An Update on Genetic Resistance of Chickpea to Ascochyta Blight

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Abstract: Ascochyta blight (AB) caused by Ascochyta rabiei (Pass.) Labr. is an important and widespread disease of chickpea (Cicer arietinum L.) worldwide. The disease is particularly severe under cool and humid weather conditions. Breeding for host resistance is an efficient means to combat this disease. In this paper, attempts have been made to summarize the progress made in identifying resistance sources, genetics and breeding for resistance, and genetic variation among the pathogen population. The search for resistance to AB in chickpea germplasm, breeding lines and land races using various screening methods has been updated. Importance of the genotype × environment (GE) interaction in elucidating the aggressiveness among isolates from different locations and the identification of pathotypes and stable sources of resistance have also been discussed. Current and modern breeding programs for AB resistance based on crossing resistant/multiple resistant and high-yielding cultivars, stability of the breeding lines through multi-location testing and molecular marker-assisted selection method have been discussed. Gene pyramiding and the use of resistant genes present in wild relatives can be useful methods in the future. Identification of additional sources of resistance genes, good characterization of the host–pathogen system, and identification of molecular markers linked to resistance genes are suggested as the key areas for future study.

Keywords: Ascochyta rabiei; breeding strategy; Cicer arietinum; disease resistance; legume

1. Introduction

Ascochyta blight (AB), caused by Ascochyta rabiei (Pass.) Labr., is an important disease of chickpea in many countries (India, Bangladesh, Algeria, Israel, Italy, Morocco, Nepal, Spain, Pakistan, Syria, Iran, USA, Australia and Canada) where cool and humid weather prevails during the flowering to podding stage [1–7]. AB severely reduces the yield of chickpea and can cause complete yield losses under favorable conditions [8–11]. The occurrence of AB has been reported in more than 40 countries across the world [12]. In most of the growing systems, chickpea is a dryland crop (rainfed) and it relies on residual soil moisture; however, in Mediterranean-like environments, the crop is sown either as a winter crop (rainfed), or as spring crop [13,14]. Winter sowing exposes chickpea to a high risk of AB and thus requires the development of resistant cultivars.

Despite worldwide recognition of the destructive potential of A. rabiei in chickpea production, very little headway in understanding pathogen biology, disease epidemiology and management has been made. Understanding pathogen population structure is important for the deployment of resistant cultivars and for developing suitable strategies to reduce disease. Managing AB of chickpea through resistance breeding has been difficult due to the evolution of new virulent pathotypes of A. rabiei [15]. Loss of resistance in highly AB-resistant chickpea varieties viz., F8 (recommended in 1940–1941), C 1234 (1950–1951) and C 235 (1968), has strongly indicated the emergence of new virulent strains of A. rabiei [16,17]. It is speculated that pathogen populations also evolve to overcome quantitative...
resistance in agro-ecosystems. However, the nature of evolution against quantitative resistance differs from the evolution against major gene resistance, and is better characterized as a process of “erosion”, rather than a process of “breakdown”. This erosion is difficult to detect because corresponding pathogen populations can display a range of pathogenicity that may vary considerably from year to year as a result of strong genotype by environment (G × E) interactions [18].

Breeding for host resistance is the most effective, efficient and environmentally friendly method to control the disease [19]. However, in pathogens like A. rabiei, monitoring of the plant pathogen population and inclusion of a threshold level of pathogen inoculum for screening of germplasm is a key component. We summarize here the progress made towards understanding variations in A. rabiei populations, genetics of resistance, resistance sources and G × E interactions.

2. Characteristics of AB in Chickpea

The causal agent of AB of chickpea exists both as an anamorph and a teleomorph. The anamorph, A. rabiei, is characterized by the formation of spherical or pear-shaped black fruiting bodies called pycnidia. A pycnidium contains numerous hyaline unicellular and occasionally bicellular spores, pycnidiospores, or conidia, developed on short conidiophores (stalks) embedded in a mucilaginous mass. Pycnidiospores are oval to oblong, straight, or slightly bent at one or both ends and measure 6–12 by 4–6 µm [20,21]. The fungus grows readily on a variety of nutrient media, the best being chickpea meal dextrose agar. A. rabiei generally produces a pale cream colored mycelium in which pale brown to black pycnidia are immersed. Cultures are variable in morphology and color, with isolates often producing a prevalence of unicellular conidia [22].

