Crop Protection xxx (2015) 1–12

Contents lists available at ScienceDirect

Crop Protection

journal homepage: www.elsevier.com/locate/cropro

Review

Fusarium wilt of chickpeas: Biology, ecology and management

Rafael M. Jiménez-Díaz [a, b, *], Pablo Castillo [b], María del Mar Jiménez-Gasco [c], Blanca B. Landa [b], Juan A. Navas-Cortés [b]

[a] Departamento de Agronomía, Universidad de Córdoba, Campus de Excelencia Internacional Agroalimentario ceiA3, Edificio C4 "Celestino Mutis", Ctra. de Madrid Km. 396, 14071 Córdoba, Spain
[b] Departamento de Protección de Cultivos, Instituto de Agricultura Sostenible (IAS), Consejo Superior de Investigaciones Científicas (CSIC), P.O. Box 4084, 14080 Córdoba, Spain
[c] Department of Plant Pathology and Environmental Microbiology, The Pennsylvania State University, University Park, PA 16802, USA

ARTICLE INFO

Article history:
Received 23 September 2014
Received in revised form
15 January 2015
Accepted 20 February 2015
Available online xxx

Keywords:
Cicer arietinum
Fusarium oxysporum f. sp. *ciceris*
Legumes
Integrated control
Epidemiology
Resistance

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 13.5×10^6 ha of chickpea are cultivated in more than 50 countries worldwide that yield nearly 13.1×10^6 t (FAOSTAT, 2014). Of that, 89.2% is grown in Asia and accounts for 84.5% of the world

* Corresponding author. Departamento de Protección de Cultivos, Instituto de Agricultura Sostenible (IAS), Consejo Superior de Investigaciones Científicas (CSIC), P.O. Box 4084, 14080 Córdoba, Spain.
E-mail address: ag1jidir@uco.es (R.M. Jiménez-Díaz).

http://dx.doi.org/10.1016/j.cropro.2015.02.023
0261-2194/

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).

Please cite this article in press as: Jiménez-Díaz, R.M., et al., Fusarium wilt of chickpeas: Biology, ecology and management, Crop Protection (2015), http://dx.doi.org/10.1016/j.cropro.2015.02.023

Díaz et al., 1989b, 1993a). This set was extended from an original one developed at the International Crop Research Institute for Semi-Arid Tropics (ICRISAT) in India to study differential isolate × cultivar interactions based on the incidence of plant mortality (Haware and Nene, 1982b). More recently, Sharma et al. (2005) developed a more concise set of eight chickpea lines comprised of four genotypes and four F_7 recombinant inbred lines as differentials for *F. oxysporum* f. sp. *ciceris* race identification (Table 2). Disease reaction of these latter differentials can differentiate races 1A through race 5 only by early appearance of symptoms and 100% wilt incidence. The two tables show conflicting reactions of line WR-315 to race 3. This race can be clearly identified by 100% wilt incidence on near-isogenic line (NIL) RIP8-94-11, which showed a susceptible reaction to races 1A through 4 (Castro et al., 2010). Recently, race 3 of *F. oxysporum* f. sp. *ciceris* was claimed to be identical to *Fusarium proliferatum* (Gurjar et al., 2009) despite that these two species are generally easy to distinguish morphologically, based on the formation of microconidia in short chains from polyphialidic conidiophores and the absence of chlamydospores in the latter species (Leslie and Summerell, 2006). This reclassification of *F. oxysporum* f. sp. *ciceris* race 3 was established on a phylogenetic analysis based on sequences of the translation elongation factor 1-α (TEF) gene using the 'race 3 standard' available from ICRISAT (assigned NRRL number 32155 by Gurjar et al., 2009), and isolate Fu-7 classified as 'race 3-like'. This is in disagreement with results of other studies using two different race 3 isolates, 8606 and 1992R3N, also from ICRISAT, which unequivocally identified them as *F. oxysporum* f. sp. *ciceris*. It was found that these latter isolates do not produce microconidia in short chains from polyphialidic conidiophores (a characteristic of *F. proliferatum*), but are *F. oxysporum* based on restriction fragment length polymorphism (RFLP) assays of the ribosomal intergenic spacer region (IGS) (RFLP-IGS) and sequence analyses using TEF and the internal transcribed spacer region of the ribosomal DNA (rDNA ITS) (Demers et al., 2014; Jiménez-Fernández et al., 2011b). In addition, the two isolates amplified a *F. oxysporum* f. sp. *ciceris*-specific PCR marker (Jiménez-Fernández et al., 2011a; Jiménez-Gasco and Jiménez-Díaz, 2003), have the same differential virulence as originally reported (Haware and Nene, 1982b; Jiménez-Gasco et al., 2001; Kelly et al., 1994), and are closely related to the other *F. oxysporum* f. sp. *ciceris* races, especially to races 2 and 4 from India (Jiménez-Gasco et al., 2001, Jiménez-Gasco et al., 2002, 2004a; Kelly et al., 1994).

Besides their identification based on biological pathotyping, identification of *F. oxysporum* f. sp. *ciceris* races 0, 5, and 6 can be made by means of a specific-PCR assay using DNA from fungal mycelia and primers FocR0-M15f/FocR0-M15r, FocR5-L10f/FocR5-L10r, or FocR6-O2f/FocR6-O2r, which selectively amplify a single 900-, 938-, and 1000-bp race markers, respectively. Furthermore, the use of primers FocR6-P18f and FocRo-M15r identifies race 1A and race 6 isolates simultaneously (Jiménez-Gasco and Jiménez-Díaz, 2003). Therefore, a positive result from this PCR assay together with a negative result from PCR assay using the race 6-specific primer pairs allow for the identification of race 1A isolates (Jiménez-Gasco and Jiménez-Díaz, 2003).

The eight *F. oxysporum* f. sp. *ciceris* races also differ in their pathotype and geographic distribution. Races 0 and 1B/C belong to the yellowing pathotype whereas races 1A through 6 belong to the wilting pathotype. Races 0, 1A, 1B/C, 5 and 6 have been reported in the Mediterranean region and in California (Halila and Strange, 1996; Jiménez-Díaz et al., 1993a; Jiménez-Gasco and Jiménez-Díaz, 2003; Jiménez-Gasco et al., 2001), while races 1A, 2, 3, and 4 have been reported in India (Haware and Nene, 1982b). Recently, races 2 and 3 were reported in Turkey (Bayraktar and Dolar, 2012; Dolar, 1997), races 2, 3, and 4 in Ethiopia (Shehabu et al., 2008), races 0, 1B/C, 5 and 6 in northwestern Mexico (Arvayo-Ortiz et al., 2011), and races 0, 1B/C, 4 and 5 in Iraq (Al-Taae et al., 2013).

Despite their extensive pathogenic variability and geographic distribution, the eight identified races of *F. oxysporum* f. sp. *ciceris* display little genetic diversity. Firstly, all isolates of *F. oxysporum* f. sp. *ciceris* studied share the same RFLP pattern for mitochondrial DNA and IGS region (Jiménez-Fernández et al., 2011b; Pérez-Artés et al., 1995), belong to the same vegetative compatibility group (Nogales-Moncada et al., 2009) and were found to have identical sequences for genes encoding TEF, β-tubulin, histone 3, actin, and calmodulin (Jiménez-Gasco et al., 2002), regardless of race, pathotype, or geographic origin. In a latter study using a larger sample of *F. oxysporum* f. sp. *ciceris* isolates, Demers et al. (2014) further confirmed such little genetic diversity; they found that all isolates of the pathogen tested share identical rDNA ITS, five mitochondrial regions previously found to be polymorphic among *F. oxysporum* populations (Cunnington, 2006), a xylanase gene (*xyl4*) and its transcriptional activator (*xlnR*), SCAR-PCR markers previously developed for identification of *F. oxysporum* f. sp. *ciceris* and of race 5 (Jiménez-Gasco and Jiménez-Díaz, 2003) and 11 microsatellites. Moreover, only a few polymorphisms were observed between and sometimes within races for the β-tubulin gene, IGS region of the rDNA, endopolygalacturonase *pg1* and exopolygalacturonase *pgx4* genes, and six microsatellite regions (Demers et al., 2014). Such a high degree of genetic similarity among races supports the monophyletic origin of this forma specialis previously reported (Jiménez-Gasco et al., 2002), as well as the stepwise pattern of evolution of the races that was first hypothesized and demonstrated by Jiménez-Gasco et al. (2004a; 2004b). In that work, the authors inferred an intraspecific phylogeny of races from DNA fingerprints generated by hybridization of restricted genomic DNA with several transposable elements, whereby each of the eight races forms a monophyletic lineage. Mapping the specific pathogenicity of races to chickpea differential cultivars onto the inferred phylogeny indicated that races appear to have evolved in a stepwise fashion, with each race evolving from another and gaining the ability to cause disease on a previously resistant chickpea cultivar according to two simplest scenarios of few parallel gains or losses of virulence. The scenario based on the gains, but not loss of virulence, is consistent with the yellowing race 0 being ancestral to wilting races and race 1B/C being its closest race in evolutionary terms. This inferred scenario would be consistent with race 0: (i) pathogenic on the fewest race-differentials of all races; (ii) being the most widespread race in the Mediterranean region, although it has not been reported from the Indian subcontinent, and (iii) showing the highest molecular diversity of all races. A second scenario of race evolution proposed that race 1A is the common ancestor of all races, which would be

Table 2
Disease reaction of selected chickpea differentials to five races of *Fusarium oxysporum* f. sp. *ciceris* (Sharma et al., 2005. Plant Dis. 89: 385–390).

