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Review

# Fusarium wilt of chickpeas: Biology, ecology and management

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ABSTRACT

Chickpeas provide high quality protein to large population sectors in South and West Asia, and the Mediterranean Basin. This crop has a significant role in farming systems as a substitute for fallow in cereal rotations. Fusarium wilt, caused by the soilborne fungus *Fusarium oxysporum* f. sp. *ciceris*, has become a major factor limiting chickpea production worldwide. The pathogen long survival in soil and high pathogenic variability, with eight races 0, 1A, 1B/C, 2, 3, 4, 5, and 6 having been identified so far, are key elements in the development and management of the disease. Development and use of high-yielding cultivars resistant to the prevalent pathogen race(s) in a given area is the most practical and cost-efficient individual disease control measure for management of the disease. Use of seeds certified free from *F. oxysporum* f. sp. *ciceris*, sanitation and cropping practices to reduce inoculum in soil, choice of sowing site and time to reduce disease potential, and protection of healthy seeds with fungicides or biocontrol agents, would be of help for the management of Fusarium wilt in chickpea in the absence of high-yielding, well-adapted resistant chickpea cultivars. Molecular protocols are available for the characterization and monitoring of *F. oxysporum* f. sp. *ciceris* populations that would help in the implementation efficiency of these disease control measures. Improvement of these disease control measures may be further realized by combining slow-wilting cultivars within an integrated management strategy.



## 1. Introduction: the host and the disease

Chickpea (*Cicer arietinum* L.) (diploid, 2n = 16) is considered to be one of the founder crops of modern agriculture (Zohary and Hopf, 2000). This plant is a member of the Papilionoid subfamily of legumes that originated from its wild *C. reticulatus* ancestor in a relatively small area in Turkish Kurdistan of the Fertile Crescent some 8000–9000 years ago (Ladizinsky and Adler, 1976; Lev-Yadun et al., 2000). Chickpea seeds are a major source of human food and animal feed because of their high content of lysine-rich protein (Jukanti et al., 2012). In addition, chickpea cultivation plays a significant role in farming systems as a substitute for fallow in cereal rotations, where it contributes to the sustainability of production and reduces the need for N fertilization through fixing atmospheric nitrogen. Those features make chickpea cultivation of particular importance to food security in the developing world.

There are two main types of chickpea germplasm, namely desi (small, angular, rugose and colored seeds) grown mainly in the Indian subcontinent and kabuli (large to medium-size, rams-head-shaped and beige to white seeds, smooth to scarcely rugose) grown mainly in the Mediterranean Basin. Consumption of desi is restricted primarily to the Middle East and Southeast Asia, whereas kabuli is a popular and valuable global commodity (Singh, 1997).

Chickpea is the second world's most important food legume crop after dry beans (*Phaseolus vulgaris* L.), grown throughout tropical, subtropical and temperate regions in South and West Asia, East and North Africa, southern Europe, North and South America, and Australia (FAOSTAT, 2014). Approximately $13.5 \times 10^6$ ha of chickpea are cultivated in more than 50 countries worldwide that yield nearly $13.1 \times 10^6$ t (FAOSTAT, 2014). Of that, 89.2% is grown in Asia and accounts for 84.5% of the world

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production. India is the leading chickpea-producing country with 73.3% of the world acreage and 67.4% of the production. Pakistan ranks second with 7.3% of world acreage and 5.7% of production, followed by Australia (4.2% acreage, 6.2% of production), Iran (4.1% acreage, 2.3% of production) and Turkey (0.3% acreage, 0.37% of production) (FAOSTAT, 2014). In much of the world, chickpea is cultivated in semi-arid environments and on soils of poor agricultural quality, which combined with yield losses caused by biotic and abiotic stresses, mainly drought, have given rise to average yields of 0.9–1.8 t/ha across these areas of cultivation, which is considerably below the theoretical potential (FAOSTAT, 2014).

Fusarium wilt is one of the most important diseases affecting chickpea worldwide. This disease was first reported in India by Butler in 1918 but its etiology was not correctly determined until 1940 by Padwick. Now, it is widespread in most chickpea growing areas in Asia, Africa, southern Europe and the Americas, but it has not yet been reported in Australia (Cunnington et al., 2007). Fusarium wilt has become a major factor limiting chickpea production in the Mediterranean Basin, the Indian subcontinent, and California (Haware, 1990; Jalali and Chand, 1992; Nene and Reddy, 1987; Trapero-Casas and Jiménez-Díaz, 1985; Westerlund et al., 1974).

Symptoms of the disease can develop at any stage of plant growth and affected plants may be grouped in patches or appear spread across a field (Haware, 1990; Nene and Reddy, 1987; Trapero-Casas and Jiménez-Díaz, 1985). Highly susceptible cultivars can show symptoms within 25 days after sowing (designated 'early wilt'), including flaccidity of individual leaves followed by a dull-green discoloration, desiccation and collapse of the entire plant. However, symptoms are usually more conspicuous at the onset of flowering, 6–8 weeks after sowing, and can also appear up to podding stage ('late wilt'). Late wilted plants exhibit drooping of the petioles, rachis and leaflets, followed by yellowing and necrosis of foliage. Initially, drooping is observed in the upper part of the plant but within few days it occurs on the entire plant. Symptoms may affect only a few branches of a plant resulting in partial wilt. Roots of affected seedlings and plants show no external root discoloration if they are uprooted before being severely affected or dried. However, the roots and stem of a plant develop a dark-brown discoloration of xylem tissues that can be seen when they are split vertically or cross-sectioned. Histological distortions occur in the vascular tissues of affected roots and stems as a result of cavity formation between phloem and xylem, xylem and medulla, and phloem and cortical parenchyma, as well as anomalous cellular proliferation in the vascular cambium. This, together with formation of optically dense gels and occlusions in xylem vessel (but not of tyloses), probably contributes to retarded vascular flow of water and nutrients as well as development of morphological symptoms (Jiménez-Díaz et al., 1989a).

