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Review Article

Integrated Strategies for the Management of Fusarium Wilt in Chickpea: A Comprehensive Review

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ABSTRACT

Fusarium wilt of chickpea, caused by Fusarium oxysporum f. sp. ciceris (Foc), is one of the most destructive soil-borne diseases limiting chickpea (Cicer arietinum L.) production worldwide. The pathogen survives for prolonged periods in soil and infected plant debris, rendering its management difficult. Infection occurs through roots via macroconidia, microconidia, and chlamydospores, followed by vascular colonization that disrupts water transport, leading to wilting and plant death. Disease severity is influenced by environmental conditions, particularly soil temperature and moisture, in addition to pathogen virulence and host susceptibility. This review critically synthesizes current knowledge on pathogen biology, pathogenic variability, and epidemiology, and evaluates integrated disease management strategies including host resistance, cultural practices, biological control agents, and chemical interventions. Particular emphasis is placed on the integration of these approaches to achieve durable, economically viable, and environmentally sustainable disease control. The review provides a consolidated framework intended to support researchers, breeders, and crop protection specialists in developing effective strategies for mitigating Fusarium wilt and enhancing chickpea productivity.

Keywords: *Biological control, Chickpea, Chlamydospores, Fusarium wilt, Host Resistance.*

INTRODUCTION

Chickpea (*Cicer arietinum* L.), the second most important pulse crop after dry beans (*Phaseolus vulgaris* L.) (Funga et al., 2017) is a climate-resilient, cool-season legume cultivated in tropical, subtropical, and temperate regions. Originating in southeastern Turkey and adjacent parts of Syria, it is a self-pollinated crop belonging to the family *Leguminaceae*, with a genome size of 738 Mb (Varshney et al., 2013). Major producers include India, Turkey, Pakistan, Australia, and Myanmar.

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The global area of chickpea is about 14 million hectares, with production of 14.25 million metric tonnes, of which India contributes 70 per cent of global production, producing 112.29 lakh tonnes of chickpea with an area of 105.61 lakh hectares (FAOSTAT, 2022). In India, the crop is grown mainly in the states of Madhya Pradesh, Rajasthan, Maharashtra, Uttar Pradesh, Haryana, Karnataka and Andhra Pradesh. Chickpea is an important source of dietary protein and provides essential minerals, calcium, and amino acids. It also contains several bioactive compounds such as phytates, lectins, and enzyme inhibitors. These compounds are associated with health benefits and help reduce the risk of chronic diseases including cardiovascular diseases, cancer, and leukoderma. Due to these nutritional and functional properties, chickpea is often classified as a "functional food" (Wallace et al., 2016).

Two major types of chickpea are cultivated worldwide: Desi and Kabuli. Desi chickpea accounts for about 90% of the total chickpea cultivation area in India and is characterized by small, dark-colored seeds with thick seed coats. In contrast, Kabuli chickpea has larger, cream-colored seeds and is widely preferred in international markets (Frimpong et al., 2009). Kabuli chickpea is globally popular and has high commercial value, whereas Desi chickpea is primarily consumed in the Middle East and South-East Asia. Chickpea also contributes to sustainable agriculture by fixing atmospheric nitrogen through symbiotic associations with *Rhizobium*, thereby reducing the need for nitrogen fertilizers.

Despite its agronomic and nutritional importance, chickpea productivity is greatly constrained by several biotic and abiotic stresses. More than 52 pathogens have been reported to infect chickpea. Among these, major diseases include Ascochyta blight, Fusarium wilt, and root rot. Fusarium wilt, caused by *Fusarium oxysporum* f. sp. *ciceris* (Foc), is one of the most prevalent and destructive diseases of chickpea, particularly in the northwestern plain zone (Garg et al., 2018).

Fusarium wilt is a major soil-borne disease occurring in chickpea-growing regions of the Mediterranean, South Asia, and East Africa. The disease causes root and stem infection that significantly reduces plant vigor and productivity (Alloosh et al., 2019). In warm and dry regions, it is considered one of the most destructive root diseases of chickpea, causing yield losses ranging from 10–40% and sometimes leading to complete crop failure (Garg et al., 2018). Disease symptoms include vascular discoloration, stunted growth, yellowing, wilting, and eventual plant death (Leitão et al., 2020). The pathogen primarily infects the roots and spreads through contaminated soil or infected seeds (Fan et al., 2022). This disease significantly reduces yield and plant vigor by affecting root development, causing foliar yellowing, and impairing plant growth and seed production.

The increasing incidence of *Fusarium oxysporum* f. sp. *ciceris* in chickpea-growing areas highlights the expansion of vulnerable regions and the need for effective disease management strategies. The widespread occurrence and economic impact of this disease necessitate a comprehensive understanding of its etiology, pathogen biology, infection mechanisms, epidemiology, genetics of pathogenesis and resistance, molecular mechanisms, and integrated disease management strategies. Therefore, this review entitled "Integrated Strategies for the Management of Fusarium Wilt in Chickpea: A Comprehensive Review" discusses the disease cycle of Fusarium wilt and highlights recent advances and integrated approaches for the effective management of this economically important disease.

DISTRIBUTION

The disease has been reported from several countries, including India, Bangladesh, Burma,

Ethiopia, Mexico, Pakistan, Syria, Tunisia, Chile, Iran, Nepal, Sudan, the United States, Peru, USSR, Malawi, Spain, Turkey and Italy. However, chickpea cultivation is greatly threatened by this disease in India, Iran, Pakistan, Nepal, Myanmar, Spain and Tunisia. It is more prevalent in lower latitudes (0-30^{0}N) where growing season is relatively drier and warmer than in higher altitudes (Arunodhayam et al., 2014).