Germplasm accession	Differential line	Pathogenic race				
		1A[a]	2	3	4	5
W6-24867	JG-62[b]	S (100)	S (94.3)	S (100)	S (100)	S (100)
W6-24868	P-2245[b]	S (100)	S (100)	S (100)	S (100)	S (100)
W6-24869	Sanford	R (0)	S (100)	S (100)	S (100)	S (95.0)
W6-24870	CRIL-1-53	S (100)	R (0)	R (0)	R (0)	R (0)
W6-24871	CRIL-1-94	R (0)	S (100)	R (0)	I (36.4)	I (30.0)
W6-24872	CRIL-1-17	R (0)	R (0)	R (0)	S (100)	R (0)
W6-24874	CRIL-1-36	I (33.3)	S (100)	S (100)	S (100)	R (0)
W6-24876	WR-315	R (0)	R (0)	R (0)	R (0)	R (0)

[a] S = susceptible, R = resistant, I = intermediate, disease incidence (%) in parentheses.
[b] JG-62 and P-2245 are resistant and susceptible, respectively, to race 0.

Please cite this article in press as: Jiménez-Díaz, R.M., et al., Fusarium wilt of chickpeas: Biology, ecology and management, Crop Protection (2015), http://dx.doi.org/10.1016/j.cropro.2015.02.023

consistent with this race being the most widespread geographically and a subsequent development of pathogenic races within the lineage (Jiménez-Gasco et al., 2004a, 2004b). Subsequently, Demers et al. (2014) found that wilting races 1A, 5 and 6 had very little intra-race diversity for two specific microsatellite markers compared with the high allele diversity shown by yellowing-inducing races 0 and 1B/C, which is consistent with the hypothesis that these races are ancestral to wilting races. It seems unlikely that the stepwise evolution of races in *F. oxysporum* f. sp. *ciceris* may have resulted from selection by specific resistance in chickpea populations based on the wide geographic distribution of races even where resistant cultivars have not been deployed. For example, a high diversity of races occurs in the Mediterranean region despite resistant cultivars generally not being used in this region (Halila and Strange, 1996; Jiménez-Díaz et al., 1993a; Jiménez-Gasco and Jiménez-Díaz, 2003; Jiménez-Gasco et al., 2001). Conversely, widespread use of race 1A-resistant cvs. ICCV-2 and ICCV-4 in India has not yet led to reports on development of race 6, which specifically overcomes that resistance and derives from race 1A. However, races 2, 3, and 4 are pathogenic to those cultivars and were reported in India before these cultivars had been released (Haware and Nene, 1982b; Kumar et al., 1985). Thus, there may have been little or no selection for resistance-breaking races of *F. oxysporum* f. sp. *ciceris*, which possibly minimizes the probabilities of resistance deployment being accompanied by development of parallel changes in virulence overcoming it.

Although the previously referenced research on genetic diversity in *F. oxysporum* f. sp. *ciceris* included few isolates from races found in India, a significant amount of research has analyzed variability in populations from India using a variety of molecular tools (Dubey and Singh, 2008; Dubey et al., 2012, 2014; Durai et al., 2012; Honnareddy and Dubey, 2006; Sharma et al., 2009, 2014). These studies show very high genetic diversity in *F. oxysporum* sampled from chickpea in India. However, some doubts exist whether the isolates studied were truly pathogenic on chickpea since the methodology used for pathogenicity tests differs significantly from the one originally described by Haware and Nene (1982b) using infested soil and controlled environmental conditions. Inoculation methods that rely solely on root-dipping in conidial suspension and incubation of the plants in greenhouse or field conditions may be inconsistent and non-reproducible (Jiménez-Díaz, *unpublished*). Furthermore, pathogenicity tests rarely included appropriate positive controls of known pathogenic races; consequently, more research is needed to make definitive conclusions about diversity of *F. oxysporum* f. sp. *ciceris* in India.

3. Pathogen biology and ecology

F. oxysporum f. sp. *ciceris* is an asexually-reproducing root-inhabiting (soil invader) fungus *sensu* Garret (1956), which survive inactive in soil by means of chlamydospores free or embedded in plant tissues. Temperature and pH ranges for *in vitro* mycelial growth of the fungus are 7.5 to 35 °C and 4 to 9.4, respectively; the optimal conditions being 25 to 27.5 °C and 5.1 to 5.9, depending upon the strain. Optimum pH for sporulation is 7.1–7.9. For a given temperature, isolates of the yellowing pathotype grow at a higher rate compared with that of wilting isolates (Duro Almazán, 2000). Chlamydospores are formed in old mycelia and infected chickpea tissues; they are smooth or rough walled, terminal or intercalary in hyphae, and may be formed single, in pairs, or in short chains.

The fungus can survive in soil and chickpea debris by means of chlamydospores for at least 6 years (Haware et al., 1996) but infection of symptomless dicotyledonous weeds can enhance survival of the pathogen in fallow soils. Thus, infested soil is a main source of primary inoculum for the development of Fusarium wilt epidemics in chickpea. Infected seeds are also a source of primary inoculum of the disease. *F. oxysporum* f. sp. *ciceris* is internally seedborne and chlamydospore-like structures have been observed in the hilum region of the seed. Plants grown from infected seeds wilt faster than infected plants that originated from healthy seeds sown in infested soil (Haware et al., 1978). Infected seeds play an important role in the long-distance dispersal of the pathogen and in its introduction into *F. oxysporum* f. sp. *ciceris*-free soils and geographic areas (Pande et al., 2007). Short-distance spread of the pathogen can also occur by dispersal of infested soil or chickpea debris through human activity, machinery, water, or wind.

Chlamydospores in soil are the primary inoculum for Fusarium wilt in chickpea, their germination being stimulated by seed and root exudates of hosts and non-hosts. *F. oxysporum* f. sp. *ciceris* gains ingress in germinating seeds and growing seedlings directly without need of wounds soon after sowing in infested soil. Invasion takes place mainly through the cotyledons and zones of the epicotyl and hypocotyl at the junction of or close to cotyledons, and to a lesser extent in the zone of root elongation and maturation (Jiménez-Díaz et al., 1989a; Stevenson et al., 1997). Later studies in infested hydroponic cultures showed that races 0 and 5 of the pathogen colonize the surface of the tap and lateral roots in both susceptible and resistant cultivars, and preferentially penetrate the meristematic cells of the root apex (Jiménez-Díaz et al., 1989a; Jiménez-Fernández et al., 2013). Then, the fungus grows in the intercellular spaces of the root cortex to reach the central root cylinder and enter into the xylem vessels. Further colonization by the pathogen takes place by means of hyphal growth and microconidia carried in the vessels by transpiration stream, as well as by lateral mycelia spread to adjacent vessels from infected ones. The systemic colonization along the plant axis (i.e., the determinative phase of pathogenesis) is then followed by development of symptoms (i.e., the expressive phase) once intense colonization of xylem vessels in root and lower stem has occurred by 10–20 days after inoculation (Jiménez-Díaz et al., 1989a; Jiménez-Fernández et al., 2013).

The rate and intensity at which the pathogen colonizes the epicotyl and stem xylem is directly related to the degree of compatibility of the *F. oxysporum* f. sp. *ciceris* race/chickpea genotype combination, being highest in the most susceptible line infected with the most virulent race (i.e., line P-2245/race 5), followed by those in line JG-62 infected by race 5, and 'P-2245' infected by the less virulent race 0 (Jiménez-Fernández et al., 2013). Abundant chlamydospores form in infected tissues as severe symptoms develop and the plant senesces. Eventually, these chlamydospores are released into the soil as infested debris decomposes. Chlamydospores may undergo cycles of renewal by limited saprophytic growth of the fungus supported by organic debris and root exudates, as well as by transient infections of hosts and non-hosts.

Compared with those compatible interactions, incompatible interactions involving same races but different chickpea lines were asymptomatic (Jiménez-Fernández et al., 2013). In these interactions, the pathogen remained either in the intercellular spaces of the root cortex failing to reach the xylem ('WR-315'/race 0), invaded the root and hypocotyl xylem vessels to a limited extent ('WR-315'/race 5) or colonized extensively the root and stem xylem vessels ('JG-62'/race 0). These reactions suggest that multiple defense mechanisms may be operating in the resistant plants (Jiménez-Fernández et al., 2013).

