Chapter 5
Disease Resistance in Cotton

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Abstract Though cotton has been adapted to subtropical and tropical regions of the world as a long season crop, it experiences a large array of diseases triggered by parasitic nematodes, fungi, bacteria and virus. Such diseases have severe impact on cotton growth and development starting from seed germination, plant growth and reproduction which ultimately lead to significant losses in fiber yield and quality. In this review chapter, the top most major diseases of cotton viz., bacterial blight, Fusarium wilt and Verticillium wilt are elaborated. Information on other cotton diseases also is referred to wherever appropriate. The objective of this chapter is to provide a comprehensive synthesis on the research progress made in the area of breeding, genetics, mapping of resistance genes or quantitative trait loci (QTLs) and marker-assisted selection for disease resistance in cotton. Considerable progress has been made in the resistance aspects of cotton's tissue structure, physiological and biochemical features, R-gene-mediated and hormone-mediated resistance through different signal pathways and their interactions. Availability of cotton whole-genome sequences and high-throughput molecular markers, offer new avenues in precisely pinpoint the site of resistant genes. As the developments in genetically modified cotton breeding germplasm is slow and problems associated to evolve a cultivar that adapts to different ecological environments still persists, modern genomics tools need to be integrated with traditional breeding methods to accelerate the process of disease resistance cotton breeding in the future.

Keywords Cotton · Diseases · Molecular breeding · Resistance sources · Marker-assisted selection

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Abbreviations

AM	Association Mapping
Avr	Avirulence Gene
BB	Bacterial Blight
BC	Back Cross
BG	Bollgard
BILs	Backcross Inbred Lines
cM	CentiMorgan
CRICPR/Cas9	Clustered Regularly Interspaced Short Palindromic Repeats/CRISPR-Associated Protein 9
CSSLs	Chromosome Segment Substitution Lines
FCF	Fungus Culture Filtrate
FOV	*Fusarium oxysporum* f. sp. *Vasinfectum*
FW	Fusarium wilt
GBS	Genotyping-By-Sequencing
GWAS	Genome-Wide Association Study
LG	Linkage Group
MAGIC	Multiparent Advanced Generation Intercross
MAS	Marker-Assisted Selection
QTL	Quantitative Trait Loci
RFLP	Restriction Fragment Length Polymorphism
RILs	Recombinant Inbred Lines
RKN	Root Knot Nematode
RR	Roundup-Ready
SNPs	Single Nucleotide Polymorphisms
SSR	Simple Sequence Repeat
VIGS	Virus Induced Gene Silencing
VW	Verticillium Wilt
Xcm	*Xanthomonas citri* pv. *malvacearum*
ZFN	Zinc Finger Nuclease

5.1 Introduction

Cotton (*Gossypium* spp.), an important economic fiber and oilseed crop with a production value of US$50 billion, is being cultivated on 2.5% of the world's arable land that spreads in more than 80 countries (http://cottonanalytics.com/cotton-in-the-world-economy/). It has been estimated that nearly two third of the global cotton production is chiefly contributed by India, China and the United States though Brazil, Pakistan, Uzbekistan, Turkey, Greece, Mexico and Argentina also contributing in significant level (https://www.statista.com/statistics/263055/cotton-production-worldwide-by-top-countries/). *Gossypium* genus has so far described with 45 diploid ($2n = 2x = 26$) and seven allotetraploid species ($2n = 4x = 52$) (Wendel and Cronn

2003). With respect to the pairing affinities of chromosomes, all the diploid species are grouped into eight genome groups viz., A to G and K (Stewart 1994).

Among the 49 *Gossypium* spp. the cultivated species are two Asiatic diploids (*G. herbaceum*, A1 and *G. arboreum*, A2 genome; $2n = 2x = 26$) and two tetraploids (*G. hirsutum*, AD1 and *G. barbadense*, AD2 genome; $2n = 4x = 52$). However, *G. hirsutum*, often referred as Upland cotton (originated in Mexico), occupies more than 95% of the global cotton production and *G. barbadense*, often called as Pima, Egyptian, or Sea-island cotton (originated in Peru) accounts for the remaining 5% of the world's raw cotton production. Asiatic cottons are cultivated on certain pockets of India and Pakistan.

It has been theorized that tetraploid cotton, including Upland and Pima, might have originated from a natural hybridization that occurred few million years ago between an Asiatic cotton and a wild D-genome species such as *G. raimondii* (Wendel and Cronn 2003). Consequently, all the seven tetraploid cottons are cross compatible and they form the primary gene pool. Species from the genomes such as A, B (dispersed in Africa) and F (found in Arabia) as well as D (distributed in the America continent) constitutes the secondary gene pool, as genes from these genomes can be transferred to the tetraploid in interspecific hybridization. On the other hand, perfect hybridization between the A and D subgenomes as well as among tertiary gene pools (such as the C (dispersed in Australia), E (spread in Arabia), G (distributed in Australia), and K (disseminated in Australia) genomes) have been found to be very poor (Stewart 1994).

Owing to its long season nature in subtropical and tropical climates, cotton is vulnerable to many diseases caused by nematodes, fungi, bacteria and virus. Cotton diseases are widespread and they limit seed germination, plant growth and reproduction and ultimately results into severe losses in final fiber yield and quality. For example, the estimated cotton yield losses due to diseases in the US alone were 11.8–12.6% in 2016–2017; even higher percentages of yield losses were noticed in some of the other states. Among the diseases, nematode complex (root knot—*Meloidogyne* spp. and reniform—*Rotylenchulus reniformis* and other nematodes) triggered the highest yield loss at estimated 4.3–4.7%, followed by boll rot (caused by pathogens such as *Rhizopus* spp.), seedling diseases (owing to complex fungal pathogens such as *Rhizoctonia solani, Pythium* spp., *Fusarium* spp. and bacterial pathogens) and Fusarium (caused by *Fusarium oxysporum* f. sp. *vasinfectum*, FOV) and Verticillium (caused by *Verticillium dahliae*) wilt. In addition to these diseases, when favourable weather conditions prevail, diseases such as root rot (*Phymatotrichopsis omnivora*), Ascochyta blight (*Ascochyta*gossypii) and leaf spots (*Alternaria* spp.) also infect cotton plants. Though cotton leaf curl virus emerges as a disease in certain pockets of Asian countries, it has been rarely reported in other cotton growing areas.

Cotton diseases are controlled by employing chemical, biological and cultural practices and the use of resistant cultivars. Among them, evolving and utilizing disease resistant cultivars is considered as affordable and efficient strategy of controlling cotton diseases. Conventional breeding for genetic improvement of cotton with improved disease resistance has been reported as early as 1910s; however, resistant

cultivars for bacterial blight was reported during 1930s in Sudan, and later in USA. Subsequently, several resistant cultivars were developed and simple management strategies were promoted (such as acid-delinting) to control this disease.

In order to combat the worldwide fibre damage caused by Fusarium and Verticillium wilt, attention was focused to evolve novel cotton cultivars with improved resistance to these wilts in 1950s. Similarly, as the incidence of root-knot nematode gradually increased in 1990s, genetic and breeding efforts on root-knot nematode resistance have been taken in USA. Such efforts were subsequently helped to identify two major resistance genes and they were used in commercial conventional and transgenic breeding programs in recent years.

This chapter highlights the progress made in the lines of genetics, breeding, mapping of resistance genes or quantitative trait loci (QTLs) and marker-assisted selection in three major cotton diseases viz., bacterial blight, Fusarium wilt and Verticillium wilt. Information on other cotton diseases have also been provided but with little details as the information are scarce on those diseases.

5.2 Bacterial Blight

5.2.1 Causal Agent and Significance

Almost all the cotton growing regions in the world are affected by Bacterial blight (BB) caused by *Xanthomonas citri* pv. *malvacearum* (*Xcm*), a gram-negative bacterium. All the aerial parts of the cotton, throughout its growth phases are vulnerable to *Xcm* infections. Water-soaked and angular-shaped lesions develops on the leaves and other vegetative parts of cotton and the nearby lesions amalgamate into bigger block spot which subsequently result into defoliation in susceptible plants (Fig. 5.1). *Xcm* infection has also been found in immature bolls, which eventually lead to boll rot (Fig. 5.1). A simple, effective and affordable management strategy of BB is use of resistant cultivars and planting with acid delinted cotton seeds. Using 11

Fig. 5.1 Symptoms of bacterial blight in cotton **a** Angular leaf spot, **b** Vein necrosis and **c** Boll rot

host differential cotton accessions, 19 *Xcm* races has been so far recognized (Hillocks 1992; Delannoy et al. 2005) and among them, race 18 is the most virulent in the USA and elsewhere in the world where cotton is cultivated (Allen and West 1991; Zhang et al. 2020b).

5.2.2 Resistant Germplasm

Cotton germplasm can be screened for BB resistance using several kinds of inoculation and evaluation methods (Thaxton and El-Zik 1993). In general, such methods should allow BB pathogen to enter into the leaves through open stomata or wounds to initiate infection (Delannoy et al. 2005). To this end, a low-pressure sprayer with organosilicone non-ion surfactants (Wheeler et al. 2007) or scratching (wounding) the lower surface of cotyledons/leaves with a toothpick (Bird 1982) or a combination of both wounding and spraying (Bourland 2018) are frequently used. Such artificial inoculations helped to evaluate in large scale cotton accessions with less labour and time. Alternatively, under laboratory conditions, syringe or vacuum infiltration have also been attempted to infect cotton seedlings with BB pathogen (Cox et al. 2019). Irrespective of the methods, the main factors that limit the development of the BB symptoms are temperature and humidity (Wheeler et al. 2007; Jalloul et al. 2015; Cox et al. 2019).

