"Unraveling the Genetic Basis of Fusarium Wilt Resistance in Chickpea (*Cicer arietinum* L.): Insights into Disease Mechanisms and Breeding Strategies for Sustainable Crop Protection."

Abstract

"Fusarium wilt poses a significant threat to global chickpea production, exacerbated by the persistent soil-borne nature of the pathogen and its diverse races. This review delves into the genetic basis of wilt resistance in chickpea, exploring disease mechanisms and breeding strategies for sustainable crop protection. Chickpea, a vital legume crop, exhibits complex genetic traits and diverse cultivars, including the kabuli and desi types. Genetic studies have identified various resistance genes against Fusarium wilt, with specific genes conferring resistance to different pathogenic races. Screening methods, both in the field and laboratory, play crucial roles in identifying wilt-resistant genotypes. Traditional breeding techniques, such as hybridization and backcrossing, alongside modern approaches like marker-assisted selection and genomic technologies, have significantly enhanced resistance breeding programs. Future prospects entail the integration of molecular markers and biocontrol treatments for more effective and sustainable management of wilt in chickpea cultivation."

Keywords: Chickpea, *Fusarium oxysporum* f. sp. *ciceris*, Pathogenic Races, Molecular markers.

INTRODUCTION

Chickpea (*Cicer arietinum* L.), often known as Bengal gram, is a self-pollinating, diploid (2n = 2x = 16) plant species, with an annual life cycle [1], and an average of 738 Mbp genome size [2], which is substantially smaller than other legume crops. It is mostly consumed by humans and is a staple meal and component of the Mediterranean diet. India is the world's largest producer of chickpeas, producing 10.13 million tonnes annually from a land area of 9.44 million hectares with a productivity of 1073 kg/ha [3].

There are 34 wild perennial species and 9 annual species [4]. The only cultivated species among the nine annual species is *Cicer arietinum* L (chickpea). Chickpea is classified into two distinct cultivated types: desi and Kabuli [31]. The desi account for over 85% of the area, and have tiny, angular-shaped, dark-colored seeds. These plants have, pink flowers, rough surface, anthocyanin coloration on the stems, and a semi-erect or semi-spreading growth habit. The Kabuli kind, which often has huge "rams head"-shaped smooth surface seeds, and a semi-spreading growth habit [32].

Annual Species (9)

C. arietinum *C. judaicum* *C. bijugum* *C. pinnatifidum* *C. chorassanicum*	*C. reticulatum* *C. cuneatum* *C. yamashitae* *C. echinospermum*

Perennial Species (34)			
C. acanthophyllum *C. macracanthum* *C. anatolicum* *C. microphyllum* *C. atlanticum* *C. mogoltavicum* *C. balcaricum* *C. montbretii* *C. baldshuanicum*	*C. multijugum* *C. canariense* *C. nuristanicum* *C. fedtschenkoi* *C. oxyodon* *C. flexuosum* *C. paucijugum* *C. floribundum* *C. laetum*	*C. pungens* *C. graecum* *C. rassuloviae* *C. grande* *C. rechingeri* *C. heterophyllum* *C. songaricum* *C. incanum* *C. spiroceras*	*C. incisum* *C. stapfianum* *C. isauricum* *C. subaphyllum* *C. kermanense* *C. tragacanthoides* *C. korshinskyi*

M. Singh *et al.*, (2014)

The fight against world hunger and malnutrition has relied heavily on bio-fortification, which combines traditional breeding with contemporary technologies to increase micro-nutrient availability in food crops.

Chickpea has the highest nutritional content of any dry legume, and contains zero anti-nutritional factors. The seed contain 23% protein, 64% carbohydrates (47% starch), 6% soluble sugar, 5% fat, 6% crude fibre, and 3% ash [6]. A 100 g serving of chickpea contains 5.2–6.0mg of iron (Fe), 2.5–5.3mg of zinc (Zn), and 15.3-56.3mg of (selenium) Se. A serving of 100 grams also comprise 732-1, 126mg of potassium (K), 125-159mg of magnesium (Mg), 93-197mg of calcium (Ca), 0.7-1.1mg of copper (Cu), and 263-370mg of phosphorus [7]. It also contains vitamins such folic acid, tocopherols, and the vitamin B complex (B2, B5, and B6).

List 1 : Amino acid composition of chickpea		
Essential amino acids	Desi	Kabuli
Arginine	8.5 ± 0.05	8.0 ± 0.06
Histidine	3.2 ± 0.03	3.0 ± 0.08
Isoleucine	4.8 ± 0.05	5.2 ± 0.06
Leucine	8.5 ± 0.09	9.5 ± 0.06
Lysine	7.0 ± 0.03	7.8 ± 0.07
Methionine	1.1 ± 0.08	1.3 ± 0.04
Phenylalanine	5.3 ± 0.06	6.2 ± 0.12
Threonine	3.0 ± 0.09	3.5 ± 0.05
Tryptophan	0.9 ± 0.03	1.1 ± 0.08
Valine	4.4 ± 0.08	5.2 ± 0.11
Non-essential amino acids	Desi	Kabuli
Alanine	5.2 ± 0.07	4.7 ± 0.06
Aspartic acid	11.5 ± 0.12	10.2 ± 0.10
Cystine	0.6 ± 0.04	0.8 ± 0.07
Glutamic acid	17.8 ± 0.07	16.5 ± 0.012
Glycine	3.6 ± 0.03	4.0 ± 0.09
Proline	4.1 ± 0.10	3.5 ± 0.05
Serine	3.5 ± 0.10	4.2 ± 0.09
Tyrosine	2.8 ± 0.09	3.1 ± 0.09

Iqbal, Amjad, *et al.*, (2006)

Chickpea has several benefits for soil health. The crop obtains 80% of its nitrogen (N) from a symbiotic rhizobial interaction, i.e fix up to 140 kg N ha^{-1} from the atmosphere [9]. It contributes much-needed organic matter to preserve and enhance long-term fertility, and sustainability soil health.The deep taproot system of chickpeas is often acknowledged for its role in soil aeration and texture improvement, benefiting subsequent crops.

Although breeding programs have significantly increased chickpea productivity, farmers still have a lot of concerns due to instability of crop production. A significant production in chickpea farming is caused by many biotic constraints, including Fusarium wilt, Ascochyta blight, root rot complex and Botrytis Grey Mould . Among them, FW and AB are especially affecting diseases, causing significant losses to chickpea production. Addressing these difficulties is critical to reducing the yield gap and ensuring sustained chickpea yields [87].

Biotic stress that impact chickpeas include, Fusarium wilt produced by F. oxysporum Schlechtend.:Fr. f. sp. ciceris has been recognized as a key yield-limiting factor in many areas [10].

Fusarium wilt

Fusarium wilt is induced by *Fusarium oxysporum* Schl. emend. Snyd. and Hans. f. sp. *ciceri* [11] is most common in hot and dry regions. It is a soil-borne pathogen, can survive without a host for up to six years [12]. Wilt can result in yield reductions ranging from 10 to 90%, and even up to 100% when relative humidity exceeds 60% [23]. Thus, under favourable conditions, the disease can lead to complete crop failure.

Chickpea wilt is a widespread problem, affecting across continents [15]. Fusarium wilt infection in chickpea plants is associated with two major symptoms: yellowing and wilting [14]. Early wilting, defined by a deep green discolouration that appears 25 days after planting. Where as signs of "late wilt," include yellowing of the leaves and drooping petioles , manifest at the podding stage [29].

