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

W. Islam[1], M. Qasim[1], A. Noman[2], A. Idrees[1] and L. Wang[*1]

[1]College of Plant Protection, Fujian Agriculture and Forestry University, Fuzhou, Fujian 350002, China
[2]Department of Botany, Government College University, Faisalabad 38040, Pakistan
[*]Corresponding Author E-mail: wang_liande@126.com

ABSTRACT

Chickpea Blight is a devastating disease of chickpea (*Cicer areitinum* L.) worldwide caused by *Ascochyta rabiei* (Pass.) Lab. The disease is more disastrous particularly in long cool and humid environmental spells. It results in huge losses by wiping off all the crop in the desert areas whenever hit 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 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 vipe 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; Shtienberg *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 *Aschochyta 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 bicellular 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* (Kovacheski) var. Arx (Syn. *Mycosphaerella rabiei* Kovacheski), 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 (Trapero-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; Rhaiem *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 (Zerrougl *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 (Trapero-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 (Lichtenzveig *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 aggression 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 breading 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 inoculums 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 Pthotype 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.

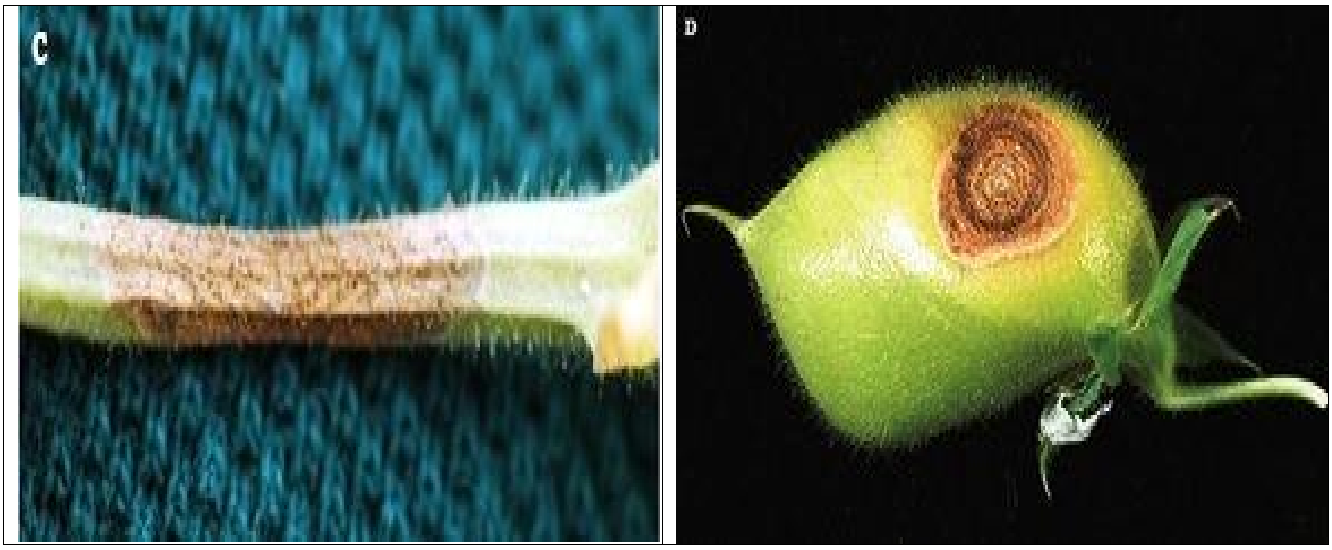

Figure 1):- Symptoms of AB disease upon different plant parts.

