agronomy

MDPI

Review

An Update on Genetic Resistance of Chickpea to Ascochyta Blight

Mamta Sharma * and Raju Ghosh

International Crops Research Institute for the Semi-Arid Tropics, Patancheru 502324, Telangana, India; r.ghosh@cgiar.org

* Correspondence: mamta.sharma@cgiar.org; Tel.: +91-40-30713493; Fax: +91-40-30713074

Academic Editors: Diego Rubiales, Maria Carlota Vaz Patto and Elena Prats

Received: 3 December 2015; Accepted: 24 February 2016; Published: 8 March 2016

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].

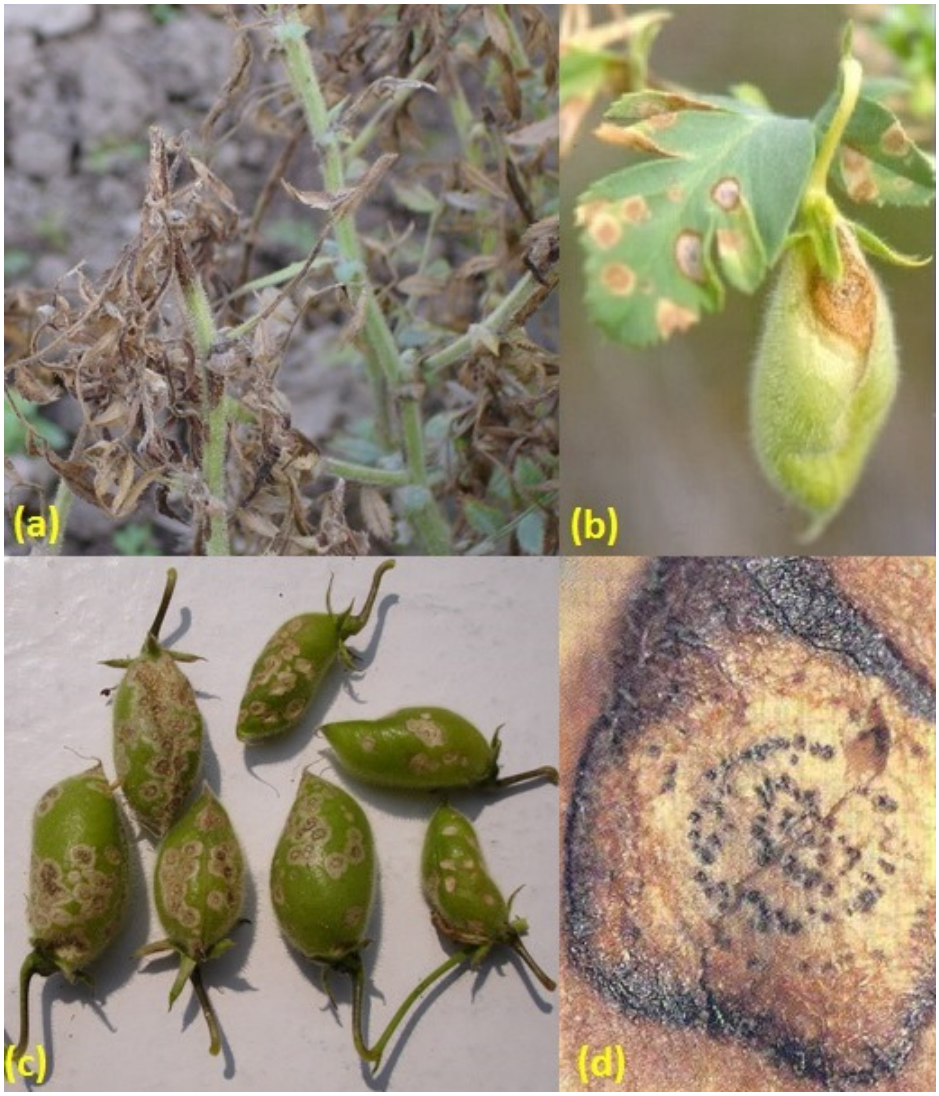

Figure 1. Ascochyta blight symptoms on chickpea plant (**a**) Severe AB infection on all aerial parts (**b**) Lesions on leaf and pods (**c**) Lesions on green pods (**d**) Pycnidial bodies arranged in concentric rings.

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. bijugam	30	-	7	20	3
C. cuneatum	3	-	1	2	-
C. echinospermum	4	-	-	3	1
C. judiacum	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, NSC 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]
RIL58-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, 53651, 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	http://www.heritageseeds.com.au;
EC 516934, ICCV 04537, ICCV 98818, EC 516850 and EC 516971	Mean disease severity $\leqslant$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]
ICC7052, 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 consistant 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 is 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). QTL2and 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 [10,82,86,88,89,92,95–102]. 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, H1G20	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/genes 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 $SCAR_{17590}$ 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.