It has been shown that quantitative resistance as well as various levels of qualitative resistance mediated by major resistance genes are hard to evaluate using toothpick scratching method. Actually, during initial studies, several numbers of major BB resistance genes were identified by employing sprayer inoculation (Zhang et al. 2020b). Though variation in BB resistance level have been documented within the genus *Gossypium*, it has been invariably found that cultivars of the diploids (such as *G. arboreum* and *G. herbaceum*) and the wild diploid (e.g., *G. anomalum*) have found to be highly resistant or immune to BB (Hunter et al. 1968; Wallace and El-Zik 1989; Hillocks 1992; Zhang et al. 2020a, b, c, d, e, f). On the other hand, tetraploids (such as *G. hirsutum*) have exhibited large variations in responses from extremely susceptible to highly resistance to BB. Nevertheless, another tetraploid species, *G. barbadense*, has always displayed susceptibility to BB (Delannoy et al. 2005; Jallou 2015), including the current commercial cultivars. Screening the cotton pre-breeding materials for BB resistance is a routine process in USA (for example, University of Arkansas regularly conducts screening program for upland and elite public breeding lines for BB resistance (Bourland 2018); Similarly, in the Texas High Plains there is an annual cotton screening program to *Xcm* race 18 under field conditions (Wheeler and Dever 2020); at New Mexico, Jinfa Zhang established a genetic and genomic research program for BB resistance. In a massive screening program, Hanan et al. evaluated more than 330 obsolete upland cotton cultivars and germplasm lines and identified several BB resistant lines. Such efforts have clearly demonstrated that resistance to *Xcm* race 18 is qualitative and can be easily fixed in breeding without a selection pressure. Further, pedigree investigation indicated that there could be

different sources of BB resistance, which can be used as potential donors in cotton breeding program to improve BB resistance.

5.2.3 Genetics of Resistance

In response to tireless efforts made in Sudan during 1930s and 1950s, nearly 10 BB resistance genes were identified (including B_1, B_2, and B_7 from Upland, B_2, B_3 and B_{10K} from *G. hirsutum* race *punctatum*, B_4 and B_6 from *G. arboreum*, B_5 from a perennial *G. barbadense*, recessive gene b_8 from *G. anomalum*, and B_{9K} from *G. herbaceum*) and were introgressed into Egyptian (*G. barbadense* L.) Sakel and other commercial Upland cotton cultivars that were cultivated in Sudan (Knight and Clouston 1939; Knight 1963).

In addition to these genes, several other BB resistance genes such as B_{9L} and B_{10L} from Upland cotton, B_{11} and another unnamed resistance gene from *G. herbaceum* which was grown in Africa, were also reported. On the other hand, such BB resistance genes were scouted only from upland cotton in USA during 1950s and 1960s and those studies were lead to identify major BB resistance genes such as B_7, B_{12}, B_{In}, B_N and B_S; besides polygene modifiers or complexes have also been reported from upland cotton (Zhang et al. 2020a, b, c, d, e, f). Though there were numerous unnamed BB resistance genes, their allelic relationships have not yet been clearly unraveled.

Bachelier et al. (1992) reported nil reciprocal effects in BB resistance as tetraploid upland cotton share a cytoplasm as that of its cytoplasm donors, *G. herbaceum* and *G. arboreum.* Nevertheless, exotic cytoplasm introgressed from distant *Gossypium* species will have its impact on cotton growth as well as responses to abiotic and biotic stresses. Despite of this fact, it has been noticed that nearly 12% of increased BB resistance in upland cotton when the *G. harknessii* cytoplasm was transferred (Mahill and Davis 1978).

The genetic basis of BB resistance was quantified using disease resistance rating scale by employing quantitative genetic designs such as F_2 and parents, generation-mean analysis (with parents and their F_1, F_2, and/or F_3, BC_1P_1 (i.e., $F_1 \times P_1$) and BC_1P_2 (i.e., $F_1 \times P_2$)) and diallel analysis (with F_1 and/or F_2) that were exposed to infections with *Xcm* 1, 2, 4, 7, 18 or 20, or a mixture of two or more races (Zhang et al. 2020a, b, c, d, e, f). It has been invariably found that resistant parents were harbouring one or more major B genes, whereas susceptible parents did not harbour such genes. Further, the heritability estimates of these studies were moderate to high (0.45–0.97; depending on crosses and studies). In general, additive effects were predominant; however, dominant effects were also reported. Consequent to the existence of residual heterozygosity in certain parents and non-uniform distribution of *Xcm*, heritability estimates of as low as 0.24 was reported for BB resistance under field conditions (Bachelier et al. 1992).

5.2.4 Resistance Breeding

Pyramiding of several BB resistance genes into the commercial *G. hirsutum* and *G. barbadense* were taken up during 1930–1950s in Sudan by Knight and his coworkers (Innes 1961, 1963, 1965, 1974, 1983; Zhang et al. 2020a, b, c, d, e, f). Similarly, during 1950–1970s, American cotton breeders, more specifically, scientists from Texas and New Mexico, have involved in evolving elite cultivars carrying one or more BB resistance genes and such cultivars have developed little or no disease symptoms during *Xcm* incidences. Thus, employing resistant cultivars with known BB resistance genes (such as BB resistant upland cultivars with the TAMCOT prefix and germplasm lines that were released from Texas A&M University (Bird 1976, 1979, 1982, 1986; El-Zik and Thaxton 1995, 1996, 1997; Thaxton and El-Zik 2004) and planting of acid delinted seeds were shown to be effective to control BB incidence during 1970s.

In the same way, during 1950s, cotton breeding for BB resistance were taken up at New Mexico State University (Zhang 2018a) and seven BB resistant cultivars (Acala 1517BR, Acala 1517BR-1, Acala 1517BR-2, Acala 1517-70, Acala 1517-77BR, Acala 1517-88, Acala 1517-95 and Acala 1517-99) were developed. The alphabets "BR" in the variety name indicates BB resistance). Both the cultivars, Acala 1517BR and its derivative Acala 1517BR-1, received their BB resistance genes from the donor parent Stoneville 20 (carrying B_7); however, they were resistant to BB race 1 but susceptible to race 2. On the other hand, Acala 1517BR-2 was resistant to both races and the resistance gene(s) to race 2 was/were donated by NM8738, which has Arizona Long Staple 120 (*G. barbadense*) genome in its background.

Acala 9136 was evolved from *G. barbadense* cv. Tanguis and it was used to evolve Acala 1517-70, which was resistant to races 1, 2, and 10. Interestingly, single plant selection in Acala 1517-70 have served to evolve several BB resistant lines. For example, Acala 1517-77BR and its cross-breeding line Acala 1517-88 were resistant to BB races 1, 2, and 10. It is also worth to mention that Acala 1517-95 was resistant to BB races 2 and 10 and Acala 1517-99 was resistant to race 1, 2, and 10, both of them have evolved their resistance from Acala 9136.

Owing to the widespread cultivation of second generation transgenic cotton since 2010, resurgence of BB was noticed in the US cotton belt. In order to combat this issue, the first generation of transgenic upland cotton cultivars with Bollgard (BG) and/or Roundup-Ready (RR) traits (for insect and herbicide resistance, respectively) were developed by backcrossing with BB-resistant conventional cultivars as recurrent parents. Despite of this effort, the disease turns out to be an issue, when replacing BG and RR cultivars with Bollgard II (BGII) and/or Roundup-Flex (RF) cultivars during 2007–2010 (Wheeler 2018). Consequently, BB triggered substantial losses in the southern US Cotton Belt (prominently in Arkansas and Mississippi, where Deltapine and Stoneville cultivars that were susceptible to BB were widely grown (Phillips et al. 2017). Therefore, almost all transgenic commercial cotton seed companies have recently designed their breeding program to incorporate BB resistance as

an important trait (Wheeler 2020). It has been reported that field screening with artificial inoculations has indicated 50–79% of the transgenic lines were BB resistant, depending on seed companies (Wheeler and Dever 2020). It has also been confirmed that 21 of 62 commercial upland cultivars from five seed companies, 11 of 150 elite breeding lines from New Mexico State University and 22 of 66 superior breeding lines from public cotton breeding programs have shown to contain different levels of BB resistance.

5.2.5 *Molecular Mapping of BB Resistance Genes*

The first report on quantitative trait locus (QTL) mapping of BB resistant genes B_2, B_3, b_6 and B_{12} was with restriction fragment length polymorphism (RFLP) markers using four F_2 populations derived by crossing susceptible *G. barbadense* Pima S-7 with four resistant upland lines: Empire B2, Empire B3, and Empire B2b6 and S295 (Wright et al. 1998). Totally seven QTLs were identified: QTLs corresponding to B_2 and B_3 on (chromosome) c20, B_{12} on chromosome c14, and four additional QTLs corresponding to the recessive b_6 alleles (b_{6a} on linkage group (LG) D02 (formerly LGU01), b_{6b} on c5, b_{6c} on c20, and b_{6d} on c14). It was indicated that the genomic region near the RFLP marker, pAR1-28 on c5, was mapped to homoeologous region, B_2 on c20. It was also speculated that this regions may be correspond to the BB resistance gene B_4 (that was identified in the diploid A genome species, *G. arboreum*) and assigned to c5 using cytological stocks.

A genome-wide association analysis using 330 US upland germplasm accessions and 26,345 single nucleotide polymorphisms (SNPs) derived from CottonSNP63K array was used to identify SNPs associated with BB race 18 resistance (Elassbli et al. 2021a, b). Among them, c5, c14 and c24 had the highest number of SNPs associated with BB resistance and those QTLs on c5, c14, and c20 were likely to be the QTLs reported by Wright et al. (1998). Thus, such studies have confirmed the consistent QTLs linked to the BB resistance in cotton germplasm.

