GENETIC RESISTANCE IN CHICKPEA AGAINST ASCOCHYTA BLIGHT: HISTORICAL EFFORTS AND RECENT ACCOMPLISHMENTS

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ABSTRACT

Chickpea Blight is a devastating disease of chickpea (Cicer arietinum L.) worldwide caused by Ascochyta rabiei (Pass.) Lab. The disease is more devastating particularly in long cool and humid environmental spells. It results in huge losses by wiping off all the crop in the desert areas whenever it hits its epidemics. To manage this disease, different management strategies are practiced. However, breeding resistance to the host is the best and environmentally safe strategy. During 1970s, the loss of host resistance against the pathogen was reported, so extra ordinary efforts were started by the scientists to enhance the host tolerance towards the pathogen. In this way, relatively simple field screening techniques were followed for breeding and identification of new resistant genotypes. The review sums up the efforts regarding host breeding against chickpea blight involving large scale field screening experiments as well as recent marker assisted breeding using the molecular mapping and QTLs against the different strains of pathogen. Moreover, the important aspects covering the related knowledge of the pathogen, its biology, variability, perpetuation, characteristics and factor affecting the disease establishment have also been summarized.

Key Words: Breeding, Disease, Fungus, Gram, Molecular marker, Screening

INTRODUCTION

Ascochyta blight (AB) of chickpea caused by Ascochyta rabiei (Pass.) Lab. is a devastating biotic factor contributing in yield reductions worldwide (Singh et al., 1994; Singh and Sharma 1998; Siddique et al., 2000, Kaiser et al., 2000; Chongo et al., 2003; Millan et al., 2003; Sarwar et al., 2012; Sharma and Ghosh, 2016). Globally, chickpea is cultivated in the rain fed areas where the disease can vire out all the crop under favorable conditions (Singh, 1997; Udupa and Baum, 2003; Pande et al., 2005; Wise et al., 2008). In 1911, AB was detected for the first time ever in Punjab province around the Attock region (Butler, 1918). Moving to the other regions of the globe, Morall and Mckenzie (1974) reported about AB in America from Saskatoon area and considered the import of the pathogen through seed. Till the eighties, the disease had prevailed around twenty six countries of the world (Nene, 1980). Now it is assumed that disease exists in more than 40 countries (Bhardwaj et al., 2010; Sharma and Ghosh, 2016).

All aerial parts of chickpea can come under symptomatic stress of AB (Nene, 1984; Shṭienberg et al., 2000) (Figure 1). Emerging seedlings show brown lesions at their base which actually happens due to infected seeds. Progressively, these lesions start increasing in size thus covering and girdling around the stem ultimately resulting in death of the plants. Leaflets bear irregular round to elongated shaped brown lesions showing dark red or dark brown margins. Usually circular lesions with darker margins become visible upon the green pods having concentrically and circularly arranged pycnidia. Involving the serious infections, the lesions prevail to the seeds inside the pods which become shriveled due to fungal fruiting bodies (Nene, 1982; Singh and Sharma, 1998; Akem, 1999). Distribution of AB is dependent upon the primary infections from diseased debris or infected seeds. The mechanism of distribution is accelerated by the highly humid and rain splashed resulting in epidemic breakdown in rain fed areas leading to complete loss of the crop (Chauhan and Sinha, 1973; Alam et al., 1987; Armstrong et al., 2001). Even the resistant cultivars cannot be regarded as symptom free as they exhibit small dark spots as well but due to the genetic resistance the spots cannot progress further (Chongo and Gossen, 2003).

AB can be managed effectively via utilization of chemicals (Rehman et al., 2013) but chemicals are harmful, have residual effects and are expensive as well (Zhang et al., 2017). So the most efficient and eco-friendly way to manage the disease involves breeding host resistance against any disease (Li et al., 2015). Breeding host resistance against AB includes conventional field screening experiments (Pande et al., 2005) as well has recent molecular breeding techniques (Labdi et al., 2013). But production of resistant genotypes has become a supreme challenge due to continuous evolving new pathotypes of A. rabiei (Singh and Reddy, 1991; Sharma and Ghosh, 2016).
The review aims to sum up all the research information about the genetic resistance in chickpea against AB which may include the valuable information about symptomology, biology, variability, life cycle and different factors affecting the AB fungus during its different life stages. Furthermore, the review summarizes almost all the field screening efforts against AB and molecular breeding techniques.