References

1. Nene, Y.L.; Reddy, M.V. Chickpea diseases and their control. In *The Chickpea*; Saxena, M.C., Singh, K.B., Eds.; CABI: Oxon, UK, 1987; pp. 233–270.
2. Kaiser, W.J.; Muehlbauer, F.J. An outbreak of ascochyta blight of chickpea in the Pacific Northwest, USA. *Intl. Chickpea News Lett.* **1989**, *18*, 16–17.
3. International Centre for Agricultural Research in the Dry Areas (ICARDA). Field Survey of Chickpea Diseases. In *Legume Annual Report*; ICARDA: Aleppo, Syria, 1996.
4. Akem, C.N. Ascochyta blight of chickpea: Present status and future priorities. *Int. J. Pest Manag.* **1999**, *45*, 131–137. [CrossRef]
5. Khan, M.S.A.; Ramsey, M.D.; Corbiere, R.; Infantino, A.; Porta-Puglia, A.; Bouznad, Z.; Scott, E.S. Ascochyta blight of chickpea in Australia: Identification, pathogenicity and mating type. *Plant Pathol.* **1999**, *48*, 230–234. [CrossRef]
6. Kaiser, W.J.; Ramsey, M.D.; Makkouk, K.M.; Bretag, T.W.; Acikgoz, N.; Kumar, J.; Nutter, F.W. Foliar diseases of cool season food legumes and their control. In *Linking Research and Marketing Opportunities for Pulses in the 21st Century*; Knight, R., Ed.; Kluwer Academic Publishers: Dordrecht, The Netherlands, 2000; pp. 437–455.
7. Chongo, G.; Buchwaldt, L.; Gossen, B.D.; Lafond, G.P.; May, W.E.; Johnson, E.N.; Hogg, T. Foliar fungicides to manage ascochyta blight (*Ascochyta rabiei*) of chickpea in Canada. *Can. J. Plant Pathol.* **2003**, *25*, 135–142. [CrossRef]
8. Singh, G.; Singh, K.; Kapoor, S. Screening for sources of resistance to Ascochyta blight of chickpea. *Int. Chickpea Newsl.* **1982**, *6*, 15–17.