HISTORY AND CLASSIFICATION OF PATHOGENS

Fusarium wilt of chickpea is incited by *F. oxysporum* Schlechtend: Fr. f. sp. *ciceris* (Padwick) Matuo & K. Sato (Jalali & Chand, 1992; Nene & Reddy, 1987). It was first reported by Butler in the book entitled "Fungi and Disease in Plants" in 1918 but its etiology was not correctly determined until 1940 by Padwick. Mckerral (1923) considered the disease to be soil borne. However, Narasimhan (1929) experimentally proved that wilt disease is caused by a species of *Fusarium*. Several workers stated that *Fusarium* wilt has become a major factor limiting chickpea production in the Indian subcontinent, the Mediterranean Basin and California. Kirk et al. (2008) placed it in Kingdom: *Fungi*, Division: *Ascomycota*, Class: *Sordariomycetes*, Order: *Hypocreales*, Family: *Nectriaceae*, Genus: *Fusarium*, Species: *F. oxysporum*, Subspecies: *F. oxysporum.* f. sp. *Ciceris*.

SYMPTOMS OF FUSARIUM WILT DISEASE OF CHICKPEA

Symptoms of Fusarium wilt in chickpea can appear at any growth stage, and infected plants may occur either in patches or scattered throughout the field (Haware, 1990). Symptom expression generally becomes visible 10–15 days after infection. Two distinct symptom types have been recognized: early wilt and late wilt. Early wilt typically appears about 25 days after sowing. It is characterized by flaccid leaves, dull-green discoloration, and gradual desiccation of the foliage, which ultimately leads to the collapse and death of the entire plant. In contrast, late wilt symptoms usually develop during the flowering stage, approximately 40–55 days after sowing. At this stage, the petioles, leaflets, and rachis begin to droop, followed by progressive yellowing and necrosis of the leaves.

The characteristic symptoms of Fusarium wilt include progressive yellowing and drying of leaves starting from the lower portions of the plant and moving upward. This is accompanied by drooping of the rachis and petioles, reduced branching, stunted plant growth, and eventual withering. Internal symptoms include brown discoloration of the vascular bundles, which becomes visible when the stem is split longitudinally. In advanced stages, the entire plant wilts and dies (Argikar, 1970) (Fig. 1). Several factors, including pathogen virulence, soil inoculum density, favorable environmental conditions, and host susceptibility influence the development of Fusarium wilt. Early symptoms often involve drooping of the upper parts of the plant, which may progress to complete wilting within a few days. In some cases, partial wilting may occur, where only certain branches of the plant are affected.

Infected seedlings may not always show external discoloration of roots. However, internal dark-brown discoloration of the xylem tissues in roots and stems becomes evident when the tissues are examined through longitudinal or cross-sectional cuts. Additional symptoms include withering of lateral roots, stunted plant growth, and reduced seed quality. Seeds produced from infected plants are often lighter in weight, discolored, and wrinkled (Jiménez-Díaz et al., 2015). Physiological changes are also associated with the disease. Infected plants exhibit reduced chlorophyll content and increased accumulation of organic acids, polyphenols, and carbohydrates, indicating metabolic alterations induced by pathogen infection.

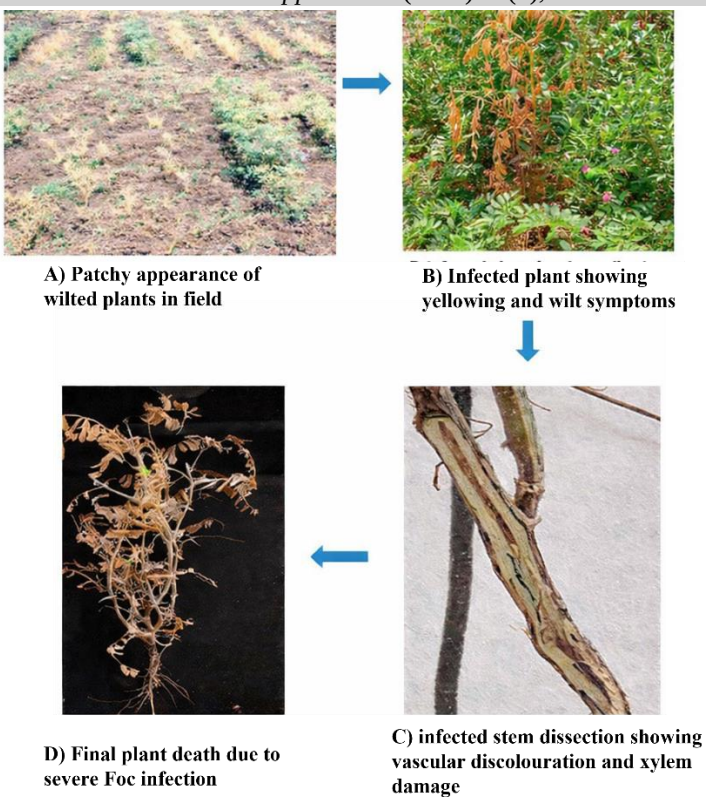

Fig 1. Symptom progression of Fusarium wilt of chickpea

DISEASES CYCLE AND EPIDEMIOLOGY

Fusarium oxysporum f. sp. *ciceris* (Foc) is an ascomycete fungus responsible for Fusarium wilt in chickpea and is known to cause significant yield losses (Pande et al., 2007). The pathogen survives in soil, seeds, and infected plant debris primarily through resting spores known as chlamydospores, which enable long-term persistence under unfavorable conditions.

On potato dextrose agar (PDA) medium, the fungal colony initially appears white and cottony, gradually turning cream to salmon in color with age. The pathogen produces three types of asexual spores: microconidia, macroconidia, and chlamydospores. Microconidia are oval to ellipsoidal and measure approximately 2.5–4.5 µm × 5–11 µm, while macroconidia are fusoid and measure 3.5–4.5 µm × 25–65 µm (Fig. 2). Chlamydospores usually develop in 15-day-old cultures, possess smooth or rough walls, and may occur singly, in pairs, or in chains (Castro et al., 2012). The hyphae are septate and profusely branched.