Fusarium wilt induced by eight different pathogenic races (0, 1A, 1B/C, 2, 3, 4, 5 and 6) and pathotypes [13]. This diversity in pathogenicity may be a result of genetic variations within the fungus. The races are categorized based on their ability to produce distinct symptoms in infected plants. The races of the fungus can elicit separate reactions in the plant, resulting in either yellowing or wilting symptoms. Yellowing is characterized by slow, progressive foliar yellowing and plant mortality. Wilting is identified by quick and severe yellowing, limpness, and premature plant death [30]. This indicates a high level of diversity in fungus, that interacts with its host plants.

Table 1 : Stage of infection and economic losses due to wilt in chickpea

Stage of infection	Economic loss (yield/value)	Reference
Seedling stage to pre- pod stage	61%	[87]
Seedling to pod-filling	10-90%	[88]
Flowering stage	43%	[87]
Early onset of wilting	77-94%	[89]
Medium onset of wilting	58-83%	[89]
Late onset of wilting	24-65%	[89]

Geographically categorised, the pathogenic races of fusarium wilt were initially identified in India. Initially, four physiological races (races 1, 2, 3, and 4) were identified using ten distinct lines [76]. There are currently eight races, including two sub-classes of race 1 (race 1A from India and race 1B/C from Spain) [77,78], race 0 from Spain [79], race 5 from Tunisia [80], and race 6 from California [52]. Additionally, Races 0 and 6 have been identified in India also [81]. While four races

(1A, 2, 3 and 4) are common on the Indian subcontinent, while other four (0, 1B/C, 5, and 6) are found in the Mediterranean and United States [80,81].

Disease cycle

Fusarium oxysporum f. sp. ciceris reproduces asexually and root-inhabiting fungus that survives inactive in soil [18]. The fungus can persist as mycelium and chlamydospores in seed, soil, crop residues, roots, and stem tissue for up to 6 years [35,36,38]. Chlamydospores can survive in soil as dormant or saprophytic without a host. The disease thrives in warm and dry soil conditions, with an ideal temperature of 22-25°C and 5 - 6.5 PH soils [43,44]. Infected seeds acts as the principal source of disease inoculation [37] and lead to disease development during the seedling and blooming stages of a plant's growth [38]. Plants developed from infected seeds wilt faster than healthy seeds germinated in affected soil [40]. Infected seeds play a crucial role in the pathogen's long-distance dispersion and introduction into wilt free regions [41]. Spores can be transported through various means like wind, water, soil, or plant detritus [45].

The fungal mycelium, referred to as the spore germ tube, penetrates the root tips of healthy plants in contaminated soil. It enters directly through wounds at the site of lateral root development. Mycelium enters xylem vessels via pits after travelling through the cortex. The pathogen primarily resides within xylem vessels, where mycelium divides and generates micro-conidia. Micro-conidia detach and travel up the vascular bundles until movement stops. At this point, they germinate, and mycelium enters the vessel wall. Lateral movement between vessels is through the pits. Blocking of vessels eventually significantly compromises the water economy of infected plants, causing stomatal closure, dark brown discoloration in vascular bundles, wilting, and leaf death, which results in death of the entire plant. The fungus then spreads throughout the plant's tissues, emerging at the surface to produce massive sporulations.

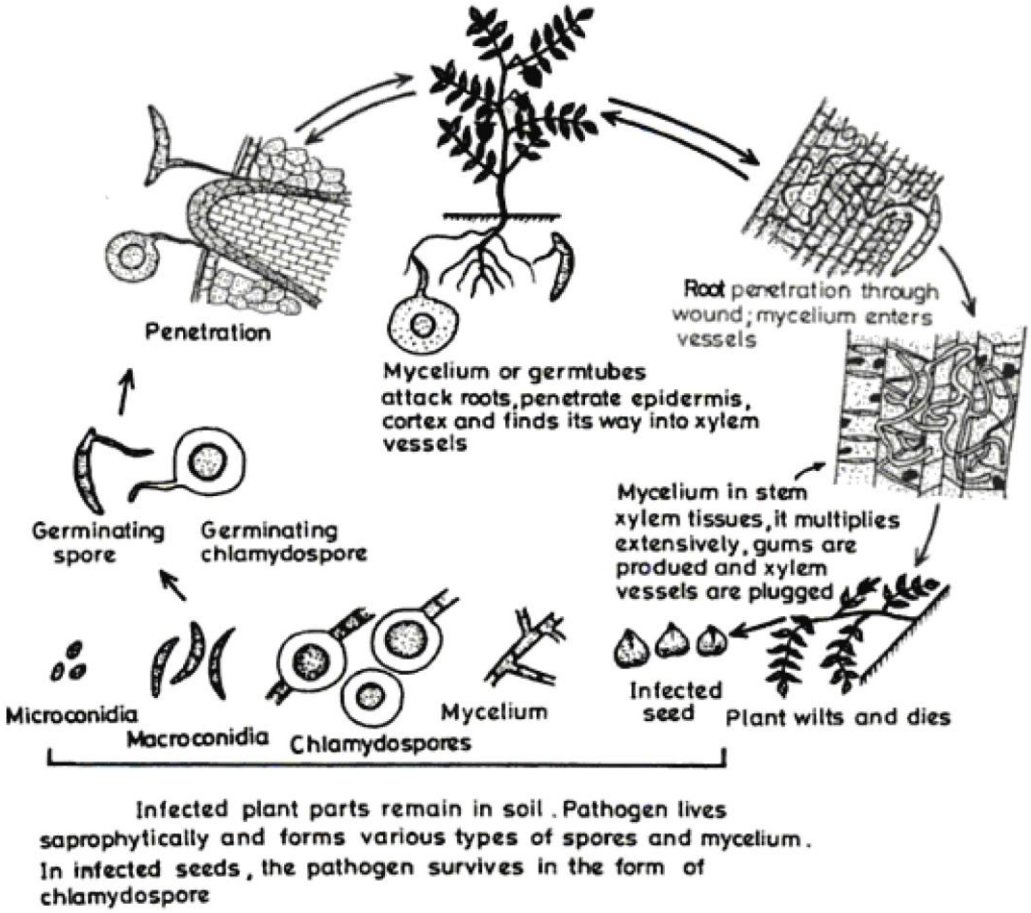

Fig 1 : Disease cycle of *F. oxysporum* f. sp. ciceris Jalali *et al.*, (1992)

Genetics of Resistance against Fusarium Wilt

Fusarium oxysporum f.sp. Ciceris exhibits diverse pathogenic races, such as races o, 1A, 1B/C, 2, 3, 4, 5 and 6 [19]. These different races likely have variations in their ability to infect and damage chickpea plants. Wilt shows two types of symptoms such as early yellowing and late wilting [14]. Early yellowing causes slower foliar yellowing, vascular discoloration, and late plant death and is associated with races 0 and 1B/C [21,22]. Severe symptoms brought on by late wilting include flaccidity, vascular discolouration, chlorosis (yellowing), and premature plant death [20] and is associated with races 1A, 2, 3, 4, 5, and 6 [21].

Resistance to race 1A can be governed by three distinct genes: h1, h2, and H3 [24,25]. Late wilting resistance can be seen in chickpea plants carrying any one of these genes (h1, h2, or H3).. Total resistance to race 1A can be achieved by having any two of these genes in combination (h1h2, h1H3, or h2H3) [24,25,26]. This means that chickpea plants carrying two of these resistance genes are fully resistant to race 1A. Resistance is typically digenic or monogenic to race 0 [83]. Monogenic resistance exists to race 3 [84], a single gene governs it. A single recessive gene is responsible for race 2 resistance [85]. Recessive and digenic resistance exists to race 4 [86]. A single gene controls resistance to race 5. Monogenic resistance controlled by a single gene, similar to race 3 and race 5 [53].