Fusarium wilt reduces chickpea production by decreasing both seed yield and seed weight (Haware and Nene, 1980; Navas-Cortés et al., 2000b). Yearly yield losses from the disease were roughly estimated at 10–15% in India and Spain (Singh and Dahiya, 1973; Trapero-Casas and Jiménez-Díaz, 1985) and 40% in Tunisia (Bouslama, 1980), but 70% to total loss of the crop can occur in years of severe outbreaks (Halila and Strange, 1996). Early wilting is reported to cause more yield loss (77–94%) than late wilting (24–65%), but seeds from late-wilted plants are lighter, rougher, and duller than those from healthy plants (Haware and Nene, 1980).

This article is not intended to be a thorough review of the literature on general aspects of Fusarium wilt of chickpea. Rather, we discuss the current prospects for its management based on the critical assessment of available knowledge on the disease etiology, epidemiology, and control strategies and measures.

## 2. Genetic and pathogenic diversity in the pathogen populations

Fusarium wilt of chickpea is caused by *Fusarium oxysporum* (Schlechtend.:Fr.) f. sp. *ciceris* (Padwick) Matuo & K. Sato. The fungus was first named *Fusarium orthoceras* Appel & Wollenw. var. *ciceri* by Padwick, and later Chattopadhyay and Sen Gupta renamed the pathogen *F. oxysporum* Schl. f. sp. *ciceri* (Padwick) Snyder & Hansen. This was accepted as the correct name of the pathogen until revised by Holliday in 1980 (Jalali and Chand, 1992; Nene and Reddy, 1987). *F. oxysporum* f. sp. *ciceris* is one of the few formae speciales of monophyletic origin in the *F. oxysporum* complex of the *Gibberella* clade, most of which are polyphyletic (Baayen et al., 2000; Demers et al., 2014; Kistler, 2001; Jiménez-Gasco et al., 2002; O'Donnell et al., 1998). This fungus is pathogenic only on *Cicer* spp. (Kaiser et al., 1994) of which chickpea is the only cultivated species. However, *F. oxysporum* f. sp. *ciceris* can also invade root tissues of other grain legumes such as bean, faba bean (*Vicia faba*), lentil (*Lens culinaris*), pea (*Pisum sativum*), and pigeonpea (*Cajanus cajans*) without causing external symptoms, thus serving as symptomless carriers of the pathogen. Other crops and dicotyledonous weeds can also serve as symptomless carriers (Haware and Nene, 1982a; Trapero-Casas and Jiménez-Díaz, 1985).

*F. oxysporum* f. sp. *ciceris* exhibits extensive pathogenic variability despite being monophyletic. Two pathotypes have been distinguished based on the distinct yellowing or wilting syndromes with brown vascular discoloration that they induce in susceptible chickpeas. The yellowing syndrome is characterized by a slow, progressive foliar yellowing and late death of the plant, while the wilting syndrome is characterized by a fast and severe chlorosis, flaccidity and early plant death (Trapero-Casas and Jiménez-Díaz, 1985). In addition to symptom types, the two pathotypes differ genetically: they can be distinguished unambiguously by random amplified polymorphic DNA (RAPD) markers (Kelly et al., 1994) as well as by specific polymerase-chain-reaction (PCR) assays using sequence characterized amplified region (SCAR) primers derived from those RAPD markers (Kelly et al., 1998). Isolates of the two pathotypes were placed in two significantly distinct groups based on RAPD and DNA fingerprinting assays (Jiménez-Gasco et al., 2001, 2004a; Kelly et al., 1994).

In addition to pathotypes, eight pathogenic races (namely races 0, 1A, 1B/C, 2, 3, 4, 5, and 6) can be identified in *F. oxysporum* f. sp. *ciceris* by the severity of disease reactions on a set of 10 differential chickpea cultivars (Table 1) (Haware and Nene, 1982b; Jiménez-

**Table 1**
Disease reaction of differential chickpea lines to pathogenic races of *Fusarium oxysporum* f. sp. *ciceris*[a].

| Differential chickpea line | Pathogenic race | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | 0 | 1A | 1 B/C | 2 | 3 | 4 | 5 | 6 |
| 12-071/10054 | S | M | S | R | R | R | R | M |
| JG-62 | R | S | S | S | S | S | S | S |
| C-104 | M | M | R/M | S | S | S | S | M |
| JG-74 | R | R | R | S | R | R | M | R |
| CPS-1 | R | R | R | S | M | M | M | R |
| BG-212 | R | R | R | S | M | M | R | R |
| WR-315 | R | R | R | R | S | R | R | R |
| ICCV-2 | R | R | R | S | S | S | S | M |
| ICCV-4 | R | R | R | S | S | S | S | M |
| P-2245 | S | S | S | S | S | S | S | S |

[a] Disease evaluated on a 0–4 severity scale depending on the percentage of affected foliar tissue (0 = 0%, 1 = 1–33%, 2 = 24–66, 3 = 67–100, 4 = dead plant) at 40 days after sowing in infested soil. Average disease reactions of <1 and >3 were considered resistant (R) and susceptible (S), respectively. Intermediate disease reactions were considered moderately susceptible (M) (Jiménez-Díaz et al., 1989b, 1993a; Jiménez-Gasco et al., 2004b).