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

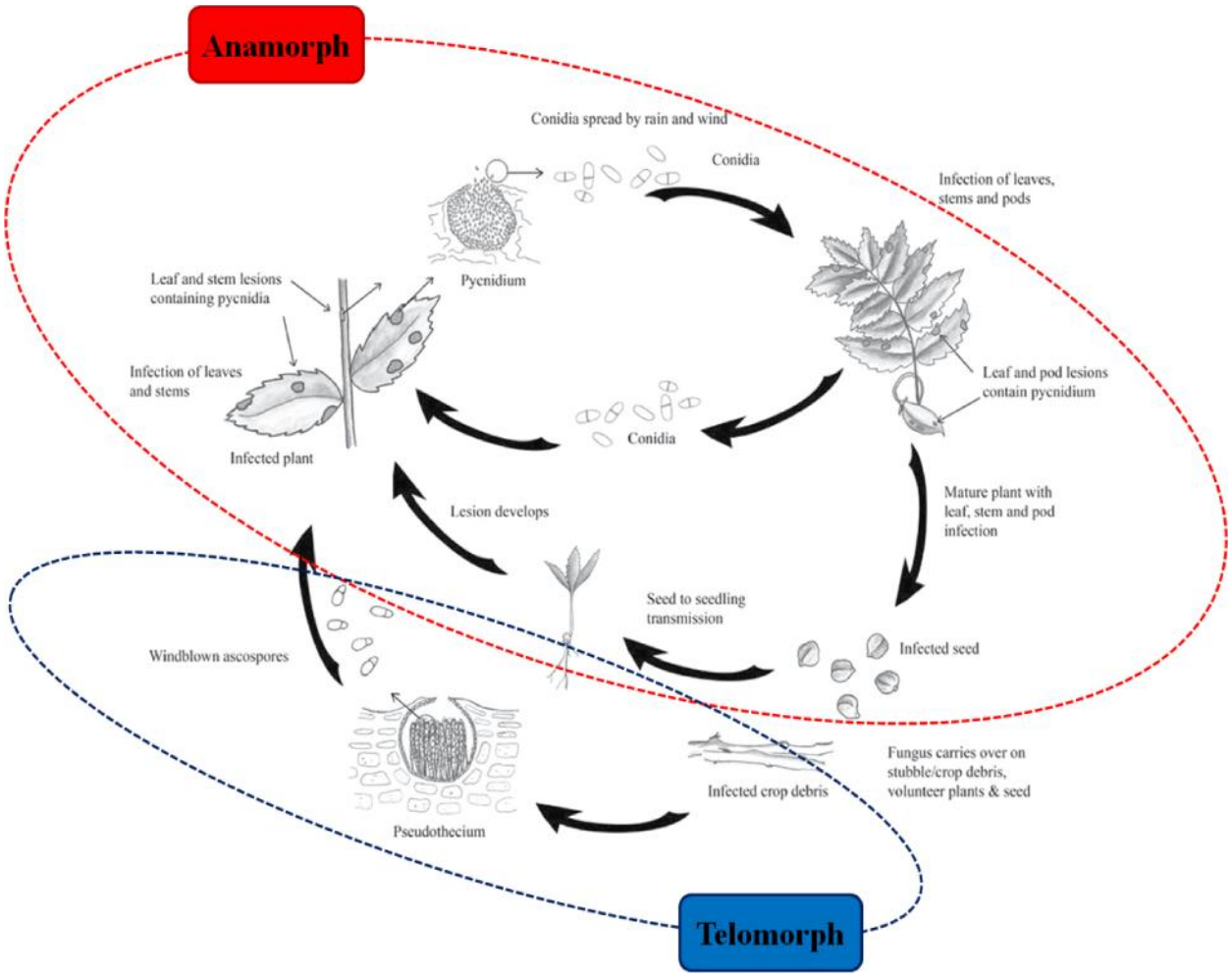

Figure 2):- Life cycle of Ascochyta blight (slightly modified; previously illustrated by Kelly Flower: http://www.croppro.com.au/crop_disease_manual/ch05s02.php).

