# Role of QTL mapping to circumscribe various diseases in different crops with special emphasis on cotton.

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## Abstract

**Cotton not only provides the world's best natural spinnable fibre but also its seed is a rich source oil and protein, which is best for food and livestock feed. A biotic and biotic stresses always a serious threat to cotton production world widely. There is a demand of such cultivars which could oppose these stresses with wide genetic variability. Molecular breeding along with marker technology opened new ways in crop improvement. Genome mapping which is the identification of genomic regions linked with different traits; using construction of linkage map and quantitative trait loci (QTL) analysis can be used to identify resistant sources with the use of marker assistant selection (MAS). Improvements in control of many biotic diseases like verticillium (V) and Fusarium Wilt (FW), Reniform and Root-knot Nematodes had been made using molecular breeding. Cotton leaf curl disease (CLCuD) is the most distressing diseases of cotton in Pakistan, north-western areas of India and some parts of Africa which could be improved using these biotechnological tools. In many agricultural crops viral diseases like tomato yellow leaf curl virus (TYLCV), tomato mosaic virus (ToMV), tomato spotted wilt virus (TSWV), rice black-streaked virus (RBSV), Maize Streak Virus (MSV), Maize dwarf mosaic virus (MDMV), Sugarcane mosaic virus (SCMV), Wheat streak mosaic virus (WSMV), Maize chlorotic dwarf virus (MCDV), Maize mosaic virus (MMV), Maize fine streak virus (MFSV), Rice Stripe Virus (RSV), Rice yellow mottle virus (RYMV), rice black-streaked dwarf virus (RBSDV) and Barley Yellow Dwarf Virus (BYDV) had been studied using molecular markers. This Review study provides explanation of molecular markers involved in crop improvement especially for disease resistance in cotton and other field crop which could carry out to find out CLCuD resistant sources in upland cotton.**

**Keywords**: MAS, QTL mapping, CLCuD, *Gossypium*, Molecular markers, Resistance.

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## Introduction

Cotton is an important cash crop with high economic significance for many countries in the tropical and subtropical regions [1]. Cotton not only provides the world's best natural spinnable fibre but also its seed is a rich source oil and protein, which is best for food and livestock feed [2,3]. In Pakistan cotton add 60% share in its foreign exchange and thus, considered as the backbone of the economy [4].

The genus *Gossypium* is a large genus comprising of 45 diploid and 5 tetraploid species. Most of the species are of wild origin and only four species are being cultivated namely: *Gossipium hirsutum* (*G. hi rsutum*), *Gossipium barbadense* (*G.barbadense*) (*tetraploid genome*) *Gossypium herbaceum* (*G.herbaceum*) *and Gossypium arboretum* (*G. arboretum*) (diploid genomea). Among the cultivated species, only *G. hirsutum* (also called upland cotton) is being cultivated on >90% global area under cotton cultivation. The reasons for this high proportion are its high quality fibre and yield attributes [5]. Although diploid cotton species including *G. arboretum* and *G. herbaceum* produce low quality fibre but still these are considered for cultivation due to their ability to cope with environmental hazards and resistance against various biotic stresses including insect, pests and diseases. Historically 1-2 million years ago allopolyploid cottons emerge to have arisen as an outcome of trans-oceanic dispersion of an A-genome taxon to an indigenous D-genome diploid followed by hybridization [6].

## Literature Review

### *Use of molecular marker for disease resistance in cotton*

The genetic loci thus identified using QTL mapping the identified regions with in the genome of individual that contains genes linked with a specific quantitative trait are recognized as quantitative trait loci or QTLs. With the distinguish understanding of many potential DNA markers in plant breeding

scientist implemented capability to enhance MAS using marker development. The identification of genomic regions linked with different traits, using construction of linkage map and QTL analysis is called QTL mapping also known as genome mapping [7-10].

The identification of the required QTLs has been speed up due to expansion of MAS and advancement in statistical modelling. The use of molecular markers to construct linkage maps for a particular trait in a particular species has made this technology vital in agricultural research programs. QTL mapping is very important in the identification of chromosomal regions involved in the inheritance of monogenic as well as polygenic traits [7,8].

In plant breeding MAS is being used as molecular tool for the utilization of DNA markers, associated with agronomical important genes and other genes kinked with biotic and abiotic stress resistances, by selecting phenotype using genotype markers [11]. This use of the molecular marker technology in plant breeding opened new ways in agricultural crop improvement is known as molecular breeding [12].

### *Genetic markers*

Genetic markers can be divided in to three types, DNA or molecular markers, morphological markers and biochemical markers. Morphological markers are basically phenotypic traits or characters these are characters which can be identified visually such as seed shape, flower colour, pigmentation or growth related traits. Biochemical markers are isozymes which are allelic variants of different enzymes that are identified by particular staining and electrophoresis [13,14].