**Biology and variability of *Ascochyta rabiei***: The existence of *A. rabiei* in nature is categorized by two stages i.e. anamorph and teleomorph. In anamorph, pear-shaped pycnidia contain unicellular or biseriell pycnidiospores (6-23 um) produced upon conidiophores (Punithalingam and Holliday, 1972; Nene, 1982). The fungus can grow upon different nutrient media producing a cream colored mycelium having immersed brown to black pycnidia or unicellular conidia inside. The teleomorph, *Didymella rabiei* (Kovachevski) var. Arx (Syn. *Mycosphaerella rabiei* Kovachevski), belongs to bipolar heterothallic ascomycete which is involved in production of pseudothecia upon over wintered filed crop residues. The particular stage came into the reports of Kovachevski (1936) in Bulgaria firstly, secondly in Russia (Kaiser and Okhovat, 1996), then Greece (Wilson and Kaiser, 1995), Hungary (Armstrong et al., 2001), Spain (Barve et al., 2003), Syria (Rhaiem et al., 2008), Iran (Taleei et al., 2008) and United States (Vail and Banniza, 2009). *A. rabiei* involves single sexual reproduction per season followed by several asexual reproductions during the parasitic phase (Trapro-Casas et al., 1996) (Figure 2). A single regulatory locus (mating-type locus) controls all the process of sexual reproduction as it acquires alternating different sequences at the mating-type loci (Barve et al., 2003; Peever et al., 2004; Rahiem et al., 2008; Taleei et al., 2008; Vail and Banniza 2009; Atik et al., 2011). The fungus penetration occurs within 24 hours of adhesion to host surface through natural openings by forming appressoria (Pandey et al., 1987; Illarslan and Dolar 2002). The penetration process completes within seven days when all the non lignified cells are destroyed (Kohler et al., 1995; Pandey et al., 1987; Illarslan and Dolar, 2002).

*A. rabiei* is highly diversified fungus having different physiological races that are becoming challenging for the breeding host resistance (Sharma and Ghosh, 2016). The difference in various isolates from different countries e.g. Pakistan, Iran, Turkey and India was recorded regarding growth, pathogenicity and colony appearance attributes (Kaiser, 1973). The various isolates of the fungus were varying in different infecting processes that may involve germ tube formation, changes in epidermal or sub epidermal cells, differing cellulose degrading enzymes etc. (Pandey et al., 1987). The genes involved in symptoms induction or infection initiation can also be variable among different isolates of the fungus as their expressions in infection generation can be slower or faster that may also be dependent upon susceptibility or resistance of the cultivar (Hanselle et al., 2001). Three different isolates were identified from Algeria (Ag1, Ag2 and Ag3) which proved much aggressive in infection creation and expression of the genes involved in quick disease establishment (Zerrougli et al., 2007). Higher genetic variability was observed when simple sequence repeat (SSR) and mating type (MAT) markers were involved in determination of genetic structure. So management of the AB is becoming difficult due to genetic diversity of the pathogen (Nourollahi et al., 2010).

**Factor affecting survival and growth rate of the fungus**: Environmental conditions are very crucial in the establishment and spread of any pathogen in nature. Similarly, the environment has weighty effects upon *A. rabiei* as because temperature around 20°C positively correlates with infection establishment (Trapro-Casas and Kaiser, 1992). Moist conditions are considered very favorable for conidia oozing out and rain splashing help in dispersal to neighboring plants (Armstrong et al., 2001). Long cool and moist spells result in disease epidemics (Rehman et al., 2013; Sharma and Ghosh, 2016). Similar findings were reported by Bedi and Aujla (1969, 1970) as they mentioned that development of fruiting bodies and fungal growth occurs better at 20°C and when pH is in between 7.6 to 8.6. Correspondingly, Chaube (1986) reported reduced survival of the fungus in infected seeds and germination of infected ones at high temperature in may up to 70% but survival rates and germination percentage recorded in lowering temperature of December was much low i.e. 30%. Dry heat treatment is also a responding factor which is helpful in pathogen eradication when applied at 55°C-65°C for 6-12 hours (Tripathi et al., 1987). The life span of the plant is also a significant aspect that may play a vital role in establishment of the disease as the fifteen days old plant are most susceptible to AB disease even if the pathogen stress is lower (Serrone et al., 1987).