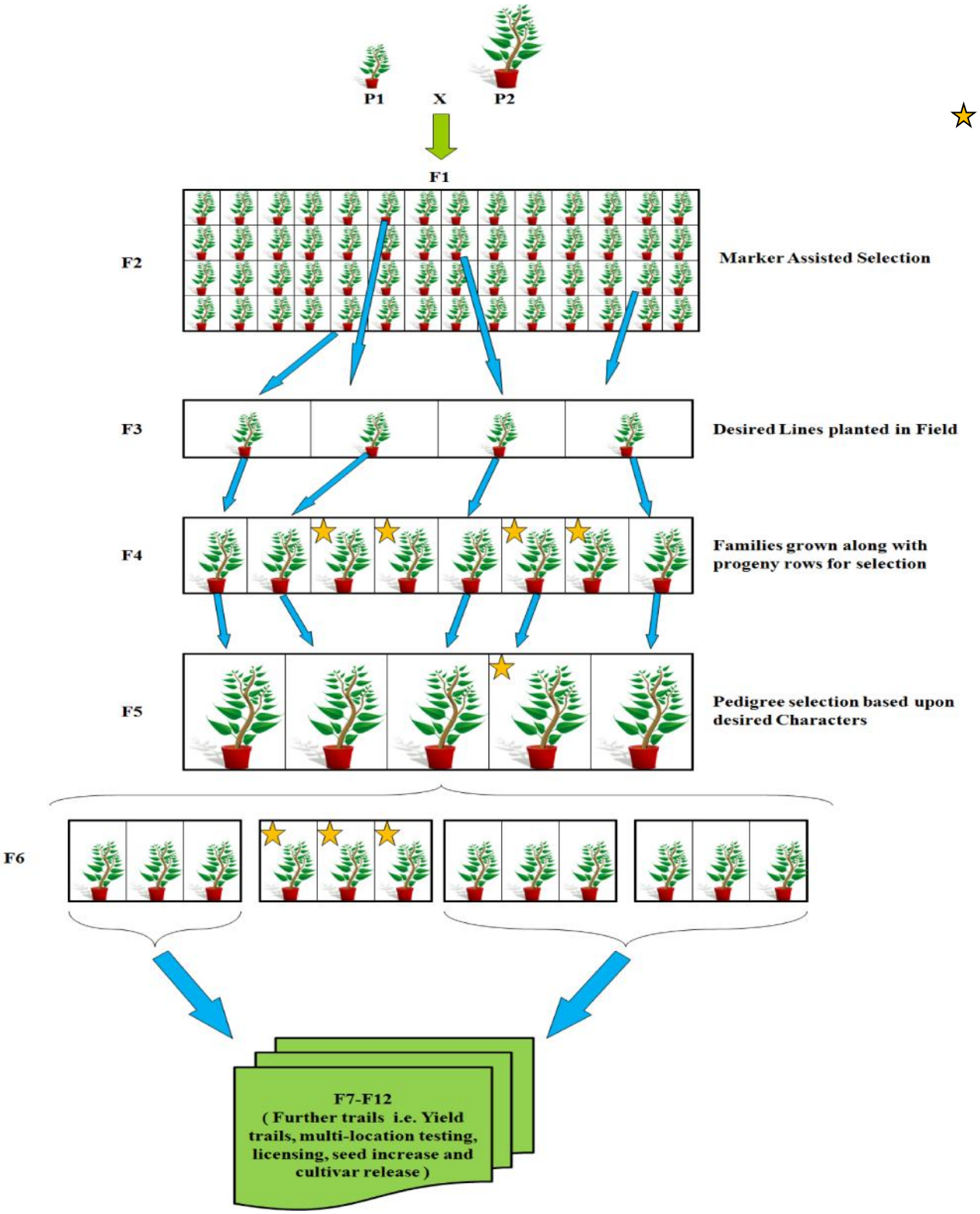

Figure 3):- Marker assisted selection (The crosses from generation one to generation six leads to different variations. From F7-F12 further multi location testing and crossing can reveal resistant cultivars with desired other characters. Also in this scheme, many lines can be discarded from earlier generations 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.