4. Epidemiology

Development of Fusarium wilt of chickpea can be influenced by the aggressiveness (defined as the amount of disease caused by a

Please cite this article in press as: Jiménez-Díaz, R.M., et al., Fusarium wilt of chickpeas: Biology, ecology and management, Crop Protection (2015), http://dx.doi.org/10.1016/j.cropro.2015.02.023

pathogen genotype on a given host genotype) of pathogenic races, inoculum density of the pathogen in soil, environmental conditions (e. g. air and soil temperature, soil moisture, soil pH, etc.) and cultivar susceptibility.

Fusarium wilt caused by an unidentified race of *F. oxysporum* f. sp. *ciceris* was reported to increase with decreasing soil matrix potential and to develop severely at 25 and 30 °C, but not at 15 and 20 °C, with an inoculum density of 500 and 1000 propagules g^{-1} soil. No disease developed at 10 °C even with an inoculum density of 5000 propagules g^{-1} soil (Bhatti and Kraft, 1992). Similarly, a threshold of 483 propagules of race 1A g^{-1} soil was reported for 100% disease incidence in 'JG-62', a highly susceptible, early wilting cultivar. This inoculum density caused no disease in late wilting cv. K-850, but 3283 propagules of race 1A g^{-1} soil were needed to cause significant disease incidence (ICRISAT, 1989).

The relationship between soil temperature and inoculum density of *F. oxysporum* f. sp. *ciceris* races 0 and 5 on disease development in chickpea cultivars differing in susceptibility was determined using quantitative nonlinear models. The models indicated a temperature x race aggressiveness (or cultivar susceptibility) interaction in Fusarium wilt. Moreover, the models estimated 22 to 26 °C as the most favorable soil temperature for infection of line P-2245 (most susceptible) and cv. PV-61 (less susceptible) by race 5, and 24 to 28 °C for infection of 'P-2245' by race 0. At 10 °C, no disease developed except in the most compatible interaction 'P-2245'/race 5. At an optimum soil temperature, maximum disease in 'P-2245' developed with 6 and 50 chlamydospores g^{-1} soil of races 5 (at 22 to 26 °C) and 0 (at 24 to 28 °C), respectively; and in 'PV-61' with 1000 chlamydospores g^{-1} soil of race 5 (at 22 to 26 °C) (Navas Cortés et al., 2007). Furthermore, at extreme temperatures, plants were either asymptomatic or developed moderate disease even when inoculum density was optimum for disease development. Similarly, at low inoculum density, no or little disease developed even at soil temperatures optimal for *F. oxysporum* f. sp. *ciceris* infection (Navas Cortés et al., 2007). Risk threshold charts indicated that limitation in disease by a deficient factor is compensated by another factor. These charts can be applied to predict the potential threat of Fusarium wilt in a geographic area based on soil temperature, the race and inoculum density in soil, and susceptibility of cultivars. The efficient application of the risk models will require the identification of the races of *F. oxysporum* f. sp. *ciceris* that prevail in a geographical area as well as the level of resistance of local or commercial cultivars to be used (Navas Cortés et al., 2007).

Also, when aggressiveness of race 1B/C (yellowing pathotype) was compared with that of races 1A and 5 (wilting pathotype) on cv. PV-61, 5000 chlamydospores g^{-1} soil of race 1B/C were needed to cause the same amount of disease that 1000 chlamydospores g^{-1} soil of race 1A. The amount of disease that developed with 5000 chlamydospores g^{-1} soil of race 1A was equal to that developed with 1000 chlamydospores g^{-1} soil of race 5 (Jiménez-Gasco et al., 2004b). Thus, the yellowing *F. oxysporum* f. sp. *ciceris* pathotype appears to be less aggressive than the wilting one, but differences in aggressiveness to a chickpea cultivar may also occur between races within a *F. oxysporum* f. sp. *ciceris* pathotype (Jiménez-Gasco et al., 2004b; Navas-Cortés et al., 2000a).

5. Management

Fusarium wilt of chickpea is a monocyclic disease in which development is driven by the pathogen's primary inoculum. Therefore, management of the disease should be targeted to exclusion of the pathogen as well as by reducing the amount and/or efficiency of the initial inoculum. Disease control measures for such aim should include: (i) use of pathogen-free seeds; (ii) site selection to avoid sowing into high risk soils; (iii) reduction or elimination of inoculum in soil; (iv) use of resistant cultivars; (v) protection of healthy seeds from resident inoculum by means of seed treatment with fungicides or biocontrol agents; and (vi) choice of cropping practices to avoid conditions favoring infection of the plant by the pathogen. Management of Fusarium wilt in chickpea would be best achieved if those disease control measures are used within an integrated management strategy whereby their use is combined either simultaneously or in a sequence (Haware et al., 1990; Jiménez-Díaz and Jiménez-Gasco, 2011).

5.1. Disease diagnosis

Early and exact diagnosis is a first step to ensure efficient management of Fusarium wilt in chickpeas. Careful examination of uprooted, affected plants for the absence of external root symptoms and presence of dark-brown discoloration in xylem tissues of roots and stem can help in the diagnosis of the disease. However, care should be taken to not confuse Fusarium wilt symptoms with leaf yellowing, wilting, and phloem discoloration that are exhibited by chickpea infected by some plant viruses (e.g., *Pea streak carlavirus*) (Kaiser and Danesh, 1971; Kaiser et al., 1993; Nene et al., 1978). Similarly, leaf yellowing and necrosis are frequently displayed by plants infected by other root fungi (e.g., *Fusarium solani* f. sp. *pisi*, *F. solani* f. sp. *eumartii*, *Macrophomina phaseolina*) (Nene et al., 1978; Trapero-Casas and Jiménez-Díaz, 1985; Westerlund et al., 1974). Moreover, care must be taken when confirming initial diagnosis by isolation in pure culture because endophytic, non-pathogenic strains of *F. oxysporum* are frequently isolated even from upper stem tissues of symptomatic chickpeas (Jiménez-Fernández et al., 2011b; Kaiser et al., 1993; Malcolm et al., 2013; Trapero-Casas and Jiménez-Díaz, 1985). In addition, morphology-based diagnosis of *Fusarium* colonies isolated from yellowing chickpeas does not easily allow differentiating *F. oxysporum* f. sp. *ciceris* from *Fusarium redolens*, which was recently demonstrated to cause symptoms on chickpea similar to those induced by the yellowing pathotype of *F. oxysporum* f. sp. *ciceris* except for the absence of vascular discoloration (Jiménez-Fernández et al., 2011b). *F. redolens*, *F. oxysporum* and *F. oxysporum* f. sp. *ciceris* can be adequately differentiated by use of molecular protocols (Bogale et al., 2007; Jiménez-Fernández et al., 2010, 2011b; Jiménez-Gasco and Jiménez-Díaz, 2003).

5.2. Exclusion and eradication of the pathogen

Effective quarantine and use of certified pathogen-free seed are essential for the management of Fusarium wilt of chickpea in areas free from *F. oxysporum* f. sp. *ciceris* (Pande et al., 2007). Healthy seed should be produced in pathogen-free areas to avoid seedborne dissemination of the pathogen. Recently, Jiménez-Fernández et al. (2011a) developed a real-time quantitative polymerase chain reaction (q-PCR) protocol that allows quantifying *F. oxysporum* f. sp. *ciceris* DNA down to 1 pg in soil as well as in root and stems of infected asymptomatic chickpea plants that may be of use for the detection and identification of the pathogen in certification programs, phytosanitary inspections, and quarantine legislation. Seedborne inoculum can be eradicated by seed dressing with Benlate® T (30% benomyl + 30% thiram) at 1.5 g kg^{-1} (Haware et al., 1978). Use of certified or fungicide-treated seed should be used in combination with choice of low disease risk soil and seed treatments with biocontrol agents (see 5.4. Combined use of choice of sowing date and treatment with biocontrol agents).

Inoculum of *F. oxysporum* f. sp. *ciceris* in soil can be reduced by sanitation, soil solarization and organic amendments. Applying these disease control measures can be costly and must therefore be

Please cite this article in press as: Jiménez-Díaz, R.M., et al., Fusarium wilt of chickpeas: Biology, ecology and management, Crop Protection (2015), http://dx.doi.org/10.1016/j.cropro.2015.02.023

considered according with disease prediction and economy of the crop harvest (see 5.4. Combined use of choice of sowing date and treatment with biocontrol agents). Nevertheless, soil solarization and organic amendments may have a non-specific effect on inoculum of soilborne fungal pathogens and plant-parasitic nematodes and thus benefit crops in rotations with chickpeas, among which the treatment costs must be distributed to be affordable. Reduction in the amount of soilborne inoculum by crop rotation is of lesser efficacy because of the capability of the pathogen to survive in soil for long periods as well as of establishing symptomless infections in asymptomatic crop and weed hosts. However, use of crop rotations in the integrated management of Fusarium wilt of chickpea should not be disregarded since this approach will help to reduce soil inoculum.

Sanitation by removal of debris from Fusarium-wilt affected chickpea crops, and burning or flaming them to achieve thermal-killing of *F. oxysporum* f. sp. *ciceris* chlamydospores, would reduce disease risk in the subsequent crop. Burning affected-crop residues has been shown to greatly reduce the amount of soil-borne inoculum of several plant pathogenic fungi (Bockus et al., 1983). Burning is contrary to longstanding conservation policy and considered a destructive practice, but similar thermo-sanitation with lesser environmental impact can be achieved by flaming the crop debris with propane or oil-fueled flamers that allow more controlled heating (Powelson and Rowe, 2008).