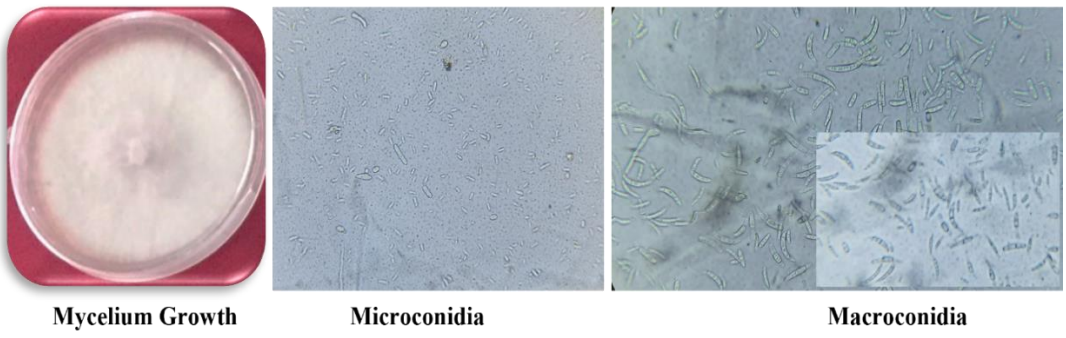

Fig 2. Mycelial colony and different type of spores of *F. oxysporum* f.sp. *ciceris*

The development of Fusarium wilt in susceptible hosts requires the presence of a virulent pathogen, favorable environmental conditions, and adequate soil inoculum (Fig. 4). Optimal growth occurs at temperatures of 25–27°C and a pH range of 5.1–5.9, although this may vary among strains. For sporulation, the optimal pH ranges between 7.1 and 7.9 (Jimenez-Diaz et al., 2011).

The disease spreads through seed transmission, contaminated soil, plant debris, and human activities (Haware et al., 1996). The pathogen may also persist in soil, seed hilum, cotyledons, and axillary tissues (Shakir & Mirza, 1994). In the disease cycle, chlamydospores act as primary inoculum, whereas macroconidia and microconidia serve as secondary inoculum. Spore germination is stimulated by carbohydrates released from decaying plant roots. Infected seeds contribute to long-distance dispersal of the pathogen (Pande et al., 2007), while short-distance spread occurs through contaminated soil, plant debris, and agricultural operations. Although conidia remain viable for a limited time, the resting spores can survive in soil until the subsequent cropping season (Chand & Khirbat, 2009).

The formation of chlamydospores is influenced by the nutrient status of the fungal inoculum, with nutrient-depleted field conditions favoring their production, whereas nutrient-rich media promote the formation of macroconidia. The pathogen exhibits strong survival ability and can colonize stems and roots of wilt-affected plants, and sometimes persists in apparently healthy plants growing in infested fields (Trapero-Casas & Jimenez-Diaz, 1985).

Dormant propagules are stimulated to germinate by carbohydrates released from decaying roots and plant tissues. These germination signals may originate from host plants, non-host plants, or plant debris (Nelson, 2012). Following germination, hyphal growth occurs rapidly, leading to the formation of conidia within 6–8 hours, while new resting spores develop within 2–3 days under favourable conditions.

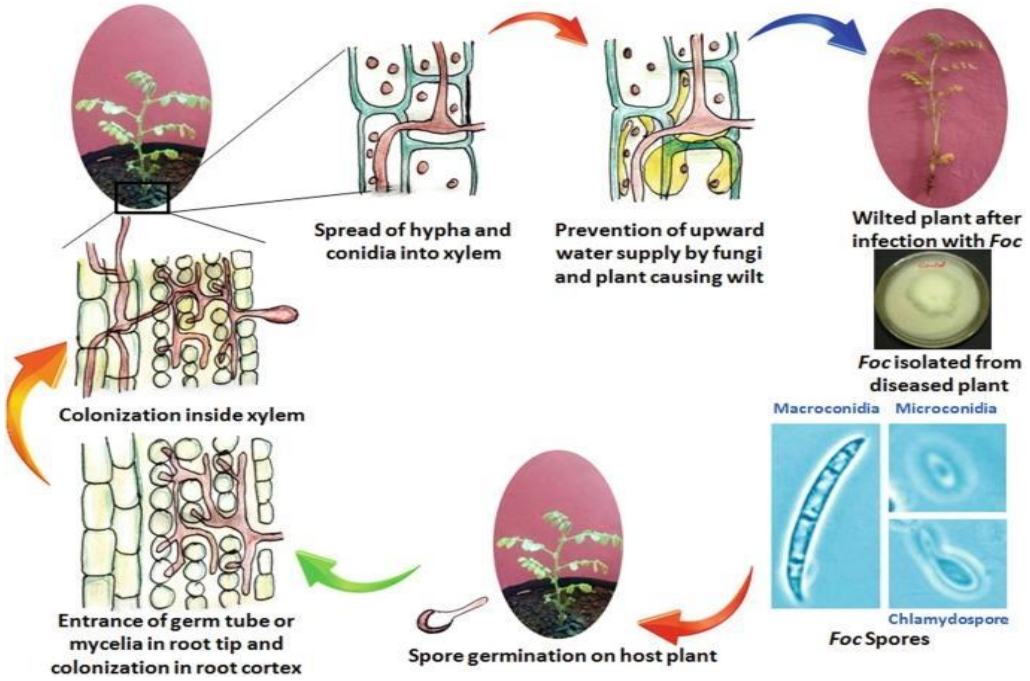

Fig. 3. Disease cycle of chickpea Fusarium wilt (Suthar et al., 2021)