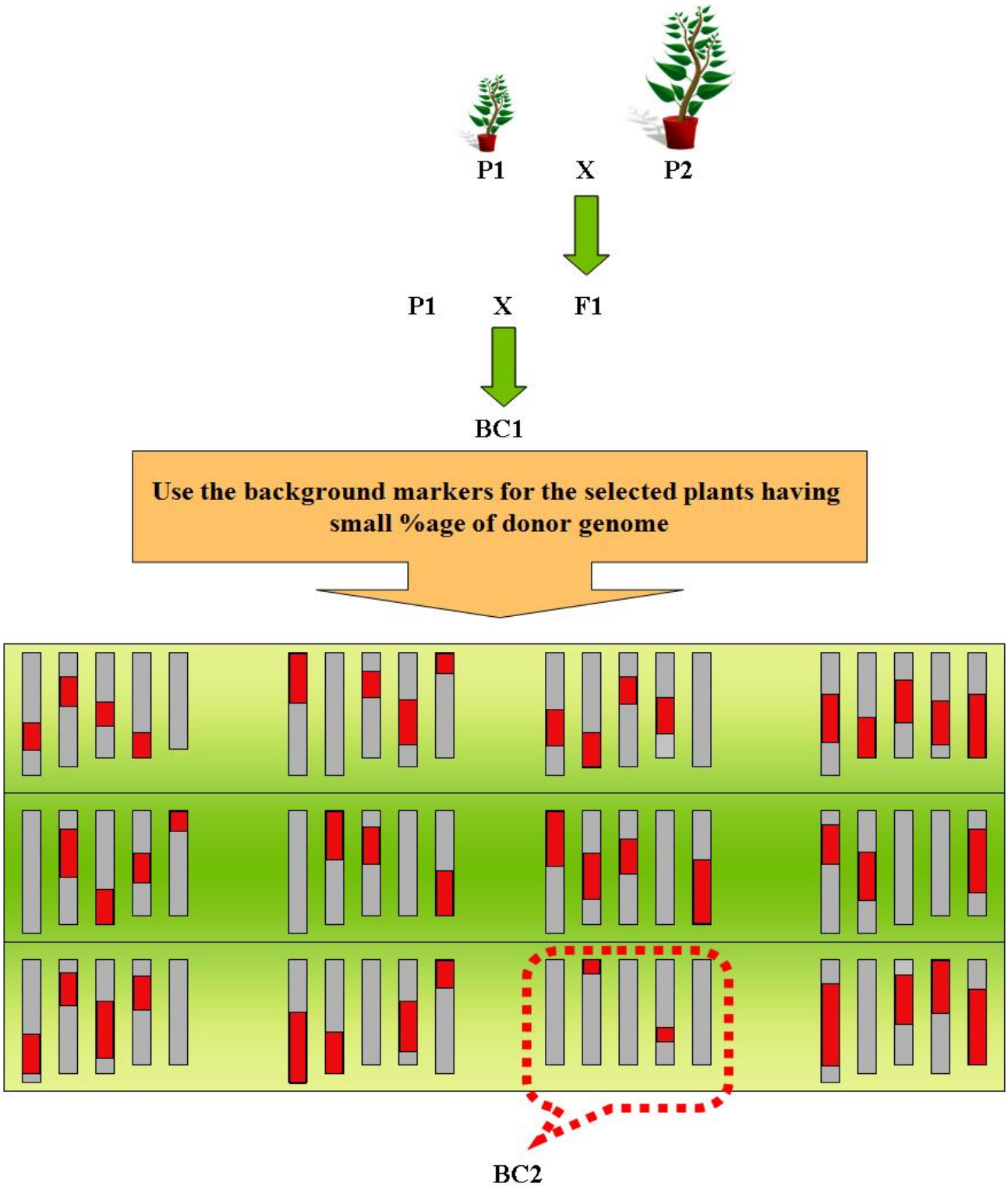

Figure 4):- Marker assisted back-crossing (It involves three steps. Step one involves the screening of targeted traits by using the markers. Second step includes selection of backcross progeny with targeted gene involving tight linked flanked markers for minimization of linkage drag. This may be called 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.