Soil solarization for 6–8 weeks during April to May successfully controlled Fusarium wilt and increased chickpea plant growth and yield in India (Chauhan et al., 1988). Soil solarization reduces the pathogen inoculum in soil mainly as a result of the increase of temperature in moist environment produced by covering a thorough tilled, moist soil with thin (25–50 μm), transparent polyethylene or polyvinyl plastic sheets tightly anchored to soil during a period of high temperature and intense solar radiation (Katan, 1981). Thus, soil solarization in areas with Mediterranean type of climate should be practiced during July and August. Solarizing the soil at sublethal temperatures results in C exudation from *F. oxysporum* f. sp. *ciceris* chlamydospores and reduction of their inoculum potential on chickpeas compared with that of unheated chlamydospores (Arora et al., 1996). These effects correspond to the 'weakening' action of sublethal heating on surviving chlamydospores of other *F. oxysporum* ff. spp. reported by Freeman and Katan (1988). Amending the soil with plant material, such as fresh broccoli or grass, before polyethylene mulching can also suppress soilborne fungal inoculum under conditions of sublethal heating of soil. This heating releases biocidal products after microbial degradation of the plant material incorporated into soil, which together with the anaerobic and strongly reducing soil conditions that develop are effective against fungal propagules (Blok et al., 2000; Kirkegaard, 2009).

Disinfestation of *F. oxysporum* f. sp. *ciceris*-infested soil can also be achieved by soil amendments with high N-containing products (e.g., animal manures, blood, bone and meat meal, fish meal, soy meal, etc.). Research by Lazarovits and co-workers (e.g., Bailey and Lazarovits, 2003; Lazarovits, 2004) has convincingly shown that activity of organic materials in the management of soilborne plant pathogens is associated with production of ammonia (NH_3) and nitrous acid (NO_2H) upon microbial degradation of N-containing products at different soil pH. NO_2H is preferentially formed in acidic soils and is more toxic than NH_3 that forms mainly in basic soils (Conn et al., 2005).

5.3. Use of resistant cultivars

Resistance to the pathogen is the most practical and cost-efficient individual disease control measure for management of Fusarium wilt of chickpea. Moreover, use of resistant cultivars would enhance the efficacy of other disease control measures in an integrated management strategy. Resistance to *F. oxysporum* f. sp. *ciceris* races has been identified mainly in desi germplasm and to a lesser extent in kabuli chickpeas, as well as in wild *Cicer* spp. Combined resistance against races 0 and 5 was identified in accessions of *C. bijugum*, *C. cuneatum*, *C. judaicum*, whereas accessions of *C. canariense* and *C. chorassanicum* were resistant to race 0 but susceptible to race 5. All accessions of *C. pinnatifidum* tested were susceptible to race 5 but some were resistant to race 0 (Kaiser et al., 1994). Resistance screening of over 13,500 desi germplasm accessions in a wilt-sick plot at ICRISAT identified 165 sources of resistance (Haware et al., 1992), some of which (ICC-2862, -9023, -9032, -10803, -11550, and -11551) proved to carry broad-base resistance in multi-location testing (Haware et al., 1990; van Rhenen et al., 1992). Likewise, 110 resistant lines were identified among 5174 kabuli germplasm accessions screened for Fusarium wilt resistance at ICARDA (International Center for Agricultural Research in the Dry Areas) (Singh, 1997). A few kabuli lines carry resistance against one or several *F. oxysporum* f. sp. *ciceris* races: 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) (Jiménez-Díaz et al., 1991; Singh and Jiménez-Díaz, 1996); lines CA-334.20.4, CA-336.14.3.0, and ICC-14216K (race 5) (Castillo et al., 2003; Navas-Cortés et al., 1998b), and line CA-2954 (races 0 and 5) (Rubio et al., 2004). Also, a few kabuli cultivars have been developed with resistance against specific races at California, India, Israel, México and Tunisia, including cvs. ICCV-2 through ICCV-6 (race 1A) (Kumar et al., 1985), Andoum 1 and Ayala (race 0) (Halila and Harrabi, 1990; Landa et al., 2006), and Gavilan, Surutato-77, Sonora-80, Tubutama, UC-15 and UC-27 (Buddenhagen et al., 1988; Helms et al., 1992; Morales, 1986). Resistance in these six later cultivars introgressed from desi line L-1186 is effective against races 0, 1A, 1B/C, 5, and 6 and it has been operative in California, México, and Spain (Jiménez-Díaz et al., 1992; 1993b; and *unpublished*). In spite of the race-specific nature of complete resistance to *F. oxysporum* f. sp. *ciceris* in chickpea, there is no evidence to date of resistance breakdown suggesting that there may be little or no selection for resistance-breaking races in this pathosystem (see 2. Genetic and pathogenic diversity in the pathogen populations).

5.3.1. Genetics and physiology of resistance

Resistance against specific races of *F. oxysporum* f. sp. *ciceris* is described as monogenic or oligogenic depending upon the race or resistance source (Sharma et al. (2005; Sharma and Muehlbauer, 2007; Singh et al., 1987a, 1987b; Upadhyaya et al., 1983a, 1983b). In early studies at ICRISAT on resistance to race 1A, differentiation among segregating chickpea genotypes according to the incubation period (IP, i.e., number of days to appearance of first disease symptoms) after artificial inoculation (early wilting, IP <20 days; late wilting >20 days) lead to hypothesize that the late wilting phenotype was controlled by three independent genes, namely h_1, h_2, H_3. Under this hypothesis, either of the genes in homozygous recessive form and the dominant allele in the third locus, independently confers late wilting resistance, but combination of any two of the late wilting genes confer complete resistance (Singh et al., 1987a, 1987b; Upadhyaya et al., 1983a, 1983b). A similar genetic system based on two (Gumber et al., 1995) or three (Kumar, 1998) independent genes was found to confer resistance to race 2 in wilt sick plot screening, which also involved late wilting or complete resistance and homozygous recessive condition. Later, Sharma et al. (2005) demonstrated that resistance to race 2 in artificial inoculation was governed by a single recessive gene. The genetic of resistance to races of *F. oxysporum* f. sp. *ciceris* was reviewed by Sharma and Muehlbauer (2007). Six single, recessive

Please cite this article in press as: Jiménez-Díaz, R.M., et al., Fusarium wilt of chickpeas: Biology, ecology and management, Crop Protection (2015), http://dx.doi.org/10.1016/j.cropro.2015.02.023

resistance genes have been identified (namely *foc-0*$_1$, *-0*$_2$, *-2*, *-3*, *-4*, and -5) that are located in two clusters on linkage group 2 of the chickpea genetic map, a region considered to be a hotspot for *F. oxysporum* f. sp. *ciceris* resistance genes. Genes *foc-0*$_1$, *-0*$_2$, *-2*, *-3*, *-4*, and -5 confer complete resistance to races 0, 2, 3, 4, and 5, respectively. Resistance to race 0 is controlled by two genes which segregate independently: *foc-0*$_1$ present in accession JG-62, and *foc-0*$_2$ present in lines CA-1938, CA-2139 and WR-315 (Halila et al., 2009, 2010). Both genes separately confer complete resistance to race 0 of the pathogen. Resistance to race 4 was monogenic recessive in some lines (Sharma et al., 2005) whereas it was digenic recessive in 'Surutato-77' (Tullu et al., 1999). Similar to races 1 and 2, the late wilting resistant phenotype was also detected for race 4. More recently, Castro et al. (2010) suggested that complete resistance to race 5 in lines ICCL-81001 and WR-315 is controlled by more than one gene, so that a combination of the gene *foc-5* and other gene/s could be required for complete resistance while the absence of these unknown genes would lead to slow wilting reactions. A race-specific slow wilting reaction in Fusarium wilt of chickpea was first observed by Sharma et al. (2005) in some chickpea lines inoculated with races 2 and 3, and later reported against race 0 in a wilt sick plot (Halila et al., 2010). In addition to race-specificity, slow wilting is characterized by a latent period, disease progress rate, and final disease severity. Compared to slow wilting, late wilting refers to susceptible lines showing a prolonged latent period that eventually show 100% wilt (Sharma and Muehlbauer, 2007). The genetics of the slow wilting reaction have not been studied yet but it has been suggested that it is controlled by minor genes. Similarly, the genetics of resistance to races 1B/C and 6 remains to be determined (Sharma and Muehlbauer, 2007).