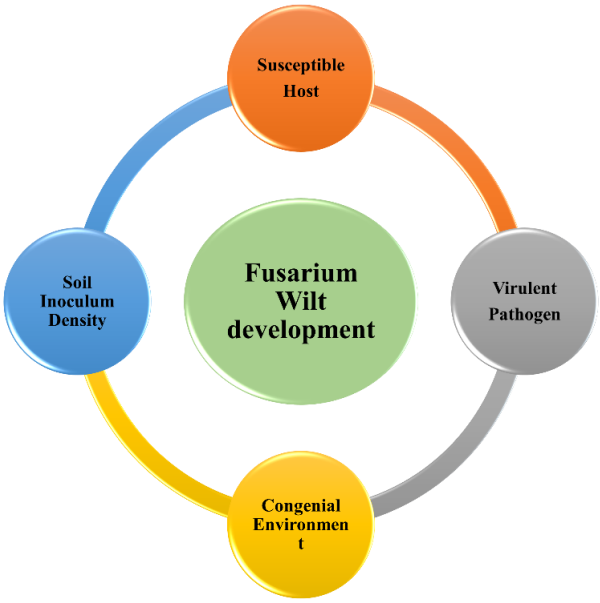

Fig. 4. Factors responsible for the development of Fusarium wilt of chickpea

Following root invasion, the pathogen penetrates epidermal cells and establishes a systemic vascular infection. Penetration may occur directly or through wounds, with root tips serving as the most common infection sites (Lucas, 1998). This process is influenced by fungal secretions, host surface structures, and regulators of spore germination (Mendgen et al., 1996). After penetration, the pathogen colonizes the root cortex and eventually enters the xylem vessels through pits, where it proliferates.

Wilting results from several interacting factors, including accumulation of fungal mycelium within xylem vessels, toxin production, and host defense responses such as the formation of gels, gums, and tyloses that obstruct water movement. In addition, the collapse of vessels due to the proliferation of adjacent parenchyma cells further contributes to the development of wilt symptoms (Fig. 3).

The pathogen can survive as mycelium or resting spores in seeds, soil, infected plant debris, and root and stem tissues, and may persist for more than six years in the absence of a host (Castro et al., 2012). Infection of symptomless dicotyledonous weeds also enhances pathogen survival in fallow soils. Consequently, infested soil serves as the primary source of inoculum for Fusarium wilt development (Al-taae et al., 2013).

PATHOGENIC AND GENETIC VARIABILITY

Pathogenic variability in Foc is evidenced by the existence of distinct pathogenic races and pathotypes. Two primary pathotypes have been identified based on symptomatology in pathogenicity assays: the wilting and yellowing pathotypes. The wilting pathotype induces rapid plant death, while the yellowing pathotype manifests as progressive foliar chlorosis, flaccidity, vascular discoloration, and eventual plant death (Jiménez-Díaz et al., 2015).

The virulence variability between Foc isolates has led to the classification of eight physiological races: races 0, 1A, 1B/C, 2, 3, 4, 5, and 6 (Sharma & Muehlbauer, 2007). These races differ in their pathogenicity on chickpea cultivars and are geographically distributed. Races 0, 1B/C, 5, and 6 are prevalent in the Mediterranean Basin and California, while races 2 and 3 are found in Ethiopia, India, and Turkey. Race 4 is reported in Ethiopia, India, and Iraq, and race 1A has been observed in India, the Mediterranean region, and the USA (Al-taae et al., 2013). The yellowing pathotype comprises races 0 and 1B/C, whereas the most virulent and economically destructive races, 1A, 2 through 6, are associated with the wilting pathotype. Plants infected by wilting races exhibit rapid symptom onset, with no

visible leaf yellowing, and complete wilting within 3-4 weeks post-inoculation. In contrast, yellowing races, such as 0 and 1B/C, cause progressive chlorosis and vascular discoloration. Late wilting, characterized by an extended latent period leading to 100% plant wilting, has been observed in some susceptible chickpea lines (Castro et al., 2012). Another phenomenon, slow wilting, involves a prolonged latent phase with a gradual disease progression. Accurate race identification is critical for breeding programs and the effective deployment of resistance genes in chickpea-growing regions. While race identification via pathogenicity tests is reliable, it requires substantial time, space, and resources. Thus, rapid, cost-effective, and reproducible molecular tools are necessary for accurate pathogen and race identification. Jiménez-Gasco et al. (2004) developed race-specific primers and polymerase chain reaction (PCR) assays to distinguish Foc and its pathogenic races 0, 1A, 5, and 6, offering a reliable alternative for race determination.

Table 1. Summary of Foc races, symptoms, and distribution

Race	Pathotype	Major symptoms	Reported distribution
0	Yellowing	Progressive chlorosis, vascular discoloration	Mediterranean region, California
1A	Wilting	Rapid collapse, severe vascular browning	India, Mediterranean, USA
1B/C	Yellowing	Chlorosis followed by wilting	Mediterranean Basin, California
2	Wilting	Severe vascular infection, rapid death	India, Ethiopia, Turkey
3	Wilting	Similar to race 2, aggressive wilt	India, Ethiopia, Turkey
4	Wilting	Highly virulent wilt, quick plant death	India, Ethiopia, Iraq
5	Wilting	Aggressive wilt symptoms	Mediterranean Basin, California
6	Wilting	Severe wilt and vascular discoloration	Mediterranean Basin, California

YIELD LOSSES IN CHICKPEA DUE TO FUSARIUM WILT DISEASE

Fusarium oxysporum f. sp. *ciceris* is one of the most destructive pathogens of chickpea, causing substantial losses in both yield and seed quality. Under favourable conditions, the disease can lead to severe epidemics and significantly reduce crop productivity. Wilt incidence caused by Foc has been reported to reach 61.0% during the vegetative stage and 43.0% during the flowering stage (Nikam et al., 2011). In Pakistan, Fusarium wilt results in annual yield losses ranging from 10–50%, depending on environmental conditions and cultivar susceptibility. Regional studies indicate varying levels of yield loss, including 10% in tropical regions such as India and Spain, 40% in Tunisia, and 17% in Iran (Karimi et al., 2012). Similarly, in Ethiopia, the disease has been reported to cause approximately 30% yield loss in chickpea crops (Meki et al., 2008).