**Breeding host resistance against Ascochyta blight**: Breeding host resistance is the most significant, efficient, eco friendly and relatively economic pathway to manage any disease (Li et al., 2015). So using resistant varieties is of paramount significance to obtain high yield (Sarwar et al., 2003; Hassan et al., 2012). But production of resistant genotypes has become a supreme challenge due to continuous evolving new pathotypes of *A. rabiei* (Singh et al., 1991; Sharma and Ghosh 2016). New strains emerge continuously in past and resulted in loss of resistance in available germplasm such as F8 (1940–1941), C 1234 (1950–1951) and C 235 (1968) lost their
resistance and were banned for cultivation previously (Bedi and Agarwal, 1962; Nene, 1982). Comparing the past with the present, durable resistance in chickpea against AB had not achieved (Lichtenzeig et al., 2002). Several structural and chemical components play vital role in building up genetic resistance in chickpea to AB. These include hydrogen-peroxide-mediated cell wall linkages, chitinase, β-1,3-glucanase and thaumatin proteins, phytoalexin accumulation & detoxification and glutathione S-transferase (Cho et al., 2004; Jayakumar et al., 2005). Variation in aggressor are approximately similar in A. rabiei populations as well as chickpea cultivars (Cho et al., 2004; Udupa and Baum, 2003; Chen et al., 2004; Chongo et al., 2004). The resistance reduces when cultivars enter their flowering and podding stage (Singh and Reddy, 1993; Chongo and Gossen, 2003). It is because the pathogen is not seemed to be interested in attacking upon the lignified tissues and pith parenchyma (Angelini et al., 1993; Illarslan and Dolar, 2002). Further studies revealed that the phenomena is due to the release of hydrogen peroxide in the apoplast, oxidases in Plasma membrane, oxalate oxidases and peroxidases in cell wall (Rea et al., 2002). Two important methods are followed for broading the host resistance to AB which has been explained extensively.

1. Field Screening Accomplishments: Exploiting host plant resistance against AB by use of various filed screening strategies which may involve controlled conditions or natural conditions was explained by Pande et al. (2005). Under field conditions, environmental factors play critical role for the disease development i.e. relative humidity (RH) and temperature. Under controlled conditions, RH can be created artificially after inoculation by use of artificial mist by irrigation foggers which leads to successful disease development (Chen et al., 2005). Other important factor for disease establishment is the use of appropriate artificial inoculum concentration. Under controlled conditions, Cut-Twig screening in metal trays and seedling screening methods are commonly used. In USA, a mini dome technique invented by Chen and Muehlbauer (2003) is also proved successful regarding AB screening.

The field screening method that is adopted worldwide for identification of resistant genetic sources against the blight at large scale was developed by ICARDA (Singh et al., 1981). In this, susceptible chickpea lines are planted at frequent intervals or all around the nursery. Nursery is inoculated with diseased debris and sprinkler irrigation is provided to create humid conditions (Udupa and Baum, 2003; Chen et al., 2005). Re-inoculation of the nursery is done with artificially prepared spore suspension (Ilyas and Khan, 1986). After the disease establishment, two methods are used to assess disease severity from which first method includes comparing with 1-9 disease rating scale (Reddy and Singh, 1984). The second method includes determination of percentage of infected leaves for each plant (Kanouni et al., 2010). This second method is thought to be more objective oriented and considered superior for disease estimation (Chen et al., 2004). These filed screening techniques have been sturdily adopted in Pakistan, India, Syria, Canada, USA, Australia and turkey for large scale experimentation.

In the early 1980s, the researchers focused upon finding genetically resistant germplasm against AB. Thousands of accessions were screened against different Ascochyta strains. Verma et al. (1981) evaluated a germplasm collection of 1258 Desi and 174 Kabuli types of chickpeas. Singh et al. (1981) conducted field screening tests of 3200 Kabuli accessions at ICARDA. Similarly, Shukla (1984) tested 1000 genotypes for resistance to AB. Furthermore, Basher and Haware (1986) screened 3360 lines while Kinaki and Dalkiran (1987) evaluated 1100 germplasm lines against AB. We reviewed that researchers from Pakistan and India conducted dozens of field screening experiments and have provided huge information regarding resistant to moderately resistant AB genotypes of chickpea. Similarly, several other scientists around the world have recorded their efforts by identification of resistant germplasm source for host breeding against Ascochyta rabiei using field screening methods. We have tried to populate the list of almost all the documented field experimentation since 1980 to date is populated (Table 1).