As indicated above (see Section 3 Pathogen biology and ecology) the complete-resistant phenotype may vary in histological expression without development of localized cell death. Thus, the reaction of line WR-315 to races 0, 1A and 2, and of line CPS 1 to races 1A and 2, characterized by inability of the pathogen to reach the xylem (Jiménez-Fernández et al., 2013; Stevenson et al., 1997), whereas race 5 invades the root and hypocotyl xylem of 'WR-315', and race 0 colonized extensively the root and stem xylem vessels of 'JG-62' (Jiménez-Fernández et al., 2013). Independent studies suggest that increased concentrations of pterocarpans phytoanticipins maackiain and medicarpin in root tissues and root exudates account at least partially for the resistance of lines CPS-1 and WR-315 to races 1, and 2, and that of cv. ICCV-2 to race 5 (Cachinero et al., 2002; Stevenson et al., 1995, 1997). In addition, some active oxygen species seem to play a role in the resistance of 'WR-315' to race 5. García-Limones et al. (2002), using whole plant extracts, reported that infection by race 5 determined an earlier increase of lipid peroxidation (malondialdehyde formation) as well as of catalase (CAT) and superoxide dismutase (SOD) activities in roots of 'WR-315' compared with that in the susceptible 'JG-62'. Conversely, activities of antioxidant enzymes ascorbate peroxidase (APX), guaiacol-dependent peroxidase (GPX), and glutathione reductase (GR) increased in roots of susceptible 'JG-62' only. Further analyses of stem extracts led the authors to conclude that the enhanced diamine oxidase (DAO) activity in stems, and earlier increases of lipid peroxidation and CAT and SOD activities in roots, can be associated with resistance to race 5 in 'WR-315' (García-Limones et al., 2002). In a follow up study using root apoplastic fluids, García-Limones et al. (2009) did not detect any CAT activity but found that GR and SOD occurred earlier, and DAO occurred in higher quantities in resistant 'WR-315' than in susceptible 'JG-62', and there was a decrease of apoplastic APX activity in this line compared with an increase in 'WR315'. This indicated that oxidative stress-related enzymes in the apoplast of infected roots have a role in Fusarium wilt pathogenesis in chickpeas, as it had been shown before to occur in pathogenesis originated by foliar pathogens (García-Limones et al., 2002, 2009). Cho and Muehlbauer (2004) used a molecular approach to unravel defence mechanisms differentially expressed in resistant 'WR-315' and susceptible 'JG-62' against infection by race 1. These authors found that phenylanine amonium lyase in isoflavonoid biosynthesis as well as APX and GR activities for detoxification of oxidative stresses were upregulated in both 'JG-62' and 'WR-315'. However, there was no significant differential expression of defense-related genes correlating with resistance in 'WR-315'. Based on that, Cho and Muehlbauer (2004) concluded that resistance to the pathogen may not require salicylic- and methyl jasmonate-mediated regulation of defense-related genes, and proposed that induction of these genes after infection by *F. oxysporum* f. sp. *ciceris* is merely an immediate response to the pathogen.

5.3.2. Abiotic and biotic factors influencing resistance to Fusarium wilt

The race-specific resistant response of chickpea cultivars to infection by *F. oxysporum* f. sp. *ciceris* races can be significantly influenced by both temperature and co-infection with plant-parasitic nematodes (Landa et al., 2006; Castillo et al., 2003). Artificial inoculation experiments showed that a 3 °C increase, from 24 to 27 °C, in the incubation temperature was sufficient for the reaction of kabuli cv. Ayala and accession PV-1 to race 1A to shift from moderately or highly resistant at constant 24 °C to highly susceptible at 27 °C. A similar but less pronounced effect was found for 'Ayala' infected with race 6 (Landa et al., 2006). However, the susceptible reaction of accession JG-62 to races 1A and 6 was not influenced by that temperature increase. This temperature effect has an impact on the use of cultural practices for management of Fusarium wilt of chickpea (see 5.3. Combined use of choice of sowing date and treatment with biocontrol agents) as shown by field experiments in Israel, whereby the high level of resistance of 'Ayala' to Fusarium wilt when sown in mid-to late January differed from a moderately susceptible reaction under warmer temperatures when sowing was delayed to late February or early March (Landa et al., 2006).

Inoculum of *F. oxysporum* f. sp. *ciceris* in soil is coincidental with that of plant-parasitic nematodes, and infection of the plant by the two pathogens may risk valuable resistance to the fungus and increase disease severity in susceptible cultivars (Castillo et al., 1998, 2003; Krishna Rao and Krishnappa, 1996; Mani and Sethi, 1987; Navas Cortés et al., 2008). The root-knot (*Meloidogyne* spp.) and root-lesion (*Pratylenchus* spp.) nematodes are among the most important nematodes damaging chickpea (Castillo et al., 2008), and several studies have addressed the influence of joint infections with *F. oxysporum* f. sp. *ciceris* on the reaction of the plant to the fungus. Studies in India showed that co-infections of wilt-resistant chickpea with *Meloidogyne incognita* or *Meloidogyne javanica* can lead to breakdown of resistance to an unidentified race of *F. oxysporum* f. sp. *ciceris* (Krishna Rao and Krishnappa, 1996; Mani and Sethi, 1987; Uma Maheswari et al., 1995, 1997). In Spain, studies focused on interactions with the cereal and legume root-knot nematode, *Meloidogyne artiellia*, which is the only one reportedly attacking chickpeas in the Mediterranean Basin (Castillo et al., 2008; Di Vito and Greco, 1988). Artificial inoculations with race 5 and two *M. artiellia* populations from Italy and Syria showed that infection by the nematode significantly increases the severity of Fusarium wilt in several chickpea lines and cultivars with late-wilting resistance to the disease, regardless of inoculum densities of race 5 (3000 or 30,000 chlamydospores g soil^{-1}), except in line CPS-1 at the lower inoculum density (Castillo et al., 2003). Also, and more importantly, infection of chickpea by *M. artiellia* can

Please cite this article in press as: Jiménez-Díaz, R.M., et al., Fusarium wilt of chickpeas: Biology, ecology and management, Crop Protection (2015), http://dx.doi.org/10.1016/j.cropro.2015.02.023

breakdown complete resistance to race 5 but this effect is influenced by the nature of the chickpea genotype, as well as by inoculum density of the pathogen. Thus, infection by *M. artiellia* overcame complete resistance to race 5 in lines CA-334.20.4 and CA-336.14.3.0, but not in line ICC-14216K at any inoculum density of the fungus. Conversely, complete resistance of cv. UC-27 was overcome by *M. artiellia* only at 30,000 chlamydospores g soil^{-1} (Castillo et al., 2003). The observed variation in the *M. artiellia*-induced breakdown of resistance to *F. oxysporum* f. sp. *ciceris* race 5 depending upon chickpea cultivars may be traced back to the nature of resistance sources. While resistance in 'UC-27' derives from resistant line L-1186 (Buddenhagen et al., 1988; Singh, 1987), resistance in lines CA-334.20.4 and CA-336.14.3.0 traces back to line ICCL-81001 (Navas-Cortés et al., 1998b). Line ICC-14216 K appears to be of Mexican origin (Pundir et al., 1988) and was obtained from ICRISAT's Genetic Resources Unit for multilocation Fusarium wilt-resistance testing in Spain. Lines ICC-14216 K, CA-334.20.4, and CA-336.14.3.0 have shown complete and consistent resistance to race 5 in repeated resistance screening under both artificial and field conditions (Navas-Cortés et al., 1998b; R.M. Jiménez-Díaz *unpublished data*). In addition to factors described above, the *M. artiellia*-induced breakdown of resistance to *F. oxysporum* f. sp. *ciceris* is also influenced by the nature of the pathogenic race. Thus, infection by *M. artiellia* had no effect on the reaction of resistant 'UC 27' and 'ICC-14216 K' to race 0, and of 'ICC-14216 K' to races 1A and 2, at inoculum densities of 20000, 3000, and 3000 chlamydospores g soil^{-1}, respectively (Navas-Cortes et al., 2008).

The mechanisms underlying the *M. artiellia*-induced breakdown of resistance to *F. oxysporum* f. sp. *ciceris* race 5 was further investigated in a proteomic approach in lines CA-336.14.3.0 and ICC-14216 that showed a differential response after co-infection with the two pathogens (Castillo et al., 2003; Palomares-Rius et al., 2011). Comparisons of the root proteomes suggested that the differential responses to race 5 following co-infection by *M. artiellia* does not appear to involve differences in constitutive protein levels in infected plants. On the contrary, the root proteomes of the two chickpea lines displayed clear differences after infection by the two pathogens. 'CA-336.14.3.0' chickpeas, whose resistance to race 5 is overcome by co-infection with *M. artiellia*, displayed a higher number of responsive proteins following infections by the pathogens compared with that of 'ICC-14216 K' in which resistance is not influenced by *M. artiellia*. That difference was due to a higher number of responsive proteins to infection by *M. artiellia*, either alone or jointly with infection by race 5. The number of proteins responsive to infection by race 5 alone was similar in the two chickpea genotypes. The higher responsiveness of 'CA-336.14.3.0' chickpeas compared with 'ICC-14216 K' may relate to a stronger metabolic re-programming in the former line during infection by the nematode, which would govern the differential responses of both genotypes to co-infection by the two pathogens without affecting their similar susceptible response to *M. artiellia*. The small number of proteins affected was common to both chickpea genotypes, but the different levels of protein in each probably plays important roles in the differential response displayed by 'CA-336.14.3.0' and 'ICC-14216 K' chickpeas following co-infections by the two pathogens. This is the case of a class I chitinase in the differential response of the two genotypes to race 5 in plants co-infected with both pathogens (Palomares-Rius et al., 2011).