The severity of yield loss largely depends on the stage of infection and environmental conditions. Early wilt infections are particularly destructive, with reported yield losses ranging from 77–94%, whereas late wilt infections may cause 24–65% losses (Haware & Nene, 1980). In several chickpea-growing regions, disease incidence has been reported to range from 14.1% to 32.0% under favourable conditions. Under highly conducive conditions, the disease may lead to extensive crop damage and substantial yield reduction (Pande et al., 2011; Sunkad et al., 2019; Dhawale & Dhale, 2021).

INTEGRATED MANAGEMENT OF DISEASE

The management of *Fusarium wilt* (*Fusarium oxysporum* f. sp. *ciceris*), one of the most destructive diseases in chickpea cultivation,

necessitates an integrated approach that combines cultural, chemical, and biological practices to mitigate disease incidence and minimize yield losses. An integrated disease management (IDM) strategy focuses on preventing pathogen establishment, controlling its spread, and enhancing plant resilience, ultimately ensuring sustainable chickpea production.

1. CULTURAL PRACTICES

Cultural practices play a pivotal role in managing *Fusarium wilt* in chickpea, offering sustainable, eco-friendly, and cost-effective strategies compatible with agro-ecosystems. These methods aim to reduce pathogen inoculum in the environment and support healthy crop growth, aligning with modern agricultural sustainability goals. Key practices include using pathogen-free, resistant seeds, implementing delayed sowing (after October 10), and adopting crop rotations with non-hosts, where excluding legumes for two years reduces wilt severity by 59–85% (Yadav et al., 2012).

Optimized nitrogen fertilization and row spacing also contribute to disease suppression. Over-fertilization, however, may exacerbate pathogen development by promoting lush, susceptible growth. Wider row spacing (50 cm) further decreased disease incidence to 17.35% compared to 29.17% at 20 cm. Additionally, delayed sowing benefits grain yield by reducing disease incidence during lower late-season temperatures (Sudharani et al., 2019). Regional variations also influence outcomes. In southern Spain, shifting sowing from early spring to early winter significantly reduced disease development.

The use of resistant cultivars is the most practical and cost-effective method for controlling Fusarium wilt. Several chickpea genotypes such as FLIP 85-20C, FLIP 85-29C, and FLIP 85-30C have been identified as resistant to Fusarium wilt. Notable resistant genotypes, such as ICCV 98505, ICCV 07105, ICCV 07111, and ICCV 07305, were identified resistant (Sharma et al., 2019). Pande et al. (2006) identified 21 FW-resistant accessions, while other genotypes like JG 315, Avrodhi, and DCP 92-3 were also found resistant. Furthermore, ICCV 05530 showed resistance to FW races 1 and 3. However, JG 62 exhibited 89–100% wilt incidence across both races. These resistant genotypes can be incorporated into breeding programs to develop varieties with improved resistance. Integrating cultural practices with other management strategies is essential to mitigate Fusarium wilt sustainably, ensuring resilient chickpea production while minimizing environmental impact.

Despite their effectiveness, these cultural practices have practical limitations under farmers' field conditions. Crop rotation is often constrained by small landholdings, market demands, and the dominance of legume-based cropping systems, which limit the feasibility of long rotation cycles. Moreover, the long survival of chlamydospores of *Fusarium oxysporum* in soil reduces the effectiveness of short-term rotations. Similarly, delayed sowing may expose crops to terminal heat stress or reduce the available growing period in some agro-climatic zones. Wider spacing and optimized fertilizer use may also conflict with farmers' yield-maximization strategies and resource availability. Therefore, while cultural practices are valuable, their adoption often depends on local socio-economic and environmental constraints.

Long-term field experiments indicate that crop rotation is most effective when implemented over multiple seasons in combination with other integrated disease management strategies. Studies have shown that sustained rotation with cereals, oilseeds, or fallow periods can gradually reduce soil inoculum density, but measurable benefits usually appear only after several cropping cycles. This highlights the importance of integrating rotation with resistant cultivars, soil health improvement, and biological control measures for durable disease suppression.

2. BIOLOGICAL CONTROL

Biological control agents (BCAs) offer a sustainable, eco-friendly alternative to chemical treatments for managing plant diseases, utilizing natural organisms or their metabolites to suppress pathogens. *Trichoderma* species are among the most studied BCAs, demonstrating high efficacy against *Fusarium oxysporum*. Suresh et al. (2022) reported that *T. viride* reduced disease incidence to 4.31%, while Kumar and Mane (2017) found that *T. harzianum* inhibited 66.67% of *Fusarium* mycelial growth. Yadav et al. (2012) observed 71.36% efficacy of the same. Rudresh et al. (2005) highlighted *T. virens* and *T. harzianum* for improving biomass, plant height, and germination, with a minimum wilt incidence of 0% at 30 DAS. Dubey et al. (2007) showed *T. viride* reduced wilt incidence to 23.6%, while Boureghda and Bouznad (2009) confirmed *T. atroviride* enhanced vegetative growth and resistance. Additionally, Zaim et al. (2018) found *T. harzianum* and *Bacillus subtilis* reduced disease incidence by up to 81.31% and 93.67%, respectively, under field conditions.