Table 1. Resistant germplasm source reported via field screening efforts against Ascochyta blight.

#	Resistant Germplasm Reported	References
1	CM-72 and CM- 86	Haq *et al.,* 1981
2	ICC-3996,ILC-4421,ICC-4475,ICC-6981,ICC-6981,ILC-2467,ICC-4324.ILC-2380,ICC-4475,ICC-6981,ICC-6988,ILC-202,ILC-2469,ILC-4421, NEC 138 and ICC-4324	Verma *et al.,* 1981
3	ICC-1257, ICC-1069 and ICC-2160	Kalia, 1984
4	ICCX830408, ICCX830602, ICCX830611, ICCX830445, ICCX830455, ICCX811174, ICCX811176, ICCX811177, ICCX811184, ICCX811186 and ICCX810623	Khirbat *et al.,*1984
5	ILC-3996	Reddy and Kabbabeh, 1985
6	ILC-72, ILC-3279 and ILC-3856	Singh *et al.,* 1984
7	ILC- 191	Puglia *et al.,* 1985
8	ILC-3279	Crino*et al.,* 1985
9	EC26446, P1252-1, BRG8, PG82-1, P919 and NEC2451	Tiwari and Pandey, 1986
10	ILC-183 and 82-11	Kinaki and Dalkiran, 1987
11	HPG-25, HPG-40, IIPG-41, HPG-69, HPG-89, HPG-85, H-75-35, H-86-100 and HPG-102	Anand and Sood, 1990
12	ILC-236, ILC-484 and ILC- 484	Reddy and Singh, 1990
13	ILC-3864, ILC-3870 and ILC-4221	Pal and Singh, 1990
14	ILC-482 and ILC-3279	Reddy and Singh, 1993
15	FLIP 90-95C, ICC-4475, ICC-12004,ICC-13269, ICC-13508 and ICC-13555	Iqbal *et al.,* 1994
16	ILC191 and CM72	Sarwar *et al.,* 1996
17	FLIP97-132C, FLIP97-227C, FLIP98-224C FLIP94-90C, FLIP95-68C, FLIP95-47C and FLIP98-231C	Iqbal*et al.,* 2002
18	F16-90 C,NCS950088,NCS950038, CMC228S,SEL96TH11488,FLIP-75C and 86135	Hussain *et al.,* 2002
19	FLIP 95-68C, FLIP 95-53C, FLIP 97-74C, FLIP 95-53C and FLIP 98-177C	Toker and Seyin, 2003
20	CM1966193, CMC77S, CM843198, CM1441198, CM1223198, CC104199, CC106199 and CC 124100	Alam *et al.,* 2003
21	Dasht	Iqbal *et al.,* 2002
22	HOO-108 and GL92024	Dubey and Singh, 2003
23	PI 559361, PI 559363 and W6 22589	Chen*et al.,* 2004
24	ILC-482 and Hashem	Younessi *et al.,* 2004
25	ATC 46934, ATC46892 and ATC 46935	Nguyen *et al.,* 2005
26	Punjab-91, Bital-98, Punjab-2000, Balkassar-2000 and Vanhar	Chaudry *et al.,* 2005
27	FLIP97-132-C, FLIP98-226C and FLIP98-231C	Iqbal *et al.,*2005
28	FLIP98-229C, FLIP82-150C, NCS 950204, NCS 950219, NSC 9903, Paidar-x, Parbat, FLIP 00-20C, FLIP 02-18C, FLIP 02-44C, FLIP 97-120C,FLIP 02-39C and FLIP 97-102C	Malik *et al.,* 2005
29	CMC44, FLIP90144C, FLIP91150C, CMC204S, CM149S, 950035, , FLIP9393C, 950072, 950248, FLIP157C, CH7/99,NCS2001,CH6/99, CM218/01 and CH30/99	Atta *et al.,* 2006
30	PB-91, E-19, F-81-312, CM-491-80 and NO. 215	Basher *et al.,* 2006
31	FLIP 03-42C, ICC 12004,ICC 3932, ICC 4033, ICC 6373, ICC6945, NCS 0507 and NCS ,AZRI-7130 and AZRI-17115	Malik *et al.,*2006
32	H97-93, 1100-256, HO1-67 and HO1-79	Waldia *et al.,* 2006
33	MCC 54, MCC 523, MCC 496, MCC 133, MCC 299, MCC528, MCC 3.11and MCC 142	Shokouhifar *et al.,* 2006
34	RIL58-ILC72/Cr5	Rubio *et al.,* 2006
35	03039, 03041, 03053, 03115, 03131, 03133, 03143, 03159, 93A-086, 93A-111 and 93A-3354	Ilyas *et al.,* 2007
36	Himachal Channa 1, Himachal Channa 2, GPF 2, HPG-17, PBG1 and PBG2	Basandrai *et al.,* 2009
37	FLIP 98-133C and FLIP 98-136C	Chandirasekaran *et al.,* 2009
38	PI 17256 and CA0090B347C	Harveson *et al.,* 2009
39	06025, 06026, 06027, 06031, 06035, 06040, 06041,06056, Vinhar, Bitter-98, Pb-	Ghazanfar *et al.,* 2010