9. Singh, G.; Verma, M.M.; Gill, A.S.; Sandhu, T.S.; Brar, H.S.; Sra, S.S.; Kapoor, S. Screening of gram varieties against Ascochyta blight. *Crop Improv.* **1984**, *11*, 153–154.
10. Udupa, S.M.; Baum, M. Genetic dissection of pathotypespecific resistance to Ascochyta blight disease in chickpea (*Cicer arietinum* L.) using microsatellite markers. *Theor. Appl. Genet.* **2003**, *106*, 1196–1202. [PubMed]
11. Pande, S.; Siddique, K.H.M.; Kishore, G.K.; Bayaa, B.; Gaur, P.M.; Gowda, C.L.L.; Bretag, T.W.; Crouch, J.H. Ascochyta blight of chickpea (*Cicer arietinum* L.): A review of biology, pathogenicity and disease management. *Aust. J. Agric. Res.* **2005**, *56*, 317–332. [CrossRef]
12. Bhardwaj, R.; Sandhu, J.S.; Kaur, L.; Gupta, S.K.; Gaur, P.M.; Varshney, R.K. Genetics of Ascochyta blight resistance in chickpea. *Euphytica* **2010**, *171*, 337–343. [CrossRef]
13. Kostrinski, J. *Problems in Chickpea Cultivation and Grain Crop Rotation in Israel*; Special publication No. 34; Agriculture Research Organization, Volcani Centre: Bet Dagan, Israel, 1974. (In Hebrew, English summary).
14. Singh, K.B.; Malhotra, R.S.; Saxena, M.C.; Bejiga, G. Superiority of winter sowing over traditional spring sowing of chickpea in the Mediterranean region. *Agron. J.* **1997**, *89*, 112–118. [CrossRef]
15. Singh, K.B.; Reddy, M.V. Advances in disease resistance breeding in chickpea. *Adv. Agron.* **1991**, *45*, 191–222.
16. Bedi, K.S.; Athwal, D.S. C 235 is the answer to blight. *Indian Fmg.* **1962**, *12*, 20–22.
17. Nene, Y.L.; Mengistu, A.; Sinclair, J.B.; Royse, D.J. An Annotated Bibliography of Chickpea Diseases 1915–1976. *Australas. Plant Pathol.* **1978**, *7*, 25–26. [CrossRef]
18. McDonald, B.A.; Linde, C. Pathogen population genetics, evolutionary potential, and durable resistance. *Ann. Rev. Phytopathol.* **2002**, *40*, 349–379. [CrossRef] [PubMed]
19. Li, H.; Rodda, M.; Gnanasambandam, A.; Aftab, M.; Redden, R.; Hobson, K.; Rosewarne, G.; Materne, M.; Kaur, S.; Slater, A.T. Breeding for biotic stress resistance in chickpea: Progress and prospects. *Euphytica* **2015**, *204*, 257–288. [CrossRef]
20. Punithalingam, E.; Holliday, P. Ascochyta rabiei. In *Descriptions of Pathogenic Fungi and Bacteria*; Commonwealth Mycological Institute: Kew, UK, 1972; Volume 34, p. 337.
21. Nene, Y.L. A review of Ascochyta blight of chickpea. *Trop. Pest Manag.* **1982**, *28*, 61–70. [CrossRef]
22. CAB International. *Crop Protection Compendium*; CAB International: Wallingford, UK, 2000.
23. Trapero-Casas, A.; Navas-Cortes, J.A.; Jimenez-Diaz, R.M. Airborne ascospores of *Didymella rabiei* as a major primary inoculum for Ascochyta blight epidemics in chickpea crops in southern Spain. *Eur. J. Plant Pathol.* **1996**, *102*, 237–245. [CrossRef]
24. Armstrong, C.L.; Chongo, G.; Gossen, B.D.; Duczek, L.J. Mating type distribution and incidence of the teleomorph of *Ascochyta rabiei* (*Didymella rabiei*) in Canada. *Can. J. Plant Pathol.* **2001**, *23*, 110–113. [CrossRef]
25. Barve, M.P.; Arie, T.; Salimath, S.; Muehlbauer, F.J.; Peever, T.L. 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. [CrossRef]
26. Peever, T.L.; Salimath, S.S.; Su, G.; Kaiser, W.J.; Muehlbauer, J. Historical and contemporary multilocus population structure *of Ascochyta rabiei* (teleomorph: *Didymella rabiei*) in the Pacific Northwest of the United States. *Mol. Ecol.* **2004**, *13*, 291–309. [CrossRef] [PubMed]
27. Rhaiem, A.; Chérif, M.; Peever, T.L.; Dyer, P.S. Population structure and mating system of *Ascochyta rabiei* in Tunisia: Evidence for the recent introduction of mating type 2. *Plant Pathol.* **2008**, *57*, 540–555. [CrossRef]
28. Vail, S.; Banniza, S. Molecular variability and mating-type of *Ascochyta rabiei* of chickpea from Saskatchewan, Canada. *Aust. J. Plant Pathol.* **2009**, *38*, 392–398. [CrossRef]
29. Atik, O.; Baum, M.; El-Ahmed, A.; Ahmed, S.; Abang, M.M.; Yabrak, M.M.; Murad, S.; Kabbabeh, S.; Hamwieh, A. Chickpea Ascochyta blight: Disease status and pathogen mating type distribution in Syria. *J. Phytopathol.* **2011**, *159*, 443–449. [CrossRef]
30. Phan, H.T.T.; Ford, R.; Bretag, T.W.; Taylor, P.W.J. A rapid and sensitive polymerase chain reaction (PCR) assay for detection of *Ascochyta rabiei*, the cause of ascochyta blight of chickpea. Australas. *Plant Pathol.* **2002**, *31*, 31–39.
31. Singh, G.; Sharma, Y.R. Ascochyta blight of chickpea. In *IPM System in Agriculture: Pulses*; Upadhyay, R.K., Mukherji, K.G., Eds.; Aditya Books Pvt. Ltd.: New Delhi, India, 1998; pp. 163–195.
32. Vir, S.; Grewal, J.S. Physiological specialization in *Ascochyta rabiei*, the causal organism of gram blight. *Indian Phytopathol.* **1974**, *27*, 265–266.