Genetic marker serves to identify difference between individual organisms, or species genetically. These markers act as flags or signs controlling different traits on the chromosomes and do not express themselves as genes. The molecular markers can be used to tag the gene of interest because these are tightly flanking those genes. Although, genetic markers are located in the flanking region of a gene but phenotypically do not have biological impact. The specific genomic location occupied by genetic markers within chromosomes is termed as "loci" Singular "locus" [7,15]. Genetic markers are very supportive to plant breeders in detection and identification of genetic variations, linkage mapping, genetic characterization, MAS, genomic fingerprinting and genotyping analysis [16,17].

Environmental effects and limited number of morphological and biochemical markers restrict their use on wide range but many markers are useful to researchers [18]. DNA markers are mostly used markers comes from DNA mutations like insertion or deletion, point mutations or errors during replication of DNA and are available in large quantity [10]. DNA markers are sited at non coding regions of DNA making them neutral and these markers could not be affected by environmental factors. In plant, breeding DNA markers not only used for construction of linkage map but also being used for many other applications like genetic diversity of large germplasm or variety identification [14,19].

Molecular markers can be divided in to three classes on the basis of different technologies. Hybridization based markers; are (RFLP) Restriction fragment length polymorphism markers are worked by studying changes in the size of DNA fragment with the use of restriction enzymes. PCR based markers; are the mostly use markers due to their high efficiency included random amplified polymorphic DNA (RAPD), amplified fragment length polymorphism (AFLP), simple sequence repeats microsatellites (SSRs) and inter-simple sequence repeats (ISSRs). DNA sequenced based markers; can identify the base sequence of a DNA fragment. The variations in single base pair (nucleotide) in sequence of individual genomic DNA are recognized as single nucleotide polymorphism (SNPs) [20-24].

### *Simple Sequence Repeats (SSRs)*

At present SSRs markers, simple sequence repeats or microsatellites and sequenced based markers SNPs are widely used by the plant breeders. SSRs or Microsatellites are stretches of di, tri, tetra or penta-tandom repeats of nucleotide units spread in eukaryotic genomes. These tandem repeats in variable numbers are known as simple sequence repeats defined as (1-6) bp are specialized by high frequency, high polymorphism, co-dominance, reproducibility and distribution. Specific pair of primers is used during amplification of each SSR locus and using gel electrophoresis amplified product is being analysed for the detection of diverse alleles of the locus. Mostly in each PCR reaction a single DNA sample is amplified using specific single SSR locus. SSRs have been developed for many crops species according to interest of the scientists and are extensively used for identification of QTLs, cultivar identification, genetic diversity, germplasm characterization, analysis of gene pool variations and MAS. SSRs are demanding markers for molecular based research from the last decade of twentieth century. In this paper, we tried to go over the main points regarding current development in plant breeding by the use of SSRs markers with special impact for the identification of QTLs for disease resistance in different crops, how can we overcome CLCuD by using this techniques [7,25-28].

### *Cotton Leaf Curl Virus (CLCuD)*

CLCuD is a one of the upsetting diseases of cotton in Pakistan, north-western areas of India and some parts of Africa [29]. The disease is being characterised by diverse symptoms including cupping of leaves, thickened primary and/or secondary veins formation of enations on the undersides of leaves. In extreme infection the proliferation of chloroplast tissues make the infected plants much greener than healthy plants. Nevertheless, disease symptoms may vary with cotton germplasm, time of sowing, whitefly (the vector) infestation, disease severity and other environmental factors. During the CLCuD attack, the older plants remain stable and hence face low yield reduction as compared to the newly germinated plantlets.

CLCuD is caused by genus *Begomovirus*, Family *Geminiviridae* and is mostly spread via whitefly *Bemisia tabaci*. *Geminiviruses* cause this disease in organization with Betasatellites (specific symptom modulating satellites) and alphasatellites (molecule of evolutionarily distinctive group of satellites) [30-32]. During last 20 years CLCuD is the mainly disturbing natural debacle that causes massive sufferers to cotton crop production in the world including Pakistan. The problem of this disease always under consideration by the researchers since it appears first time in 1967. In Pakistan CLCuD appeared in epidemic form during 1992-93 and 1993-1994 causing yield loss of 9.05 million bales

and 8.04 million bales respectively. Along with huge decline to cotton production CLCuD also demur the quality of lint by effecting traits like fibre uniformity index, Ginning out turn percentage, fiber fineness, staple length, maturity ration and fibre strength. Lint traits decrease their quality due to variation in construction of fiber components like cellulose protein and wax [29]. Losses like production of seed cotton yield and other agronomical traits has been estimated by many workers and studies showed that there is an average reduction in plant height about 40.6%, number of bolls/plant 72.5%, boll weight 33.8%, 3.9%, ginning out turn, 3.4% fiber length, 0.7% fiber strength and 23.8% fiber fineness [33,34].