2. Marker assisted breeding: Marker assisted selection (MAS) is the best way regarding single-gene traits but it can be utilized for breeding the multiple and complex traits as well but still it seemed quite difficult as fewer success has been attained in this regard (Ribaut and Hoisington, 1998; Young, 1999; Bouchez et al., 2002; Lecomte et al., 2004)(Figure 3). Disease resistance program can be accelerated by MAS and their application by DNA markers linked to resistance genes (Michelmore, 1995; Hammond-Kosack and Jones, 1997). Historical studies revealed that AB resistance is controlled by a single dominant gene (Hafiz and Ashraf, 1953; Vir et al., 1975; Eser, 1976). But Tekeoglu et al., (2000) mentioned that two quantitatively inherited recessive genes and several minor genes play vital role against AB resistance. It is not known that these two genes exhibit the same loci or different. Further details of host pathogen interactions and resistance inheritance mechanism have been explained by Pande et al. (2005, 2010). Interaction between multiple recessive genes and a couple of dominant complementary genes were confirmed regarding inherited resistance in race 4 of Ascochyta rabiei upon fifteen chickpea genotypes (Labdi et al., 2013).
Molecular mapping of AB resistance conferring genes is an important but complex aspect to create resistant varieties. Regarding this, identification of several QTLs have been confirmed with respect to multiple linkage groups against the AB resistance. Among them, linkage group 2 bears two most important QTLs at loci GA16 and TA37 which are thought to be managing the resistance against AB pathotype 1. Linkage group 4 inhibits a couple of QTLs which are linked to TA72, CaETR. Regarding pathotype II, upon linkage group 2, a specific TA46 marker was identified for resistance in FLIP 84-92c under field conditions (Cho et al., 2004). Under controlled conditions, association of different loci i.e. TS12b and STMS28 was found upon linkage group 1 (Flandez-Galvez et al., 2003a). Further studies were conducted to understand the position of different QTLs upon the chromosomes and found that QTL1 was located upon chromosome-C, QTL2,3 upon chromosome-H and QTL4,5,6 upon chromosome-B (Bian et al., 2007). All these QTLs acquire resistance against AB Phtotype I and II and are responsible for phenotypic variation. A few years ago, fourteen micro markers linking to QTLs Ar2a,c ;Ar3c ; Ar4a,b ; Ar6b and Ar8a upon LG2,3,4,6 and LG were identified (Hamwieh et al., 2013).

Allele specific markers (CaETR, TA72, SCY17, GAA47, SCAR17590) have been proven successful for AB resistance (Castro et al., 2015; Bouhadida et al., 2013). In the same infection lesion or same crop field a lot of pathotypes of AB can be coexisting which complicate the overall picture (Peever et al., 2004). This may lead to the random mating of these pathotypes thus enhancing the genetic diversity (Barve et al., 2003). AB resistance is a quantitative trait so two disease scoring systems have been evolved for different QTLs, measuring disease reaction (Flandez-Galvez et al., 2003a). QTL1 and QTL 2 were reported for involvement in Ascochyta resistance showing 50 and 45% of blight variation within two successive years while their association with genomic regions on LG1, LG2, LG3 and LG4 is also conferred (Santra et al., 2000; Flandez-Galvez et al., 2003b; Udupa and Baum, 2003; Taran et al., 2007; Aryamanesh et al., 2010). Among all, the significance of LG4 regarding resistance against the pathogen was reported by many scientists (Kanouni et al., 2009). All the documented findings regarding QTLs, representative linkage groups and their phenotypic variations have been described (Table 2).