33. Udupa, S.M.; Weigand, F.; Saxena, M.C.; Kahl, G. Genotyping with RAPD and microsatellite markers resolves pathotype diversity in the Ascochyta blight pathogen of chickpea. *Theor. Appl. Genet.* **1998**, *97*, 299–307. [CrossRef]
34. Vail, S.; Banniza, S. Structure and pathogenic variability in *Ascochyta rabiei* populations on chickpea in the Canadian prairies. *Plant Pathol.* **2008**, *57*, 665–673. [CrossRef]
35. Varshney, R.; Pande, S.; Kannan, S.; Mahendar, T.; Sharma, M.; Gaur, P.; Hoisington, D. Assessment and comparison of AFLP and SSR based molecular genetic diversity in Indian isolates of *Ascochyta rabiei*, a causal agent of Ascochyta blight in chickpea (*Cicer arietinum* L.). *Mycol. Prog.* **2009**, *8*, 87–97. [CrossRef]
36. Atik, O.; Ahmed, S.; Abang, M.M.; Imtiaz, M.; Hamwieh, A.; Baum, M.; El-Ahmed, A.; Murad, S.; Yabrak, M.M. Pathogenic and genetic diversity of *Didymella rabiei* affecting chickpea in Syria. *Crop Prot.* **2013**, *46*, 70–79. [CrossRef]
37. Imtiaz, M.; Abang, M.M.; Malhotra, R.S.; Ahmed, S.; Bayaa, B.; Udupa, S.M.; Baum, M. Pathotype IV, a new and highly virulent pathotype of *Didymella rabiei*, causing Ascochyta blight in chickpea in Syria. *Plant Dis.* **2011**, *95*, 1192. [CrossRef]
38. Bayaa, B.; Udupa, S.M.; Baum, M.; Malhotra, R.S.; Kabbabeh, S. Pathogenic variability in Syrian isolates of *Ascochyta rabiei*. In Proceedings of the 5th European Conference on Grain Legumes, Dijon, France, 7–11 June 2004.
39. Geistlinger, J.; Weising, K.; Winter, P.; Kahl, G. Locus-specific microsatellite markers for the fungal chickpea pathogen *Didymella rabiei* (anamorph) *Ascochyta rabiei*. *Mol. Ecol.* **2000**, *9*, 1919–1952. [CrossRef]
40. Phan, H.T.T.; Ford, R.; Taylor, P.W.J. Population structure of *Ascochyta rabiei* in Australia based on STMS fingerprints. *Fungal Divers.* **2003**, *13*, 111–129.
41. Bayraktar, H.; Dolar, F.S.; Tör, M. Determination of genetic diversity within *Ascochyta rabiei* (Pass.) Labr. the cause of Ascochyta blight of chickpea in Turkey. *J. Plant Pathol.* **2007**, *89*, 341–347.
42. Fondevilla, S.; Krezdorn, N.; Rotter, B.; Kahl, G.; Winter, P. In planta Identification of Putative Pathogenicity Factors from the Chickpea Pathogen *Ascochyta rabiei* by De novo Transcriptome Sequencing Using RNA-Seq and Massive Analysis of cDNA Ends. *Front. Microbiol.* **2015**, *6*, 1329. [CrossRef] [PubMed]
43. Turgeon, B.G. Application of mating type gene technology to problems in fungal biology. *Annu. Rev. Phytopathol.* **1998**, *36*, 115–137. [CrossRef] [PubMed]
44. Turgeon, B.G.; Berbee, M.L. Evolution of pathogenic and reproductive strategies in Cochliobolus and related genera. In *Molecular Genetics of Host-Specific Toxins in Plant Disease*; Kohmoto, K., Yoder, O.C., Eds.; Kluwer Academic Publishers: Dordrecht, The Netherlands, 1998; pp. 153–163.
45. Poeggeler, S. Phylogenetic relationships between mating-type sequences from homothallic and heterothallic ascomycetes. *Curr. Genet.* **1999**, *36*, 222–231.
46. Singh, K.B.; Malhotra, R.S.; Halila, M.H.; Knights, E.J.; Verma, M.M. Current status and future strategy in breeding chickpea for resistance to biotic and abiotic stresses. *Euphytica* **1994**, *73*, 137–149. [CrossRef]
47. Chen, W.; Mcphee, K.E.; Muehlbauer, F.J. Use of a mini-dome bioassay and grafting to study resistance of chickpea to ascochyta blight. *J. Phytopathol.* **2005**, *153*, 579–587. [CrossRef]
48. Pande, S.; Sharma, M.; Gaur, P.M.; Tripathi, S.; Kaur, L.; Basandarai, A.; Khan, T.; Gowda, C.L.L.; Siddique, K.H.M. Development of screening techniques and identification of new sources of resistance to Ascochyta blight disease of chickpea. *Australas. Plant Pathol.* **2011**, *40*, 149–156. [CrossRef]
49. Chen, W.; Muehlbauer, F.J. An improved technique for virulence assay of *Ascochyta rabiei* on chickpea. *Int. Chickpea Pigeonpea Newsl.* **2003**, *10*, 31–33.
50. Singh, K.B.; Hawtin, G.C.; Nene, Y.L.; Reddy, M.V. Resistance in chickpeas to *Ascochyta rabiei*. *Plant Dis.* **1981**, *65*, 586–587. [CrossRef]
51. Pande, S.; Sharma, M.; Gaur, P.M.; Gowda, C.L.L. *Host Plant Resistance to Ascochyta Blight of Chickpea*; Information Bulletin No 82; International Crops Research Institute for the Semi-Arid Tropics: Patancheru, India, 2010; p. 40.
52. Ahmad, F.; Gaur, P.M.; Croser, J. Chickpea (*Cicer arietinum* L.). In *Genetic Resources, Chromosome Engineering and Crop Improvement*; Singh, R.J., Jauhar, P.P., Eds.; CRC Press: Boca Raton, FL, USA, 2005; pp. 187–217.
53. Collard, B.; Ades, P.; Pang, E.; Brouwer, J.; Taylor, P. Prospecting for sources of resistance to ascochyta blight in wild *Cicer* species. *Australas. Plant Pathol.* **2001**, *30*, 271–276. [CrossRef]