The teleomorph, Didymella rabiei (Kovacheski) var. Arx (Syn. Mycosphaerella rabiei Kovacheski), is a bipolar heterothallic ascomycete and is characterized by pseudothecia developing on chickpea crop residues that have over-wintered in the field. Pseudothecia are dark brown to black, subglobose, 120–270 µm in diameter, erupting from the host tissue and without a conspicuous ostiole. Binucleate asci are cylindrical to subclavate surrounded by paraphyses and contain eight hyaline unequally bicellular ascospores. Ascospores are ellipsoid to biconic with a constriction at the septum and measure 9.5–16 by 4.5–7 µm. The life cycle of D. rabiei consists of a single sexual generation per season, which develops on infected over-wintering chickpea debris, followed by several asexual generations during the parasitic phase of the disease cycle [23]. Sexual reproduction is controlled by a single regulatory locus referred to as a mating-type locus and alternate sequences at the mating-type locus are completely dissimilar and code for different regulatory genes. The presence of opposite mating types (MAT1-1 and MAT1-2) and the teleomorph have been reported from some chickpea-growing regions in the world [24–29]. The morphological characteristics of A. rabiei and Phoma medicaginis var. pinodella are similar, which makes it difficult to distinguish between the 2 species. However, a PCR test developed by Phan et al. [30] can be used to detect and confirm the identity of A. rabiei.

Symptoms of AB can develop on all aerial parts of a plant. Plants are attacked at any growth stage, depending on the inoculum availability. However, AB is most prominent during the flowering to early podding growth stages. Air-borne conidia and ascospores infect younger leaves and produce small water-soaked necrotic spots that rapidly enlarge and coalesce. Conidia may also be water-borne and splash dispersed to infect foliage tissue on the same or nearby plants. Subsequently, symptoms spread rapidly to all aerial parts including leaves, petioles, flowers, pods, branches, and stems, which leads to rapid collapse of tissues and death of the plant (Figure 1). Development of pycnidia in concentric rings on lesions is the characteristic symptom of A. rabiei infection. Lesions that develop on leaves and pods appear circular with brown margins and a grey center that contains pycnidia, whereas lesions developing on petiole, stems, and branches are elongated. The lesions that develop on apical twigs, branches, and stems differ in size and in later stages girdle the affected plant parts. The regions above the girdled portion are killed and may break off. Diseased pods with visible blight symptoms often fail to develop any seed. Pod infection often leads to seed infection through the testa and cotyledons. Infected seed can be discolored and possess deep, round, or irregular cankers, sometimes bearing
pycnidia visible to the naked eye. Infection during the pod maturation stage often results in shrivelled and infected seed [4,31].

Figure 1. Ascochyta blight symptoms on chickpea plant (a) Severe AB infection on all aerial parts (b) Lesions on leaf and pods (c) Lesions on green pods (d) Pycnidial bodies arranged in concentric rings.

3. Pathogen Variability

*A. rabiei* shows a high degree of pathogenic and genetic variability, and AB-resistant chickpea cultivars have become susceptible in some countries [26–28]. The presence of a teleomorph (*D. rabiei*) in the *A. rabiei* life cycle contributes to variability within the pathogen population, which may generate a new combination of virulence genes and the development of new pathotypes [11]. In *A. rabiei* of chickpea, a number of pathotypes were reported; for instance, more than ten pathotypes by Vir and Grewal, [32]; five pathotypes by Nene and Reddy, [1] and three pathotypes by Udupa *et al.* [33]. Udupa *et al.* [33] reported the occurrence of three pathotypes; pathotype I (less aggressive), pathotype II (aggressive) and pathotype III (most aggressive) as revealed by molecular markers [26,34–36]. A new *A. rabiei* pathotype (pathotype IV) was reported in Syria that is capable of affecting the highly resistant chickpea genotypes (ICC-12004 and ICC-3996) known for their resistance to pathotypes I, II and III. Breeding materials at ICARDA are being screened against this new pathotype IV, and so far low levels of resistance have been observed [37,38]. High genetic diversity has also been reported...
from USA, Tunisia and Canada where popular varieties have become susceptible to new aggressive pathotypes [26–28]. Microsatellite markers revealed high levels of polymorphism among isolates from Tunisia, Australia, USA, Turkey, Pakistan, Syria and India [26,27,35,39–41]. Further, very little information about A. rabiei is available at the genomic level. Recently, Fondevilla et al. [42] reported a comprehensive A. rabiei transcriptome and identified several putative pathogenicity factors specifically induced during infection.