A cluster of genes on linkage group 2 of the chickpea map, comprising foc-0, foc-1, foc-2, foc-3, foc-4 and foc-5, provide Fusarium wilt-resistance to chickpeas [91].
Resistance to race 0 is regulated by two separate genes: Foc01/foc01, flanked by markers $OPJ20_{600}$ and TR 59 [97], which are located on linkage group 3, equivalent to linkage group 2 [91]. The resistant gene, Foc02/foc02, located on Linkage Group 2, is flanked by STMS markers TS 47 and TA 59. A wilt resistance gene, H1, targeting race 1 [92], was first identified and found to be located 7.0 cM from the RAPD markers $CS\text{-}27_{700}$ and $UBC\text{-}170_{550}$ [93]. Two additional QTLs (FW-Q-APR-6-1 and FW-Q-APR-6-2) were mapped for race 1A using the $F_{2:3}$ mapping population of 'C 214' x 'WR 315' [98]. ISSR markers $UBC\text{-}855_{500}$ [94] and $UBC\text{-}825_{1200}$ [95] were associated with Race 4's resistance gene. Co-segregation of $UBC\text{-}855_{500}$ and $CS\text{-}27_{700}$ revealed a tight relationship between resistance genes for race 4 and race 1 [96]. An intraspecific RIL population developed by crossing WR-315 (not susceptible to races 1A, 2, 3, 4, and 5) with C-104 (susceptible) [99] utilised to identify a cluster of five resistance genes (foc-1, foc-2,foc-3, foc-4, and foc-5). Apart from *foc-01* and the two QTLs for race 1A, all other resistance genes against wilt pathogens are in linkage group 2 [93].

The five genes were grouped together and spread across 8.2 cM on linkage group 2 of the garbanzo linkage map. The resistance gene cluster was 2.952 Mb, based on an estimate of 360 kb per cM [91]. Among the five genes, foc-1 and foc-5 were 2.0 cM apart, whereas foc-5 was flanked by foc-3 at 3.4 cM distance. The separation between foc-1 and foc-3 was calculated to be 5.4 cM. Foc-3 and foc-2 were separated by 1.0 cM, whereas foc-2 and foc-4 were separated by 1.8 cM. The distance between two genes (foc-1 and foc-4) at the cluster's opposing ends was 8.2 cM.Two sub-clusters may be distinguished within the resistance gene cluster. Genes foc-4, foc-2, and foc-3 clustered together at 2.8 cM, while foc-5 and foc-1 clustered at 2.0 cM each. The two subclusters were separated by 3.4 cM [93].

Table 2 : The Source of resistance to different races:

Fusarium Race	Name of Resistance Gene	Number and Nature of Wilt Resistance Gene	Effect of Resistance Gene on Wilting	Symptoms	Linkage Group/ Chromosome	Markers Linked to the Resistance Genes/QTLs
0	*foc-0_1/Foc-0_1* [48,70] *foc-0_2/Foc-0_2* [64,69]	Monogenic or digenic	Complete resistance	Yellowing	LG5 [47] LG2 [64]	OPJ20600 (3.0) and TR59 (2.0) ; H2I20, CaGM20820, CaGM20889 and TS43. TA59 and TS47 [49,64,70]
1A	*h1 (syn foc-1)* *h2* [55,56] *H3*[54]	Trigenic	Late wilting Late wilting Late wilting	Wilting	LG2 [46]	H3A12 (3.9) and TA110 (2.1) ; TA59 (4.4), TA96 (4.9), TA27 (4.9) and CS27A (4.9) ; TR19, TA194 and TA660 [49,69]
1B/C	-	-	-	Yellowing	-	-

2	*foc-2* [50]	Monogenic	Complete resistance	Wilting	LG2 [65]	TA96 (1.5), TA27 (1.5), TR19 (4.9) and CS27A (1.5). H3A12 (2.7); TA110 and TA37 [49,65]
3	*foc-3/Foc-3* [53]	Monogenic	Complete resistance	Wilting	LG2 [46]	TA59 (0.5), TA96 (0.5), TA27 (0.5) and CS27A (0.5) ; H1B06y (0.2) and TA194 (0.7) [49,65]
4	*foc-4* [50] Two recessive genes [51]	Monogenic recessive	Complete resistance	Wilting	LG2 [64]	TA59 (3.8), TA96 (3.3), TA27 (3.3) and TR19 (3.1). CS27 (3.7). R-2609-1 (2.0) and OP-U17-1 (4.1) [66,68]
5	*foc-5/Foc-5* [52]	Monogenic	Complete resistance	Wilting	LG2 [66]	TA59 (2.4), TA96 (2.9), and CS27A (2.9); TA27 (2.9) ; SPA and PRP-RGA1 [66,67]
6	-	-	-	Wilting	-	-

Strategies for Screening to Identify Wilt-Resistant Genotypes

Identifying wilt-resistant genotypes in chickpeas involves a combination of field-based and laboratory screening strategies. Wilt diseases in chickpeas are often caused by soil borne pathogens like Fusarium oxysporum and result in significant yield losses. These are some of the widely recognised screening techniques for identification of wilt-resistant genotype .

Field screening

Resistance to wilt genotypes is commonly screened using the sick plot approach of wilt because of its ability to examine many genetic materials in the field [100]. The major criteria for evaluation are disease symptoms, which are confirmed by re-isolating the organism that causes it. Inoculum requirements vary depending on race, habitat, and crop maturity.

In field screening for wilt resistance, test genotypes are planted next to a susceptible cultivar ('JG 62') as a line of indication that appears after every two to four test entries to ensure uniformity of the inoculum. In addition, resistant chickpea genotypes such as 'WR 315' (ICC 11322) and 'JG 74' are interspersed every ten rows to identify possible confounding infections. 'WR 315' resists other FOC races except race 3, and 'JG 74' resists all races except race 2, making them significant resistance sources [101,102]. By providing a thorough evaluation of resistance in natural environments, this method helps to identify robust genotypes. It provides genotype performance data under various pathogen strains and inoculum concentrations, which is essential for breeding programs aimed at improving chickpea cultivar resistance to Fusarium wilt.
The combination of vulnerable and resistant checks enhances the reliability and precision of field screening results, enabling more effective resistance breeding techniques [103,104].

Screening under controlled conditions

Green houses screening

A standardised pot culture approach has been developed for screening chickpea germplasm in greenhouses, [105] ensuring 90% wilt in susceptible lines. However, frequent watering can cause soil compaction, which can impair the association with field performance. Sub-irrigation reduces compaction and keeps the surface soil dry, simulating field conditions [106]. Perlite can be used to replace soil in pots, and all test plants can be infected simultaneously by root cutting and dipping in spore solution [107]. Root dip inoculation is another method [108,109]. The categorization of wilted plants into early, late, and resistant groups is made difficult by the challenging task of maintaining equal inoculum density in each sick plot.

Laboratory screening

The technique of artificial screening for chickpeas [110,111] was established to provide uniform inoculum loads at the identical stages of vegetative development. Root damage prior to inoculation ensures that all plants have equal infection potential [112]. Additionally, it has been suggested that a pollen bioassay is a quick and easy method of identifying resistant, late wilting, and susceptible genotypes.

Enhancing chickpea resistance genetically through breeding techniques.

Breeding methods for crop improvement in chickpea

Breeding efforts have significantly reduced the impact of Fusarium wilt on chickpea crops. Typically, chickpea breeding programs encompass three key phases:

1. Genetic studies, which serves as the foundation of the breeding program.
2. selection for disease resistance and desirable plant varieties within that variety.
3. The selected lines are evaluated for commercial production [113].