The information concerning heritage of CLCuD always essential for plant researchers but still a widespread opinion had not been established about the inheritance of CLCuD [35]. Collection of minor genes by recurrent selection for the development of CLCuD resistant material has been achieved by Rahman et al. [36]. It is also reported that resistance is based on major dominant genes which may affect by the evolution of pathogen [37]. During a study by crossing *G. hirsutum* and *G. barbadense*, established the possessions of a single dominant gene controlling this disease [38]. The $F_1$ plant progenies of crosses obtained between highly resistant LRA-5166 variety with highly susceptible variety S-12 found virus free plants and progenies in $F_2$ showed 1:3 ratios which reveal a single gene is responsible in the inheritance of CLCuD [39,40]. Phenomenon of controlling resistance to CLCuD there is contribution of dominant epistasis along with two dominant genes [41]. Dhaliwal et al. [42] Stated that association of three genes in *G. hirsutum* involved controlling resistance to CLCuD, two for resistance (R1CLCuDhir and R2CLCuDhir), third gene work as suppressor of resistance (sCLCuDhir).

CLCuD disease severity is directly related to population of vector and climatic conditions like temperature, humidity, rainfall and wind. Rainfall at seedling stage may increase population of white fly due to prevalence of food sources [43]. Alternate plants of weeds and cultivated host crops also contributed as a cause of virus during the off season of cotton. Virus vector infects other fields to develop infection sites along with cotton fields. Regression analysis on air temperature, rainfall, relative humidity and wind movement association with CLCuD severity has been studied in different genotypes of cotton. Maximum and minimum temperature range of 33ºC and 25 increased disease infestation. Their results also showed that there is a reduced correlation of humidity and rainfall with disease incidence and non-significant correlation whitefly population and CLCuD severity on all germplasm under study [44].

Biotechnology introduced *Bt* technology with insecticidal protein for the control of lepidopteron and dipteran insects of cotton and other crops. More than two hundred different types of *Bt* protein have been used as toxicity which are isolated from many strains of *Bacillus thuringiensis*. After widespread adaptation crops containing *Bt* toxic protein against chewing insects there is another class of insects known as sucking insects including whitefly, aphid, jassid and mealy bug emerged as serious threat to different crops including cotton. Whitefly previously taken as a minor pest is now becomes an important pest of different crops world widely. It is the vector to transfer more than two hundred harmful viruses to the plants along with damage to the plant by attacking on the phloem tissues. Therefore, there is need to develop such methods which limits the population of such insects, to avoid extensive influx of viral disease on different crops along with source of resistant varieties [45-48].

### *Molecular markers techniques for disease resistance*

Molecular markers have achieved a key role in crop breeding for cultivar improvement for agronomic attributes as well as resistance against insects, pests and diseases. A large number of QTLs have been available now depending upon the choice of experimental design the objectives of the research program. During past couple of decades, QTL mapping technique has been improved enormously and as a result thousands of QTLs are now available for major as well as minor crops [49]. There are many reviews already available describing in detail the application of QTL mapping in various crops [6,14,26,50-55] but, this area is not well covered for disease resistance in *G. hirsutum*. In this review, we will highlight the applications of QTL mapping for resistance against major disease of cotton.

The cotton cultivation has been under continuous threat of different economically important diseases caused by viruses, bacteria, fungi and nematodes. Apart from chemical control of their causal organisms, the most effective way to counter these diseases is the development of genetic resistance in crop plants. The discovery of QTL mapping ease the breeders work to identify and characterise the genetics of disease resistance in crops particularly cotton.

### *QTLs mapping for resistance of Fusarium Wilt (FW) in Cotton*

FW is one of injurious diseases of cotton caused by *Fusarium oxysporum* f. sp. *Vasinfectum* which is a soil borne fungus. In the United States five important FW resistant sources (Cook 307-6, Coker Clevewilt, Dillon, Dixie Triumph, and Wild) and in China three (CRI 12, Chuan 57-681 and Chuan 52-128) have been identified using persistent pedigree analysis. Study on many early segregating populations revealed predominant type of gene action involved on FW resistance but due to high errors in experiments, heritability low down frequently. In cotton many resistance genes had been identified with the use of molecular markers and segregating analysis [56]. Genetic mapping studies identified about 40 QTLs on 19 chromosomes. In Upland and Pima cotton five resistant genes FwR, Fw1, Fw2, FOV1 and FOV4, in many qualitative genetic studies (Table 1).