Mating type distribution is one of the important factors that contributes to variation in any pathogen population. The MAT-specific markers have been used in rapid determination of mating type ratios in A. rabiei populations and detection of introduction of a second mating type into an area [25,26,41]. Further, MAT genes have been proposed as potentially useful regions of the genome for phylogenetic reconstruction and genetic variability studies [43,44]. MAT genes appear to evolve more quickly than other regions of the genome but are highly conserved within species, making them useful for phylogenetic analysis of closely related species [43–45]. The mating type distribution of A. rabiei has not been thoroughly determined, although it is assumed that A. rabiei ascospores from the sexual stage represent recombinant progeny that could contribute to increased genotypic diversity in A. rabiei populations. This variation is potentially adaptive, allowing the pathogen to evolve increased virulence on resistant cultivars and/or to develop resistance to fungicides.

Studying the genetic diversity of A. rabiei isolates infecting wild Cicer spp. is very important to compare pathogen movement between wild and cultivated chickpea species. Understanding A. rabiei gene/genotype flow is especially relevant in a country such as Syria that lies in the center of the origin of chickpeas. Comparing the population structure of the pathogens isolated from wild and cultivated chickpeas using DNA markers allow the estimation of gene flow among populations from different hosts and geographic regions [46].

4. Genetic Resources of AB Resistance

Development and use of reliable and repeatable resistance screening techniques to exploit host plant resistance for any disease is a prerequisite. A number of screening techniques under field and controlled environments have been reported for AB as reviewed in Pande et al. [11]. Temperature and relative humidity are critical factors in AB establishment. A high level of relative humidity during the first 24 h post-inoculation period is critical. Growth chambers where relative humidity can be controlled are useful; however, additional steps, such as use of foggers or mist irrigation immediately after inoculation can help maintain relative humidity at high levels and ensure successful infection [10,47]. Spore concentration in the inoculum is also a significant factor, with the ideal level being the lowest spore concentration that causes sufficient disease in a majority of host genotypes. This facilitates the greatest discrimination among the lines in a trial. Screening techniques such as seedling screening and cut twig screening using excised trays are routinely being used to screen chickpea genotypes against AB in the controlled environment growth chamber facility at ICRISAT, Patancheru India [48]. Chen and Muehlbauer [49] developed a mini-dome technique for AB resistance screening and this technique is in use at Pullman, WA, USA. Field screening techniques for AB resistance in chickpea were initially developed by Singh et al. [50] in India. Screening was carried out in areas where the prevailing weather conditions were conducive to the development of disease and preferably where natural inoculum was abundant. The procedure consists of planting susceptible check plants every two or four tested entries, scattering infected plant debris collected in the previous season, maintaining high humidity through sprinkler irrigation, and, if needed, spraying the test entries with a spore suspension of a virulent isolate of A. rabiei. A resistant check/ susceptible was included in order to compare resistance of test entries with that of known resistant material [51]. A positive correlation between a field and controlled environment screening technique for AB was reported by Pande et al. [48].

A number of AB resistant sources have been identified and used in breeding programs although none possessed complete resistance [11,49]. Pande et al. [48] reported 29 lines with resistance to AB
and these lines are being exploited in breeding programs at ICRISAT. Most of these AB resistant lines have a wide range of maturity (112–142 days). Breeding for resistance to AB has been a major focus in chickpea breeding programs in many countries, such as India, Syria, Canada, USA, Australia, Turkey and Pakistan [52]. The most widely used sources of resistance have been supplied by ICRISAT, India and ICARDA, Syria (presently at Lebanon). In Australia, the first variety released with a moderate level of resistance to AB was “Howzat” in 2001, and breeders have since selected a number of desi and kabuli lines with higher levels of resistance from ICRISAT and ICARDA breeding lines, as well as existing Australian varieties. Three new AB-resistant varieties (Ambar, Neelam and PBA Striker) have recently been released in Australia (http://www.news.uwa.edu.au/201310256190/business-and-industry/ tough-new-varieties-set-revive-profitable-chickpea-industry).