	2000 and Paidar-91	
40	53628, 53225, 53227, 53230, 53231, 53233, 53235 ,53244, 53380,53436,53643, 54247, 53045, 53217, 53218, 53323, 53651 and 53398	Iqbal *et al.,* 2010
41	ICCV 98813, Flipper, ICCV 05111, ICCV 98801 and Jimbour #1	Du*et al.,* 2012
42	FLIP 97-121C	Kaur *et al.,* 2012
43	Ambar	www.heritageseeds.com.au
44	08006, 08016, 08017, 08024, 08026, 08030, 08050, 08051, 08052, 08053, 08054, 07006, 07008, 07009, 07010, 07025, 07045, 07057, 07058, 06001, 06004, 06024, 06040, 06052, 05007, 05030, 09001, 09030, 09005, 09006, 09007, 09009, 09011, 09013, 09015, 09016, 09019, 09020, 09021, 09022, 09023, 09025, 09027, 09028, 09029, 09031, 09032, 09033, 09034, 09035, 09036, 09038, 09039, 09041, 09042, 09047, 09048, 06A003, 06A004, 05A020, 07A005, 09AG005, 93A111, 09AG009, 09AG012, 07A003 and 07A008	Hasan *et al.,* 2012
45	04A09, 06A083 and 07A006	Sahi *et al.,* 2012
46	ILC182, ILC200, ILC3279, ILC3912, ILC3919, ILC3274,ILC3856, ILC7374, ILC7795, PHC 15,ICC12004 and ICC12004	Kaur *et al.,*2013
47	CH32/02 and CH9/02, Pb2008,CM2008, CH87/02(B8/02), CH7/02, CH31/02, CH34/03, CH4/02 and CH88/03	Sarwar *et al.,* 2012
48	EC 516934, ICCV 04537, ICCV 98818, EC 516850 and EC 516971	Pande *et al.,* 2013
49	FLIP 4107, FLIP 1025 and FLIP 10511	Benzohra *et al.,* 2013
50	PB-101 and PB-620	Ali *et al.,* 2013
51	Thal-2006, Dasht and Vanher-2000	Rehman *et al.,*2013
52	ICC7052, ICC4463, ICC4363, ICC2884, ICC7150, ICC15294 and ICC11627	Kimurto *et al.,* 2013
53	K-60013, K-98008, D-97092, K-96001, K-96022, D-91055, D90272, D-96050,D-Pb2008 and D-Pu502-362	Ahmad *et al.,* 2013
54	ILC 200, ILC 5921, ILC 6043, ILC6090, ILC 202, ILC 2956, ILC 5586, ILC 2506, ILC3279, ILC 3856 ,ILC 4421, ILC 72, ILC 182 , ILC 187, ILC72, ILC182, ILC200, ILC442 and ILC6090	Labdi *et al.,* 2013
55	8032, Thal-2006, 06001 and 5CC-109	Rashid *et al.,* 2014
56	CM-98, 1848, 6003 and 7050	Aslam *et al.,* 2014
57	10A and 28B	Duzdemir *et al.,*2014
58	k01208,K-01209, K-01212 and K-01213	Jabbar *et al.,* 2014
59	ILC72, ILC182, ILC187, ILC200 and ILC202	Benzohra *et al.,* 2015
60	ILC 8068, ICC 4475, ILC 200, ILC 7374 and ILC 7795.	Labdi *et al.,*2015
61	Alef. PI 383626	Armstrong-Cho *et al.,*2015
62	K0010-09, K0021-09, K0025-09, K0030-09, K0051-09, K0054-09, K0057-09, K0058-09,K0062-09, K0066-09, BKK17124, BKK07151, D080-09, D084-09, D089-09, D090-09, D094- 09, D095-09, BK07A005, BK96A2055, BK05A015, BK04A013 and FG-0908	Shah *et al.,* 2015
63	CICA1007, CICA0912 and Genesis™ 425	Moore *et al.,* 2016, https://grdc.com.au