5.4. Combined use of choice of sowing date and treatment with biocontrol agents

Date of sowing is a key factor in determining yield of chickpea crops (Landa et al., 2004b; Navas-Cortés et al., 1998a, 2000b; Singh and Saxena, 1993). In the Mediterranean region, chickpea is traditionally sown in the spring, and the crop develops on the residual moisture in soil from winter rains. As the season proceeds, the crop experiences rising temperatures and increasing soil moisture stress that shorten the vegetative and reproductive periods and decrease yields (Singh and Saxena, 1993). Fusarium wilt incidence and severity are enhanced by warm, dry soils occurring in spring-sown crops (Gupta et al., 1987; Trapero-Casas and Jiménez-Díaz, 1985; Westerlund et al., 1974). Conversely, winter sowing enables matching of crop growth stages with optimum environmental conditions and increases yield through better use of available water in soil (Singh and Saxena, 1996). Choice of sowing time has been recommended for management of Fusarium wilt of chickpea. Experiments conducted in India showed that Fusarium wilt intensity decreased and chickpea seed yield increased in plantings advanced to mid-October (Jalali and Chand, 1992; Saraf, 1974). The effects of sowing date in the management of Fusarium wilt of chickpea under Mediterranean conditions was addressed by Navas-Cortés et al. (1998a, 2000b) in a 3-year study in southern Spain, which also determined the influence of virulence of the pathogen race and cultivar susceptibility. In this study, advancing the sowing date from early spring to early winter significantly delayed epidemic onset, slowed down epidemic development, and reduced the final disease incidence and severity, and yield loss. However, the net effects of advancing chickpea sowing on control of Fusarium wilt varied with the susceptibility of the cultivar and virulence of the *F. oxysporum* f. sp. *ciceris* race; i.e., the overall benefit for disease management that results from early sowing diminishes if a highly susceptible cultivar is used, a highly virulent race prevails in soil, or both (Navas-Cortés et al., 1998a, 2000b). Indeed, for each sowing date, seed yield loss was determined primarily by virulence of the *F. oxysporum* f. sp. *ciceris* race, and to a lesser extent by susceptibility of the chickpea cultivar. Seed yield loss was higher with the highly virulent race 5 than with the less virulent race 0. This loss in seed yield was greater in line P-2245 (highly susceptible to both races) than in cvs. PV-60 and PV-61 (both susceptible to race 5 and moderately resistant to race 0). Seed yield loss caused by race 5 averaged over sowing dates in the 3 years of study was highest (99.7%) in the most susceptible 'P-2245' and lowest (81.9%) in the least susceptible 'PV-61'. Similarly, seed yield loss caused by race 0 ranged from 65.6 to 30.6% for 'P-2245' and 'PV-61', respectively. Moreover, yield reduction by Fusarium wilt was also associated with poor seed size and quality (Navas-Cortés et al., 2000b).

When using the choice of sowing date as a disease control strategy for management of Fusarium wilt of chickpeas care should be taken to avoid a negative influence on the effects of biological control practices derived from temperatures prevailing in the chosen sowing dates (Landa et al., 2004b) (see below).

Fusarium wilt of chickpea can be controlled by the treatment with different bacterial or fungal biocontrol agents (e.g., *Bacillus* spp., nonpathogenic *F. oxysporum*, *Pseudomonas* spp., and *Trichoderma harzianum*). However, disease suppression by these microbial agents have been shown to be influenced by: i) the inoculum density of the pathogen, ii) the race, strain or isolate of the pathogen, and iii) the environmental conditions prevailing when biocontrol activity should operate (e.g., Hervás et al., 1997, 1998; Landa et al., 1997, 2001).

Chickpea genotype has been shown to play a significant role in supporting populations of biocontrol agents in the plant rhizosphere as well as their activity against *F. oxysporum* f. sp. *ciceris*. For instance, Hervás et al. (1997, 1998) found that two chickpea cultivars ('PV-61', 'ICCV-4') with different genetic background but susceptible to highly virulent *F. oxysporum* f. sp. *ciceris* race 5 varied in the level of wilt suppression achieved on them when their roots were colonized by different biocontrol agents, including a

Please cite this article in press as: Jiménez-Díaz, R.M., et al., Fusarium wilt of chickpeas: Biology, ecology and management, Crop Protection (2015), http://dx.doi.org/10.1016/j.cropro.2015.02.023

nonpathogenic *F. oxysporum* isolate, *T. harzianum* and *Bacillus* spp.. Thus, the extent of protection from Fusarium wilt was always higher and more consistent in cv. PV-61 than in cv. ICCV-4 even though the root system of both cultivars was colonized by the biocontrol agents to the same extent.

The antagonistic potential of four biocontrol agents (*Pseudomonas chlororaphis* 30-84, *Bacillus circulans* RGAF6a, and *Bacillus megaterium* RGAF12 and RGAF51) was shown to vary with the race and geographical origin of *F. oxysporum* f. sp. *ciceris* isolates. Interestingly, *Bacillus* isolates obtained from the chickpea rhizosphere differed in their antagonistic activity and inhibited mycelial growth of *F. oxysporum* f. sp. *ciceris* to a lower extent compared with that of *F. oxysporum* isolates originating from plants other than chickpeas (Landa et al., 1997).

The amount of Fusarium wilt suppression in chickpeas achieved by introduced biocontrol agents has been shown to be influenced also by the inoculum density of the pathogen. Under optimal environmental conditions for disease and in the absence of biocontrol agents, Fusarium wilt development in chickpea was greater at 250 to 1000 chlamydospores g soil^{-1} compared to that reached at 25 to 100 chlamydospores g^{-1} soil. However, seed and soil treatments with *P. fluorescens* RG26 and RGAF19 as biocontrol agents only suppressed Fusarium wilt development at an inoculum density of the pathogen below 250 chlamydospores g^{-1} soil. This suggests that pathogen inoculum potential at high inoculum density is just too high to be counteracted by the biocontrol agents (Landa et al., 2001).

Activity of biocontrol agents in the suppression of Fusarium wilt of chickpea may also be greatly influenced by temperature. Landa et al. (2004a) demonstrated that treatment of soil or chickpea seeds with four biocontrol bacteria (*P. fluorescens* RGAF19 and RG26, *B. megaterium* RGAF51, and *Paenibacillus macerans* RGAF101) delayed chickpea seedling emergence but increased chickpea growth. However, the extent of plant growth promotion decreased and emergence increased as incubation temperature was raised from 20 to 30 °C. Furthermore, these four biocontrol agents colonized the chickpea rhizosphere and grew as endophytes within chickpea stem tissues at 20, 25 and 30 °C. However, while the rhizospheric bacteria population increased with a significant linear trend as temperature increased from 20 to 30 °C, endophytic stem colonization by bacteria was highest at 25 °C. Moreover, incubation conditions strongly interacted in modulating the extent of Fusarium wilt suppression in chickpea by the four biocontrol bacteria referred above (Landa et al., 2001). Interestingly, the disease was suppressed by these bacteria only at 20 or 30 °C, but not at 25 °C, the temperature at which disease developed more severely (Landa et al., 2001). Thus, disease suppression by the bacteria decreased as conditions became more favorable for disease development, to the extent that at 25 °C the disease potential was too high to be counteracted by the biocontrol agents (Landa et al., 2001).

The efficacy of biocontrol agents in the management of Fusarium wilt can be enhanced if combined with choice of sowing date. Landa et al. (2004b) carried out a 3-year study under field conditions in soils infested with *F. oxysporum* f. sp. *ciceris* race 5 in southern Spain, which was aimed to assess the efficacy of combining the use of partially-resistant chickpea genotypes, choice of sowing date and treatments with biological control agents (*P. fluorescens* RG 26, *B. megaterium* RGAF51, *Bacillus subtilis* GB03, and *F. oxysporum* Fo 90105, nonpathogenic to chickpea), in the management of Fusarium wilt. Although Fusarium wilt epidemics developed earlier and faster as mean temperature increased (i.e., delayed sowing date) regardless of biological treatments, the increase in chickpea seed yield was the most consistent effect of the biocontrol agents. However, that effect was primarily influenced by sowing date (i.e., temperature regimes), which also determined disease development. Nevertheless, the biocontrol agents delayed the onset of disease as well as increased seedling emergence. Although treatments with the biocontrol agents provided a moderate level of Fusarium wilt suppression, a significant increase in chickpea seed yield was obtained only with sowing dates that promoted environmental conditions moderately conducive for disease development, indicating a potential benefit from those bacterial strains if combined with other disease control measures in an integrated management strategy.