Developing chickpea varieties with high levels of resistance to AB has been a challenging proposition because of the following factors: (i) paucity of high levels of resistance in the primary gene pool; (ii) complex genetic basis of resistance conferred by several quantitative trait loci (QTLs); (iii) a highly variable pathogen population; and (iv) the emergence of new pathotypes due to natural recombination through sexual reproduction in the AB life cycle. Since an adequate level of genetic resistance is not available in the cultivated genotypes, different gene pools of Cicer species such as C. bijugum, C. echinospermum, C. pinnatifidum, C. judaicum and C. montbretii have been exploited for AB resistance. Two C. echinospermum accessions classified as resistant to AB [53] and being cross compatible with C. arietinum, could provide valuable sources of resistance [54] to AB. Wild accessions of C. judiacum such as ATC 46934, ICC 17211, IG 69986, IG 70030, IG 70037 and IG 70038 were reported resistant to AB [55–57]. The reaction of few wild species to AB in controlled environment screening at ICRISAT is provided in Table 1. Some wild accessions have shown resistance to more than one stress, for example, ILWC 7-1 of C. bijugum showed resistance to ascochyta blight, fusarium wilt, leaf miner, cyst nematode and cold, and ILWC 33/S-4 of C. pinnatifidum showed resistance to ascochyta blight, fusarium wilt, seed beetle and cyst nematode [46,52]. The feasibility of introgression from the tertiary to the domestic gene pool and access to these novel sources of resistance is an important priority for chickpea breeders [58]. An updated list of resistant cultivars to AB in chickpea is provided in Table 2.

Table 1. Reaction of wild Cicer species to Ascochyta blight in controlled environment screening at ICRISAT, Patancheru, India.

| Wild Cicer species | Accessions Screened | Reaction to Ascochyta Blight Infection * |
|-------------------|---------------------|----------------------------------------|
|                   |                     | R  | MR | S  | HS |
| C. bijugum        | 30                  | -  | 7  | 20 | 3  |
| C. cuneatum       | 3                   | -  | 1  | 2  | -  |
| C. echinospermum  | 4                   | -  | -  | 3  | 1  |
| C. judaicum       | 47                  | 5 **| 34 | 8  | -  |
| C. pinnatifidum   | 27                  | -  | 13 | 13 | 1  |
| C. reticulatum    | 31                  | -  | -  | 15 | 16 |
| C. yamashitae     | 6                   | -  | -  | -  | 6  |

* Based on the disease score, the wild accessions were categorized for their reaction to Ascochyta blight infection as follows: 1.0–3.0 = resistant (R), 3.1–5.0 = moderately resistant (MR), 5.1–7.0 = susceptible (S) and 7.1–9.0 = highly susceptible (HS). ** ICC 17211, IG 69986, IG 70030, IG 70037 and IG 70038.
Table 2. An updated list of Ascochyta blight-resistant chickpea sources (2000–2015).