54. Ahmad, F.; Slinkard, A.E. The extent of embryo and endosperm growth following interspecific hybridization between *Cicer arietinum* L. and related annual wild species. *Genet. Resour. Crop Evol.* **2004**, *51*, 765–772. [CrossRef]
55. Nguyen, T.T.; Taylor, P.W.J.; Redden, R.J.; Ford, R. Genetic diversity in *Cicer* using AFLP analysis. *Plant Breed.* **2004**, *123*, 173–179. [CrossRef]
56. Pande, S.; Kishore, G.K.; Upadhyaya, H.D.; Narayana Rao, J. Identification of sources of multiple disease resistance in mini-core collection of chickpea. *Plant Dis.* **2006**, *90*, 1214–1218. [CrossRef]
57. Pande, S.; Ramgopal, D.; Kishore, G.K.; Mallikarjuna, N.; Sharma, M.; Pathak, M.; Narayana Rao, J. Evaluation of wild *Cicer* species for resistance to Ascochyta blight and Botrytis grey mold in controlled environment at ICRISAT, Patancheru, India. *J. Sat. Agric. Res.* **2006**, *2*, 1–3.
58. Mallikarjuna, N. Ovule and embryo culture to obtain hybrids from interspecific incompatible pollinations in chickpea. *Euphytica* **1999**, *110*, 1–6. [CrossRef]
59. Iqbal, S.M.; Hussain, S.; Bakhsh, A.; Bashir, M. Sources of resistance in chickpea against ascochyta blight disease. *Int. J. Agri. Biol.* **2002**, *4*, 488–490.
60. Dubey, S.C.; Singh, B. Evaluation of chickpea genotypes against Ascochyta blight. *Indian Phytopath.* **2003**, *56*, 505.
61. Chen, W.; Coyne, T.C.J.; Peever, T.L.; Muehlbauer, F.J. Characterization of chickpea differentials for pathogenicity assay of Ascochyta blight and identification of chickpea accessions resistant to *Didymella rabiei*. *Plant Pathol.* **2004**, *53*, 759–769. [CrossRef]
62. Malik, S.R.; Iqbal, S.M.; Iqbal, U.; Ahmad, I.; Haqqani, A.M. Response of Chickpea Lines to *Ascochyta rabiei* at Two Growing Stages. *Caspian J. Env. Sci.* **2005**, *3*, 173–177.
63. Shokouhifar, F.; Bagheri, A.A.R.; Rastgar, F.M. Identification of resistant chickpea lines against pathotypes causing ascochyta blight disease in Iran. *Iranian J. Biol.* **2006**, *19*, 29–42.
64. Rubio, J.; Moreno, M.T.; Moral, A.; Rubiales, D.; Gil, J. Registration of RIL58-ILC72/Cr5, a chickpea germplasm line with rust and Ascochyta blight resistance. *Crop Sci.* **2006**, *46*, 2331–2332. [CrossRef]
65. Ilyas, M.B.; Chaudhry, M.A.; Javed, N.; Ghazanfar, M.U.; Khan, M.A. Sources of resistance in chickpea germplasm against ascochyta blight. *Pak. J. Bot.* **2007**, *39*, 1843–1847.
66. Chandirasekaran, R.; Warkentin, T.D.; Gan, Y.; Shirtliffe, S.; Gossen, B.D.; Taran, B.; Banniza, S. Improved sources of resistance to ascochyta blight in chickpea. *Can. J. Plant Sci.* **2009**, *89*, 107–118. [CrossRef]
67. Iqbal, S.M.; Ali, S.; Ghafoor, A. Development of resistance in chickpea to Ascochyta blight. *Mycopath.* **2010**, *8*, 61–64.
68. Kaur, l.; Sandhu, J.S.; Malhotra, R.S.; Imtiaz, M.; Sirari, A. Sources of stable resistance to Ascochyta blight in exotic kabuli chickpea. *J. Food Legumes* **2012**, *25*, 79–80.
69. Pande, S.; Sharma, M.; Gaur, P.M.; Basandrai, A.K.; Kaur, L.; Hooda, K.S.; Basandrai, D.; Kiran Babu, T.; Jain, S.K.; Rathore, A. Biplot analysis of genotype × environment interactions and identification of stable sources of resistance to Ascochyta blight in chickpea (Cicer arietinum L.). *Australas. Plant Pathol.* **2013**, *42*, 561–571. [CrossRef]
70. Benzohra, I.E.; Bendahmane, B.S.; Labdi, M.; Benkada, M.Y. Sources of Resistance in Chickpea Germplasm to Three Pathotypes *of Ascochyta rabiei* (Pass.) Labr. In Algeria. *World Appl. Sci. J.* **2013**, *21*, 873–878.
71. Kimurto, P.K.; Towett, B.K.; Mulwa, R.S.; Njogu, N.; Jeptanui, L.J.; Rao, G.N.V.P.R.; Silim, S.; Kaloki, P.; Korir, P.; Macharia, J.K. Evaluation of chickpea genotypes for resistance to Ascochyta blight (*Ascochyta rabiei*) disease in the dry highlands of Kenya. *Phytopathol. Mediterr.* **2013**, *52*, 212–221.
72. Ahmad, S.; Khan, M.A.; Sahi, S.T.; Ahmad, R. Evaluation of chickpea germplasm against *Ascochyta rabiei* (pass) lab. *J. Animal Plant Sci.* **2013**, *23*, 440–443.
73. Duzdemir, O.; Selvi, B.; Yanar, Y.; Yildirimi, A. Sources of resistance in chickpea (*Cicer arietinum* L.) land races against Ascochyta rabiei causal agent of ascochyta blight disease. *Pak. J. Bot.* **2014**, *46*, 1479–1483.
74. Benzohra, I.E.; Bendahmane, B.S.; Benkada, M.Y.; Labdi, M. Screening of 15 chickpea germplasm accessions for resistance to *Ascochyta rabiei* in North West of Algeria. *American-Eurasian J. Agric. Environ. Sci.* **2015**, *15*, 109–114.
75. Lichtenzveig, J.; Shtienberg, D.; Zhang, H.B.; Bonfil, D.J.; Abbo, S. Biometricanalyses of the inheritance of resistance to *Didymella rabiei* in chickpea. *Phytopathology* **2002**, *92*, 417–423. [CrossRef] [PubMed]
76. Gan, Y.; Gossen, B.D.; Li, L.; Ford, G.; Banniza, S. Cultivar type, plant population and ascochyta blight in chickpea. *Agron. J.* **2007**, *99*, 1463–1470. [CrossRef]