Hybridization crosses have been widely adopted in chickpea breeding programs, especially when Desi and Kabuli types hybridise intraspecifically with distinct genetic backgrounds [114]. In Kabuli-type breeding programmes, Desi parents are used to add crucial genes that resist Fusarium wilt, while Kabuli parents contribute to enhancing seed size and quality in Desi breeding initiatives [115].

ICRISAT found 165 sources of resistance after screening approximately 13,500 Desi germplasm accessions for Fusarium wilt resistance [116]. Similarly, 5174 Kabuli germplasm accessions were evaluated for resistance by ICARDA, identifying 110 resistant lines [117].

Primarily, resistance to Foc races has been found in wild Cicer spp. and Desi germplasm. Accessions of C. bijigum, C. cuneatum, and C. judaicum were shown to have combined resistance against races 0 and 5, but accessions of C. canariense and C. chorassanicum demonstrated resistance to race 0 but sensitivity to race 5. Additionally, many C. pinnatifidum accessions were resistant to race 0 but sensitive to race 5 [118].

Conventional Breeding for Wilt Resistance:

Chickpea, being a highly self-pollinated crop, lends itself well to conventional breeding methods. The simple inheritance pattern of wilt resistance makes conventional techniques such as back-crossing and recombination breeding effective. Recombination breeding involves controlled crossing between superior genotypes and wilt-resistant donors, followed by pedigree selection across segregating generations. This approach has been widely employed to incorporate wilt resistance (WR) into chickpeas.

However, transferring desirable alleles can be challenging due to the complexity of tracking desired and undesired alleles in breeding lines. Advanced-backcross QTL-based breeding (AB-breeding) offers a solution by facilitating the controlled introduction of novel alleles from wild relatives into cultivated varieties [119].

Marker-assisted gene pyramiding enables the combining many beneficial wilt-resistant genes into one genotype, leveraging the established tight association between markers and target traits in chickpeas [120].

Incorporating Genomic Techniques into Strategies for Breeding Chickpeas:

Genetic Maps:

Chickpea genetic mapping is based on the segregation and recombination concepts found in Mendelian genetics. In the beginning, isozymes from F2 populations developed through interspecific crossings [121] were analyzed in order to create genetic maps. Together with Quantitative Trait Loci linked to traits including blooming duration, agronomics, and resistance to the ascochyta blight [125,126], these maps discovered genes controlling a variety of phenotypes, including growth habit, fusarium wilt resistance, double podding, and flower color [122,123,124]. Most of the maps, which are abundant with markers, were generated from populations obtained via crosses with C. reticulatum [127].

Microsatellite markers were used in populations from interspecific crosses in subsequent research to harness more genetic variability among genotypes of chickpeas [128]. Next-generation sequencing technology enabled the first transcriptome analysis of the chickpea genome [129], which also made it possible to generate extensive genetic maps with a vast array of molecular markers derived from transcriptome data [130]. For the purpose of classifying, describing, and screening infections and illnesses, molecular markers are essential. It is common practice to classify and identify fungus using internal transcribed spacer markers [131].

Marker-Assisted Breeding:

Marker-assisted selection is a powerful tool that can improve genetic variations and simplify the selection process for complex traits. It is particularly beneficial for prolonged phenotypic examinations. MAS improves the selection of desirable traits, which accelerates rapid variety development, particularly in disease resistance, which is notably a quantitative traits [132]. By pyramiding resistance genes, methods such as marker-assisted backcrossing (MABC) speed up genetic recovery and variety development [133]. Foreground selection, background selection, and recombinant

selection are important phases in MABC that facilitate the transmission of gene of interest or QTLs (quantitative trait loci) while eliminating undesirable traits.

Future breeding operations depend critically on maintaining stability across many genotypes to choose disease-resistant and high-yielding chickpea lines. Although chickpea genotypes have a high degree of resistance to wilt disease, a unique strategy utilizing PCR-based markers in association with field screening aims to discover and assess wilt-resistant lines against the parasite F. oxysporum sp. Ciceris [134]. Extensive insights were obtained from germplasm classified according to disease susceptibility in adult and seedling stages. This revealed greater susceptibility to wilt disease, maybe due certain accessions possessed slow wilting resistance.

Significant differences were found in the genotypes of chickpeas from both native and foreign sources at the seedling and reproductive stages, according to statistical analysis. In order to more effectively identify robust lines against Fusarium wilt disease, gene pyramiding and molecular breeding [135] can be enabled by the use of molecular markers for chickpea screening. Using a variety of markers (RAPD and SSR) [136], previous research has determined the genetic linkage of resistance genes for distinct FOC races (FOC 1, 2, 3, 4, and 5) in inbred chickpea lines derived from resistant and susceptible paternal combinations.

Chickpea breeding projects might be expedited by combining MAS with molecular markers, especially for improving disease resistance to Fusarium wilt. The use of marker-assisted methods into breeding strategies is essential for sustainable crop improvement since it not only accelerates variety production but also improves precision and efficiency in choosing desirable characteristics..

Conclusion

Fusarium wilt is a serious threat to worldwide chickpea production due to the pathogen's persistent existence in soil and the presence of diverse races. Disease severity is determined by inoculum density, warm soil temperatures, and chickpea susceptibility. Sustainable management incorporates measures including optimal planting dates, host resistance, and early disease detection. Molecular techniques help in diagnosis, whereas resistant chickpea cultivars are still a cost-effective management tool. Advances in discovering resistant germplasm and understanding race-specific resistance genetics enable the creation of cultivars that are more stable and resistant to a variety of illnesses and environmental challenges. Biocontrol treatments, along with pre-planting procedures like pathogen-free seeds and sanitation, improve integrated wilt management.

References:

1. Jendoubi W, Bouhadida M, Boukteb A, Béji M, Kharrat M. Fusarium wilt affecting chickpea crop. Agriculture. 2017 Mar 9;7(3):23.
2. Varshney RK, Song C, Saxena RK, Azam S, Yu S, Sharpe AG, Cannon S, Baek J, Rosen BD, Tar'an B, Millan T. Draft genome sequence of chickpea (Cicer