| Resistance Source | Remarks | Country Reported | Year | References |
|-------------------|---------|------------------|------|------------|
| FLIP94-90C, FLIP95-68C, FLIP95-47C, FLIP97-132C, FLIP97-227C, FLIP98-224C and FLIP98-231C | - | Pakistan | 2002 | [59] |
| HOO-108 and GL92024 | - | India | 2003 | [60] |
| PI 559361, PI 559363 and W6 22589 | Showed a high level of resistance to two pathotypes | USA | 2004 | [61] |
| FLIP98-229C, FLIP82-150C, NCS 950204, NCS 950219, NCS 9903, PaidarxParbat, FLIP 00-20C, FLIP 02-18C, FLIP 02-44C, FLIP 97-120C, FLIP 02-39C and FLIP 97-102C | Showed resistance in both green house and field | Pakistan | 2005 | [62] |
| MCC 54, MCC 523, MCC 496, MCC 133, MCC 299, MCC528, MCC 3.11 and MCC 142 | Two desi accessions and six Kabuli accessions were resistant against six pathotypes | Iran | 2006 | [63] |
| RILS8-ILC72/Cr5 | - | Spain | 2006 | [64] |
| 03039, 03041, 03053, 03115, 03131, 03133, 03143, 03159, 93A-086, 93A-111 and 93A-3354 | Germplasm lines | Pakistan | 2007 | [65] |
| FLIP 98-133C and FLIP 98-136C | Showed strong resistance to AB on leaves, stems and pods, in addition to having high yield | Canada | 2009 | [66] |
| 53628, 53225, 53227, 53230, 53231, 53233, 53235, 53244, 53380,53436, 53643, 54247, 53045, 53217, 53218, 53323, 53631, 53398 | Germplasm lines with disease score 1 to 3 at seedling stage | Pakistan | 2010 | [67] |
| FLIP 97-121C | Disease rating ranged from 2 to 3.5. | India | 2012 | [68] |
| Ambar | Desi chickpea variety that combines early flowering, competitive yield | Australia | 2012 | [69] [http://www.heritageseeds.com.au] |
| EC 516934, ICCV 04537, ICCV 98818, EC 516850 and EC 516971 | Mean disease severity ≤3.0 on the 1–9 scale and the reactions were consistent in multi-environments | India | 2013 | [69] |
| FLIP 4107, FLIP 1025 and FLIP 10511 | Exhibited highly resistant response against three pathotypes | Algeria | 2013 | [70] |
| ICC7952, ICC4463, ICC4363, ICC2884, ICC7150, ICC15294 and ICC11627 | - | Kenya | 2013 | [71] |
| K-60013, K-98008, D-97092, K-96001, K-96022, D-91055, D-90272, D-96050, D-Pb2008 and D-Pu502-362 | - | Pakistan | 2013 | [72] |
| 10A and 28B | - | Turkey | 2014 | [73] |
| ILC72, ILC182, ILC187, ILC200 and ILC202 | Exhibited highly resistant response against three pathotypes | Algeria | 2015 | [74] |
5. Genotype × Environment and AB Interactions

Genotype × environment (G × E) interaction is an important component in breeding for disease resistance because pathogens may vary in their aggressiveness under different environments, and thus physiological races may differ across environments. Further, the growth, development and physiological status of host genotypes may change across environments. G × E interaction studies are very useful in identifying stability of genotypes across environments. There is a paucity of information regarding the G × E and AB interaction in chickpea. Pande et al. [69] identified five genotypes with consistent resistant reaction to AB (EC 516934, ICCV 04537, ICCV 98818, EC 516850 and EC 516971) using the genotype and genotype × environment (GGE) biplot analyses of multi-environment data. Multi-environment testing revealed not only significant genotypic effects but also significant effects of the environment and the G × E interaction for AB severity. Significant G × E was expected as AB in chickpea is largely affected by environment [75,76]. However, Chandirasekaran et al. [66] observed the relative ranks of varieties in all site-years for leaf area under the disease progression curve (LAUDPC) and the stem area under the disease progression curve (SAUDPC), and means of the pod disease ratings (POD) did not vary significantly in the absence of cross-over interactions. GGE biplot analysis has been widely used in recent years to determine the stability of disease resistance through multi-location trials in order to identify stable resistant genotypes [77,78].

6. Inheritance and Marker Assisted Breeding for AB Resistance

Classical genetic studies of AB resistance have shown it to be governed by a single dominant gene [79–82]. Few studies had conferred AB through the combination of a recessive and a dominant gene [83]. Details of the genetic basis of host pathogen interactions have been provided by Pande et al. [11,51]. In the recent study conducted by Labdi et al. on the inheritance of resistance to race 4 of A. rabiei on 15 chickpea accessions, resistance was reported to be governed in different genotypes by a single recessive gene, two recessive complementary genes, two dominant complementary genes and two recessive genes with epistasis interaction [84]. Resistance in ILC 3279, ILC 3856 and ILC 4421 was controlled either by three recessive genes or two recessive duplicated genes, and in ILC 72, ILC 182 and ILC 187, resistance was polygenic in nature.