77. Rubiales, D.; Avila, C.M.; Sillero, J.C.; Hybl, M.; Narits, L.; Sass, O.; Flores, F. Identification and multi-environment validation of resistance to *Ascochyta fabae* in faba bean (*Vicia faba*). *Field Crop Res.* **2012**, *126*, 165–170. [CrossRef]
78. Sharma, M.; Kiran Babu, T.; Gaur, P.M.; Ghosh, R.; Rameshwar, T.; Chaudhary, R.G.; Upadhyay, J.P.; Gupta, Om.; Saxena, D.R.; Kaur, L.; *et al*. Identification and multi-environment validation of resistance to *Fusarium oxysporum* f. sp. *Ciceris* in chickpea. *Field Crop Res.* **2012**, *135*, 82–88. [CrossRef]
79. Hafiz, A.; Ashraf, M. Studies on inheritance of resistance to Mycosphaerella blight in gram. *Phytopathology* **1953**, *43*, 580–581.
80. Vir, S.; Grewal, J.S.; Gupta, V.P. Inheritance of resistance to Ascochyta blight in chickpea. *Euphytica* **1975**, *24*, 209–211. [CrossRef]
81. Eser, D. *Heritability of Some Important Plant Characters, Their Relationships with Plant Yield and Inheritance of Ascochyta Blight Resistance in Chickpea (Cicer arietinum L.)*; Ankara University, Faculty of Agriculture Publications No. 620: Ankara, Turkey, 1976.
82. Taleei, A.; Kanouni, H.; Baum, M. Genetical analysis of ascochyta blight resistance in chickpea. In *Bioscience and Biotechnology, Communications in Computer and Information Science*; Slezak, D., Arsalan, T., *et al*, Eds.; Springer-Verlag: Berlin, Germany, 2009.
83. Singh, K.B.; Reddy, M.V. Inheritance of resistance to ascochyta blight in chickpea. *Crop Sci.* **1983**, *23*, 9–10. [CrossRef]
84. Labdi, M.; Malhotra, R.S.; Benzohra, I.E.; Imtiaz, M. Inheritance of resistance to *Ascochyta rabiei* in 15 chickpea germplasm accessions. *Plant Breed.* **2013**, *132*, 197–199. [CrossRef]
85. Varshney, R.K.; Mohan, S.M.; Gaur, P.M.; Gangarao, N.V.P.R.; Pandey, M.K.; Bohra, A.; Sawargaonkar, S.L.; Chitikineni, A.; Kimurto, P.K.; Janila, P.; *et al*. Achievements and prospects of genomics-assisted breeding in three legume crops of the semi-arid tropics. *Biotechnol. Adv.* **2013**, *3*, 1120–1134. [CrossRef] [PubMed]
86. Cho, S.; Chen, W.; Muehlbauer, F.J. Pathotype-specific genetic factors in chickpea (*Cicer arietinum* L.) for quantitative resistance to Ascochyta blight. *Theor. Appl. Genet.* **2004**, *109*, 733–739. [CrossRef] [PubMed]
87. Winter, P.; Benko-Iseppon, A.M.; Hüttel, B.; Ratnaparkhe, M.; Tullu, A.; Sonnante, G.; Pfaff, T.; Tekeoglu, M.; Santra, D.; Sant, V.J.; *et al*. A linkage map of the chickpea (*Cicer arietinum* L.) genome based on recombinant inbred lines from a *C. arietinum* × *C. reticulatum* cross: localization of resistance genes for Fusarium wilt races 4 and 5. *Theor. Appl. Genet.* **2000**, *101*, 1155–1163. [CrossRef]
88. Flandez-Galvez, H.; Ford, R.; Pang, E.C.K.; Taylor, P.W.J. An intraspecific linkage map of the chickpea (*Cicer arietinum* L.) genome based on sequence-tagged microsatellite site and resistance gene analog markers. *Theor. Appl. Genet.* **2003**, *106*, 1447–1456. [PubMed]
89. Flandez-Galvez, H.; Ades, R.; Ford, R.; Pang, E.; Taylor, P. QTL analysis for ascochyta blight resistance in an intraspecific population of chickpea (*Cicer arietinum* L.). *Theor. Appl. Genet.* **2003**, *107*, 1257–1265. [CrossRef] [PubMed]
90. Bian, X.Y.; Ford, R.; Han, T.R.; Coram, T.E.; Pang, E.C.K.; Taylor, P.W.J. Approaching chickpea quantitative trait loci conditioning resistance to Ascochyta rabiei via comparative genomics. *Australas. Plant Path.* **2007**, *36*, 419–423. [CrossRef]
91. Iruela, M.; Castro, P.; Rubio, J.; Cubero, J.I.; Jacinto, C.; Millan, T.; Gil, J. Validation of a QTL for resistance to Ascochyta blight linked to resistance to Fusarium wilt race 5 in chickpea (*Cicer arietinum* L.). *Eur. J. Plant Pathol.* **2007**, *119*, 29–37. [CrossRef]
92. Hamwieh, A.; Imtiaz, M.; Hobson, K.; Kemal, S.A. Genetic diversity of microsatellite alleles located at quantitative resistance loci for Ascochyta blight resistance in a global collection of chickpea germplasm. *Phytopathol. Mediterr.* **2013**, *52*, 191–199.
93. Madrid, E.; Chen, W.; Rajesh, P.N.; Castro, P.; Millan, T.; Gil, J. Allele-specific amplification for the detection of Ascochyta blight resistance in chickpea. *Euphytica* **2013**, *189*, 183–190. [CrossRef]
94. Varshney, R.K.; Mir, R.R.; Bhatia, S.; Thudi, M.; Hu, Y.; Azam, S.; Zhang, Y.; Jaganathan, D.; You, F.M.; Gao, J.; *et al*. Integrated physical, genetic and genome map of chickpea (*Cicer arietinum* L.). *Funct. Integr. Genomics* **2014**, *14*, 59–73. [CrossRef] [PubMed]
95. Tekeoglu, M.; Rajesh, P.N.; Muehlbauer, F.J. Integration of sequence tagged microsatellite sites to the chickpea genetic map. *Theor. Appl. Genet.* **2002**, *105*, 847–854. [PubMed]