arietinum) provides a resource for trait improvement. Nature biotechnology. 2013 Mar;31(3):240-6.
3. Kushwaha K, Tyagi PK, Shridhar AK, Tyagi A. Effect of Nipping and Varieties on Yield Attributes and Yield of Chickpea (Cicer arietinum L.).
4. Malhotra RS, Pundir RP, Kaiser WJ. Cicer species-conserved resources, priorities for collection and future prospects. InLinking Research and Marketing Opportunities for Pulses in the 21st Century: Proceedings of the Third International Food Legumes Research Conference 2000 (pp. 603-611). Springer Netherlands.
5. DS IS, Dilipbhai IK. Introduction to Legumes: Overview and its Importance for Food Security.
6. Asati R, Tripathi MK, Tiwari S, Yadav RK, Tripathi N. Molecular breeding and drought tolerance in chickpea. Life. 2022 Nov 11;12(11):1846.
7. Thavarajah D, Thavarajah P, Gupta DS. Pulses biofortification in genomic era: multidisciplinary opportunities and challenges. Legumes in the omic era. 2014:207-20.
8. Jukanti AK, Gaur PM, Gowda CL, Chibbar RN. Nutritional quality and health benefits of chickpea (Cicer arietinum L.): a review. British Journal of Nutrition. 2012 Aug;108(S1):S11-26.
9. Verma S, Madala N, Halavath SK, Suresh BG, Lavanya GR. Evaluation of chickpea (Cicer arietinum L.) germplasm for yield and yield attributing traits in Eastern Plain Zone of Uttar Pradesh. Int. J. Curr. Microbiol. Appl. Sci. 2020;9(10):1944-56.
10. Reddy MV, Haware MP, Nene YL, McDonald D. Recent Advances In Fungal Diseases Of Chickpea And Pigeonpea.
11. Gopalakrishnan S, Pande S, Sharma M, Humayun P, Kiran BK, Sandeep D, Vidya MS, Deepthi K, Rupela O. Evaluation of actinomycete isolates obtained from herbal vermicompost for the biological control of Fusarium wilt of chickpea. Crop Protection. 2011 Aug 1;30(8):1070-1078.
12. Jaiswal RK, Kirar BS, Mishra RK. Fusarium Wilt Disease of Chickpea (Cicer arietinum L.). Diseases of Pulse Crops and their Management.:1.
13. del Mar Jiménez-Gasco M, Pérez-Artés E, Jiménez-Diaz RM. Identification of pathogenic races 0, 1B/C, 5, and 6 of Fusarium oxysporum f. sp. ciceris with random amplified polymorphic DNA (RAPD). European Journal of Plant Pathology. 2001 Feb;107:237-48.
14. Sharma KD, Muehlbauer FJ. Fusarium wilt of chickpea: physiological specialization, genetics of resistance and resistance gene tagging. Euphytica. 2007 Sep;157:1-4.
15. Singh PK, Singh M, Agnihotri VK, Vyas D. Arbuscular mycorrhizal fungi: biocontrol against Fusarium wilt of chickpea. International Journal of Scientific Research Publication. 2013;3:1-5.
16. Sharma M, Nagavardhini A, Thudi M, Ghosh R, Pande S, Varshney RK. Development of DArT markers and assessment of diversity in Fusarium oxysporum f. sp. ciceris, wilt pathogen of chickpea (Cicer arietinum L.). BMC genomics. 2014 Dec;15:1-4.
17. Achari SR, Mann RC, Sharma M, Edwards J. Diagnosis of Fusarium oxysporum f. sp. ciceris causing Fusarium wilt of chickpea using loop-mediated isothermal amplification (LAMP) and conventional end-point PCR. Scientific Reports. 2023 Feb 14;13(1):2640.

18. Yadav RK, Tripathi MK, Tiwari S, Tripathi N, Asati R, Patel V, Sikarwar RS, Payasi DK. Breeding and genomic approaches towards development of Fusarium wilt resistance in chickpea. Life. 2023 Apr 11;13(4):988.
19. Roy S, Kumar U, Kumari M, Kumari D, Yadav M, Prasad R, Kumar P, Kumari P. A review on fusarium wilt disease of chickpea and different strategies for its management. Journal of Pharmacognosy and Phytochemistry. 2017;6(6):2605-13.
20. Jaiswal RK, Kirar BS, Mishra RK. Fusarium Wilt Disease of Chickpea (Cicer arietinum L.). Diseases of Pulse Crops and their Management.:1.
21. Achari SR, Mann RC, Sharma M, Edwards J. Diagnosis of Fusarium oxysporum f. sp. ciceris causing Fusarium wilt of chickpea using loop-mediated isothermal amplification (LAMP) and conventional end-point PCR. Scientific Reports. 2023 Feb 14;13(1):2640.
22. Sharma KD. Fusarium Wilt of Chickpea: Breeding and Genomic Approaches for Designing Wilt-Resistant Chickpea. InDiseases in Legume Crops: Next Generation Breeding Approaches for Resistant Legume Crops 2023 Oct 26 (pp. 61-84). Singapore: Springer Nature Singapore.
23. Mazen MM, Ibrahim SD. Biodiversity of Fusarium oxysporum isolated from diseased chickpea and detection of resistance sources to some Egyptian chickpea cultivars. Egyptian Journal of Phytopathology. 2021 Jun 12;49(1):182-96.
24. Upadhyaya HD, Smithson JB, Haware MP, Kumar J. Resistance to wilt in chickpea. II. Further evidence for two genes for resistance to race 1. Euphytica. 1983 Nov;32(3):749-55.Warda J, Mariem B, Amal B, Mohamed B, Mohamed K. Fusarium wilt affecting chickpea crop. Agriculture. 2017;7:23.
25. Warda J, Mariem B, Amal B, Mohamed B, Mohamed K. Fusarium wilt affecting chickpea crop. Agriculture. 2017;7:23.
26. Singh H, Kumar J, Haware MP, Smithson JB. Genetics of resistance to Fusarium wilt in chickpeas. Genetics and plant pathogenesis. Blackwell, Oxford. 1987:339-42.
27. Yadav RK, Tripathi MK, Tiwari S, Tripathi N, Asati R, Patel V, Sikarwar RS, Payasi DK. Breeding and genomic approaches towards development of Fusarium wilt resistance in chickpea. Life. 2023 Apr 11;13(4):988.
28. Saraf CS, Rupela OP, Hegde DM. Biological nitrogen fixation and residual effects of winter grain legumes in rice and wheat cropping systems of the Indo-Gangetic plain.
29. Jha UC, Bohra A, Pandey S, Parida SK. Breeding, genetics, and genomics approaches for improving Fusarium wilt resistance in major grain legumes. Frontiers in Genetics. 2020 Oct 23;11:560972.
30. Jiménez-Díaz RM, Castillo P, del Mar Jiménez-Gasco M, Landa BB, Navas-Cortés JA. Fusarium wilt of chickpeas: Biology, ecology and management. Crop Protection. 2015 Jul 1;73:16-27.
31. Pundir RP, Rao NK, Van den Maesen LJ. Distribution of qualitative traits in the world germplasm of chickpea (Cicer arietinum L.). Euphytica. 1985 Nov;34:697-703.
32. Ahmad F, Gaur PM, Croser J. Chickpea (Cicer arietinum l.). Genetic resources, chromosome engineering, and crop improvement-grain legumes. 2005 Mar 16;1:187-217.
33. Jiménez-Díaz RM, Castillo P, del Mar Jiménez-Gasco M, Landa BB, Navas-Cortés JA. Fusarium wilt of chickpeas: Biology, ecology and management. Crop Protection. 2015 Jul 1;73:16-27.