5.5. Conclusions and future prospects

Fusarium wilt is a major constrain to chickpea production in most areas of cultivation worldwide except Australia, where the pathogen has not been reported to date. Development of the disease is favored by the long survival of the pathogen in soil and the occurrence of at least eight pathogenic races in its populations. These races differ in virulence on chickpea genotypes as well as in aggressiveness on susceptible cultivars, the latter being correlated with the amount of inoculum and environmental conditions required for severe disease. Disease incidence and severity are driven by pathogen inoculum density and warm temperature in soil, and chickpea cultivar susceptibility.

Effective management of Fusarium wilt in chickpea is best achieved by means of integrated disease management strategies, a prerequisite of which is the accurate and quick diagnosis of the pathogen and its pathogenic races. Molecular protocols have been developed that would be of much help for that purpose. Use of high-yielding, well-adapted chickpea cultivars resistant to the prevalent pathogen race(s) is the most practical and cost-efficient individual disease control measure for the management of Fusarium wilt. Significant progress has been made in the identification of 'desi' and 'kabuli' chickpea germplasm lines, as well as in the development high-yielding 'kabuli' cultivars carrying complete resistance to one or more races of the pathogen. Also, significant progress has been made in the unraveling of genetics of race-specific resistance. This will allow further progress in pyramiding multiple race-specific resistance in chickpea cultivars that would enhance multilocation stability, and potentially to combine that with resistance to other important diseases (e.g., Ascochyta blight, root knot and cyst nematodes) and tolerance to environmental stresses (e.g., drought). However, since use of race-specific resistant cultivars has not given rise to resistance breakdown to date, pre-planting diagnosis of the existing *F. oxysporum* f. sp. *ciceris* race(s) by means of molecular protocols would be of help in avoiding risky soils. Slow wilting resistance has also been identified in chickpea germplasm. Combining the use of this resistance with other pre-panting disease control measures (including pathogen-free seed, sanitation 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 enhance efficiency in the integrated management of Fusarium wilt in chickpeas. The pre-planting decision-taking process for efficient integrated disease management requires skillful assistance to famers and involvement of well-trained professional plant pathologists. Declining or even despairing University education in Plant Pathology and the loss of extension-related activities in commercial agriculture are placing a threat of erosion at the top of the trickle-down structure responsible for knowledge transfer to the field required for the practice of efficient integrated disease management.

Acknowledgments

The authors are member of the research group "Sanidad Vegetal-AGR-136". We acknowledge grant AGR-136 from 'Consejería de

Economía, Innovación y Ciencia', Junta de Andalucía, and the European Union, Fondo Europeo de Desarrollo regional, "Una manera de hacer Europa".

References

Al-Taae, A.K., Hadwan, H.A., Al-Jobory, S.A.E., 2013. Physiological races of *Fusarium oxysporum* f. sp. *ciceris* in Iraq. J. Life Sci. 7, 1070–1075.

Arora, D.K., Pandey, A.K., Srivastava, A.K., 1996. Effects of heat stress on loss of C, germination and pathogenicity from chlamydospores of *Fusarium oxysporum* f. sp. *ciceri*. Soil Biol. Biochem. 28, 399–407.

Arvayo-Ortiz, R.M., Esqueda, M., Acedo-Felix, E., Sanchez, A., Gutierrez, A., 2011. Morphological variability and races of *Fusarium oxysporum* f. sp. *ciceris* associated with chickpea (*Cicer arietinum*) crops. Amer. J. Agric. Biol. Sci. 6, 114–121.

Baayen, R.P., ÓDonnell, K., Bonants, P.J.M., Cigelnik, E., Kroon, L.P.N.M., Roebroeck, E.J.A., Waalwijk, C., 2000. Gene genealogies and AFLP analyses in the *Fusarium oxysporum* complex identify monophyletic and nonmonophyletic formae speciales causing root rot and wilt diseases. Phytopathology 90, 891–899.

Bailey, K.L., Lazarovits, G., 2003. Suppressing soil-borne diseases with residue management and organic amendments. Soil Till. Res. 72, 169–180.

Bhatti, M.A., Kraft, J.M., 1992. Effects of inoculum density and temperature on root rot and wilt of chickpea. Plant Dis. 76, 50–54.

Bayraktar, H., Dolar, F.S., 2012. Pathogenic variability of *Fusarium oxysporum* f. sp. *ciceris* isolates from chickpea in Turkey. Pak. J. Bot. 44, 821–823.

Blok, W.J., Lamers, J.G., Termorshuizen, A.J., Bollen, G.J., 2000. Control of soilborne plant pathogens by incorporating fresh organic amendments followed by tarping. Phytopathology 90, 253–259.

Bockus, W.W., O'Connor, J.P., Raymond, P.J., 1983. Effect of residue management method on incidence of *Cephalosporium* stripe under continuous winter wheat production. Plant Dis. 67, 1323–1324.

Bogale, M., Wingfield, B.D., Wingfield, M.J., Steenkamp, E.T., 2007. Species-specific primers for *Fusarium redolens* and a PCR-RFLP technique to distinguish among three clades of *Fusarium oxysporum*. FEMS Microbiol. Lett. 271, 27–32.

Bouslama, M., 1980. Chickpea Improvement in Tunisia, in: Proc. Int. Workshop Chickpea Improvement. International Crops Research Institute for the Semi-Arid Tropics (ICRISAT), Patancheru, India, pp. 277–280.

Buddenhagen, I.W., Workneh, F., Bosque-Pérez, N., 1988. Chickpea improvement and chickpea diseases in California. Intern. Chickpea Newslett. 19, 9–10.

Cachinero, J.M., Hervás, A., Jiménez-Díaz, R.M., Tena, M., 2002. Plant defense reactions against fusarium wilt in chickpea induced by incompatible race 0 of *Fusarium oxysporum* f. sp. *ciceris* and nonhost isolates of *F. oxysporum*. Plant Pathol. 51, 765–776.

Castillo, P., Mora-Rodriguez, Mª.P., Navas-Cortés, J.A., Jiménez-Díaz, R.M., 1998. Interactions between *Pratylenchus thornei* and *Fusarium oxysporum* f. sp. *ciceris* on chickpea. Phytopathology 88, 836–844.

Castillo, P., Navas-Cortés, J.A., Gomar-Tinoco, D., Di Vito, M., Jiménez-Díaz, R.M., 2003. Interactions between *Meloidogyne artiellia*, the cereal and legume root-knot nematode, and *Fusarium oxysporum* f. sp. *ciceris* race 5 in chickpea. Phytopathology 93, 1513–1523.

Castillo, P., Navas-Cortes, J.A., Landa, B.B., Jiménez-Díaz, R.M., Vovlas, N., 2008. Plant-parasitic nematodes attacking chickpea and their in planta interactions with rhizobia and phytopathogenic fungi. Plant Dis. 92, 840–853.

Castro, P., Pistón, F., Madrid, E., Millán, T., Gil, J., Rubio, J., 2010. Development of chickpea near-isogenic lines for Fusarium wilt. Theor. Appl. Genet. 121, 1519–1526.

Chauhan, Y.S., Nene, Y.L., Johansen, C., Haware, M.P., Saxena, N.P., Sardar, S., Sharma, S.B., Sahrawat, K.L., Burford, J.R., Rupela, O.P., Kumar Rao, J.V.D.K., Sithanantham, S., 1988. Effects of soil solarization on pigeon pea and chickpea. Res. Bull. nº 11. International Crops Research Institute for the Semi-Arid Tropics (ICRISAT), Patancheru, A.P., India.

Cho, S., Muehlbauer, F.J., 2004. Genetic effect of differentially regulated fungal response genes on resistance to necrotrophic fungal pathogens in chickpea (*Cicer arietinum* L.). Physiol. Mol. Plant Pathol. 64, 57–66.

Conn, K.L., Tenuta, M., Lazarovits, G., 2005. Liquid swine manure can kill *Verticillium dahliae* microsclerotia in soli by volatile fatty acids, nitrous acid, and ammonia toxicity. Phytopathology 95, 28–35.

Cunnington, J., 2006. Novel primers developed from mitochondrial intergenic spacers are useful for multi-locus sequence typing of *Fusarium oxysporum* strains. Eur. J. Plant Pathol. 116, 77–80.

Cunnington, J., Lindbeck, K., Jones, R.H., 2007. National Diagnostic Protocol for the Detection of Fusarium Wilt of Chickpea (*Fusarium Oxysporum* f. sp. *ciceris*). Plant Health Australia, Camberra, Australia.

Demers, J.E., Garzón, C.D., Jiménez-Gasco, M.M., 2014. Striking genetic similarity between races of *Fusarium oxysporum* f. sp. *ciceris* confirms a monophyletic origin and clonal evolution of the chickpea vascular wilt pathogen. Eur. J. Plant Pathol. 139, 303–318.

Di Vito, M., Greco, N., 1988. The relationship between initial population densities of *Meloidogyne artiellia* and yield of winter and spring chickpea. Nematol. Mediterr. 16, 163–166.

Dolar, F.S., 1997. Determination of the races of *Fusarium oxysporum* f. sp. *ciceris* in Ankara province. Turk. J. Turk. Phytopathol. 26, 11–15.

