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Biology, Ecology, and Management of Fusarium Wilt in Chickpeas

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Abstract:

Chickpeas serve as an essential source of high-quality protein for large populations across South and West Asia and the Mediterranean region. In farming systems, they play a crucial role by replacing fallow land in cereal crop rotations. However, Fusarium wilt, caused by the soilborne fungus *Fusarium oxysporum* f. sp. *ciceris*, poses a significant threat to global chickpea production. The pathogen's prolonged survival in soil and considerable pathogenic variability-comprising eight identified races (0, 1A, 1B/C, 2, 3, 4, 5, and 6)-complicate disease management. The most effective and economical strategy for controlling Fusarium wilt is the development and adoption of high-yielding cultivars resistant to the dominant pathogen race(s) in a given region. Additional management strategies include using certified disease-free seeds, implementing sanitation and agronomic practices to minimize soil inoculum, selecting optimal sowing sites and timings to lower disease risk, and protecting healthy seeds with fungicides or biocontrol agents. In the absence of well-adapted resistant cultivars, these approaches can help mitigate disease impact. Molecular techniques are available to characterize and monitor *F. oxysporum* f. sp. *ciceris* populations, improving the effectiveness of disease control measures. Further advancements in managing Fusarium wilt could be achieved through an integrated strategy that incorporates slow-wilting cultivars alongside other control methods.

Introduction:

Chickpea (*Cicer arietinum* L.), a diploid species (2n = 16), is recognized as one of the foundational crops of modern agriculture. It belongs to the Papilionoid subfamily of legumes and traces its origins to its wild ancestor, *Cicer reticulatus*, which was domesticated in the Turkish Kurdistan region of the Fertile Crescent around 8,000–9,000 years ago. Rich in lysine-based protein, chickpea seeds are a crucial food source for

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humans and livestock. Additionally, chickpea cultivation enhances farming systems by replacing fallow land in cereal rotations, contributing to sustainable production, and reducing nitrogen fertilizer dependency through atmospheric nitrogen fixation. These attributes make chickpea a vital crop for food security in developing countries. There are two primary types of chickpea germplasm: desi and kabuli. Desi chickpeas, which have small, angular, wrinkled, and colored seeds, are primarily cultivated in the Indian subcontinent, whereas kabuli chickpeas, characterized by large to medium-sized, rams-head-shaped, smooth or slightly wrinkled beige to white seeds, are predominantly grown in the Mediterranean region. While desi chickpeas are mainly consumed in the Middle East and Southeast Asia, kabuli chickpeas are valued as a global commodity. Chickpea is the second most important food legume worldwide after dry beans (*Phaseolus vulgaris* L.). It is cultivated across tropical, subtropical, and temperate regions, including South and West Asia, East and North Africa, southern Europe, North and South America, and Australia. The crop is grown on approximately 13.5 million hectares in over 50 countries, producing nearly 13.1 million tons annually. The majority (89.2%) of global chickpea cultivation takes place in Asia, accounting for 84.5% of total production. India is the leading producer, contributing 73.3% of global chickpea acreage and 67.4% of total production. Pakistan follows with 7.3% of the acreage and 5.7% of production, while Australia, Iran, and Turkey also contribute to global chickpea cultivation. Chickpea is primarily grown in semi-arid regions with poor soil conditions, where it faces significant yield losses due to biotic and abiotic stresses, particularly drought. As a result, average yields range between 0.9–1.8 t/ha, falling significantly below the crop's theoretical potential.

Fusarium wilt, a devastating disease affecting chickpea worldwide, was first reported in India by Butler in 1918 but was not correctly identified until Padwick's work in 1940. The disease is now widespread in chickpea-growing regions across Asia, Africa, southern Europe, and the Americas, though it has not been reported in Australia. Fusarium wilt has become a significant limitation to chickpea production in key growing areas such as the Mediterranean Basin, the Indian subcontinent, and California. The disease can manifest at any stage of plant growth, with affected plants appearing in patches or scattered across a field. Highly susceptible cultivars may exhibit symptoms within 25 days of sowing, known as "early wilt." This includes leaf flaccidity, dull-green discoloration, desiccation, and plant collapse. More commonly, symptoms appear around

