Review Article,

"Genetic Insights into Fusarium Wilt Resistance in Chickpea: Mechanisms and Sustainable Breeding Strategies"

Abstract

"*Fusarium oxysporum* f. sp. *ciceris*, the agent responsible for Fusarium wilt, poses a serious risk to the global production of chickpeas (*Cicer arietinum* L.). There are different races of this soil-borne pathogen, and each one has a different amount of virulence, which makes crop resilience difficult. Early yellowing and late wilting are two indicators of chickpea wilt that cause significant output losses. Complex genetic interactions are involved in chickpea resistance to Fusarium wilt. Resistance against particular pathogen races is conferred by a number of resistance genes, including h1, h2, and h3. Breeding procedures include both cutting-edge genomic techniques like marker-assisted selection (MAS) and traditional techniques like hybridization and backcrossing. By facilitating the accurate identification and stacking of resistance genes, MAS speeds up breeding. To treat this illness, it is essential to comprehend the genetic diversity of Fusarium wilt races, figure out the genetic basis of resistance, and use efficient breeding techniques. The goal of developing resilient chickpea varieties through the integration of genomic techniques and traditional breeding is to provide sustainable crop production in the face of changing disease problems".

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)

<table>
<tr><td colspan="2"><i>C. arietinum</i>
<i>C. judaicum</i>
<i>C. bijugum</i>
<i>C. pinnatifidum</i>
<i>C. chorassanicum</i></td><td colspan="2"><i>C. reticulatum</i>
<i>C. cuneatum</i>
<i>C. yamashitae</i>
<i>C. echinospermum</i></td></tr>
<tr><td colspan="4">Perennial Species (34)</td></tr>
<tr><td><i>C. acanthophyllum</i>
<i>C. macracanthum</i>
<i>C. anatolicum</i>
<i>C. microphyllum</i>
<i>C. atlanticum</i>
<i>C. mogoltavicum</i>
<i>C. balcaricum</i>
<i>C. montbretii</i>
<i>C. baldshuanicum</i></td><td><i>C. multijugum</i>
<i>C. canariense</i>
<i>C. nuristanicum</i>
<i>C. fedtschenkoi</i>
<i>C. oxyodon</i>
<i>C. flexuosum</i>
<i>C. paucijugum</i>
<i>C. floribundum</i>
<i>C. laetum</i></td><td><i>C. pungens</i>
<i>C. graecum</i>
<i>C. rassuloviae</i>
<i>C. grande</i>
<i>C. rechingeri</i>
<i>C. heterophyllum</i>
<i>C. songaricum</i>
<i>C. incanum</i>
<i>C. spiroceras</i></td><td><i>C. incisum</i>
<i>C. stapfianum</i>
<i>C. isauricum</i>
<i>C. subaphyllum</i>
<i>C. kermanense</i>
<i>C. tragacanthoides</i>
<i>C. korshinskyi</i></td></tr>
</table>

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.

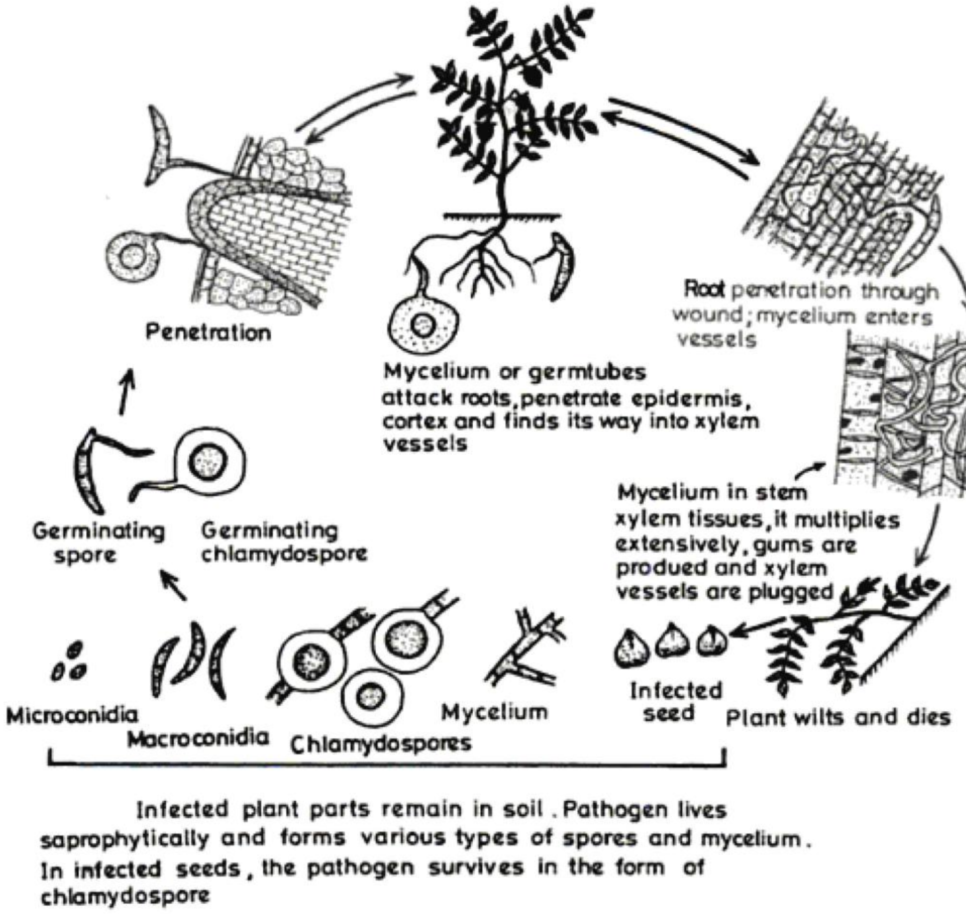

Fig 1 : Disease cycle of *F. oxysporum* f. sp. ciceris Jalali *et al*., (1992)

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-27$_{700}$ and UBC-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-855$_{500}$ [94] and UBC-825$_{1200}$ [95] were associated with Race 4's resistance gene. Co-segregation of UBC-855$_{500}$ and CS-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.

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