonwealth mycological institute: Kew, UK., 34: p337.

rabiei on chickpea. Int. Chickpea Pigeonpea Newsletter. 10: 31–33.

Rashid, A., Younas, M. U., Ehetisham, M., Farooq, M., Waris, I. H., Perveez, E., Ahmad, F., Latif, M., & Ahmad. M. (2014). Screening of chickpea (*Cicer arietinum* l.) varieties against *Ascochyta blight* and its management through biopesticides. Arch. Phytopathol. Pl. Protect. 47 (8), 938-943.

Reddy, M. V., & Kabbabeh, S. (1985). Pathogenic variability in *Ascochyta rabiei* (Pass.) Lab. In Syria and Lebanon. Phytopathol. *Mediterrenean*. 24: 265-266.

Rubiales, D., Fondevilla, S., Chen, W., & Davidson, J. (2018). Editorial: Advances in Ascochyta Research. *Front. Plant Sciences*. 9, 22.

Sahi, M. S. T., Burhan, M., Iqbal, T., & Sarwar, N. (2012). Screening of chickpea breeding material and varieties/elite lines against blight (*Ascochyta rabiei*). *Journal of Agricultural Research,* 50(4), 517-523.

Shah, T. M., Imran, M., Atta, B., Shafiq, M. M., Aslam, M., & Hussain, K. (2015). Screening of chickpea advanced lines for sources of resistance against blight and wilt two major diseases of chickpea. *Pakistan Journal of Botany,* 47(6), 2443-2448.

Sharma, M., & Ghosh, R. (2016). An update on genetic resistance of chickpea to *Ascochyta blight*. Agronomy. 6: 18.

Singh, G., & Sharma, Y. R. (1998). The *Ascochyta blight* of chickpea. In IPM System in the Agriculture: Pulses

Tripathi, H. S., Singh, R. S., & Chaube, H. S. (1987). Effect of sun drying on the recovery of *Ascochyta rabiei* from infected chickpea seeds. *Chickpea Newsletter*, 16:13-14.

Upadhyay, R.K., Mukherji, K.G., Eds.; Aditya Books Pvt. Ltd.: New Delhi, India, pp. 163–195.

Upadhyaya, H. D., Dwivedi, S. L., Gowda, C., & Singh, S. (2007). Identification of diverse germplasm lines for agronomic traits in a chickpea (*Cicer arietinum* L.) core collection for use in crop improvement. *Field Crops Research*, 100, 320–326

Verma, M. M., Singh, G., Sandhu, T. S., Singh, H., Sandhu, S. S., Singh, K., & Bhullar, F. (1981). Sources of resistance to gram blight and grey mold. *Chickpea newsletter*, 4, 14-16