34. Jimenez-Diaz RM, Trapero-Casas A, de La Colina JC. Races of Fusarium oxysporum f. sp. ciceri infecting chickpeas in southern Spain. InVascular Wilt Diseases of Plants: Basic Studies and Control 1989 (pp. 515-520). Berlin, Heidelberg: Springer Berlin Heidelberg.
35. Kukreja S, Salaria N, Thakur K, Goutam U. Fungal disease management in chickpea: current status and future prospects. Fungi and their role in sustainable development: current perspectives. 2018:293-309.
36. Singh A, Srivastava S, Singh HB. Effect of substrates on growth and shelf life of Trichoderma harzianum and its use in biocontrol of diseases. Bioresource technology. 2007 Jan 1;98(2):470-3.
37. Zaidi RK, Pathak N. Evaluation of seed infection of fungi in Chickpea.
38. Jaiswal RK, Kirar BS, Mishra RK. Fusarium Wilt Disease of Chickpea (Cicer arietinum L.). Diseases of Pulse Crops and their Management.:1.
39. KUSHWAHA DA. FUNGI (Viruses, Bacteria and Mycoplasma). Dr. AK KUSHWAHA; 2020 Apr 21.
40. Haware MP, Nene YL, Rajeshwari R. Eradication of Fusarium oxysporum f. sp. ciceri transmitted in chickpea seed.
41. Mawar R, Mathur M, Tomer AS. Diseases of Arid Legumes: Extent and Management. Diseases of Pulse Crops and their Management. 2021;381.
42. Jalali, BHUSHAN L., and H. A. R. I. Chand. "Chickpea wilt." *Plant diseases of international importance* 1 (1992): 429-444.
43. Agrios GN. Environmental effects on the development of infectious plant disease. Plant Pathology. 2005;5:249-64.
44. Singh GU, Chen W, Rubiales DI, Moore KE, Sharma YR, Gan Y. Diseases and their management. InChickpea breeding and management 2007 (pp. 497-519). Wallingford UK: CABI.
45. Rafiq CM, Mahmood MT, Ahmad M, Ali I, Kaukab S, Shafiq M, Saleem M, Iqbal U. Fusarium wilt's pathogenic studies and disease management: A review. Genetics and Molecular Research. 2020;19.
46. Halila I, Cobos MJ, Rubio J, Millán T, Kharrat M, Marrakchi M, Gil J. Tagging and mapping a second resistance gene for Fusarium wilt race 0 in chickpea. European Journal of Plant Pathology. 2009 May;124:87-92.
47. Cobos MJ, Fernández MJ, Rubio J, Kharrat M, Moreno MT, Gil J, Millan T. A linkage map of chickpea (Cicer arietinum L.) based on populations from Kabuli× Desi crosses: location of genes for resistance to fusarium wilt race 0. Theoretical and Applied Genetics. 2005 May;110:1347-53.
48. Rubio J, Hajj- Moussa E, Kharrat M, Moreno MT, Millan T, Gil J. Two genes and linked RAPD markers involved in resistance to Fusarium oxysporum f. sp. ciceris race 0 in chickpea. Plant Breeding. 2003 Apr;122(2):188-91.
49. Sampaio AM, Araujo SD, Rubiales D, Vaz Patto MC. Fusarium wilt management in legume crops. Agronomy. 2020 Jul 25;10(8):1073.
50. Sharma KD, Chen W, Muehlbauer FJ. Genetics of chickpea resistance to five races of Fusarium wilt and a concise set of race differentials for Fusarium oxysporum f. sp. ciceris. Plant disease. 2005 Apr;89(4):385-90.
51. Tullu A, Kaiser WJ, Kraft JM, Muehlbauer FJ. A second gene for resistance to race 4 of Fusarium wilt in chickpea and linkage with a RAPD marker. Euphytica. 1999 Sep;109:43-50.
52. Tullu AB, Muehlbauer FJ, Simon CJ, Mayer MS, Kumar JA, Kaiser WJ, Kraft JM. Inheritance and linkage of a gene for resistance to race 4 of fusarium wilt and RAPD markers in chickpea. Euphytica. 1998 Jul;102:227-32.

53. Tekeoglu M, Tullu A, Kaiser WJ, Muehlbauer FJ. Inheritance and linkage of two genes that confer resistance to Fusarium wilt in chickpea. Crop science. 2000 Sep;40(5):1247-51.
54. Sharma KD, Muehlbauer FJ. Fusarium wilt of chickpea: physiological specialization, genetics of resistance and resistance gene tagging. Euphytica. 2007 Sep;157:1-4.
55. Upadhyaya HD, Haware MP, Kumar J, Smithson JB. Resistance to wilt in chickpea. I. Inheritance of late-wilting in response to race 1. Euphytica. 1983 Jun;32:447-52.
56. Singh H, Kumar J, Smithson JB, Haware MP. Complementation between genes for resistance to race 1 of Fusarium oxyspomm f. sp. ciceri in chickpea. Plant pathology. 1987 Dec;36(4):539-43.
57. Anjaiah V, Cornelis P, Koedam N. Effect of genotype and root colonization in biological control of fusarium wilts in pigeonpea and chickpea by Pseudomonas aeruginosa PNA1. Canadian Journal of Microbiology. 2003 Feb 1;49(2):85-91.
58. Joseph B, Patra RR, Lawrence R. Characterization of plant growth promoting rhizobacteria associated with chickpea (Cicer arietinum L.). International Journal of Plant Production. 2007 Sep 1;1(2):141-52.
59. Fridlender M, Inbar J, Chet I. Biological control of soilborne plant pathogens by a β-1, 3 glucanase-producing Pseudomonas cepacia. Soil Biology and Biochemistry. 1993 Sep 1;25(9):1211-21.
60. Dehariya K, Shukla A, Sheikh IA, Vyas D. Trichoderma and arbuscular mycorrhizal fungi based biocontrol of Fusarium udum butler and their growth promotion effects on pigeon pea. Journal of Agricultural Science and Technology. 2015 Mar 10;17(2):505-17.
61. Shukla A, Dehariya K, Vyas D, Jha A. Interactions between arbuscular mycorrhizae and Fusarium oxysporum f. sp. ciceris: effects on fungal development, seedling growth and wilt disease suppression in Cicer arietinum L. Archives of Phytopathology and Plant Protection. 2015 Feb 7;48(3):240-52.
62. Fravel D, Olivain C, Alabouvette C. Fusarium oxysporum and its biocontrol. New phytologist. 2003 Mar;157(3):493-502.
63. Larkin RP, Fravel DR. Efficacy of various fungal and bacterial biocontrol organisms for control of Fusarium wilt of tomato. Plant disease. 1998 Sep;82(9):1022-8.
64. Halila I, Cobos MJ, Rubio J, Millán T, Kharrat M, Marrakchi M, Gil J. Tagging and mapping a second resistance gene for Fusarium wilt race 0 in chickpea. European Journal of Plant Pathology. 2009 May;124:87-92.
65. Gowda SJ, Radhika P, Kadoo NY, Mhase LB, Gupta VS. Molecular mapping of wilt resistance genes in chickpea. Molecular breeding. 2009 Sep;24:177-83.
66. Winter P, Benko-Iseppon AM, Hüttel B, Ratnaparkhe MI, Tullu AB, Sonnante GA, Pfaff TH, Tekeoglu MU, Santra DI, Sant VJ, Rajesh PN. A linkage map of the chickpea (Cicer arietinum L.) genome based on recombinant inbred lines from a C. arietinum× C. reticulatum cross: localization of resistance genes for fusarium wilt races 4 and 5. Theoretical and Applied Genetics. 2000 Nov;101:1155-63.
67. Cobos MJ, Winter P, Kharrat M, Cubero JI, Gil J, Millan T, Rubio J. Genetic analysis of agronomic traits in a wide cross of chickpea. Field Crops Research. 2009 Mar 15;111(1-2):130-6.
68. Benko-Iseppon AM, Winter P, Huettel B, Staginnus C, Muehlbauer FJ, Kahl G. Molecular markers closely linked to fusarium resistance genes in chickpea show