Table (2) Markers/QTLs identified for genetic resistance of chickpea against Ascochyta blight.

#	Markers/QTLs Identified	Linkage Group	References
1	UBC733b, UBC181a and Dia4	LG1 and LG6	Santra *et al.,* 2000
2	GAA47	LG4	Tekeoglu *et al.,* 2002
3	STMS11, GA2 and TR20	LG4	Collard *et al.,* 2003
4	TS45, TA146 and TA130	LG1, LG2 and LG3	Flandez-Galvez *et al.,* 2003
5	SC/OPK13-003	LG4	Millan *et al.,* 2003
6	STMS11, GA2, GAA47 and TR20	LG4	Rakshit *et al.,* 2003
7	Ta20,TA72 and ar1	LG2 and LG4	Udapa *et al.,* 2003
8	GA16,GA24,GAA47 and Ta46	LG2, LG4 and LG6	Cho *et al.,* 2004
9	H3C041, TA2, H1A12/H1H13, H1G20, H1C092 and TA3/H3C11a	LG4 and LG8	Lichtenzveig *et al.,* 2006
10	OPAI09746 and UBC881621	LG2	Cobos *et al.,* 2006

11	TA194	LG4	Iruela *et al.,* 2006
12	TA34 and TA142	LG3	Aryamanesh *et al.,*2007
13	TA64, TS54 and TA176	LG3, LG4 and LG6	Tar'an *et al.,* 2007
14	TR19, TS54, TA132, TS45 and TA64	LG2, LG3, LG4 and LG8	Anbessa *et al.,* 2009
15	TA125, TA72 and GA26	LG3, LG4 and LG6	Kanouni *et al.,* 2009
16	TA34, TA142, STMS11, TAA170, H3D09 and H1A12	LG3 and LG4	Aryamanesh *et al.,* 2010
17	STMS11, Ta106 and CaM0244	LG4, LG5 and LG6	Sabbavarapu *et al.,* 2013
18	SNP_40000185, TA146 and TA72	LG4	Stephens *et al.,* 2014
19	CaETR and GAA47	LG4	Castro *et al.,*2015
20	CAV1SC21.1P149511,scaffold905p1129574,CAV1 SC48.1P396061,CAV1SC2.1P308242,CAV1SC1.1p 494014,CAV1sc445.1p9288, CAV1SC102.1P54882 and scaffold1567p981540	LG1,LG3,LG4,LG5, LG6, LG7 and LG8	Daba *et al.,* 2016

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 inbread 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.

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