Though it was originally hypothesized that CS50, an Australian resistant upland cultivar, possessed the $B_2B_3B_7$ and B_{Sm} genes on c20, later it was shown by making interspecific cross with susceptible Pima S-7 that the BB resistance was due to single dominant locus. It was also observed that such resistance locus was co-segregated with a RFLP marker on c14, which was associated with B_{12}, an African cotton cultivar's gene that confirms broad-spectrum resistance (Rungis et al. 2002).

Structural position and validation of B_{12} on c14 was also demonstrated by developing intraspecific upland cross consisting 285 $F_{4:5}$ families derived from Australian Delta Opal (which was resistant to BB race 18) and susceptible DP 388 (Xiao et al. 2010). Such effort has identified closely linked simple sequence repeat (SSR) markers (viz., CIR246, BNL1403, BNL3545, and BNL 3644), which had 5.6 cM distance to the resistance gene, B_{12}. Subsequently, this genomic region was further fine mapped with SNP markers (viz., NG0207069, NG0207155, NG0210142, and NG0207159) which had 3.4 cM to B_{12}.

Another study in Brazil has employed bulk segregant analysis using 127 F_2 population obtained from Delta Opal × BRAS ITA 90 and revealed that 80-bp SSR marker, amplified by BNL 2643, was linked to BB resistance in Delta Opal (Marangoni et al. 2013). Similarly, Silva et al. (2014) demonstrated that a 146-bp SSR marker amplified by CIR246 was associated with B_{12} in S295 and Delta Opal; besides it was also shown to be linked with other genes such as B_2B_3 (in 101-102B) and $B_{9L}B_{10L}$ (in Guazuncho-2). Accordingly, it can be concluded that B_{12} in S295 may be intimately associated with B_2B_3 locus, which in turn might be homologous to or co-segregates with $B_{9L}B_{10L}$. Therefore it was resolved that though CIR246 may be useful to identify resistance to BB race 18, but it cannot be used to differentiate gene(s) within the same chromosomal region that confirms BB resistance.

In an attempt to identify the candidate genes around the B_{12} region, Yang et al. (2015) described a 354-kb region containing 73 putative plant disease resistance genes by employing an interspecific F_2 population derived from S295 × Pima S-7 and the genome sequence of *G. raimondii*. Another similar attempt has delineated B_{12} gene and described few putative disease resistance genes using 550 multi-parent advanced generation intercross (MAGIC) lines and 500,000+ genotyping-by-sequencing based SNPs (Thyssen et al. 2019; Zhang et al. 2020a, b, c, d, e, f).

5.2.6 Marker-Assisted Selection (MAS)

As outlined above, CIR246, a breeder friendly SSR marker, reported by Xiao et al. (2010) has found to be helpful in cotton molecular breeding to evolve BB resistance cultivars using MAS. Xiao et al. (2010) have clearly showed that among the three alleles (146, 156, and 166-bp) amplified by CIR246, the cotton lines with the homozygous 146-bp alleles were found to be resistant; cotton genotypes with heterozygous alleles, 146-bp and 156-bp or 146-bp and 166-bp were found to be segregating for BB resistance; and homozygous cotton accessions with 156-bp allele (including Acala Maxxa) or 166-bp allele or heterozygous 156-bp and 166-bp alleles were found to be susceptible. It has also been demonstrated that the above resistant 146-bp allele was consistent with the SNP haplotype (ACTT) in the nine resistant lines. On the other hand, the susceptible allele, 155-bp or 165-bp, was consistent with the SNP haplotype (GGCA) in the nine susceptible lines.

Consistent of this SSR marker for its association with BB resistance has also further confirmed by Silva et al. (2014): CIR246 amplified the 146-bp allele in BB race 18 resistant lines S295 (carrying B_{12}), Delta Opal (carrying B_{12}), 101-102B (carrying $B_2B_3B_{sm}$) and Guazuncho-2 (carrying $B_{9L}B_{10L}$); alternatively 156-bp allele was amplified in BB race 18 susceptible lines Memane B1 (carrying B_2B_{sm}) and ST 2B-S9 (carrying B_{sm}). Similarly, 166-bp allele was amplified in susceptible Acala 44, deficient of known *B* genes. It was hypothesized that owing to outcrossing over generations and artificial development of advanced breeding lines as well as commercial cultivars, those alleles were no longer be homozygous for resistance.

Use of SSR marker CIR 246 (1.8 cM from B_{12}) and SNP marker NG0207155 (0.6 cM from B_{12}) for screening three Tanzania and four Brazilian Upland cultivars has revealed a differential allele frequency and resistance and showed that the cultivars were not homozygous at B_{12} (Faustine et al. 2015). Presence of 8–15% of water-soaked symptoms in the seedlings of resistant cultivars (such as PHY 375WRF, FM 1830GLT and FM 2484B2F) has also been described under greenhouse screening conditions. Thus, a vigilant inference on use of markers in evolving BB resistant cotton cultivars is also warranted while doing the MAS.

5.3 Verticillium Wilt

5.3.1 Causal Agent and Significance

A soil born fungal pathogen, *Verticillium dahliae* Kleb., causes a serious disease called Verticillium wilt (VW) in several cotton production countries and resulted drastic yield reduction. The first report on VW was noticed in Virginia during 1914 (Carpenter 1914) and now it is being reported in almost all the global cotton cultivating zones. With respect to its impact on defoliation, the strains of *V. dahliae* is grouped as two pathotypes: defoliating and non-defoliating (Schnathorst and Mathre 1966). It has also been grouped in to three categories with respect to vegetative compatibility based on complementation with auxotrophic nitrate non-utilizing (*nit*) mutants: VCG 1, 2 and 4 (Daayf et al. 1995). Besides, few reports such as Hu et al. (2015) have categorized it as race 1 and 2 based on its virulence. It is also interesting to note that VCG 1 found to be defoliating pathotype and race 2; on the other hand, VCG 2 and VCG 4 are non-defoliating pathotypes and race 1 (Daayf et al. 1995; Hu et al. 2015).

Primarily VW disease happens during the vegetative stage of the cotton as microsclerotia of *V. dahliae* invade the roots profusely and dwell in root vascular system (Fig. 5.2). Such invasion in root system seriously upset the uptake of water and nutrients from the soil, which ultimately lead to xylem discoloration, leaf necrosis, chlorosis and shedding, plant wilting and stunted growth and finally death of the plant. Therefore, there would be a significant fiber yield loss. It has been reported that cotton yield reduction to the tune of 0.5–3.5% in USA (Blasingame and Patel 2013) and 7.9% in other countries (Karademir et al. 2012; Hu et al. 2015) was due to VW. If VW develops during reproductive and boll setting stage, the fibre quality traits such as 50% span length and micronaire were found to be largely affected (Bell 1992, 2001; Zhang et al. 2012).

In order to control VW, several tested strategies such as crop rotation, judicial application of fungicides and use of endophytic and arbuscular mycorrhizal fungi have been proposed. However, evolving VW resistant cotton cultivars continues to be the most efficient and affordable method.

Fig. 5.2 Symptoms of Verticillium Wilt in cotton **a** Interveinal chlorosis and necrosis, **b** Dark brown streaks in the stem

5.3.2 Screening Techniques and Resistance Sources

In general, the cotton field that have shown severe VW symptoms can be selected for screening of newly evolved cotton lines as they serve as natural reservoir of the VW pathogen, *V. dahliae*. Indeed, such strategy has long been used to identify and release VW resistant cultivars in USA (Bell 1992).

Another strategy would be to successive cultivation of VW susceptible cultivars in the field that has been ear marked for VW disease resistance screening. By doing so, the field will be enriched with the *V. dahliae* inoculum; besides the field inoculum can also be supplemented with artificial inoculation or applications of crop residues and gin trash from heavily infected cotton plants. It has also been suggested that addition of excess nitrogen fertilizer with more irrigation water fasten the occurrence of VW incidence.

Bell (1992) opined that VW develops effortlessly when the boll setting stage coincide with mid-summer as there would be increased soil temperature that favours disease development. Therefore, scoring of VW disease severity would be better, if it collected during boll development stage. It should also be noted that short season or early-maturing cotton genotypes which has the capacity to produce heavy bolls during summer may show increased susceptibility when compared with late-maturing cotton genotypes (as they have more vegetative growth and less fruiting during this season). Consequently, when VW resistance rating is practiced at boll harvest stage, there would be a confuse in separating the pre-mature boll opening due to VW infection and early maturation of bolls in those short duration cotton genotypes. Hence, it would be better to avoid screening the cotton genotypes for VW resistance when the plants are in maturity phase (Bell 1992).

Usually, field VW resistance in cotton is decided by several factors including resistance genes, environmental features, inoculum density and host–pathogen interactions. Invariably, it has been realized that difficulty in maintaining high and uniform

level of pathogen inoculum in the field greatly limits the reliable screening for disease resistance. Further, changes in environmental factors and weather across the cotton growing seasons and locations affects VW screening procedure. In addition, genotype responses to the VW infection also varies. For example, individuals of the given genotypes, including susceptible cotton accessions, may produce different morphological features ranging from no symptom to severe disease symptoms. Such variations have also been reported even after artificial inoculation of *V. dahliae.* Consequently, it is suggested that field screening of VW resistance may be carried out on case by case i.e., on a population basis.