significant alignments to pathogenesis-related genes located on Arabidopsis chromosomes 1 and 5. Theoretical and Applied Genetics. 2003 Jul;107:379-86.
69. Mayer MS, Tullu A, Simon CJ, Kumar J, Kaiser WJ, Kraft JM, Muehlbauer FJ. Development of a DNA marker for Fusarium wilt resistance in chickpea. Crop Science. 1997 Sep;37(5):1625-9.
70. Cobos MJ, Fernández MJ, Rubio J, Kharrat M, Moreno MT, Gil J, Millan T. A linkage map of chickpea (Cicer arietinum L.) based on populations from Kabuli× Desi crosses: location of genes for resistance to fusarium wilt race 0. Theoretical and Applied Genetics. 2005 May;110:1347-53.
71. Singh DK, Jha MM. Effect of fungicidal treatment against chickpea wilt caused by Fusarium oxysporum f. sp. ciceri.
72. Haware MP, Nene YL, Rajeshwari R. Eradication of Fusarium oxysporum f. sp. ciceri transmitted in chickpea seed.
73. Pande S, Rao JN, Sharma M. Establishment of the chickpea wilt pathogen Fusarium oxysporum f. sp. ciceris in the soil through seed transmission. The Plant Pathology Journal. 2007;23(1):3-6.
74. Animisha A, Zacharia S, Jaiswal KK, Pandey P. Integrated management of chickpea wilt incited by Fusarium oxysporum f. sp. ciceris.
75. Gaur PM, Jukanti AK, Varshney RK. Impact of genomic technologies on chickpea breeding strategies. Agronomy. 2012 Aug 23;2(3):199-221.
76. Haware MP, Nene YL. Races of Fusarium oxysporum f. sp. ciceri. Plant disease. 1982;66(9):809-10.
77. Trapero Casas A, Jiménez Díaz RM. Fungal wilt and root rot diseases of chickpea in southern Spain.
78. Jimenez-Diaz RM, Alcala A, Hervás A, Trapero-Casas JL. Pathogenic variability and host resistance in the Fusarium oxysporum f. sp. ciceris/Cicer arietinum pathosystem.
79. Cabrera De La Colina J, Trapero-Casas A, Jiménez-Díaz RM. Races of Fusarium oxysporum f. sp. ciceri in Andalucía, southern Spain. International chickpea newsletter. 1985.
80. Halila MH, Strange RN. Identification of the causal agent of wilt of chickpea in Tunisia as Fusarium oxysporum f. sp. ciceri race 0. Phytopathologia mediterranea. 1996 Aug 1:67-74.
81. Rahman ML, Haware MP, Mian IH, Akanda AM. Races of Fusarium oxysporum f. sp. ciceri causing chickpea wilt in India.
82. del Mar Jiménez-Gasco M, Pérez-Artés E, Jiménez-Diaz RM. Identification of pathogenic races 0, 1B/C, 5, and 6 of Fusarium oxysporum f. sp. ciceris with random amplified polymorphic DNA (RAPD). European Journal of Plant Pathology. 2001 Feb;107:237-48.
83. Tekeoglu M, Tullu A, Kaiser WJ, Muehlbauer FJ. Inheritance and linkage of two genes that confer resistance to Fusarium wilt in chickpea. Crop science. 2000 Sep;40(5):1247-51.
84. Sharma M, Telangre R, Ghosh R, Pande S. Multi-environment field testing to identify broad, stable resistance to sterility mosaic disease of pigeonpea. Journal of General Plant Pathology. 2015 May;81:249-59.
85. Gowda SJ, Radhika P, Kadoo NY, Mhase LB, Gupta VS. Molecular mapping of wilt resistance genes in chickpea. Molecular breeding. 2009 Sep;24:177-83.
86. Tullu AB, Muehlbauer FJ, Simon CJ, Mayer MS, Kumar JA, Kaiser WJ, Kraft JM. Inheritance and linkage of a gene for resistance to race 4 of fusarium wilt and RAPD markers in chickpea. Euphytica. 1998 Jul;102:227-32.

87. Choudhary AK, Kumar S, Patil BS, Sharma M, Kemal S, Ontagodi TP, Datta S, Patil P, Chaturvedi SK, Sultana R, Hegde VS. Narrowing yield gaps through genetic improvement for Fusarium wilt resistance in three pulse crops of the semi-arid tropics. SABRAO Journal of Breeding and Genetics. 2013;45(03):341-70.
88. Srivastava SK, Singh SN, Khare MN. ASSESSMENT OF YIELD LOSSES IN SOME PROMISING GRAM CULTEV ARS DUE TO FUSAJ (JUM WILT. Indian J. Plant Proto. 1984;12:125-6.
89. Choudhary AK, Kumar S, Patil BS, Sharma M, Kemal S, Ontagodi TP, Datta S, Patil P, Chaturvedi SK, Sultana R, Hegde VS. Narrowing yield gaps through genetic improvement for Fusarium wilt resistance in three pulse crops of the semi-arid tropics. SABRAO Journal of Breeding and Genetics. 2013;45(03):341-70.
90. Singh S. PM Gaur, SK Chaturvedi, NP Singh, and JS Sandhu. Broadening the genetic base of grain legumes. 2014 Oct 28;51.
91. Winter P, Benko-Iseppon AM, Hüttel B, Ratnaparkhe MI, Tullu AB, Sonnante GA, Pfaff TH, Tekeoglu MU, Santra DI, Sant VJ, Rajesh PN. A linkage map of the chickpea (Cicer arietinum L.) genome based on recombinant inbred lines from a C. arietinum× C. reticulatum cross: localization of resistance genes for fusarium wilt races 4 and 5. Theoretical and Applied Genetics. 2000 Nov;101:1155-63.
92. Mayer MS, Tullu A, Simon CJ, Kumar J, Kaiser WJ, Kraft JM, Muehlbauer FJ. Development of a DNA marker for Fusarium wilt resistance in chickpea. Crop Science. 1997 Sep;37(5):1625-9.
93. Sharma KD, Muehlbauer FJ. Fusarium wilt of chickpea: physiological specialization, genetics of resistance and resistance gene tagging. Euphytica. 2007 Sep;157:1-4.
94. Ratnaparkhe MB, Santra DK, Tullu A, Muehlbauer FJ. Inheritance of inter-simple-sequence-repeat polymorphisms and linkage with a fusarium wilt resistance gene in chickpea. Theoretical and Applied Genetics. 1998 Mar;96(3):348-53.
95. Ratnaparkhe MB, Tekeoglu M, Muehlbauer FJ. Inter-simple-sequence-repeat (ISSR) polymorphisms are useful for finding markers associated with disease resistance gene clusters. Theoretical and Applied Genetics. 1998 Sep;97:515-9.
96. Choudhary AK, Kumar S, Patil BS, Sharma M, Kemal S, Ontagodi TP, Datta S, Patil P, Chaturvedi SK, Sultana R, Hegde VS. Narrowing yield gaps through genetic improvement for Fusarium wilt resistance in three pulse crops of the semi-arid tropics. SABRAO Journal of Breeding and Genetics. 2013;45(03):341-70.
97. Cobos MJ, Fernández MJ, Rubio J, Kharrat M, Moreno MT, Gil J, Millan T. A linkage map of chickpea (Cicer arietinum L.) based on populations from Kabuli× Desi crosses: location of genes for resistance to fusarium wilt race 0. Theoretical and Applied Genetics. 2005 May;110:1347-53.
98. Sabbavarapu MM, Sharma M, Chamarthi SK, Swapna N, Rathore A, Thudi M, Gaur PM, Pande S, Singh S, Kaur L, Varshney RK. Molecular mapping of QTLs for resistance to Fusarium wilt (race 1) and Ascochyta blight in chickpea (Cicer arietinum L.). Euphytica. 2013 Sep;193:121-33.
99. Sharma KD, Muehlbauer FJ. Genetic mapping of Fusarium oxysporum f. sp. ciceris race-specific resistance genes in chickpea (Cicer arietinum L.). InAbstract