In a FW resistance mapping population of $F_{2:3}$ of susceptible *G. barbadense* Xinhai14 x FW resistant Upland 98134 in Xinjiang, 91 markers obtained from 22 linkage groups covering 1362 cm was identified. Four QTLs associated with FW resistance were identified amplifying 12.4, 21.0, 4.7 and 11.9% of the phenotypic variance (PV) on c3, c15, c23 and c26 chromosomes respectively [57]. In two intraspecific population of $F_2$ Upland cotton, consists of 79 and 154 individuals four QTL identified explaining (13.1-45.9)% of PV, located on c17 or c2 linked with CIR305 and JESPR304 [58]. Seven QTLs associated with FW resistance was identified on c3, c12, c17 (D13), c18 (D3), c19 (D5) and c24 (D8) each with 2.9 to 6.6% of PV [59]. In an $F_2$ mapping population of susceptible Xinhai 21 × resistant HK237,

SSR markers NAU3240 linked to chromosome c21 (D11) was identified at genetic distance of 19 cm [60]. About 12 QTLs associated with FW resistance identified on c1, c3, c8, c12, c16 and c26 in which three of these c1, c3, c8, c12, c16 and c26 was studied in three environments [61].

In three $F_2$ intraspecific populations (*G. barbadense* × *G. barbadense*), (*G. hirsutum* × *G. hirsutum*), five $F_2$ interspecific populations (*G. hirsutum* × *G. barbadense*) and recombinant inbred line (RIL) population of *G. barbadense* 3-79 × *G. hirsutum* TM-1 to identify QTL resistance to race 4. Many QTLs also linked with c3, c8, c14, c7, c19 and c25 were found governing inheritance of resistance [62].

### *QTLs mapping for resistance of Verticillium Wilt (VW) in cotton*

VW is one of the destructive diseases of cotton and cause huge loss to cotton production caused by throughout the world. This disease is caused by Verticillium dahlia which is a soil borne fungus. Due to symptoms of VW like necrosis or wilting, leaf chlorosis, plant death and boll abscission this disease cause considerable reduction in cotton yield and deteriorate lint quality as well.

For disease resistance in cotton numerous QTLs have been mapped, some consistent QTLs from these were recognized for use in plant breeding and genomic research. In our study population of backcross inbred line (BIL) with 4 year replicated trials were used to identify QTLS. About 10 QTLs resistance to this disease were mapped using 392 SSR markers on 2895 cm linkage map. These identified QTLs were sited on consensus map with other QTLS like QTLs7 RN, QTL 75 RKN s, QTLs 182 VW, and QTLs 27 FW which were reported from 32 different publications. Another meta-analysis for QTLs also identifies 13 hotspots for resistance to VW in 28 QTL clusters. Many QTLs and cluster of QTL on chromosome significantly correlated with nucleotide binding site (NBS) genes particularly in A-sub genome. These VW resistance QTLs recognized in this study could provide basics for understanding of genetic basis of this disease. These 28 QTLS, 24 hotspots give imperative information for high resolution mapping and MAS [63-65].

Were constructed a $F_2$ mapping population obtained from the interspecific cross of Pima S-7 (*G. barbadense*) a highly tolerant to VW and Acala 44 (*G. hirsutum*) a susceptible variety to VW [66]. Phenotypic observation for disease severity and incidence was recorded from both the lines. About 3 weeks after inoculation data was recorded from each plant infected with the disease along with some growth parameters like total shoot weight, number of healthy leaves, stem weight, leaf weight and number of nodes. Phenotypic distribution of $F_2$ for these parameters suggested polygenic inherited type of resistance. Both the progenies (10 plants each) were screened with 255 SSR primers and 60 markers were selected to identify QTLs. 11 linkage groups were recognized using 35 markers and straddling 531 cm having average distance of 15.17 cm. Map QTL and QTL Cartographer were used for the analysis of QTL and recognize that 15 markers were in significant linkage association and other are linked to 10, 11, 12 and 25 chromosomes. It was found that 3 loci 3147-2, STS1, CM12 had huge effect on resistance to VW. One locus was located on LG-2 and two loci were positioned on LG-1 and linkage groups positioned on chromosome number 11 also identify $QTL_s$ associated with VW resistance with interspecific chromosome segment introgression lines (CSILs) [67]. About 42 QTLS identified with 23 for resistance increasing influence and 19 with resistance decreasing influence against different three isolates. Identified QTLs were mapped on about 18 chromosomes with LOD values in the range of 3.00 to 9.29. These pyramiding resistance QTLs could be used in cotton breeding programme with broad spectrum resistance to VW.