As expected, irrespective of cotton genotypes or pathogen strains, all the genetic evaluations reports indicated the quantitative inheritance of VW resistance. Thus, it is essential to test the cotton accessions, that were selected for field VW resistance, under replicated field trials and confirm its VW resistance across the seasons and locations. Therefore, it would be desirable to screen multiple entries of the genotype of the germplasm under field conditions across the season and locations with two or more replications in order to reduce the experimental error. Such effort would increase the reliability of process that favours the selection of germplasm line with sufficient VW resistance under field conditions. Besides, this will also facilitate the cataloguing of the germplasm lines according to their response to VW.

On the other hand, VW resistance screening under field conditions possesses the following limitations: There would be different races of the pathogen with changing distribution pattern, which make the field extremely heterogenous. As larger area is required to screen large number of cotton accessions with minimum two replications at boll development stage or maturity (over a period of five to six months), the field heterogeneity further increases. Requirement of large field area also generate additional challenges during crop, soil, and irrigation management practices, which may ultimately affect the reliability of VW resistance screening procedure. To circumvent these issues, artificial inoculation of *V. dahliae* under controlled conditions (such as greenhouses) have been proposed for screening VW resistance in cotton.

Artificial screening under greenhouse conditions involves inoculation of cotton roots (when the plants are in two to four leaf stage) with *V. dahliae* by employing root-dipping or root-ball techniques (Bell 1992). Cotton can also be grown in bottomless plastic or paper pots on a soil bed and roots are wounded when they are in young seedlings stage; further *V. dahliae* spore suspension is also irrigated to the root zones. In order to make it as an effortless and inexpensive procedure, it has been proposed to wound the roots before or immediately after root inoculation. Nevertheless, damaging the roots may neutralize the first line of defenses against VW infection in cotton. Therefore, it would not mimic the natural field screening resistance.

Artificial screening under greenhouse conditions can be effectively and simply performed by supplying conidial suspensions directly onto the surface of potted soil and it does not require root wounding. By maintaining optimal temperature conditions, it may take ~15 days to develop VW symptoms. Though identifying number of plants infected with VW symptoms can be easily done, it may not provide the disease severity (and thus it will not be useful in categorizing the germplasm as VW resistance). Alternatively VW disease severity can be collected

by using arbitrary scale of 0–5 (0 = no visible leaf symptoms (healthy plant); 1 = <25% chlorotic/necrotic leaves (mild to moderate leaf symptoms); 2 = 25–50% chlorotic/necrotic leaves and little defoliation (severe leaf symptoms); 3 = 50–75% chlorotic/necrotic leaves or defoliation; 4 = >75% chlorotic/necrotic leaves or up to 90% defoliation/terminal dieback/stunted plant; 5 = complete defoliation/stems dying back to ground level). Replications-wise scores have to be collected from each investigated cotton germplasm lines, which can be used to calculate the average VW severity rate as sum of rating × number of plants divided by the total number of plants evaluated (Zhang et al. 2012; Fang et al. 2013). Relative disease index (%) can also be calculated from disease severity rating on a 0–100% scale by calculating the ratio between the average rating and the highest rating (5), multiplied by 100.

Few alternative screening methods have also been reported for VW evaluation. For example, the soil infested with *V. dahliae* that were grown on wheat bran and corn meal or with microsclerotia (@10^3 microsclerotia g^{-1} soil) can be used for VW resistance screening. However, they were shown to be less effective when compared with root-dipping method. Bell (1992) proposed a stem-puncture method to inoculate *V. dahliae* suspensions (@ 2–3 × 10^6 mL^{-1} per plant) under both greenhouse and field conditions. Though such effort was labourious and lengthy, it conciliates the resistance mechanisms provided by the root. Therefore, visual symptoms of VW are more obvious in stem-puncture method when compared with root inoculation technique.

Alternatively other screening strategies such as culturing cotton accessions in a hydroponics system or in tissue culture and then subsequently inoculating with *V. dahliae* conidial suspension (@10^7 spores mL^{-1}) by 40 min of root-dipping have also been reported (Peng et al. 2008). It has also been proposed to culture the cotton seedlings or callus tissues in a plant tissue culture medium containing toxin isolated from *V. dahliae*. Though such strategies offer accelerated identification of VW resistance lines within a month period, such bioassay techniques are not commonly reported in the literature.

Though *G. barbadense* has shown highest level resistance to VW when compared with other three cultivated cotton species (Zhang et al. 2012, 2014a, b), the resistance mechanism has not been efficiently introgressed into commercial Upland cotton (Zhang et al. 2012). As majority of upland cotton breeding programs were conducted under non-VW infected fields, the lines evolved from these programs were shown to be susceptible to VW. Hence, it is essential to identify resistant upland cotton lines by evaluating under field (natural infections) and/or greenhouse (artificial inoculation) conditions. Such screening is a routine program since 2000 in USA, China and in other countries and commercial cultivars are now released only after screening for VW resistance (Wheeler and Schuster 2006; Wheeler and Woodward 2016; Zhang et al. 2012; Zhou et al. 2014). Since 1980, several national and provincial level field screening facilities have been established in China to evaluate cultivars and advanced breeding lines for VW resistance (Zhang et al. 2014a).

5.3.3 Resistance Breeding

Efforts in evolving novel cotton lines with VW resistance has been tried in the farms of New Mexico and California (Smith et al. 1999). Pedigree selection was used to release the first VW resistance upland cotton during 1940s and reselections from the Acala 1517 (which was commercialized in 1939) have identified VW tolerant Acala 4-42 and Acala 1517WR, which were released in California and New Mexico, respectively during mid-1940s. Subsequently, in 1960 the first VW resistant line Acala 1517D was commercialized in New Mexico. Additionally, in 1967, with substantial introgression of genetic elements from *G. barbadense* into AxTE-1, a new improved VW resistance line Acala SJ-1 was released in California. Subsequently, Acala SJ-2 was evolved through pedigree selection from Acala SJ-1 in 1973.

After 1970, cross breeding has been used as a major breeding strategy to evolve VW resistance cotton lines and varieties such as Acala 1517 V, 1517-70, 1517-75, and 1517-91 were released using this method in New Mexico. When Acala 1517-70 (which has a parentage of Hopcala with Tanguis (*G. barbadense*)) crossed with NM 8874 (which was derived from 1517 V) resulted into VW resistant Acala 1517-91. During mid-1970's–1990, Mexico VW resistant lines were used in California crossing program to evolve VW resistant Acala cultivars such as Acala SJ-3, Acala SJ-4, Acala SJ-5, SJC-1, GC-510, and Acala Maxxa. Further, realizing the importance in VW resistance in cotton cultivars, several other VW resistant cultivars were commercialized (to name a few: Kings M-5, GC-356, Acala Prema, Acala Royale, and DL 6166). At the same time, few cultivars such as Deltapine 20, Deltapine 50, Deltapine 51, DP Acala 90, Stoneville 495, Delcot 344, Paymaster 147, Paymaster 303, Paymaster 404, and Paymaster HS 26 shown to possess moderate resistance (Bell 1992; Smith et al. 1999). Using the breeding history of VW resistance Acala cultivars, Bell (1992) hypothesized that VW resistance might be derived from *G. barbadense*.

After 1995, transgenic insect resistant and/or herbicide tolerant cotton become widespread in USA cotton belt and it gradually replaced conventional cotton cultivars in subsequent years. Field and greenhouse experiments conducted by Zhang et al. (2012) confirmed that most of the Acala cotton cultivars had moderate levels of VW resistance; besides few of the transgenic cultivars commercialized by key seed companies in the USA also shown similar levels of VW resistance. Thus, Bell (1992) and Zhang et al. (2012) concluded that cotton breeding has made significant achievement in increasing or maintaining the resistant level to VW. Zhang et al. (2012) have shown the first line of evidence that backcross breeding can be used to transfer VW resistance from Pima S-7 parent (*G. barbadense*) to upland cotton.

Though several other cotton producing countries have also pursued breeding for VW resistance in cotton, recent publication on these efforts were rarely available. A comprehensive review made by Ma and Chen indicated that selections under natural field conditions (showing VW symptoms) during 1950–1970 resulted into the identification of VW resistant/tolerant cultivars such as Zhong 8004, Zhong 7327, Zhong 3474, Liaomian 5, and Shan 1155 in China. Similarly, cross breeding has also

been used to release CCRI 12 in 1987 in China. It is a high-yielding, highly resistant to Fusarium wilt and tolerant to VW cultivar and in 1990, it occupied 20% of the China's cotton area. Subsequently, cultivars such as Yumian 19, 86-6, BD18, Chuan 737, and Chuan 2802 were also released which had VW resistance (Ma et al. 1997, 2002). Though the genetics of VW resistance in CCRI 12 and Chuan 2802 were found to be controlled by major genes, it was found that these cultivars were shown to contain moderate resistance and hence VW is a most serious disease in China in certain seasons.

5.3.4 Genetics of Resistance

As *V. dahliae* race 2 (defoliating pathotype) is more virulent when compared with *V. dahliae* race 1 (non-defoliating), screening cotton lines for VW resistance generally employs race 1 under field or greenhouse conditions. Detailed review on genetic studies using segregating populations of F_2 or BC_1F_1 derived from interspecific or intraspecific crosses involving *G. barbadense* and upland cotton lines have been shown that VW resistance was qualitatively inherited (Zhang et al. 2014a). On the other hand, so far no major VW resistance gene has been reported using Mendelian segregation or QTL mapping with the help of molecular marker. However, Guo et al. (2016) and Zhang et al. (2016a, b) have shown marker-assisted selection to genetically improve VW resistance in cotton.

As there is no direct connection between a disease resistance gene (R) in cotton and an avirulence gene (*Avr*) in VW has been reported, it is speculated that cotton may have a quantitative trait to response to VW infection. Therefore, conventional as well as modern genomics for quantitative genetic analysis have been chiefly used to unravel the genetic basis of VW resistance in cotton (Zhang et al. 2014a).