of the International food legume research conference—IV, Indian Agricultural Research Institute, New Delhi, India 2005 (pp. 18-22).
100. Infantino A, Kharrat M, Riccioni L, Coyne CJ, McPhee KE, Grünwald NJ. Screening techniques and sources of resistance to root diseases in cool season food legumes. Euphytica. 2006 Jan;147:201-21.
101. Nene YL, Haware MP. screening chickpea for resistance to Wilt. Plant Disease. 1980 Jan 1;64(4):379-80.
102. Sharma KD, Muehlbauer FJ. Genetic mapping of Fusarium oxysporum f. sp. ciceris race-specific resistance genes in chickpea (Cicer arietinum L.). InAbstract of the International food legume research conference—IV, Indian Agricultural Research Institute, New Delhi, India 2005 (pp. 18-22).
103. Deshmukh RB, Patil JV, Mhase LB, Kamble MS, Deshmukh DV, Jamadagni BM. Digvijay—An High Yielding, Wilt Resistant Chickpea Variety for Maharashtra State. J. Maharashtra agric. Univ. 2010;35:367-70.
104. Babu DR, Ravikumar RL. Genetic evidence for resistance to Fusarium wilt of pollen grains in chickpea (Cicer arietinum L.). Current Science. 2009 Mar 25:811-5.
105. Landa BB, Navas-Cortés JA, Jiménez-Díaz RM. Integrated management of Fusarium wilt of chickpea with sowing date, host resistance, and biological control. Phytopathology. 2004 Sep;94(9):946-60.
106. Phillips JC. A distinct race of chickpea wilt in California.
107. Ratnaparkhe MB, Santra DK, Tullu A, Muehlbauer FJ. Inheritance of inter-simple-sequence-repeat polymorphisms and linkage with a fusarium wilt resistance gene in chickpea. Theoretical and Applied Genetics. 1998 Mar;96(3):348-53.
108. Pande S, Sharma M, Avuthu N, Telangre R. High Throughput Phenotyping of Chickpea Diseases: Stepwise Identification of Host Plant Resistance. Information Bulletin No. 92. International Crops Research Institute for Semi-Arid Tropics; 2012.
109. Pande S, Sharma M, Avuthu N, Telangre R. High Throughput Phenotyping of Chickpea Diseases: Stepwise Identification of Host Plant Resistance. Information Bulletin No. 92. International Crops Research Institute for Semi-Arid Tropics; 2012.
110. Tullu A. Genetics of fusarium wilt resistance in chickpea. Washington State University; 1996.
111. Sharma KD, Chen W, Muehlbauer FJ. Genetics of chickpea resistance to five races of Fusarium wilt and a concise set of race differentials for Fusarium oxysporum f. sp. ciceris. Plant disease. 2005 Apr;89(4):385-90.
112. Jiménez-Díaz RM, Castillo P, del Mar Jiménez-Gasco M, Landa BB, Navas-Cortés JA. Fusarium wilt of chickpeas: Biology, ecology and management. Crop Protection. 2015 Jul 1;73:16-27.
113. Muzquiz M, Wood JA, Yadav S, Redden B, Chen W, Sharma B. Chickpea breeding and management. CABI International: Wallingford, UK. 2007.
114. Berrada AF, Shivakumar BG, Yaduraju NT. Chickpea in cropping systems. Chickpea breeding and management. 2007 May 9:193.
115. Yadav SS, Chen W, editors. Chickpea breeding and management. CABI; 2007.
116. Haware MP, Nene YL, Pundir RP, Rao JN. Screening of world chickpea germplasm for resistance to fusarium wilt. Field Crops Research. 1992 Jul 1;30(1-2):147-54.

117. Singh KB. Chickpea (Cicer arietinum L.). Field crops research. 1997 Jul 1;53(1-3):161-70.
118. Kaiser WJ, Alcala-Jimenez AR, Hervas-Vargas A, Trapero-Casas JL, Jimenez-Diaz RM. Screening of wild Cicer species for resistance to races 0 and 5 of Fusarium oxysporum f. sp. ciceris.
119. Tanksley SD, Nelson JC. Advanced backcross QTL analysis: a method for the simultaneous discovery and transfer of valuable QTLs from unadapted germplasm into elite breeding lines. Theoretical and Applied Genetics. 1996 Feb;92:191-203.
120. Kumar J, Choudhary AK, Solanki RK, Pratap A. Towards marker- assisted selection in pulses: a review. Plant breeding. 2011 Jun;130(3):297-313.
121. Gaur PM, Slinkard AE. Genetic control and linkage relations of additional isozyme markers in chick-pea. Theoretical and Applied Genetics. 1990 Nov;80:648-56.
122. Sharma KD, Muehlbauer FJ. Fusarium wilt of chickpea: physiological specialization, genetics of resistance and resistance gene tagging. Euphytica. 2007 Sep;157:1-4.
123. Kazan KM, Muehlbauer FJ, Weeden NE, Ladizinsky G. Inheritance and linkage relationships of morphological and isozyme loci in chickpea (Cicer arietinum L.). Theoretical and Applied Genetics. 1993 May;86:417-26.
124. Simon CJ, Muehibauer FJ. Construction of a chickpea linkage map and its comparison with maps of pea and lentil. Journal of Heredity. 1997 Mar 1;88(2):115-9.
125. Cobos MJ, Rubio J, Fernández- Romero MD, Garza R, Moreno MT, Millán T, Gil J. Genetic analysis of seed size, yield and days to flowering in a chickpea recombinant inbred line population derived from a Kabuli× Desi cross. Annals of Applied Biology. 2007 Aug;151(1):33-42.
126. Cobos MJ, Winter P, Kharrat M, Cubero JI, Gil J, Millan T, Rubio J. Genetic analysis of agronomic traits in a wide cross of chickpea. Field Crops Research. 2009 Mar 15;111(1-2):130-6.
127. Winter P, Benko-Iseppon AM, Hüttel B, Ratnaparkhe MI, Tullu AB, Sonnante GA, Pfaff TH, Tekeoglu MU, Santra DI, Sant VJ, Rajesh PN. A linkage map of the chickpea (Cicer arietinum L.) genome based on recombinant inbred lines from a C. arietinum× C. reticulatum cross: localization of resistance genes for fusarium wilt races 4 and 5. Theoretical and Applied Genetics. 2000 Nov;101:1155-63.
128. Hajibarat Z, Saidi A, Hajibarat Z, Talebi R. Characterization of genetic diversity in chickpea using SSR markers, start codon targeted polymorphism (SCoT) and conserved DNA-derived polymorphism (CDDP). Physiology and molecular biology of plants. 2015 Jul;21:365-73.
129. Garg R, Patel RK, Jhanwar S, Priya P, Bhattacharjee A, Yadav G, Bhatia S, Chattopadhyay D, Tyagi AK, Jain M. Gene discovery and tissue-specific transcriptome analysis in chickpea with massively parallel pyrosequencing and web resource development. Plant Physiology. 2011 Aug 1;156(4):1661-78.
130. Hiremath PJ, Kumar A, Penmetsa RV, Farmer A, Schlueter JA, Chamarthi SK, Whaley AM, Carrasquilla- Garcia N, Gaur PM, Upadhyaya HD, Kavi Kishor PB. Large- scale development of cost- effective SNP marker assays for diversity assessment and genetic mapping in chickpea and comparative mapping in legumes. Plant biotechnology journal. 2012 Aug;10(6):716-32.

131. Yadav RK, Tripathi MK, Tiwari S, Tripathi N, Asati R, Patel V, Sikarwar RS, Payasi DK. Breeding and genomic approaches towards development of Fusarium wilt resistance in chickpea. Life. 2023 Apr 11;13(4):988.
132. Calonnec A, Wiedemann- Merdinoglu S, Deliere L, Cartolaro P, Schneider C, Delmotte F. The reliability of leaf bioassays for predicting disease resistance on fruit: a case study on grapevine resistance to downy and powdery mildew. Plant Pathology. 2013 Jun;62(3):533-44.
133. Gaur PM, Jukanti AK, Varshney RK. Impact of genomic technologies on chickpea breeding strategies. Agronomy. 2012 Aug 23;2(3):199-221.
134. Yu KhinHnin YK, Su Thein ST. Pot screening of chickpea germplasm lines against wilt.
135. Soregaon CD, Ravikumar RL. Marker assisted characterization of wilt resistance in productive chickpea genotypes. Electronic Journal of Plant Breeding. 2010;1(4):1159-63.
136. Sharma KD, Muehlbauer FJ. Fusarium wilt of chickpea: physiological specialization, genetics of resistance and resistance gene tagging. Euphytica. 2007 Sep;157:1-4.