In terms of molecular mapping, a considerable number of QTLs have been identified in many studies with respect to several linkage groups (LG 2, 3, 4, 6, and 8) for AB resistance [85]. Two major QTLs on LG 2, close to the GA16 and TA37 loci, control resistance to A. rabiei pathotype I [86]. Two QTLs for pathotype II are located on LG 4, one is linked to CaETR or GAA47 and the other is linked to TA72/ScY17 [10,86,87]. Cho et al. [86] identified an additional SSR marker (TA46) located on LG 2 that was strongly associated with the resistance derived from FLIP 84-92C to Pathotype II. This marker explained between 59 and 69% of the variations for resistance using different isolates under controlled environments. Furthermore, loci TS12b and STMS28 on LG 1, TS45 and TA3b on LG 2, were significantly associated with the disease reaction under controlled environments [88,89]. Bian et al. [90] compared three chickpea LGs, harbouring six QTLs conditioning resistance against A. rabiei with the most comprehensive chickpea map (W-Ca-LG) and found that QTL1 (LG 3) was located in the subcentromere region of the chickpea W-Ca-LG3 (chromosome C). QTL2 and QTL3 (LG 8) were located on the long arm of the W-Ca-LG8 (chromosome H) and QTL4, QTL5 and QTL6 (LG 4) were located in the subcentromere region of the W-Ca-LG4 (chromosome B). However, the majority of AB-resistance QTLs were reported mainly on two LG, CaLG02 and CaLG04. For instance, AB resistance QTL ar1 (LG 2) and ar2a (LG 2), identified by Udupa and Baum, [10], and QTLAR3 identified by Iruela et al. [91], are present in the same genomic region mainly flanked by GA16 and TA110 (LG 2) markers on CaLG02. The QTLs present in this genomic region confer resistance to both Pathotype I and II of A. rabiei and contribute up to 20% phenotypic variation. More recently, Hamwieh et al. [92] identified 14 microsatellite markers that were linked to seven QTLs for A. rabiei resistance (Ar2a, Ar2c, Ar3c, Ar4a, Ar4b, Ar6b and Ar8a) on the five chickpea linkage groups (LG 2, LG 3, LG 4, LG 6 and LG 8). Madrid et al. [93] also reported development of a co-dominant marker (CaETR) based on allele
sequence length polymorphism in an ethylene response gene located in the QTLAR1 region (LG 4). Varshney et al. [94] developed a physical map of chickpea, locating an AB-resistance QTL region. Among 306 genes, genes like the BED finger-nbs resistance protein and the gene with a leucine-rich repeat domain were typically involved in host resistance mechanisms, such as DNA-directed RNA polymerase subunit beta, receptor-like protein kinase and Ser-Thr protein kinase. Further, this region also harbors the NAC domain protein for systemic acquired resistance as well as the NB-LRR-type disease resistance protein. In summary, QTLs contributing to A. rabiei (Ar) resistance were identified by many research groups—14 Ar loci located on eight chickpea LGs, named as Ar1a, Ar2a, Ar2b, Ar2c, Ar3a, Ar3b, Ar3c, Ar4a, Ar4b, Ar5a, Ar6a, Ar6b, Ar7a, and Ar8a [10,82,86,89,92,95–102]. These markers will be important for enabling the pyramiding of resistance genes from diverse sources to reduce the time required to generate resistant cultivars. An updated list of QTLs and markers identified for AB is provided in Table 3.

### Table 3. The QTLs or genes identified for chickpea host resistance to Ascochyta blight.

| Marker/QTL        | Linkage group | Phenotypic variation (%) | References |
|-------------------|---------------|--------------------------|------------|
| UBC733b, UBC181a, Dia4 | LG1, LG6      | 50.3 and 45              | [103]      |
| TS45, TA146, TA130 | LG1, LG2, LG3 | 76                       | [89]       |
| Ta20, TA72, ar1   | LG2, LG4      | 35.9                     | [10]       |
| GA16, GA24, GAA47, Ta46 | LG2, LG4, LG6 | 69.2                     | [86]       |
| H3C041, TA2       | LG4           | 14.4                     | [97]       |
| H1A12/H1H13, H1H20 | LG4           | 42                       | [97]       |
| H1C092, TA3/H3C11a | LG8           | 16                       | [97]       |
| OPAi09746, UBC881621 | LG2          | 28.0                     | [104]      |
| TA194             | LG4           | 55.0                     | [96]       |
| TA64, TS54, TA176 | LG3, LG4, LG6 | 56                       | [98]       |
| TR19, TS54        | LG2, LG4      | 48                       | [99]       |
| TA132, TS45       | LG4, LG8      | 38                       | [99]       |
| TA64              | LG3           | 14                       | [99]       |
| TA125, TA72, GA26 | LG3, LG4, LG6 | 46.5                     | [105]      |
| TA34, TA142       | LG3           | 49                       | [106]      |
| STMS11, TAA170    | LG4           | 49                       | [106]      |
| H3D09, H1A12      | LG4           | 49                       | [106]      |
| STMS11, Ta106, CaM0244 | LG4, LG5, LG6 | 41.6                     | [101]      |
| SNP_40000185      | LG4           | 45                       | [102]      |
| TA146, TA72       | LG4           | 59                       | [102]      |
| CaETR, GAA47      | LG4           | 34                       | [107]      |