As indicated by majority of the studies that were conducted on interspecific crosses derived from *G. barbadense* and *G. hirsutum* (Pan et al. 1994; Ma et al. 2000), a dominant or partially dominant gene was involved in VW resistance and allelic nature of the resistance genes from different sources of *G. barbadense* have also been established (Ma et al. 2000). It has also been shown in intraspecific hybrids of upland cotton that inheritance of VW resistance is more complex.

Thus, there is no concrete evidence (whether VW resistance is a qualitative trait controlled by one or two major genes or a quantitative trait regulated by minor polygenes, and whether the resistance is dominant or recessive) that supports VW resistance in cotton. Different resistance sources and homozygosity of genes in VW resistance, virulence and inoculum levels of pathogen, evaluation methods, environmental factors (especially soil temperature and moisture), and plant maturity level at the time of evaluation were found to confound to draw a solid inference on inheritance of VW resistance in cotton.

So far reports have supported resistance to VW in cotton was both qualitative (governed by single dominant gene) (Mert et al. 2005) and quantitative (conditioned

by multiple genes) trait (Roberts and Staten 1972; Wang et al. 2005). Addtionally, there are reports that supports the VW resistance is controlled by two or more dominant genes (Zhang et al. 2000).

There are also studies that provided evidence to state that VW resistance in upland cotton was controlled by dominant gene and two additive genes and the additive effects were vital in imparting resistance to VW. It has been shown that the cotton lines with both additive genes were resistant, whereas lines with only one of the additive genes displayed tolerance and lines with neither of the two genes were susceptible. By employing the progenies derived from VW-resistant cultivar Zhongzhimian KV-3 and VW susceptible cotton line KV9-1, similar kind of additive gene actions has also been reported.

However, in another study conducted by Jiang et al. VW resistance in upland cotton lines was controlled by two dominant major genes with additive-dominance-epistatic effects. This study was conducted on F_2 and $F_{2:3}$ progenies derived from the resistant upland cotton line 60,182 and susceptible upland cultivar Junmian 1 by inoculating with individual *V. dahliae* isolates BP2 (non-defoliating), VD8 (defoliating), T9 (defoliating) and a mixture of these isolates.

Reports have also supported that VW resistance in cotton is race or pathotype specific. For example, Pan et al. (1994) shown that the VW resistance in the progenies derived from *G. barbadense* × *G. hirsutum* was conditioned by a major gene when inoculated with a single *V. dahliae* race or strain; on the other hand, VW resistance was controlled by quantitative genes when inoculated with mixed races or strains. Similarly, Mert et al. (2005) reported that the VW resistance in upland cotton to a defoliating (D) pathotype was governed by a single dominant gene. However, dominant alleles at two loci were involved in imparting resistance to a non-defoliating (ND) pathotype. Such variations in the genetic studies have been attributed to the vigor, development and maturity of cotton plants and temperature, virulence and inoculum level of the pathogen (Bell 1992) which can affect their responses to VW infection.

Other quantitative genetic studies that were conducted on diallel crosses among 10 selected lines of upland cotton (Verhalen et al. 1971) and half-diallel design using five cotton cultivars Aguado et al. have also reported that VW resistance was a quantitative trait, and additive genetic variance component was more predominant in governing the resistance trait.

In contrast, recessive gene mediated VW also reported in few cotton genetic studies. For example, Roberts and Staten (1972) has concluded the VW resistance was recessive based on their investigation on F_2 and F_3 populations derived from 8229 × Lankart 57, 8076 × Lankart 57, and 8861 × Lankart 57. It should also be noted that this study has reported heritability which was varied from 0 to 0.83 depending on exposure level, generation tested, and type of parental tolerance. Similarly, generation mean analysis conducted on seven generations of a crosses (derived from VW resistant cultivar Acala SJC-1 with the susceptible lines S5971, Acala 4-42, and Deltapine 70) have indicated that more than one gene were involved in controlling VW resistance and the resistance was recessive (Devey and Roose 1987).

5.3.5 *Mapping of Verticillium Wilt Resistance QTLs*

Zhang et al. (2015a) have documented a total of 193 QTLs linked to VW resistance in cotton from meta-analysis 13 published reports, which comprised seven early segregating populations (F_2, BC_1S_1 and $F_{2:3}$) and five recombinant inbred lines (RILs) and backcross inbred lines (BILs) including chromosome segment substitution lines (CSSLs) and one association mapping (AM) panel. Similar kind of meta-analysis has reported 201 QTLs for VW resistance (Abdelraheem et al. 2017).

Recent studies have actually reported additional QTLs (more than 400) associated with VW resistance. For example, Palanga et al. (2017) found 119 QTLs using 196 intra-upland RIL population that were tested in five environments. Similarly, Shi et al. (2016) found 48 QTLs using BC_1F_1, BC_1S_1 and BC_2F_1 derived from an interspecific *G. barbadense* × *G. hirsutum.* By employing early segregating biparental populations, main-effect QTLs and QTL hotspots for VW resistance have also been reported (Zhang et al. 2014a, 2015a; Abdelraheem et al. 2017; Zhao et al. 2018).

Several studies (Zhao et al. 2014, 2017, 2018; Baytar et al. 2017; Li et al. 2017; Sun et al. 2019) have also used AM panel that comprises different cotton germplasm lines to identify VW resistance genomic regions. Totally, 26 SSRs were found to be associated with VW resistance in a study conducted at Turkey that used a panel of 108 elite upland germplasm lines and 967 SSRs (Baytar et al. 2017). In another study conducted at China using a panel of 158 upland lines, Zhao et al. (2014) documented that 42 out of 212 SSRs were strongly linked to VW resistance. In an extended effort by the same crew, 192 SNPs were found to be linked to VW resistance out of 18,726 SNPs (Zhao et al. 2017). They have further shown that three clustered QTLs on D09 (c23) is likely to be the main-effect QTL VW-D09-1 reported by Ning et al. (2013).

Consistent QTL (on chromosome A10 (c10)) across three environments for VW resistance along with 16 other QTLs have been reported by Li et al. (2017) using genotyping-by-sequencing (GBS) based SNPs. They have also shown that the gene, TIR-NBS-LRR on A10 would be a likely candidate gene that confer VW resistance in cotton. Similarly, other candidate genes such as *GbTMEM214* and *GbCYP450* on chromosome D04 (c22) would be representing two main-effect VW resistance QTLs (Zhao et al. 2018). This study has employed QTL mapping that used a segregating population derived from a CSSL and its recurrent parent and virus induced gene silencing (VIGS). Though there are VW responsive genes were identified (Li et al. 2017; Shaban et al. 2018), they were not shown to have direct genetic relationship with any VW resistance genes or QTLs that have been reported so far.

Identification of consistent QTLs linked to VW resistance by testing same segregating plant materials across the environments and seasons have been reported (Fang et al. 2013, 2014; Zhang et al. 2014a; Li et al. 2017; Palanga et al. 2017; Zhao et al. 2017). Similarly, QTLs that were consistently identified across segregating populations that employed different parents were also documented (Zhang et al. 2015a; Abdelraheem et al. 2017). Despite of this effort, owing to use of relatively small mapping populations (usually 100–200 progeny) and a low number of markers (usually 200–300), QTL mapping resolution was low. Alternatively, it is proposed to

use high-throughput sequencing- or chip- based SNP markers and a large mapping population to fine map the identified QTL regions. Such effort will not only enable the location of candidate genes that governs the natural variation in VW resistance but also will help to clone VW resistance genes.

5.3.6 Marker-Assisted Selection for VW Resistance

The first report on MAS for introgression of VW resistance was by Du et al. and they used the SSR marker, BNL 3556, on c8 or A02, which explained 50% of the phenotypic variance for VW resistance. Nevertheless, this marker was withdrawn and a new SSR viz., BNL 3255_208 on the same chromosome was found to be linked with the VW resistance gene at a genetic distance of 13.7 cM. Further studies on this marker established that it was found in 85–89% of the interspecific F_2 or BC_1F_1 progenies that have shown VW resistance.

Among the 18 SSRs linked to VW resistance, Kong et al. (2010) reported that three SSRs (viz., BNL1721, BNL2733, and BNL3452) on c8 were useful to select cotton progenies with VW resistance. Similarly, Wang et al. documented two VW resistance QTLs on c16 and reported that two SSR markers (NAU 751 and BNL 195) were useful in increasing VW resistance; especially when both the markers were used, the resistance level was increased. Li et al. (2013) has surveyed 39 SSR markers linked to VW resistance among the progenies derived from a composite cross and its reciprocal cross made in upland cotton. Such effort has concluded that BNL 3241 (on c17), NAU 1225 (on c5 or c19), NAU 1230 (on c5 or c19), JESPR 153 (on c1 or c18), and BNL 3031 (on c9 or c23) were substantially enhanced VW resistance and selection of progenies that has two marker combinations, especially JESPR 153 and BNL 3031 were found to be superior in VW resistance. In another interesting study, Qi et al. shown that SSR markers NAU 1225, NAU 828, and NAU 1269 (all on D5) generated specific PCR products that can discriminate VW resistant and susceptible cultivars.

Thus, a large array of reports on QTLs linked to VW is available for MAS. However, MAS has not yet been shown as an effective procedure to introgress the VW resistance in routing cotton breeding program due to several limitations including validating the markers for their utility. For example, though c8 carries VW resistance, use of those flanking SSR markers in MAS provided variation in resistance level when it was used in interspecific and intraspecific crossings (Kong et al. 2010). Similarly, the usefulness of the two SSRs on c16 and other five SSRs reported by Li et al. (2013) yet to be validated for MAS towards genetic improvement of VW resistance.