flowering (6–8 weeks post-sowing) and may extend to podding, referred to as "late wilt." Late-wilted plants display drooping of petioles, rachis, and leaflets, followed by yellowing and necrosis. Initially, symptoms are observed in the upper part of the plant before spreading throughout. In some cases, only certain branches are affected, leading to partial wilting. Infected roots may not show discoloration in early stages, but as the disease progresses, dark-brown xylem discoloration becomes evident when the roots or stems are split open. Histological changes include cavity formation between phloem and xylem, as well as cellular proliferation in the vascular cambium. These structural disruptions, along with xylem occlusions caused by dense gels (but not tyloses), contribute to impaired water and nutrient transport, leading to visible disease symptoms. Fusarium wilt significantly reduces chickpea yield by decreasing seed production and seed weight. Annual yield losses are estimated at 10–15% in India and Spain and up to 40% in Tunisia. However, in years of severe outbreaks, yield losses can reach as high as 70% or even lead to complete crop failure. Early wilt is more destructive, causing yield losses between 77–94%, whereas late wilt results in losses ranging from 24–65%. Additionally, seeds from late-wilted plants tend to be lighter, rougher, and duller compared to those from healthy plants. Given the severity of Fusarium wilt, implementing effective disease management strategies—such as breeding resistant cultivars, adopting agronomic practices to minimize soil inoculum, and using biocontrol agents—is critical for sustaining global chickpea production.

Pathogen biology and ecology

Fusarium oxysporum f. sp. *ciceris* is an asexually reproducing, soil-inhabiting fungus that survives in the soil through dormant chlamydospores, which may be free or embedded in plant tissues. The fungus thrives within a temperature range of 7.5 to 35°C and a pH range of 4 to 9.4, with optimal growth occurring at 25 to 27.5°C and pH 5.1 to 5.9, depending on the strain. Sporulation is most favorable at pH 7.1 to 7.9. Among the two pathotypes, isolates of the yellowing type exhibit faster growth than those of the wilting type at any given temperature. Chlamydospores, formed in aged mycelia and infected chickpea tissues, can be smooth or rough-walled, terminal or intercalary in hyphae, and may occur singly, in pairs, or in short chains. These structures enable the pathogen to persist in the soil and chickpea debris for at least six years. Furthermore, symptomless infection of dicotyledonous weeds can extend the pathogen's survival in fallow soils, making infested soil a major source of primary inoculum for *Fusarium* wilt

outbreaks in chickpeas. Infected seeds also serve as a primary inoculum source, with chlamydospore-like structures found in the hilum region of the seed. Plants grown from infected seeds wilt more rapidly than those grown in infested soil from healthy seeds. Additionally, infected seeds facilitate long-distance dispersal of the pathogen, introducing it into previously unaffected soils and regions. Short-distance spread occurs through the movement of contaminated soil or plant debris via human activities, machinery, wind, or water.

Chlamydospores in the soil germinate in response to seed and root exudates from both host and non-host plants. The pathogen infects germinating seeds and young seedlings directly, without requiring wounds, shortly after sowing in infested soil. Entry points include the cotyledons, the epicotyl-hypocotyl junction, and to a lesser extent, the root elongation and maturation zones. Studies in hydroponic systems have shown that races 0 and 5 colonize the surfaces of tap and lateral roots in both susceptible and resistant cultivars, preferring to penetrate meristematic cells at the root apex. Once inside, the fungus spreads intercellularly within the root cortex, reaching the central cylinder and entering xylem vessels. Further pathogen movement occurs through hyphal growth and the transport of microconidia via the transpiration stream, as well as lateral spread between xylem vessels. The pathogen's systemic colonization along the plant axis marks the determinative phase of pathogenesis. This is followed by the expressive phase, in which symptoms become visible. By 10 to 20 days post-inoculation, extensive colonization of xylem vessels in the root and lower stem leads to the development of wilt symptoms. The extent and speed of *Fusarium oxysporum* f. sp. *ciceris* colonization in the epicotyl and stem xylem depend on the compatibility between the pathogen race and the chickpea genotype. The highest colonization occurs in the most susceptible combination—line P-2245 infected with the highly virulent race 5—followed by line JG-62 with race 5 and P-2245 with the less virulent race 0. As infection progresses and severe symptoms develop, abundant chlamydospores form in plant tissues. Upon plant senescence, these chlamydospores are released into the soil as infected debris decomposes. The pathogen can persist in the soil through cycles of chlamydospore renewal, supported by limited saprophytic growth on organic debris and root exudates, as well as transient infections of both host and non-host plants. In contrast, incompatible interactions between the same pathogen races and resistant chickpea lines result in asymptomatic responses (Jiménez-Fernández et al., 2013). In these cases, the pathogen either remains

confined to the intercellular spaces of the root cortex without reaching the xylem (e.g., WR-315/race 0), invades the root and hypocotyl xylem to a limited extent (WR-315/race 5), or extensively colonizes the root and stem xylem (JG-62/race 0). These varied responses indicate that multiple defense mechanisms are likely at play in resistant chickpea plants.