### *QTLs mapping for resistance of the root-knot nematode in cotton*

The root-knot nematode (RKN Meloidogyne incognita) is an endoparasitic pest of upland cotton, along with loss from parasitism, severity and incidence of FW, also greatly increased by nematodes. In a recent study for the detection of resistance to root-knot nematode, linkage mapping were developed using latest available SSR markers from a mapping population of M-120RNR X Pima S-6. The QTL analysis revealed two regions which were considerably linked to resistance phenotype. A major QTL was recognized on chromosome number 14 (qMi-C14 along with confirmation of a QTL previously detected on Chromosome number 11 (qMi-C11). The identified locus qMi-C14 derived from resistant parent Mexico Wild Jack Jones, while the qMi-C11 derived from Cleve wilt parent. The locus qMi-C14 showed 45% of phenotype variation in production of egg which had had logarithms of odds LOD-17. In alliance with galling index, percent variation of 6% proposed that the locus qMi-C11 found greater effect on suppression of root gall than egg production, while the locus qMi-C14 locus had greater influence on egg production. The identified markers strongly associated with qMi-C11 and qMi-C14 loci could be used in marker assistant breeding for the development of root-knot nematode resistance sources in cotton [68] (Table 1) worked on a mapping population of highly resistant *G. hirsutum* cultivar M-120 RNR with Auburn 623 RNR to recognize the inheritance of genetic resistance of nematode [69]. With the use of two $F_2$ interspecific populations derived from the cross of M-120 RNR with *G. barbadense* (Pima S-6). Genetic mapping were showed considerable association with resistance phenotype on chromosome number 1 and chromosome number 11 with the use of RFLP markers. By using SSR markers this association was confirmed with the finding of two QTL on chromosome number 7 and 11. In the combined study of two different populations, the QTL on chromosome number 11 named Mi-C11 had major effect with LOD score 19.21 (for population 1 LOD 9.69, for population 2 LOD 9.61) and responsible for total phenotypic variation of 63.7% (for population 1, 52.6%, for population 2, 65.56%). The QTL number the chromosome number 7 Mi1-CO7 had minor affect with LOD score of 3.48 and accountable for 7.7% phenotypic variation. The alleles from the parents M-120 RNR and Pima S-6 were responsible to enhance resistance in the Mi-C11 locus and Mi-C07 locus respectively. The locus Mi-C11 of allele M-120 RNR imitative from the Auburn 623 RNR seems to be come from the source of Cleve wilt 6 Cultivar. These finding will help to recognize genotypes resistant to M. Incognita with SSR markers CIR316 instead of laborious screening in the green house.

During a later study on two DNA pools comprises of 5 individuals

***Table 1.*** *List of recognized QTLs or genes associated with various disease resistances in cotton along with chromosomal position.*

| Disease | QTLs/ gene | Chromosome No. | Associated Marker | Population/Variety | Reference |
|---|---|---|---|---|---|
| Root-knot Nematodes | Mi-C11 | Chromosome 11 | SSRs (CIR316) | $F_2$ populations M-120 RNR | [69] |
| | Mi1-C07 | Chromosome 07 | | | |
| | Mi-C11 (Fine mapping) | Chromosome 11 | SSRs (CIR316, CIR069) AFLP (E14M27-375) | $F_2$ populations M-120 RNR | [70] |
| | qMi-C11 qMi-C14 | Chromosome 11 | SSRs | $F_2$ populations Clevewilt parent, Mexico Wild Jack Jones. | [68] |
| | | Chromosome 14 | | | |
| | RKN | Chromosome 11 | SSRs | CS-B22Lo, CS-B04, CS-B18 Chromosome substitution (CS) lines | [2] |
| | | Chromosomes 4 and 22 | | | |
| | RKN | Chromosome 11 | SSR-CIR-316 | -- | [86,87] |
| | RKN | Chromosome 11 | SSR-MUCS088 | -- | [88] |
| | RKN | Chromosome 11, 21 | SSR-1231 | -- | [86,89] |
| Reniform Nematodes | REN | Chromosome 11, 21 | SSR-BNL3279 | -- | [90,91] |
| FW (*Fusarium wilt*) | FW | chromosomes 16 | SSRs | CS-B16 | [2] |
| Fusarium oxysporum f. sp. Vasinfectum Fusarium wilt (FW) | Fw1, Fw2, FwR 45, 46 (c17), FOV1 (c16), FOV4 (c14). | Chromosomes 17 | AFLP, SSRs | $F_2$ populations $F_3$, BC1, BC2 | [57] |
| | | Chromosomes 16 | | | |
| | | Chromosomes 14 | | | |
| Fusarium wilt (FW) | 10 QTLs | Chromosome 2, 4, 9, 12, 13, 21, 22, 23 | SSRs | Backcross inbred line (BIL) | [63] |
| | 5 QTLs | Chromosome 2, 17 | SSRs JESPR304 CIR305 | $F_2$ populations | [92] |
| Verticillium wilt (*Verticillium dahliae*) | 3 QTLs | Chromosome 11 LG1, LG2 | SSRs (CM12, STS1, 3147-2) | $F_2$ population Pima $S_{-7}$ (*Gossypium barbadense* cv.) | [66] |
| | 42 QTL, | Chromosomes A1, A3, A4, A5, A7, A8, A9, A12, A13, D1, D2,D3, D4, D5, D7, D8, D11, D12 | SSRs | Interspecific chromosome segment introgression lines (CSILs) | [67] |
| | QTLs (q-5, q-6, q-8, q-13) | A6/D6 A7/D7 A11/D11 | SSRs, NAU905 NAU2754 NAU3053 NAU2508 | 155 cotton inbred lines | [93] |
| | 21 (QTLs) quantitative trait loci were identified | Ch 8, 11 15, 17, 19, 21, 26 | SSRs, AFLP | recombinant inbred mapping population | [94] |
| | 42 QTLS Identified | 15 chromosomes | SSRs | Association mapping. (158 elite cotton germplasm) | [95] |
| | 3 QTLs Detected | Chromosome c4, c19 | RGA (resistance gene-analog), RGA-AFLP (targeted amplified fragment length polymorphism) markers | mapping population of Interspecific backcross inbred lines | [96] |