5.4 Fusarium Wilt

5.4.1 Causal Agent and Significance

Another most serious disease in cotton caused by caused by *Fusarium oxysporum* f. sp. *vasinfectum* Atk. Sny & Hans (FOV) is Fusarium wilt (FW), which is a soil-borne fungal disease. This disease occurs almost in every cotton growing areas in the world and it attacks young roots. Especially, the vascular systems of stems and roots are heavily infected with fungus toxic compounds which limits the uptake of water and nutrients and lead to xylem discoloration, leaf necrosis/chlorosis/shedding (Fig. 5.3), stunting, wilting and finally heavy yield losses (Zhang et al. 2015a, b, c; Sanogo and Zhang 2016).

FW symptoms development is heavily dependent on genotype, the pathotype and inoculum load, environmental factors such as temperature, moisture and nematodes. For example, the FW pathotype, California isolate FOV4, develops symptoms independent of nematodes whereas other pathotypes need heavy fungal inoculum and nematode infection that cause root wounding. It has also been reported that crop management practices including planting date and application of fertilizers also influence the FW occurrence. In USA alone, annual cotton yield loss in cotton due FW was reported to be in the range of 0.19–1.36% (Blasingame and Patel 2013).

Globally eight pathogenic FOV races have been reported and among them races 1, 2, 3, 4 and 8 were identified in the cotton belts of USA (Kim et al. 2005; Holmes et al. 2009; Cianchetta et al. 2015). Other races were reported in other parts of global cotton growing regions: race 3 in Egypt and Israel, race 4 in India, race 5 in Sudan, race 6 in Brazil, and races 7 and 8 in China (Armstrong and Armstrong 1960; Hillocks 1992; Davis et al. 2006; Sanogo and Zhang 2016; Bell et al. 2017). As the recent molecular evidences cannot distinguish the races 3 and 5 as well as races 4 and 7, the FOV race classifications were recently revised (Davis et al. 2006; Halpern et al.

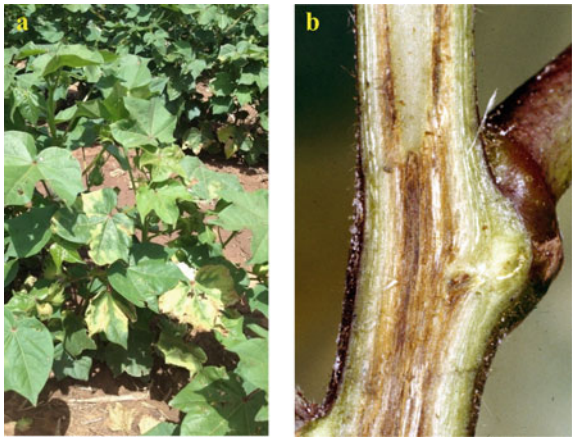

Fig. 5.3 Symptoms of Fusarium Wilt in cotton **a** Plants with yellowing leaves and **b** Stem showing browning symptoms in vascular system of the cotton stem

2020). During the recent past decade, it has been found that FOV race 4 has become a serious threat to cotton productivity in the west and southwest US Cotton Belt (Halpern et al. 2018; Zhu et al. 2020a, b).

5.4.2 Sources of Resistance

As that of VW, screening for FW resistance in cotton can be conducted using both greenhouse and field experiments. The cotton fields showing heavy infestation of FOV due to continuous cropping of susceptible cotton cultivars can be employed to select cotton accessions having FW resistance. Such kind of field has been effectively established since 1952 in the National Fusarium Wilt Nursery in Auburn University at Tallassee, AL. In order to increase the inoculum load, the crop residues of heavily infected cotton plants may be ploughed back in to the soil. Employing such kind of natural field conditions have the advantages of subjecting the testing cotton accessions under a set of physical and biological environments that favours the FW. However, it will be difficult to maintain uniform inoculum load throughout the field and hence each investigated cotton line may be exposed to different levels of FOV inoculum. Screening under natural field conditions may also experiences the other limitations such as presence of mixed FW pathotypes, plant infection with other soil pathogens, and variable soil and crop conditions which may lead to experimental errors.

In order to assess the stability of the genotype for its resistance to FOV, each genotype has to be tested across the locations and seasons with multiple replications. Evaluation of the genotypes under controlled greenhouse conditions offers unique advantages such as replicated screening of large number of genotypes within limited space and effective control on specific inoculum type and dose, method of inoculation, and phenological stage of the plant.

First report on FOV infection was documented by Atkinson in USA and it was reported that variation in response to FW (specifically to race 1) among the cotton cultivars. Such variations were invariably found in both upland and pima cotton and hence elaborate FW resistance breeding efforts were taken up. As a result, FW resistance in the newly evolved cultivars were documented but with poor yield. For example, though Auburn 56, Auburn M, and Dixie King II were shown resistance to FOV consequently for four years, they possess poor yield. Similarly, though Coker 310 and Stoneville 603 were high yielding with good fiber quality, they have moderate resistance to FW. Results on the field screening at the National Fusarium Wilt Nursery in Auburn University at Tallassee, AL for FOV-RKN disease complex have been published by Verticillium Wilt and Fusarium Wilt Committee in various issues of the Proceedings of Beltwide Cotton Conference or special reports from the Alabama Agricultural Experiment Station.

Report summarized by Kappelman indicated that among 2208 cotton cultivars and lines evaluated in the National Fusarium Wilt Screening Test, Tallassee, AL, between 1967 and 1974, few of them were promising. Interestingly, Auburn 623

RNR, Auburn BR1 and Auburn BR2 were found to be resistant consequently for six years of experiments. Similarly, Dixie King II, Auburn 56, Auburn M and Rex Smoothleaf-66 exhibited FW resistance for eight successive years, and McNair 511 and Delcot 277 were found to be tolerant to FW for four years. However, few of them have exhibited intermediate tolerance (for example, Deltapine 25, Coker 312, Coker 310 and Dixie King III) during two to five years of investigation.

In general, lines developed after 1965 had higher FW resistance than the previously released cultivars and further genetic improvement of FW resistance was made in breeding lines after 1975. Kappelman documented that Reba B-50, a commonly grown cultivar in Paraguay, was highly resistant to FW. Another report by Kappelman indicated that Deltapine 55, McNair 220, Delcot 311, Rex 713 and Stoneville 603 were highly tolerant to the FOV-RKN complex, but none was as resistant as the Auburn BR2 check. Both under field and greenhouse conditions, several commercial and experimental cotton materials were screened for FOV race 4 in California, New Mexico and California (Hutmacher et al. 2013; Zhang et al. 2020a, b, c, d, e, f).

Extensive screening of large set of cotton germplasm lines (over 3700 accessions) against FOV race 7 have been conducted in China and reported that 0.62% were immune, 4.42% highly resistant, and 5.04% resistant. It is generally believed that *G. arboreum* was extremely resistant to FOV. However, few upland cotton cultivars that have shown their resistance against FW were collected from naturally infected fields and used as donors in cotton biotic stress resistance breeding.

In another report, which published after a decade, it was highlighted that among the 1211 cotton accessions tested for FOV race 7 resistance, 0.25% were immune and 9.66% were highly resistant. It has also been shown that primitive landraces of upland cotton found to be resistant to race 1 and race 7. After 1980, screening the cotton prebreeding materials at national and provincial level against FW as well as VW have become a regular activity.

Variations in FW resistance among cultivated diploid Asiatic cottons have also been documented. Evaluation of two cultivated diploid species (*G. arboreum* and *G. herbaceum*) in India for FW (race 4) resistance has indicated that though *G. arboreum* was not shown immunity, LD 212, LD 224, LD 231, LD 252, LD 254 and LD 258 were exhibited resistance with lesser (2.2 to 7.3%) disease incidences. In another report by Leena and Kshirsagar, it has been indicated that among the 12,983 diploid genotypes (*G. arboreum* and *G. herbaceum*) screened at the AICCIP center at Pune, India, 979 genotypes were found to be resistant with 0.01–10.00% disease incidence and 1,205 genotypes exhibited moderate resistance with 10.01–30.00% disease incidence.

Similar kinds of large-scale screening for FW resistance have been conducted in other global regions including the former Soviet Union, Uganda, Sudan, Tanzania, Egypt, Peru, Brazil, Argentina, Australia (Hillocks 1992). *G. barbadense* lines including Bahtim 185, and Giza 67 were found to be resistant to FOV (race 3). In Sudan, screening has indicated that few upland cultivars exhibited no resistance to race 1; however, they were almost immune to race 5. Similarly, totally 10 *G. barbadense* were found to be resistant to race 1 and they have also shown mixed

reactions to race 5 (i.e., response was ranged from highly susceptible to highly resistant).

Experiments with cotton wild species indicated that *G. australe* Mueller and *G. sturtianum* Willis have also shown mixed responses to a highly virulent FOV strain (race not classified). They have also shown that none of the cotton lines have shown resistance to this FOV strain.

5.4.3 Resistance Breeding

Cotton accessions that have shown FW resistance when they had grown in FOV—infested soils were selected and they formed the earliest FW resistant cotton cultivars. Later, in 1920–1930's several other resistant cultivars including Cook 307-6, Wilds, and Coker Clevewilt were evolved and they occupied huge cotton growing zones of Southeast USA. When Wilds was crossed with Coker 100-Wilt, it resulted into Coker 413 and its reselection Coker 421. In Central African Republic, several FW resistant cotton cultivars were developed using Coker 100-Wilt as FW resistance source. Similarly, Cook 307-6 was used as resistance source to evolve Empire, Delcot 277, Hartsville, and McNair 220. Auburn 56 was developed by crossing Cook 307-6 and Coker Clevewilt and further reselections resulted into release of Auburn M, McNair 511, and McNair 1032. Interestingly, Auburn 56 has also been employed to develop RKN resistant cultivars such as Auburn 634RNR in USA and FW resistant cultivars in Brazil. As infection by RKN enhance the chance of FW development, breeding for RKN resistance in cotton would increase the possibility of developing FW resistance or at least reduce the FW disease severity.