Management

Disease diagnosis

Timely and accurate diagnosis is the initial step to guarantee effective management of Fusarium wilt in chickpeas. A meticulous analysis of uprooted, diseased plants for the lack of outward root symptoms and the presence of dark-brown staining in the xylem tissues of roots and stems might facilitate the diagnosis of the disease. Nonetheless, caution must be used to avoid conflating the signs of Fusarium wilt with the leaf yellowing, wilting, and phloem staining observed in chickpeas infected by some plant viruses (e.g., Pea stripe carlavirus). Plants infected by several root fungus, such as Fusarium solani f. sp. pisi, F. solani f. sp. eumartii, and Macrophomina phaseolina, commonly exhibit leaf yellowing and necrosis. Furthermore, caution is essential when validating the initial diagnosis through isolation in pure culture, as endophytic, non-pathogenic strains of F. oxysporum are often isolated from the upper stem tissues of symptomatic chickpeas. The morphology-based diagnosis of Fusarium colonies isolated from yellowing chickpeas does not readily distinguish F. oxysporum f. sp. ciceris from Fusarium redolens, which has recently been shown to produce symptoms in chickpeas akin to those caused by the yellowing pathotype of F. oxysporum f. sp. ciceris, with the exception of vascular discoloration. F. redolens, F. oxysporum, and F. oxysporum f. sp. ciceris can be effectively distinguished by molecular techniques.

Exclusion and eradication of the pathogen

For the control of Fusarium wilt of chickpea in regions devoid of F. oxysporum f. sp. ciceris, effective quarantine and the use of certified pathogen-free seed are crucial. To prevent the spread of the infection through seed, healthy seed should be grown in regions free of pathogens. In order to detect and identify the pathogen in certification programs, phytosanitary inspections, and quarantine laws, Jimenez-Fernandez et al. (2011a) recently developed a real-time quantitative polymerase chain reaction (q-PCR) protocol that enables quantifying F. oxysporum f. sp. cereis DNA down to 1 pg in soil as well as in the roots and stems of infected asymptomatic chickpea plants. According to Haware et al. (1978), seed dressing with Benlate® T (30% benomyl þ 30% thiram) at 1.5 g kg~1 can eliminate seedborne inoculum. In addition to selecting low disease risk soil and applying

seed treatments with biocontrol agents, it is recommended to utilize certified or fungicide-treated seed. Combined use of choice of sowing date and treatment with biocontrol agents. By using organic amendments, soil solarization, and cleanliness, the inoculum of F. oxysporum f. sp. ciceris in soil can be decreased. Since implementing these disease control strategies might be expensive, they should be evaluated in light of crop harvest economy and disease prediction. Combined use of choice of sowing date and application of biocontrol agents). However, organic amendments and soil solarization might affect the inoculum in a non-specific way. Crop rotation should not be overlooked in the integrated management of chickpea Fusarium wilt, nevertheless, since it will aid in lowering soil inoculum. The risk of illness in the following crop would be decreased by sanitizing Fusarium-wilt-affected chickpea crops by clearing away their waste and burning or igniting them to thermally kill F. oxysporum f. sp. ciceris chlamydospores. It has been demonstrated that burning crop leftovers can significantly lower the quantity of soil-borne inoculum of a number of plant pathogenic fungus. Although burning is regarded as a destructive practice and goes against long-standing conservation policy, crop debris can be similarly thermo-sanitized with less of an impact on the environment by using oil- or propane-fueled flamers that enable more controlled heating.