Mapping of important QTL/gene responsible for AB resistance for molecular breeding in chickpea has been considered an important input for present day breeding programs. The strategies for molecular breeding of complex traits such as AB resistance can be taken further by selecting the QTL in segregating progeny [108]. One strategic approach is to simultaneously monitor restoration of the genetic background with QTL introgression and select progeny with recombination events in critical chromosome positions, known as marker-assisted backcrossing (MABC). MABC aims at conversion of targeted lines with respect to one or two traits without disturbing all other native traits of the target cultivar [109]. The MABC has been successfully employed recently to introgress AB resistance with double-podding traits in chickpea cultivars CDC Xena, CDC Leader, and FLIP98-135C [110], and a QTL-hotspot containing QTLs for root traits and abiotic stress tolerance in JG 11, a leading chickpea cultivar from India [111]. Varshney et al. [112] demonstrated the use of MABC to develop superior lines resistant to AB. To develop resistant lines, two QTL regions for AB, ABQTL-I and ABQTL-II, were targeted for introgression. Foreground selection with eight markers linked to QTL regions was used for selection of plants with desirable alleles in different segregating generations. In addition to the foreground, back-ground selection was performed for selection of plants with high recurrent parent genome recovery, with evenly distributed 40 SSR markers. After three backcrosses and three rounds of
selfing, 14 MAB lines were generated for AB [112]. Phenotyping of these lines has identified seven resistance lines for AB.

Although the use of marker assisted selection (MAS) is mostly straightforward for manipulating single-gene traits, its potential for breeding complex traits has also been recognized [113,114]. However, it should be noted that the use of markers for polygenic trait improvement remains difficult, with few success stories reported to date [115,116]. For instance, Castro et al. [107] reported the usefulness of allele specific markers (CaETR and GAA47) for MAS and also reported that markers TA72 and SCY17 could be useful for MAS but the high distorted segregation towards the susceptible parent in the region where these markers are located could explain their low effectiveness. Bouhadida et al. [117] also used one allele specific marker (CaETR) and one codominant SCAR marker and reported that these two markers contributed efficiently to the selection of new chickpea varieties with better combinations of alleles to ensure durable resistance to AB.

7. Conclusions

Considerable progress has been made in the last decade in understanding the AB pathogen and its genetics of resistance in chickpea. Resistance to AB has been found in chickpea and breeding for resistance is making progress by identifying new resistance genes. Molecular tools are being integrated with conventional breeding approaches to speed up the process of introgressing genes into chickpea elite genotypes. Molecular markers associated with major QTLs conferring resistance to AB have been located on linkage maps, and these markers can be used for efficient pyramiding of the traits of interest. Stability, effectiveness, and usefulness of the recently introgressed and pyramided resistances remain to be determined across greenhouse and field environments against A. rabiei isolates of varying aggressiveness and their deployment in cultivar development. Efforts, therefore, need to continue to combine high levels of AB resistance with other desirable traits for incorporation into future releases as promising cultivars of different market classes of chickpea in AB-prone environments.

Acknowledgments: The author is thankful to several colleagues at ICRISAT and collaborators from many NARS programs from India. The work reported in this article was undertaken as a part of the CGIAR Research Program on Grain Legumes. ICRISAT is a member of the CGIAR.

Conflicts of Interest: The authors declare that they have no competing interests.

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