Despite of these efforts, earlier attempts in evolving FW resistant cultivars usually have poor agronomic trait expressions and low yield in the fields that has shown no FOV inoculum. At this juncture, the first cotton line that has both wilt resistance and good agronomic traits was Coker 100 Wilt which was commercialized in 1942.

Though it is cumbersome to combine both wilt resistance and desirable agronomic properties, there was a slow progress in genetic improvement cotton for improved FW resistant, several cotton cultivars with FW resistant and good quality have been developed since 1967. Later, host plant resistance mechanisms were utilized in development of cotton for FW resistance in public cotton breeding programs conducted in Arkansas, Louisiana, Alabama, New Mexico and Texas (Bourland 2018).

Commercial cotton seed companies always focus in introgressing FW resistance in their cultivars or hybrids and it can be exemplified with current commercial cultivars. As new virulent strains are evolving recently, such as FOV race 4 in California, New Mexico and Texas, molecular breeding has addressed this problem and released FOV race 4 resistant transgenic Pima cotton cultivars. On the other hand, available current commercial upland cultivars are susceptible to FOV race 4.

China has the most successful and extensive breeding programs for FW resistance. Upland cotton was first introduced from the USA for direct commercial production in the early 1900s until 1950s. In the 1950s, selection for FW resistance in

heavily FOV-infested production fields resulted in the development of highly resistant Chuan 52-128 from Delfos 531 and Chuan 57-681 from Deltapine 15 in Shichuan Province. Chuan 52-128, Chuan 57-681 and introgression lines with resistance from *G. arboreum* have become the major sources of FW resistance for the development of more than one hundred of FW-resistant cultivars in China (Feng et al. 1996b). In the 1960s, of the 13 FW resistant cultivars released, the most notable was the development of many FW resistant cultivars including Shaanmian 4, Shaanmian 112, Shaanmian 401, Shaanmian 5245, Shaanmian 3563, and Shaanmian 65-141 by using these two resistant sources in cross breeding in Shaanxi Province. These in turn gave rise to many wilt resistant cultivars including 86-1 and Lukang 1 in the 1970s (of a total of 29 FW resistant cultivars released) and Jimian 7 (of 48 wilt resistant ones released) in the 1980s. In the 1980s, however, the most significant progress was the release of Zhongmian (CRI) 12 in 1986, derived from a Uganda cotton × Xingtai 6871, which in turn gave rise to more than 40 cultivars in the 1990s. The Uganda cotton was first introduced from the U.S. in the early twentieth century with origin from Allen and Sunflower. Selections and breeding produced the long staple Uganda Bukalasa Pedigree Albar (BPA) in production and short to medium staple Serere Albar Type Uganda (SATU). Xingtai 6871 was a selection from Xuzhou 1818 which was also selection from Xuzhou 209, and Xuzhou 209 was directly selected from Stoneville 2B. Therefore, CRI 12 and Chuan 57-681 (from Deltapine 15) shared Stoneville 2B and Allen in their origins, so both understandably shared the same FW resistance gene *Fw2* based on an allelic test (Feng et al. 1998). Since CRI 12 combined high yield with high resistance to FW, tolerance to VW and wide adaptations, it continued to be the single most dominant commercial cultivar until the mid-1990s when more than 20% of cotton acreage in China was grown to this cultivar. In the 1990s, more than 100 wilt resistant cultivars were released including Zhongmian 24, 27, 35, 36 and 41, and Yumian 19. In the 2000s, numerous FW resistant cultivars and hybrids have been released for commercial production.

In China, improvement of FW resistance is one of the breeding objectives in almost all the cotton breeding programs. Presently, most released cultivars carry FW resistance. The cotton acreage in China grown with FW resistant cultivars was increased from 44% in the early 1990s to more than 80% in the early 2000s. FW is no longer a problem in cotton production in China. Based on the National Cotton Variety Tests on FW and VW resistance over 16 years (1973–88), 74 wilt-resistant cultivars were identified and 24 of them were released for commercial production. However, a pedigree analysis indicated that majority of the FW resistant sources were from Delfos 531, Deltapine 15, *G. arboreum* and Uganda cotton (Feng et al. 1996b). The narrow genetic basis was confirmed by Wen et al. and Wang et al. based on SSR and AFLP markers on 54 Chinese germplasm with FW and VW resistance.

Many other cotton-growing countries such as Argentina, Australia, Brazil, Egypt, Israel, Sudan, Tanzania, and Uzbekstan also have had successful breeding programs for enhancing resistance to FW in Upland and *G. barbadense*. For example, FW was a serious problem in India in *G. arboreum* and *G. herbaceum*, but it is controlled by the release and use of many FW resistant cultivars including Jirila, H420, Suyog, G. Cot 11, and G Cot 15.

5.4.4 Genetics of Resistance

Studies on genetic analysis of FW resistance using Mendelian and quantitative genetic approaches have shown that it was both a qualitative and a quantitative trait (Zhang et al. 2015a). Initial attempts on genetic studies in response to FOV were conducted under natural field conditions as there is no proper screening techniques under greenhouse conditions. In 1930–40s, it has been established in India that resistance in *G. arboreum* to race 4 was controlled by two complementary dominant genes and a third inhibitory gene; however, in *G. herbaceum* it was by one dominant gene. Fahmy indicated that in Egyptian *G. barbadense,* the resistance to race 3 was governed by one dominant major gene with few minor genes. In another study conducted in segregating populations of a cross derived from susceptible Pima S-5 (*G. barbadense*) and the resistant upland cultivar Acala SJ-2 at Israel disclosed that the resistance to race 3 was determined by a dominant gene. Similarly, Gridi-Papp et al. and Zhang et al. (2015a, b, c) have shown in Brazil that field resistance to race 6 in IAC 17 cultivar was due to a major gene which was derived from Auburn 56.

Studies in F_2 and BC_1F_1 derived from resistant upland cultivars Empire, Delfos 425 and Cook 307–6 in USA highlighted that a major dominant or recessive gene involvement in resistance development to race 1 or 2. In Sea-Island Seabrook (*G. barbadense*), it was established that the resistance to race 1 or 2 was determined by two dominant genes with an additive effect. A major dominant gene (*FOV1*) which provide resistance to race 1 in Pima S-7 (*G. barbadense*) has been identified under greenhouse conditions and mapped to c16 or D07 using SSRs (Wang and Roberts 2006; Ulloa et al. 2011).

Similarly in another study by Ulloa et al. (2013), a major resistance gene (*FOV4*) to race 4 was identified in Pima S-6 and mapped to c14 or D02 using SSRs. Though wwo major RKN resistance genes were mapped on c14 or D02 (Niu et al. 2007; He et al. 2014), it is not yet revealed that whether *FOV4* and one of the two RKN resistance genes are the same or different (Wubben et al. 2019).

Feng et al. (1996a) in China have employed several upland cotton germplasms to study the genetics of FW resistance to race 7 and reported that a major dominant resistance gene was involved in four resistant lines including CRI 12. Additionally, it has further been shown that the resistance gene (named as Fw_1) in Chuan 52–128 was allelic or same as that of Shaan 1155 and 86–1 resistance gene; similarly, the resistance gene (named as Fw_2) in Chuan 57–681 was allelic or same as that of CRI 12 (Feng et al. 1998).

Another effort in China using two $F_{2:3}$ populations derived from resistant CRI 35 and Sumian 10 have shown that a dominant resistance gene (Fw^R) on c17 or D03 was involved in FW resistance (Wang et al. 2009). However, the inheritance of this gene and the other two genes (Fw_1 and Fw_2) reported by Feng et al. (1998) is yet to be established. SSRs were also used to map a single dominant resistance gene on c21 or D11 using progenies derived from susceptible Xinhai 21 and highly resistant HK 237. Hence, it can be concluded that the two major resistance genes for race 1 (mapped on D07 from Pima S-7) and race 4 (located on D02 from Pima S-6) might

be dissimilar to two major resistance genes identified for race 7 (which were mapped as one each on D03 from Upland and on D11 from *G. barbadense*). Du et al. (2018) have reported a main-effect QTL on c11 or A11 for FOV7 resistance using GWAS that employed 215 Chinese *G. arboreum* accessions. They also identified a candidate gene *Ga11G2353* encoding for a glutathione S-transferase in that region.

Apart from these efforts, quantitative inheritance of FW resistance in cotton has also been shown in several occasions. In general, FW resistance was halfway in F_1 when compared with parents and displayed a continuous variation among the progenies irrespective of F_2, F_3, and BC_1 populations. FW resistance to FOV race 1 or 2, it has been shown that there may 2–3 major resistance genes. Additive effects with greater degree were usually reported than those of dominance or epistasis. Wang et al. have confirmed by employing generation-mean analysis that significant additive effect to FOV7 resistance was exist, whereas dominance and epistatic effects were also found in a few crosses.