Advanced studies, such as those by Thangavelu and Gopi (2015), demonstrated complete suppression of Fusarium wilt using a synergistic approach with rhizospheric and endophytic *Trichoderma*. Similarly, Younesi et al. (2021) identified *T. longibrachiatum* KT8 and *T. harzianum* KT9 and KT10 as the most effective isolates in Iran. These findings emphasize the potential of BCAs in integrated disease management (IDM), where their combination with cultural practices and non-antibiotic strategies enhances plant resistance and pathogen suppression (Collinge et al., 2022).

The integration of BCAs into IDM frameworks is essential for sustainable agriculture, as they promote enhanced defense mechanisms in plants, ensuring long-term pathogen resistance and reduced reliance on chemical controls. Field-level inconsistency in the performance of *Trichoderma* spp. against chickpea wilt is largely attributed to spatiotemporal variability in soil physicochemical properties, indigenous microbiome dynamics, and abiotic stress regimes that modulate rhizosphere competence, root colonization efficiency, and secondary metabolite expression. Ecological buffering by native microbial communities and differential pathogen inoculum density further constraint establishment and persistence of introduced strains. Additionally, formulation-associated limitations including reduced propagule viability, inadequate shelf stability, carrier incompatibility, and inconsistent colony-forming unit (CFU) thresholds—impair field reliability and scalability (Woo et al., 2014). Thus, deployment of genomically characterized, stress-resilient strains integrated with advanced encapsulation technologies and stringent quality assurance frameworks is imperative to achieve predictable and durable biological suppression.

3. BOTANICALS

Suresh et al. (2022) demonstrated that 30 days post-diagnosis, treatments with neem cake (8.88%) and castor cake (9.30%) achieved the lowest wilt incidence. Similarly, neem cake reduced disease incidence to 11.23% and 20.19% in studies by Patra et al. (2017) and Mukesh et al. (2017), outperforming other treatments. Allium sativum (garlic) bulb extract significantly lowered wilt incidence from 65.9% in untreated seeds to 23.6% (Hari Chand & Singh, 2005). Singh and Hari Chand (2004) also found that *Azadirachta indica* (neem) leaf extract at 100% concentration completely inhibited pathogen spore germination. Neem oil and garlic extracts have proven effective in producing disease-free seedlings (Singh et al., 2004). Further studies confirmed the antifungal efficacy of *A. indica* leaf extract, *Ocimum sanctum* (holy basil), and *Curcuma longa* (turmeric) against wilt pathogens (Singh et al., 2008). These botanical treatments exhibit promising antifungal activity, offering eco-friendly alternatives for managing Fusarium wilt in chickpeas. Integrating these strategies into management programs can enhance sustainability and reduce reliance on synthetic fungicides.

Table 2. Integrated management strategies for Fusarium wilt of chickpea and their effectiveness

Component	Management Strategy	Details / Examples	Effectiveness / Remarks
Cultural Practices	Resistant varieties	FLIP 85-20C, FLIP 85-29C, FLIP 85-30C; ICCV 98505, ICCV 07305; JG 315, Avrodhi, DCP 92-3; ICCV 05530	Most economical and practical method; reduces disease significantly
	Avoid susceptible varieties	JG 62 (highly susceptible)	89–100% wilt incidence reported
	Crop rotation	Avoid legumes for 2 years; rotate with non-host crops	Reduces wilt severity by 59–85%
	Delayed sowing	Sowing after October 10	Reduces disease incidence and improves yield
	Optimum row spacing	50 cm spacing	Disease reduced to 17.35% vs 29.17% at 20 cm
Biological Control	*Trichoderma* spp.	*T. viride, T. harzianum, T. virens, T. atroviride, T. longibrachiatum*	4–71% disease reduction; improved plant growth
	Bacillus subtilis	Field application	Up to 93.67% disease reduction
Botanicals	Neem cake / Castor cake	Soil amendment	Wilt incidence reduced to 8.88–20.19%
	Garlic extract	Seed treatment (*Allium sativum*)	Wilt reduced from 65.9% to 23.6%
	Neem leaf extract	*Azadirachta indica* (100% concentration)	Complete inhibition of spore germination
	Tulsi / Turmeric extracts	*Ocimum sanctum, Curcuma longa*	Effective antifungal activity
Chemical Control	Carbendazim	Seed treatment	88.7% wilt reduction
	Carbendazim + Mancozeb (0.1%)	Seed and soil treatment	Up to 100% reduction in field trials
	Tricyclazole	In vitro treatment	100% fungal growth inhibition
	Thiram / Copper oxychloride / Fosetyl-Al	Systemic and protective fungicides	Reduced seedling mortality and wilt incidence
	Salicylic acid, BABA, Zinc chloride	Defense-inducing chemicals	Reduced disease incidence
Molecular Approaches	Transcriptomics	Identification of resistance genes and SNP markers	Useful for marker-assisted breeding
	Proteomics	Higher ROS enzymes in resistant genotypes	Enhanced innate immunity
	Metabolomics	Phytoalexins (luteolin, genistein), chitinases	Strengthens plant defense response

4. CHEMICAL CONTROL

Chemical control is a key component of integrated management for chickpea Fusarium wilt. Tricyclazole and carbendazim have shown 100% inhibition of fungal growth in vitro (Surnar et al., 2022), while seed treatments with carbendazim or carbendazim + mancozeb (0.1%) reduced wilt incidence by 88.7% and 100%, respectively, in field trials (Mohan et al., 2017). Soil treatment with carbendazim + mancozeb reduced wilt incidence by 73.2% under pot conditions (Golakiya et al., 2018). Other effective treatments include carbendazim, fosetyl-Al, and combinations such as thiram + carbendazim (Kumar and Mane, 2017).