(homozygous for parental alleles), a bulk segregation analysis was conducted to build up fine map in the target region. About 9 AFLP markers were identified which were linked to target region during survey of 1152 AFLP primers. With the screening of 1221, $F_2$ additional individuals derived from basic mapping population, the locus Mi-C11 surrounded to a 3.6 cm distance flanked by the AFLP marker E14M27-375 and SSR marker CIR069. These results clarify the fine genetic structure of locus MiC11 could be used to isolate resistance gene for nematode in M-120 RNR using fine mapping [70].

### *QTLs mapping for viral disease resistance in tomato, potato and tobacco*

TYLCD is a severe danger to production of tomato in tropic and subtropics areas by harming its production badly. TYLCD is caused by *Begomovirus* of the *Geminiviridae* family; this is a single stranded DNA virus which is spread by a vector white fly [71]. In order to recognize QTLs associated with tomato yellow leaf curl Thailand virus Taiwan strain (TYLCTHV-TW) a highly resistant germplasm of tomato FLA456 was used in scientific study. From a cross of FLA456 (resistant) x CLN1621L (susceptible) an $F_6$ RIL, four QTLs, qTy4.1, qTy6.1, qTy10.1 and qTy11.1 were identified on chromosome TYLCD 4, 6 10 and 11 respectively. On the basis of disease reaction of gene action of identified QTLs was recessive. On chromosome 4 the markers SLM4-34 and SINAC1 flanked locus qTy4.1 and SLM11-17 and SLM11-12 distinct locus qTy11.1 which were co-located already recognized loci Ty-2 and Ty5 respectively. The QTLs qTy10.1 flanked by markers SLM10-80-SLM10-46 and qTy6.1 by SLM6-55 and TES-0014