3. Marker assisted back cross breeding: Marker assisted back cross breeding (MABCB) involves the selection of genotypic progeny is done by monitoring the genetic restoration along with the recombining events upon chromosomal positions (Sharma and Gosh, 2016) (Figure 4). The technique involves the modification of only the target genes without any disruption towards the neighboring genetic traits (Varshney et al., 2009). For example, the AB resistance has been introgressed into FLIP98-135C, CDC Leader and Xena cultivars by using this technique (Taran et al., 2013). Similarly in India, root traits have been incorporated for tolerance against abiotic factors in cultivar JG11 (Collard et al., 2003). Furthermore, the technique was used by Varshney et al. (2016) for the development of AB resistant genotypes by incorporation of QTL-1 and QTL-II. A sum of eight molecular marker linked to QTLs were involved as foreground selection for desired alleles while background selection was based upon usage of high genomic recovering parents along with forty diverse SSR markers. Fourteen MAB genotypes which were resistant to AB were evolved after three selfing and backcrossing rounds successively.
Figure 1):- Symptoms of AB disease upon different plant parts.

A. Seeds  
B. Leaves  
C. Stem  
D. Pods

Figure 2):- Life cycle of Ascochyta blight (slightly modified; previously illustrated by Kelly Flower: http://www.croppro.com.au/crop_disease_manual/ch05s02.php).
Figure 3):- Marker assisted selection (The crosses from generation one to generation six leads to different variations. From F7-F12 further multi location testing and crossing can reveal resistant cultivars with desired other characters. Also in this scheme, many lines can be discarded from earlier generations leading to the evaluation of fewer lines in the coming generations). Here indicates non desired plants. So these plants are not continued for further trails from F7-F12. P1 indicates progeny1 while P2 states for progeny2.
Marker assisted back-crossing (It involves three steps. Step one involves the screening of targeted traits by using the markers. Second step includes selection of backcross progeny with targeted gene involving tight linked flanked markers for minimization of linkage drag. This may be called recombinant selection. Third and final step refers to the selection of backcross progeny with background markers). Here P1 and P2 are indicators of Progeny 1 and 2 while BC1 and BC2 states Back Cross 1 and 2.

Figure 4):- Marker assisted back-crossing
Table 1. Resistant germplasm source reported via field screening efforts against Ascochyta blight.

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<th>Resistant Germplasm Reported</th>
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<td>2</td>
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<td>ILC-3996, ILC-3279 and ILC-3856</td>
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<td>ILC-191, ILC-3279</td>
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<td>EC26446, P1252-1, BRG8, PG82-1, P919 and NEC2451</td>
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<td>ILC-3864, ILC-3870 and ILC-4221</td>
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<td>ILC-482 and ILC-3279</td>
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Table (2) Markers/QTLs identified for genetic resistance of chickpea against Ascochyta blight.

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<td>1</td>
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Conclusions and Future Prospects: Managing the genetic resistance in chickpea against AB is a challenge because of high level of diversity in primary gene pool of host, complexity in molecular bases in QTLs and variable pathogen population with continuously emerging new pathotypes. Extraordinary efforts of scientist since 1980’s to date has enabled us understanding the pathogen, its genetics, variability, diversity, lifecycle, perpetuation, biology, factors contributing in its survival and host screening techniques regarding resistance. Dozens of experiments were conducted in which thousands of inbred lines and cultivars were tested in different countries of the world by the scientists to keep a close eye upon the resistant germplasm sources so that the resistant genes may be identified and incorporated for durable resistance against the pathogen.

Recently, some new molecular techniques have got highlighted such as RNA interference (RNAi), next generation sequencing (NGS) and CRISPR-Cas9. These techniques are relatively simple, fast and efficient to quickly perform genetic mutations (Islam et al., 2017). Utilization of these techniques can lead towards the development of AB resistant varieties through incorporation of resistant genes from the wild types. Integration of molecular tools along with the conventional breeding approaches can boost up the gene introgression into new genotypes. Upon the linkage maps, resistance related molecular markers assisted with major QTLs have been identified which are beneficial for pyramiding the unique traits. But still we are far behind in achieving a complete AB prone environment so efforts are needed to be continued in the very way to defeat the pathogen and acquiring the desirable results.

Acknowledgements: We acknowledge the anonymous reviewers for their critical suggestions to improve the review. Furthermore, we tried to include almost all the available data regarding breeding resistance to chickpea against AB via field screening efforts and recent molecular breeding techniques however we apologize to all the other authors whose contributions have not been included in this review.

Author Contributions: WI and MQ collected, surveyed the research data and compiled the MS. AN helped in compiling tables, AI suggested about the figures while LW reviewed the article and approved for publication.

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