Systemic fungicides like thiram and copper oxychloride also significantly reduced wilt incidence and seedling mortality (Maitlo et al., 2014). Carbendazim enhanced seed germination and yield, comparable to Trichoderma-based treatments (Mahajan et al., 2020). Non-conventional chemicals such as salicylic acid, BABA, and zinc chloride also effectively reduced disease incidence (Zope et al., 2018). Despite its effectiveness, overreliance on chemical control risks fungicide resistance and environmental concerns. Combining chemical treatments with biological and agronomic measures offers a more sustainable strategy for managing Fusarium wilt in chickpeas.

Despite their effectiveness, chemical approaches must be used judiciously. Continuous or indiscriminate use of systemic fungicides, particularly benzimidazoles such as carbendazim, can lead to the development of resistant pathogen populations. In addition, excessive chemical inputs may adversely affect soil microflora, reduce populations of beneficial antagonists, and raise environmental and food-safety concerns. Residue accumulation, input costs, and regulatory restrictions on certain fungicides further limit their long-term sustainability. Therefore, chemical control should be viewed as a supportive rather than a standalone strategy. Integration of fungicide seed treatment with resistant cultivars, crop rotation, biological control agents, and soil health management provides a more durable and environmentally sound approach for managing Fusarium wilt of chickpea.

5. Multi-Omics Approaches

Recent advances in omics technologies have greatly accelerated chickpea research by enabling integrated analyses of the genome, transcriptome, proteome, metabolome, ionome, and phenome. These approaches provide a systems-level understanding of molecular responses to environmental and physiological stresses and support crop improvement strategies (Muthamilarasan et al., 2019). Rapid progress in next-generation sequencing and high-throughput analytical platforms has generated large datasets that facilitate the identification of regulatory networks controlling stress responses and disease resistance.

By linking genes, proteins, and metabolites with plant phenotypes**, multi-omics integration** offers deeper insight into mechanisms underlying disease resistance, stress adaptation, and yield stability. The combination of omics datasets with bioinformatics and computational tools enables the identification of candidate genes and pathways governing important agronomic traits. Consequently, multi-omics strategies are increasingly being applied to accelerate the development of chickpea cultivars with improved resilience to biotic stresses and to promote sustainable crop production.

a) Transcriptomics

Transcriptomic studies have substantially improved the understanding of chickpea responses to *Fusarium oxysporum* f. sp. *ciceris* infection by identifying genes associated with host defense, signaling pathways, and stress adaptation. Comparative transcriptome analyses between infected and healthy plants have revealed numerous differentially expressed genes (DEGs) **t**hat represent potential targets for resistance breeding.

High-throughput gene expression platforms such as microarrays and RNA sequencing (RNA-seq) have enabled detailed characterization of transcriptional dynamics during host–pathogen interactions. Recent

studies have emphasized the role of resistance-associated gene families, particularly NBS-LRR proteins and WRKY transcription factors, in regulating immune responses against Fusarium wilt (Priyadarshini et al., 2023). In addition, genomic analyses of chickpea cultivars including JG 62, ICCV 2, K 850, and WR 315 have identified significant single nucleotide polymorphisms (SNPs) and insertion/deletion (InDel) variants associated with resistance to Fusarium wilt (Caballo et al., 2019).

Genetic investigations using crosses such as ILC 3279 × WR 315 have validated genomic regions controlling resistance to Foc race 5 and facilitated the development of resistant and susceptible near-isogenic lines (Aizat and Hassan, 2018). Differential expression analyses have further identified several candidate genes linked to wilt resistance, including LOC101495941 (MATE transporter protein), LOC101509359 (MADS-box transcription factor), and LOC101510206 (serine-related enzyme), along with previously reported defense-associated chaperonin genes LOC101490851 and LOC101499873. Moreover, multiple transcription factor transcripts exhibited altered expression patterns in cultivars JG 62 and WR 315 following infection with Foc race 1, indicating activation of metabolic and transport pathways involved in plant defense (Castillejo et al., 2015). Collectively, transcriptomic analyses provide a powerful framework for identifying resistance-related genes, mapping quantitative resistance loci, and supporting marker-assisted breeding for durable wilt resistance.

b) Proteomics and Metabolomics

Proteomic and metabolomic approaches complement transcriptomic studies by revealing functional proteins and metabolites directly involved in defense responses and host–pathogen interactions**.** Protein profiling in chickpea has identified several defense-related proteins, including syntaxins**,** subtilisin-like proteases, chitinases, β-1,3-glucanases, proteinase inhibitors, leucine-rich-repeat proteins, and pathogenesis-related (PR) proteins**,** which contribute to restricting pathogen colonization and enhancing host immunity.

Comparative proteomic analyses between the susceptible genotype JG 62 and the resistant genotype Digvijay have revealed clear differences in defense responses. The resistant cultivar showed higher abundance of reactive oxygen species (ROS)-associated enzymes**,** including glutaredoxin, glutathione peroxidase, ascorbate peroxidase, and peroxiredoxin, which are involved in oxidative burst signaling and enhanced resistance to pathogen invasion (Palomares-Rius et al., 2011). Increased accumulation of PR proteins in resistant plants further highlights their role in limiting pathogen proliferation.

Metabolomics provides additional insights into biochemical reprogramming during pathogen infection by analyzing the complete set of metabolites within plant tissues. Integration of metabolite profiling with next-generation sequencing and mass spectrometry-based techniques has improved understanding of metabolic responses to pathogen stress (Behmand et al., 2023). Resistant chickpea plants often accumulate metabolites associated with defense activation, including sugars such as hexokinase-related intermediates, trehalose, sucrose synthase products, and glucose-6-phosphate, which support oxidative burst responses, energy metabolism, and lignification required for structural defense (Upadhyay et al., 2023).

Proteomic and metabolomic investigations of the resistant cultivar Digvijay have also identified several defense-related compounds, including endo-β-1,3-glucanase, chitinases, and phytoalexins such as luteolin and genistein**.** In contrast, susceptible cultivars show depletion of certain sugars, amino acids, and carbohydrates, which facilitates pathogen colonization and accelerates disease development (Palomares-Rius et al., 2011).