96. Iruela, M.; Rubio, J.; Barro, F.; Cubero, J.I.; Millan, T.; Gil, J. Detection of two quantitative trait loci for resistance to Ascochyta blight in an intra-specific cross of chickpea (*Cicer arietinum* L.): Development of SCAR markers associated with resistance. *Theor. Appl. Genet.* **2006**, *112*, 278–287. [CrossRef] [PubMed]
97. Lichtenzveig, J.; Bonfil, D.J.; Zhang, H.B.; Shtienberg, D.; Abbo, S. Mapping quantitative trait loci in chickpea associated with time to flowering and resistance to *Didymella rabiei* the causal agent of Ascochyta blight. *Theor. Appl. Genet.* **2006**, *113*, 1357–1369. [CrossRef] [PubMed]
98. Tar'an, B.; Warkentin, T.D.; Tullu, A.; Vanderberg, A. Genetic mapping of Ascochyta blight resistance in chickpea (*Cicer arietinum*) using a simple sequence repeat linkage map. *Genome* **2007**, *50*, 26–34. [PubMed]
99. Anbessa, Y.; Taran, B.; Warkentin, T.D.; Tullu, A.; Vandenberg, A. Genetic analyses and conservation of QTL for Ascochyta blight resistance in chickpea (*Cicer arietinum* L.). *Theor. Appl. Genet.* **2009**, *4*, 757–765. [CrossRef] [PubMed]
100. Kottapalli, P.; Gaur, P.M.; Katiyar, S.K.; Crouch, J.H.; Buhariwalla, H.K.; Pande, S.; Gali, K.K. Mapping and validation of QTLs for resistance to an Indian isolate of Ascochyta blight pathogen in chickpea. *Euphytica* **2009**, *165*, 79–88. [CrossRef]
101. Sabbavarapu, M.M.; Sharma, M.; Chamarthi, S.K.; Swapna, N.; Rathore, A.; Thudi, M.; Gaur, P.M.; Pande, S.; Singh, S.; Kaur, L.; *et al*. Molecular mapping of QTLs for resistance to Fusarium wilt (race 1) and Ascochyta blight in chickpea (*Cicer arietinum* L.). *Euphytica* **2013**, *93*, 121–133. [CrossRef]
102. Stephens, A.; Lombardi, M.; Cogan, N.O.I.; Forster, J.W.; Hobson, K.; Materne, M.; Kaur, S. Genetic marker discovery, interspecific linkage map construction and quantitative trait locus analysis of ascochyta blight resistance in chickpea (*Cicer arietinum* L.). *Mol. Breed.* **2014**, *33*, 297–313. [CrossRef]
103. Santra, D.K.; Tekeoglu, M.; Ratnaparkhe, M.; Kaiser, W.J.; Muehlbauer, F.J. Identification and mapping of QTLs conferring resistance to ascochyta blight in chickpea. *Crop Sci.* **2000**, *40*, 1606–1612. [CrossRef]
104. Cobos, M.J.; Rubio, J.; Strange, R.N.; Moreno, M.T.; Gil, J.; Millan, T. A new QTL for Ascochyta blight resistance in an RIL population derived from an interspecific cross in chickpea. *Euphytica* **2006**, *149*, 105–111. [CrossRef]
105. Kanouni, H.; Taleei, A.; Peyghambari, S.A.; Okhovat, S.M.; Baum, M.; Abang, M. QTL analysis for ascochyta blight resistance in chickpea (*Cicer arietinum* L.) using microsatellite markers. *J. Agric. Res.* **2009**, *25*, 109–127.