Nan has shown the additive variance of 0.348 (i.e., narrow-sense heritability) for phenotypic variation of FW disease index caused by FOV7 using a RIL population from a cross of HS46 × MARCABUCAG8US-1-8. Additive effects on FOV resistance have also been investigated in *G. arboreum* (to race 4) in India, *G. barbadense* (to race 3) in Egypt, and *G. hirsutum* (to race 7) in China (Xiao 1985, 1988, 1992; Zhang et al. 1994, 1995; Feng et al. 1996a, b, c, 1998). These studies were consistently shown that additive variances (general combining ability) were greater than dominance variances (specific combining ability) in several investigations; however, narrow-sense heritability estimates were varied from 0.2 to 0.9 which were due to different diallel crosses and evaluation conditions used in those studies.

5.4.5 Molecular Mapping of FW Resistance Quantitative Trait Loci

Both biparental and association mapping strategies were applied to identify QTLs linked to the FW resistance developed by FOV races 1, 4 and 7 as well as Australian FOV (for a review, see Zhang et al. 2015a). In another attempt, Zhang et al. (2015b) have utilized meta-analysis of six different mapping studies that were conducted with different types of segregating populations including RILs and declared 33 QTLs associated with FW. Abdelraheem et al. (2017) also employed meta-analysis and reported 47 QTLs related to FW resistance in cotton. They have noticed that most of those QTLs were found on five chromosomes viz., c6 (A06), c14 (D02), c17 (D03), c22 (D04), and c25 (D06).

Wang et al. (2018) used 367 SSRs to genotype RILs derived from an interspecific cross (Pima S-7 (resistant to FOV race 1 and susceptible to FOV race 4) × Acala 991 NemX (susceptible to FOV race 1 and tolerant to FOV race 4)) and located six major QTLs on c1 (A01), c2 (A02), c12 (A12), c15 (D01), and c21 (D11) linked to

resistant to FOV race 1 and two major QTL on c14 (D02) and c17 (D03) associated with resistant to FOV race 4.

Abdelraheem et al. conducted GWAS in USA upland cotton germplasms using 25,677 SNPs derived from CottonSNP63K array and reported 13 QTLs for FOV race 4 resistance on six chromosomes viz., c8 (A08), c14 (D02), c17 (D03), c19 (D05), c16 (D07), and D13. In another study, the same team has utilized 163 RILs derived from FOV race 4-resistant *G. barbadense* Pima S-6 and susceptible 89,590 using 403 SSRs and identified seven QTLs for FW resistance (c14 (D02), c17 (D03), c19 (D05), c25 (D06), c24 (D08) and c21 (D11) under greenhouse conditions.

Thus, it has been concluded that though there were different QTLs identified for various races (1, 4, and 7) and an Australian strain of FOV, the major QTLs for FW resistance were found to be localized in only few chromosomes such as c4 (A04) and c20 (D10) (Zhang et al. 2015a, b; Abdelraheem et al. 2017).

It is obvious that the cotton lines that are resistant to different FOV races might be susceptible to the Australia FOV strains. However, there are multiple sources for the FW resistance such as MCU-5. It has been shown that the major FW resistance gene(s) in MCU-5 would be different from those resistant genes found in upland cottons of China and *G. barbadense* (which were resistant to race 7), American Pima S-6 (resistant to race 4) and Pima S-7 (resistant to race 1).

Though common and consistent QTLs across the biparental mapping populations, environments/locations and seasons were reported (Zhang et al. 2015a, b; Abdelraheem et al. 2017), such QTL detections were hindered by the genetic variation contributed by the two parents used in the mapping population development. Further, use of small numbers of molecular markers (100–300) result into poor genome coverage and thereby led to lower resolution in QTL detection process. Though recent developments in sequencing- or chip- based SNP markers have increased the availability of large numbers of SNPs, better increased size of mapping population (such as >500) is required to identify the finely mapped QTL (which has flanking markers with a distance of <0.1 cM. Identification of such fine mapped QTLs allows documentation of candidate genes underlying the natural variation in FW resistance as well as the cloning of FW resistance genes.

5.4.6 Molecular Breeding Techniques

Biochemical assays have helped to identify secondary metabolites as well as enzymatic activities that were involved in FW resistance. For example, electrophoretic mobility of polyphenol oxidase (or catechol oxidase) obtained from germinating seeds of upland cotton cultivars that differ for FW and VW resistance showed that cultivars that has higher level of resistance to FW and VW possess more and stronger polyphenol oxidase bands than susceptible lines and they have shown that selection of breeding materials based on this biochemical assay actually speed up the selection process with lowest cost.

Plant tissue culture technique also favours the selection of FW resistant cultivars as shown by Wang et al. They indicated that the growth of resistant cultivars was less affected by the presence of crude extracts of FOV and resistant lines developed more proficient calli even after several incremental increase of the FOV inoculum over generations of subculture. In another attempt, the FOV toxin (fusaric acid) was employed to screen calli from FOV treated explants of Coker 201, and identified FW tolerant cell lines which was then completely regenerated into cotton plants (Zhang et al. 1994). Ganesan and Jayabalan regenerated plantlets from somatic embryos that were cultured on medium mixed with 40% FOV fungus culture filtrate (FCF), which were isolated from embryogenic calli cultured on 5–50% FCF callus induction medium. Such effort has helped to identify regenerated plants with FW tolerance when those plants were grown in pots inoculated with 1×10^5 spores mL^{-1} of FOV.

In spite of the successful progress in identifying DNA markers linked to FW resistant genes or QTLs with major effects, utilization of these markers in MAS has not been reported in cotton biotic stress resistance breeding. On the other hand, genetic engineering approaches have been tried to impart FW resistance in cotton. Exogenous DNA was introduced into susceptible cotton cultivar via called pollen tube pathway and generated FW resistance. Specifically, single resistance gene from the resistant cultivar Chuan 52-128 was transferred into the ovaries of the FW susceptible upland cultivars Jiangsu 1 and Jiangsu 3 and two promising resistant lines viz., 3049 (from Jiangsu 3) and 3072 (from Jiangsu 1), were generated.

Similarly, Cheng et al. transformed cotton with genes that code for *chitinase* and *β-1,3-glucanase* (which are involved in the plant defense system with synergistic effects) through the pollen-tube pathway method. Screening of the transgenic plants under greenhouse and wilt-infected field nurseries in successive four years helped to identify transgenic cotton lines with improved FW and VW resistance. Ganesan and Jayabalan also highlighted that transfer of chitinase resulted into enhanced resistance to FW as well as to *Alternaria macrospora.* Further transgenic development in upland cotton cultivar SVPR2 with *chitinase* II under the control of the CaMV 35S promoter exhibited resistant to FW and Alternaria leaf spot. It has also been shown by Emani et al. that transgenic cotton developed with cDNA that encode 42 kDa *endochitinase* from the mycoparasitic fungus, *Trichoderma virens*, has exhibited substantial resistance against a soil-borne pathogen, *Rhizoctonia solani* and a foliar pathogen, *Alternaria alternata.* However, its responses to FW were not documented.

Non-expressor of pathogenesis-related (*NPR*) genes usually involved in plant defense system. Parkhi et al. obtained *Arabidopsis thaliana NPR1* gene and transferred to cotton and documented the transgenic cotton's resistance against FW, VW, *Rhizoctonia solani*, *Alternaria alternata*, and reniform nematodes. Similarly, Lei et al. documented that the transfer of *SNC1* (*suppressor of npr1-1, constitutive 1*) from *A. thaliana* into upland cottons, Zhongmian (CRI) 35 and Junmian 1, has enhanced FW resistance when the transgenics were exposed to FOV by the root-dipping method. Gaspar et al. has reported the effectiveness of the plant defensin *NaD1*, from *Nicotiana alata*, against FW and VW in transgenic cotton.

Kristyanne et al. indicated that the antifungal activity of magainin 2 (obtained from the skin of the clawed frog *Xenopus* laevis) on five species including FOV.

Rajasekaran et al. shown that expression of synthetic *D4E1* in transgenic upland cotton plants inhibited FW and VW development. Besides, these transgenic seedlings also developed lesser disease symptoms of black root caused by *Thielaviopsis basicola,* synonym. *Chalara elegans.*

5.5 Future Prospects

Primary and secondary metabolites produced by cotton against environmental stresses including disease causing pathogens helps them to combat the ill-effects caused by these pathogens. Production of these metabolites are usually regulated by gene expression especially at transcriptional and post-transcriptional level. Identification and characterization of these genes and their alleles through genomics assisted allele mining strategies will be useful to unravel the molecular mechanisms involved in cotton disease resistance. Besides such efforts would be used to conserve the cotton biotic stress resistant germplasm resources. Ultimately these efforts would be useful to design a sound molecular breeding strategy that focus the genetic improvement of cotton for improved disease resistance.

Recent developments have indicated even the complex agronomic traits can be manipulated by simply generating point mutations or single base changes in a certain gene. Such small nucleotide changes can be easily executed with gene editing technology which uses artificial engineered and modified nucleases (such as ZFN and CRICPR/Cas9). Genome editing has shown to possess wider applications in cotton as this tool is simple to operate with high efficiency and less cost. Even though realizing the impact of genome editing in cotton for disease resistance is currently limited, its applications in wheat, rice and corn provide strong evidences for its role in cotton disease resistance breeding. Though considerable progress has been made in the aspects of cotton's tissue structure resistance, physiological and biochemical resistance, R-gene-mediated resistance, hormone-mediated disease resistance signal pathways and their interactions, still we don't have clear understanding on the complete disease resistance mechanisms in cotton, which warrants further research in this direction. Availability of cotton whole-genome sequences and high-throughput molecular markers (Boopathi 2020), offer new avenues in finely pinpoint the site of resistant genes. As the developments in genetically modified cotton breeding germplasm is slow and problems associated to evolve a line that adapts to different ecological environments, modern genomics tools need to be integrated with traditional breeding methods to accelerate the process of disease resistance cotton breeding in the future.

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