Collectively, integrated transcriptomic, proteomic, and metabolomic analyses highlight the critical roles of PR proteins, ROS-generating enzymes, transcription factors, flavonoids, and phenolic

compounds in enhancing resistance to Fusarium wilt (Miedaner et al., 2020).

These multi-omics approaches provide a comprehensive framework for identifying resistance genes, elucidating defense pathways, and accelerating the development of wilt-resistant chickpea cultivars.

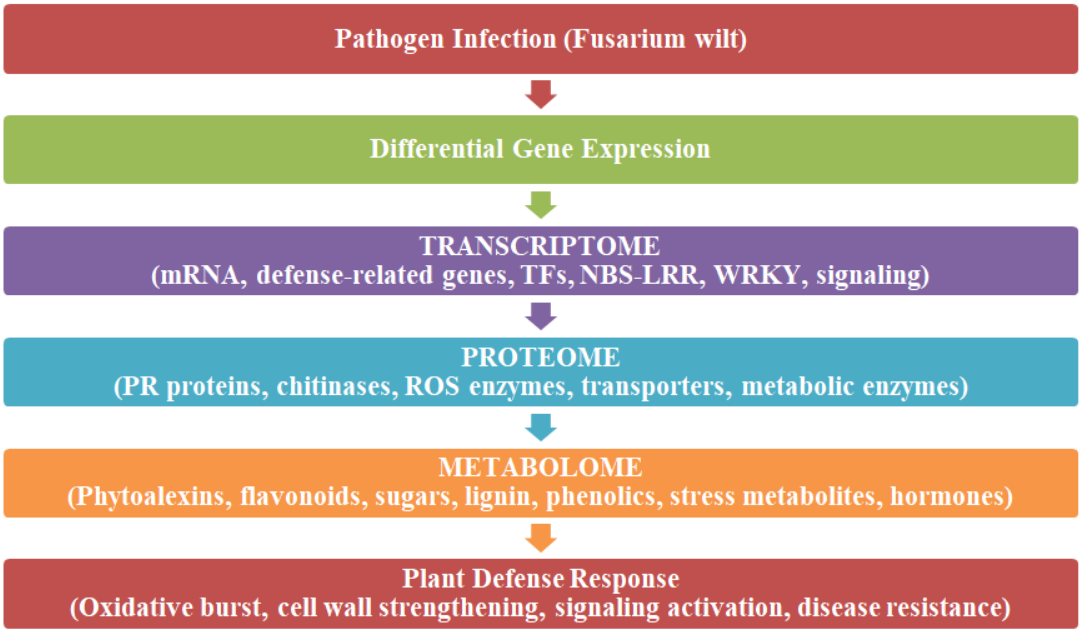

Fig. 5. Multi-omics cascade in chickpea defense against Fusarium wilt

EMERGING CHALLENGES AND NOVEL APPROACHES

The recent knowledge of Fusarium wilt in populous crops such as chickpea has added new dimensions in the disease dynamics. The underpinning of Fusarium wilt epidemics in the region appears to be complicated and may involve several effectors and mechanisms. Therefore, to address this problem and opportunity, it becomes necessary to identify preferred, successful, and well-suited strategies to manage this disease in chickpea. Various useful landmarks may be picked up for developing an effective disease management strategy in the light of current understanding, research literature, and available reports. At present, integrated management of chickpea wilt would be the best choice for controlling this important disease. Another avenue to control chickpea wilt can be tackling the pathogen effectors, and a variety of candidate pathogen effectors from the wilt pathogen have been identified using DNA-based techniques. Subsequently, the underlying mechanism can be identified and valued to design better resistance. Conversely, if numerous panels of races discovered showing the broad range of chickpea wilt pathogen distributed in different wilt zones, then conventional breeding for resistance is a totally effector-based strategy. In addition, omics-based research is very nascent to unveil the underlying information about population structures and genomic adaptability of this important pathogen. Potential transcriptomics revelation studies of chickpea wilt pathogen are towards an approach to perceive wilt pathogen behavior in chickpea growing soils or along with chickpea root systems. Conceptualizing the disease as effectors mediated, it may be redressed through effectors-based strategies or via breeding activities. Therefore, design new venues for future research on current approaches for precise modification of chickpea wilt in the future.

CONCLUSION

Fusarium wilt of chickpea, caused by *Fusarium oxysporum* f. sp. *ciceris*, is a major constraint to chickpea production worldwide due to its soil persistence, seedborne nature, and ability to infect plants at multiple growth stages. Disease development is strongly influenced by environmental factors such as temperature, soil moisture, and pH. Integrated management remains the most effective strategy, combining resistant cultivars, crop rotation with non-host crops, biological control agents, and cultural practices to reduce soil inoculum and disease incidence. Advances in genomics, molecular markers, and gene-editing technologies are accelerating the identification and deployment of durable resistance in chickpea breeding programs.

Future efforts should focus on improving the field performance of biological control agents, monitoring pathogen race diversity, and integrating climate-based forecasting and decision-support tools to assist farmers in timely disease management. A coordinated approach linking breeding, molecular research, quality seed systems, and effective extension services will be essential for sustainable management of Fusarium wilt and improved chickpea productivity.

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All authors declared that there is no conflict of interest.

Author's Contribution:

The authors contributed to the preparation of this manuscript in different capacities. **Deepak Kumari** was responsible for the collection of relevant articles and the writing of the original manuscript. **N. K. Ydaav** contributed to drafting, evaluation, and approval of the manuscript. **Ajeev Kumar** assisted in drafting the manuscript, provided supervision, and approved the final content. **Garima** was involved in manuscript reading and formatting. **Pratibha** carried out verification of references and plagiarism checking. **Rubleen Kaur** performed the final review of the manuscript before submission.

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