Use of resistant cultivars

The most effective and economical individual disease control strategy for Fusarium wilt of chickpeas is resistance to the pathogen. Furthermore, in an integrated management approach, the introduction of resistant cultivars would improve the effectiveness of other disease control techniques. Desi germplasm and, to a lesser extent, Kabuli chickpeas and wild Cicer spp. have been found to be resistant to F. oxysporum f. sp. races of the bacterium. Accessions of C. bijugum, C. cuneatum, and C. judaicum showed combined resistance against races 0 and 5, while accessions of C. canariense and C. chorassanicum showed resistance to race 0 but susceptibility to race 5. While some C. pinnatifidum accessions were resistant to race 0, all evaluated were vulnerable to race 5. 165 sources of resistance were found through resistance screening of more than 13,500 Desi germplasm accessions in a wilt-sick plot at ICRISAT. ICC-2862, -9023, -9032, -10803, -11550, and -11551 were among those that demonstrated broad-base resistance in multi-location testing. Similarly, out of 5174 Kabuli germplasm accessions examined for Fusarium wilt resistance at ICARDA (International Center for Agricultural Research in the Dry Areas), 110

resistant lines were found. Resistance to one or more races of F. oxysporum f. sp. ciceris is present in a small number of Kabuli lines: line ILC 9784 (races 0, 1A, and 5); lines ILC 9785, ILC 9786, FLIP 86-93C, FLIP 87-33C, and FLIP 87-38C (races 0 and 1A); lines CA-334.20.4, CA-336.14.3.0, and ICC-14216K (race 5); and line CA-2954 (races 0 and 5). Additionally, some Kabuli cultivars have been created that are resistant to particular races in California, India, Israel, Mexico, and Tunisia. These include the cvs. ICCV-2 through ICCV-6 (race 1A), Andoum 1 and Ayala (race 0), and Gavilan, Surutato-77, Sonora-80, Tubutama, UC-15, and UC-27. In California, Mexico, and Spain, resistance in these six later cultivars that were introgressed from Desi line L-1186 has been successful against races 0, 1A, 1B/C, 5, and 6. There is no evidence of resistance breakdown to yet, despite the fact that chickpeas have race-specific full resistance to F. oxysporum f. sp. ciceris. This suggests that there may be little to no selection for resistance-breaking races in this pathosystem.

Conclusions and future prospects

With the exception of Australia, where the disease has not yet been identified, Fusarium wilt is a significant barrier to chickpea output in the majority of agricultural regions across the world. The pathogen's lengthy survival in soil and the presence of at least eight pathogenic races in its populations promote the development of the disease. Both virulence toward chickpea genotypes and aggressiveness toward susceptible cultivars vary across these races; the latter is connected with the quantity of inoculum and environmental factors necessary for severe illness. The density of pathogen inoculum, warm soil temperatures, and chickpea cultivar vulnerability all influence the incidence and severity of disease. Accurate and prompt detection of the pathogen and its pathogenic races is a prerequisite for integrated disease management strategies, which are the most effective way to manage Fusarium wilt in chickpea. For such, molecular methods have been devised that would be very beneficial. The most feasible and economical individual disease control strategy for managing Fusarium wilt is the use of high-yielding, well-adapted chickpea cultivars that are resistant to the prevalent pathogen race or races. The identification of "desi" and "kabuli" chickpea germplasm lines, as well as the creation of high-yielding "kabuli" cultivars with total resistance to one or more disease races, have advanced significantly. Significant advancements have also been achieved in the understanding of the genetics underlying race-specific resistance. This will make it possible to advance the pyramiding of multiple race-specific resistance in chickpea cultivars, which would improve multilocation stability. It may

also be possible to combine this resistance with tolerance to environmental stressors, such as drought, and resistance to other significant diseases, such as Ascochyta blight, root knot, and cyst nematodes. Preplanting diagnosis of the current F. oxysporum f. sp. ciceris race(s) using molecular techniques may be helpful in avoiding hazardous soils, nevertheless, as the deployment of race-specific resistant cultivars has not yet resulted in resistance breakdown. Chickpea germplasm has also been found to exhibit resistance to slow wilting. The efficiency of the integrated management of Fusarium wilt in chickpeas would be increased by combining the use of this resistance with other preempting disease control measures (such as pathogen-free seed, sanitation to lower inoculum in soil, selection of sowing location and time to lower disease potential, and protection of healthy seeds with fungicides or biocontrol agents). For effective integrated disease management, the preplanting decision-making process necessitates the participation of qualified expert plant pathologists and skillful support for farmers. The top of the trickle-down structure that transfers knowledge to the field needed for the practice of effective integrated disease management is in danger of eroding due to declining or even declining university education in plant pathology and the decline of extension-related activities in commercial agriculture.

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