106. Aryamanesh, N.; Nelson, M.N.; Yan, G.; Clarke, H.J.; Siddique, K.H.M. Mapping a major gene for growth habit and QTLs for Ascochyta blight resistance and flowering time in a population between chickpea and *Cicer reticulatum*. *Euphytica* **2010**, *173*, 307–319. [CrossRef]
107. Castro, P.; Rubio, J.; Madrid, E.; Fernández-Romero, M.D.; Millán, T.; Gil, J. Efficiency of marker-assisted selection for ascochyta blight in chickpea. *J. Agric. Sci.* **2015**, *153*, 56–67. [CrossRef]
108. Gupta, P.K.; Varshney, R.K. The development and use of microsatellite markers for genetic analysis and plant breeding with emphasis on bread wheat. *Euphytica* **2000**, *113*, 163–185. [CrossRef]
109. Varshney, R.K.; Nayak, S.N.; May, G.D.; Jackson, S.A. Next generation sequencing technologies and their implications for crop genetics and breeding. *Trends Biotechnol.* **2009**, *27*, 522–530. [CrossRef] [PubMed]
110. Taran, B.; Warkentin, T.D.; Vandenberg, A. Fast track genetic improvement of Ascochyta blight resistance and double podding in chickpea by marker-assisted backcrossing. *Theor. Appl. Genet.* **2013**, *126*, 1639–1647. [CrossRef] [PubMed]
111. Collard, B.C.Y.; Pang, E.C.K.; Ades, P.K.; Taylor, P.W.J. Preliminary investigations of QTL associated with seedlings resistance to Ascochyta blight from *Cicer echinospermum*, a wild relative of chick pea. *Theor. Appl. Genet.* **2003**, *107*, 719–729. [CrossRef] [PubMed]
112. Varshney, R.K.; Mohan, S.M.; Gaur, P.M.; Chamarthi, S.K.; Singh, V.K.; Srinivasan, S.; Swapna, N.; Sharma, M.; Singh, S.; Kaur, L.; *et al*. Marker-Assisted Backcrossing to Introgress Resistance to Fusarium Wilt Race 1 and Ascochyta Blight in C 214, an Elite Cultivar of Chickpea. *Plant Genome* **2014**, *7*. [CrossRef]
113. Bouchez, A.; Hospital, F.; Causse, M.; Gallais, A.; Charcosset, A. Marker-assisted introgression of favorable alleles at quantitative trait loci between maize elite lines. *Genetics* **2002**, *162*, 1945–1959. [PubMed]
114. Lecomte, L.; Duffé, P.; Buret, M.; Servin, B.; Hospital, F.; Causse, M. Marker-assisted introgression of five QTLs controlling fruit quality traits into three tomato lines revealed interactions between QTLs and genetic backgrounds. *Theor. Appl. Genet.* **2004**, *109*, 658–668. [CrossRef] [PubMed]
115. Ribaut, J.M.; Hoisington, D. Marker-assisted selection: new tools and strategies. *Trends Plant Sci.* **1998**, *3*, 236–239. [CrossRef]

116. Young, N.D. A cautiously optimistic vision for marker-assisted breeding. *Mol. Breeding* **1999**, *5*, 505–510. [CrossRef]
117. Bouhadida, M.; Benjannet, R.; Madrid, E.; Amri, M.; Kharrat, M. Efficiency of marker-assisted selection in detection of ascochyta blight resistance in Tunisian chickpea breeding lines. *Phytopathol. Mediterr.* **2013**, *52*, 202–211.