***Table 2.*** *Recently Identified QTLs or genes associated with resistance to viral diseases in different crops.*

| Crop/species | Disease | QTLs/ gene | Marker | Population | Reference |
|---|---|---|---|---|---|
| Tomato (*Solanum lycopersicum*) | Tomato Yellow Leaf Curl Virus (Tylcv) | Ty4 | SSR | $F_2$ | [97] |
| | Tomato Yellow Leaf Curl Virus (Tylcv) | Ty3-HRM1<br>Ty3-HRM2<br>Ty3-SCAR1 | high-resolution melting (HRM) markers, sequence characterized amplified region (SCAR) marker | $F_2$ | [77] |
| | (TYLCTHV-TW) Tomato Yellow Leaf Curl Thailand Virus Taiwan Strain | qTy4.1<br>qTy6.1<br>qTy10.1<br>qTy11.1 | SSRs<br>RFLP | (RIL) population | [72] |
| | Resistance to *Bemisia tabaci* | TG313<br>C2_At2g41680<br>TG523/<br>TG400/cLEG-37- G17 | cleaved amplified polymorphic markers (CAPs), sequence characterized amplified region (SCAR) markers, conserved orthologous sequence (COS), conserved orthologous sequence II (COSII) | Interspecific $F_2$ population | [98] |
| | TOMV (Tomato Mosaic Virus) | Tm-1 / SCN20 | SCAR | Interspecific $F_2$ population | [99] |
| | | Tm2/ OPK6<br>Tm2/ OPA-12<br>Tm2/SCB12<br>Tm2/SCE16<br>Tm2/SCGO9<br>Tm2/SC118<br>Tm2/SCN13 | RAPD<br>SCAR | Interspecific $F_2$ population | [100] |
| Tomato (*Solanum lycopersicum*) | TOMV (Tomato Mosaic Virus) | TM2-748<br>TM22 SNP 2494<br>TM2-SNP901mis<br>TMV-2262 | SNP | $F_2$ | [99] |
| | Tomato Spotted Wilt Virus (TSWV) | Sw-5/ ZUP641 | SCAR | $F_2$ | [101] |
| | | Sw-5/ Sw5b-1RR<br>Sw-5/CT220 | CAPS | $F_2$ | [102] |
| | Tomato Spotted Wilt Virus (TSWV) | Sw-5/G5<br>Sw-5/ K16<br>Sw-5/S12 | RAPD | $F_2$ | [103] |
| Tobacco (Nicotiana) | Tobacco Leaf Curl Disease (Tlcd) | SC230<br>SC379 | SC230<br>SC379 | $F_2$ | [78] |
| Maize | Rice Black-Streaked Virus | qMRD2, qMRD6, qMRD7, qMRD8 and qMRD10 | SSR | $F_{7:9}$ RILs | [79] |
| | Maize Streak Virus | Msv1 (Fine mapping) | SNP | $F_{2:3}$ family | [82] |
| Maize (*Zea mays*) | MDMV (Maize Dwarf Mosaic Virus), SCMV (Sugarcane Mosaic Virus), WSMV (Wheat Streak Mosaic Virus), MCDV (Maize Chlorotic Dwarf Virus), MMV (Maize Mosaic Virus), MFSV (Maize Fine Streak Virus) | 17 QTLs linked with 6 viruses. In the region of chromosome 2, 3, 6, and 10. | SSR | $F_2$ (Filial 2) RIL (Recombinant inbred line) | [85] |
| Rice *(Oryza sativa* L.) | Rice Stripe Virus | qSTV4, qSTV11.1 and qSTV11.2, | SSR | RIL population | [104] |
| | Rice Yellow Mottle Virus (RYMV) | QTL12 and QTL7 | RFLP, SSR | $F_2$;$F_3$, BC1, BC2 | [105] |
| | Rice Black-Streaked Dwarf Virus (RBSDV) | qRBSDV-3, qRBSDV-10 and qRBSDV-11 | SSR | $F_{2:3}$ families | [104] |
| Barley (*Hordeum vulgare L.)* | Barley Yellow Dwarf Virus | Yd2 | STS | | [104] |
| | Barley Yellow Mosaic Virus | rym1, rym5, rym11 | RFLP, CAPS | $F_2$ | [104] |
| | Barley Yellow Mosaic Virus | rym4, rym9, rym11 | RAPD, SSR | $F_1$-derived doubled haploids | [104] |

markers on chromosomes 6 and 10 with LOD values ranged from 2.79 to 13.76. For the identified QTLs together make a contribution of 60.5% of phenotypic variation in TYLCTHV-[TW] resistance. The identified QTLs from FLA456 would be utilized in deployment of virus resistance tomato varieties along with *Begomoviruses* diversity which cause TYLCD in different areas [71,72] (Table 2).

About five genes controlling TYLCD resistance had been mapped these genes are Ty-11, Ty-2, Ty-3, Ty-4 and Ty-5 which were derived using S chilense accessions LA2779 and LA1969 which were mapped on chromosome number 6 [73]. The gene Ty-1 is dominant gene for TYLCV resistance and their location at chromosome number 6 is close to Mi locus flanked with RFLP molecular markers TG97 and TG297 [74]. But, in a latest study concluded that gene Ty-1 and Ty-3 are allelic or very close located at the long arm of chromosome number 6 [75]. Another gene ty-5 which showed recessive gene action also identified near the Ty-5 locus from the "Tyking" variety of tomato [76].

In a latest study a segregating population was developed from a cross of resistant to TYLCV line A45 (*S. lycopersicum*) with the susceptible line A39 (*S. lycopersicum*) to identify gene based markers associated with Ty-3 gene. In order to test TYLCV resistance from segregating population *Agrobacterium*-mediated screening was used and the disease severity was screened with scoring method and analyses evaluation using polymerase chain reaction. A marker (Ty3-SCAR1) sequence characterized amplified region (SCAR) and two high-resolution melting markers (HRM) (Ty3-HRM2 and Ty3-HRM1) were build up by evaluating resistant and susceptible lines at sequences of the Ty-3 genes. These gene-based markers could be utilized to improve TYLCV resistance using MAS in plant breeding programs [77]. Tobacco leaf curl disease (TLCD) is a severe disease of tobacco originated by *Begomoviruses* in *Geminiviridae*, lower down the production by showing symptoms like downward curling of leaf, thinking of leaf and swelling of leaf vein [78].

### *QTLs mapping for diseases resistance in maize (Zea mays L.)*

Maize rough dwarf ailment (MRDD) has become a virus disease of maize and is curtailed maize production globally. In past bug affected rice crop which was known as RBSDV in China. MRDD has prevailed and to affect production in China straight away. Some researchers has searched solution of this disease and resilient to MRDD. In the present study, researcher want to identify QTLs linked to resistance of this disease. Two elite lines of maize '90110' highly resistant and 'Ye478' which is susceptible to MRDD were used to develop F2 segregated population. The $F_2$ and BC1 were being utilized for BSA known as bulk sergeant enquiry. Their main purpose was to highlight resistance related markers associated with this disease. QTL has been checked by various experiments over 3 years while many more $F_7$ 9 RILs has used for QTLs. In the experiments QTL qMRD2, qMRD6, qMRD7, qMRD8 and qMRD10 have been deliberated. The qMRD8 on chromosome 8 has analysed to be single key QTL. Consulting struggle to RBSDV ailment in virtually all characters and atmospheres which explains (12-28.9)% the phenotypic variance for disease sternness in this study. Another scientist studied 87-1 resistant line with group inoculation in net house and identified two SSR markers that might be linked to resistant genes controlling MRDD [79,80].

GLS (Gray Leaf Spot) is one of the severe diseases all around the world where maize has been cultivating since years. In sub Saharan Africa or Brazil the situation is very critical while US the situation is reverse due to climate changes but it can become cause of destruction of corn production in US owe to thin hereditary ignoble of North American resilient germplasm. The only way to control this disease was the development of resistant variety. We did compile together the inordinate QTL finding influence of inherited connection with the extraordinary determination influence of genome wide association study. We recognized four GLS confrontation QTL on chromosomes 1 6 7 and 8, while the resolution increased severely due to genome-wide association study (GWAS). Linkage mapping results showed that a novel QTL (QTLGLSchr8a) is linked GLS resistance which verify the locations of already identified QTLs (QTLGLSchr8b, QTLGLSchr1, QTLGLSchr6 and QTLGLSchr7 [81] (Table 2).

MSV is another distressing disease of maize causes significant yield loss in the Sub-Saharan Africa areas. Molecular mapping analysis in previous studies identified one of the major QTL (Msv1) on chromosome number 1 linked to MSV resistance. This Msv1 QTL was mapped using QTL isogenic recombinant strategy for fine mapping using a large F2 segregating population of CML312 × CML206 on chromosome number with 0.87 cm interval. GBS markers were used for high density genotyping of 278 tropical and subtropical lines obtained from CIMMYT along with Genome-wide association study that were accomplish in the Drought Tolerant Maize for Africa (DTMA). On chromosome number 119 SNPs were identified in 82 to 93 Mb region which correspond the fine mapping of Msv1 [82]. Many other researchers also identified QTLs linked to MSV resistance [83,84] were conducted molecular mapping analysis in inbred line of maize which was multiple virus-resistant and studied six virus diseases resistance [85-104]. The inbred line of maize Oh1VI were used which is resistant to about ten different viruses. In order to identify genetic inheritance linked to multiple virus resistance F2 segregating population and another population of RIL were obtained from Oh1VI inbred line x Oh28 inbred line (virus susceptible). These progenies were screened for their symptoms for MMV, chlorotic dwarf virus, Maize chlorotic dwarf virus, WSMV, MDMV and SMV. As a result of QTL mapping on RIL population recognized about 17 QTLs linked with six different viruses. From this identified QTLs 15 were clustered in the region of chromosome number 2, 3, 6 and 10. It is hard to say whether, which QTL is linked to a single or may be multiple virus resistance but this combination of linkage of genes associated with resistance to many viruses could be utilized to facilitated breeding of multi virus resistant crops.

## Discussion and Conclusion

For abundant diseases molecular markers associated with QTLs or genes have been identified in many crops. These molecular markers had been utilized in development of the resistant cultivars using MAS. In cotton many diseases related QTLs had been reported as for simple inherited traits molecular breeding for resistance could be more precise than improvements of

complex traits. In order to locate QTLs or genes for CLCuD resistance in cotton using molecular techniques widespread approach of work is needed, because diseases with similar pathogens had been successfully find out disease resistant genes or markers as discussed in this study.

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