Dubey, S.C., Priyanka, K., Singh, V., Singh, B., 2012. Race profiling and molecular diversity analysis of *Fusarium oxysporum* f. sp. *ciceris* causing wilt in chickpea. J. Phytopathol. 160, 576–587.

Dubey, S.C., Priyanka, K., Singh, V., 2014. Phylogenetic relationship between different race representative populations of *Fusarium oxysporum* f. sp. *ciceris* in respect of translation elongation factor 1- α, β-tubulin, and internal transcribed spacer region genes. Arch. Microbiol. 196, 445–452.

Dubey, S.C., Singh, S.R., 2008. Virulence analysis and oligonucleotide fingerprinting to detect diversity among Indian isolates of *Fusarium oxysporum* f. sp. *ciceris* causing chickpea wilt. Mycopathologia 165, 398–406.

Durai, M., Dubey, S.C., Tripathi, A., 2012. Analysis of virulence and its region-based genetic variability among the Indian populations of *Fusarium oxysporum* f. sp. *ciceris* causing chickpea wilt. J. Plant Pathol. 94, 651–662.

Duro Almazán, M.D., 2000. Influencia de la Temperatura y el pH en el Desarrollo *in vitro* de Razas de *Fusarium oxysporum* f. sp. *ciceris*. M. Sc. thesis. Universidad de Córdoba, Córdoba, Spain (in Spanish).

FAOSTAT (Food and Agriculture Organization of the United Nations Statistical Database), 2014. FAOSTAT Production Statistics of Crops. Available: http://faostat.fao.org/site/567/default.aspx#ancor (accessed 10.08.14.).

Freeman, S., Katan, J., 1988. Weakening effects on propagules of *Fusarium* by sub-lethal heating. Phytopathology 78, 1656–1661.

García-Limones, C., Dorado, G., Navas-Cortés, J.A., Jiménez-Díaz, R.M., Tena, M., 2009. Changes in the redox status of chickpea roots in response to infection by *Fusarium oxysporum* f. sp. *ciceris*: apoplastic antioxidant enzyme activities and expression of oxidative stress-related genes. Plant Biol. 11, 194–203.

García-Limones, C., Hervás, A., Navas-Cortés, J.A., Jiménez-Díaz, R.M., Tena, M., 2002. Induction of an antioxidant enzyme system and other oxidative stress markers associated with compatible and incompatible interactions between chickpea (*Cicer arietinum* L.) and *Fusarium oxysporum* f. sp. *ciceris*. Physiol. Mol. Plant Pathol. 61, 325–337.

Garret, S.D., 1956. Biology of Root Infecting Fungi. Cambridge University Press, Cambridge, UK.

Gumber, R.K., Kumar, J., Haware, M.P., 1995. Inheritance of resistance to Fusarium wilt in chickpea. Plant Breed. 114, 277–279.

Gupta, O., Kotasthane, S.R., Khare, M.N., 1987. Factors influencing epidemiology of vascular wilt of chickpea. Proc. Natl. Acad. Sci. India 57, 86–91.

Gurjar, G., Barve, M., Giri, A., Gupta, V., 2009. Identification of Indian pathogenic races of *Fusarium oxysporum* f. sp. *ciceris* with gene specific, ITS and random markers. Mycologia 101, 484–495.

Halila, I., Cobos, M.J., Rubio, J., Millán, T., Kharrat, M., Marrakchi, M., Gil, J., 2009. Tagging and mapping a second resistance gene for Fusarium wilt race 0 in chickpea. Eur. J. Plant Pathol. 124, 87–92.

Halila, M.H., Harrabi, M.M., 1990. Breeding for dual resistance to Ascochyta and wilt diseases in chickpea. Options Mediterr. Sér. Sémin. 9, 163–170.

Halila, I., Rubio, J., Millán, T., Gil, J., Kharrat, M., Marrakchi, M., 2010. Resistance in chickpea (*Cicer arietinum*) to Fusarium wilt race 0. Plant Breed. 129, 563–566.

Halila, H.M., Strange, R., 1996. Identification of the causal agent of wilt of chickpea in Tunisia as *Fusarium oxysporum* f. sp. *ciceris* race 0. Phytopathol. Mediterr. 35, 67–74.

Haware, M.P., 1990. Fusarium wilt and other important diseases of chickpea in the Mediterranean area. Options Mediterr. Sér. Sémin. 9, 61–64.

Haware, M.P., Jiménez-Díaz, R.M., Amin, K.S., Phillips, J.C., Halila, M.H., 1990. Integrated management of wilt and root rots of chickpea. In: Chickpea in the Nineties (Ed.), ICRISAT (International Crops Research Institute for the Semi Arid Tropics). ICRISAT Center, Patancheru, India, pp. 129–133.

Haware, M.P., Nene, Y.L., 1980. Influence of wilt at different stages on the yield loss in chickpea. Trop. Grain Legume Bull. 19, 38–40.

Haware, M.P., Nene, Y.L., 1982a. Symptomless carriers of the chickpea wilt Fusarium. Plant Dis. 66, 250–251.

Haware, M.P., Nene, Y.L., 1982b. Races of *Fusarium oxysporum* f. sp. *ciceris*. Plant Dis. 66, 809–810.

Haware, M.P., Nene, Y.L., Natarajan, M., 1996. Survival of *Fusarium oxysporum* f. sp. *ciceri* in the soil in the absence of chickpea. Phytopathol. Mediterr. 35, 9–12.

Haware, M.P., Nene, Y.L., Pundir, R.P.S., Narayana Rao, J., 1992. Screening of world chickpea germplasm for resistance to Fusarium wilt. Field Crops Res. 30, 147–154.

Haware, M.P., Nene, Y.L., Rajeswari, R., 1978. Eradication of *Fusarium oxysporum* f. sp. *ciceris* transmitted in chickpea seed. Phytopathology 68, 1364–1368.

Helms, D., Pamella, L., Buddenhagen, I.W., Workneh, F., Tucker, C.L., Foster, K.W., Gepts, P.L., 1992. Registration of 'UC 15' chickpea. Crop Sci. 32, 500.

Hervás, A., Landa, B.B., Jiménez-Díaz, R.M., 1997. Influence of chickpea genotype and *Bacillus* sp. on protection from Fusarium wilt by seed treatment with nonpathogenic *Fusarium oxysporum*. Eur. J. Plant Pathol. 103, 631–642.

Hervás, A., Landa, B.B., Dafnoff, L., Jiménez-Díaz, R.M., 1998. Effects of host plant resistance, and commercial and indigenous microorganisms on Fusarium wilt development in chickpea. Biol. Control 13, 166–176.

Honnareddy, N., Dubey, S.C., 2006. Pathogenic and molecular characterization of Indian isolates of *Fusarium oxysporum* f. sp. *ciceris* causing chickpea wilt. Curr. Sci. 91, 661–666.

ICRISAT., 1989. Annual Report 1988. ICRISAT, Patancheru, India.

Jalali, B.L., Chand, H., 1992. Chickpea wilt. In: Singh, U.S., Mukhopadhayay, A.N., Kumar, J., Chaube, H.S. (Eds.), Plant Diseases of International Importance, Diseases of Cereals and Pulses, vol. I. Prentice Hall, Englewood Cliffs, NJ, USA, pp. 429–444.

Jiménez-Díaz, R.M., Alcalá-Jiménez, A.R., Hervás, A., Trapero-Casas, J.L., 1993a. Pathogenic variability and hosts resistance in the *Fusarium oxysporum* f. sp.

ciceris/*Cicer arietinum* pathosystem. In: Proc. 3rd Eur. Semin. Fusarium Mycotoxins, Taxonomy, Pathogenicity and Host Resistance. Hodowsla Ròslin Aklimatyazacja i Nasiennictwo. Plant Breeding and Acclimatization Institute, Radzikóv, Poland, pp. 87–94.

Jiménez-Díaz, R.M., Basallote-Ureba, M.J., Rapoport, H., 1989a. Colonization and pathogenesis in chickpea infected by races of *Fusarium oxysporum* f. sp. *ciceris*. In: Tjamos, E.C., Beckman, C. (Eds.), Vascular Wilt Diseases of Plants, vol. H28. Springer-Verlag, Berlin, Germany, pp. 113–121.

Jiménez-Díaz, R.M., Crinó, P., Halila, M.H., Mosconi, C., Trapero-Casas, A., 1993b. Screening for resistance to Fusarium wilt and Ascochyta blight in chickpea. In: Singh, K.B., Saxena, M.C. (Eds.), Breeding for Stress Tolerance in Cool-season Food Legumes. John Wiley & Sons, Chichester, UK, pp. 77–96.

Jiménez-Díaz, R.M., Jiménez-Gasco, M.M., 2011. Integrated management of Fusarium wilt diseases. In: Alves-Santos, F.M., Díez, J.J. (Eds.), Control of Fusarium Diseases. Research Signpost, Kerala, India, pp. 177–215.

Jiménez-Díaz, R.M., Singh, K.B., Trapero-Casas, A., Trapero-Casas, J.L., 1991. Resistance in kabuli chickpea to Fusarium wilt. Plant Dis. 75, 914–918.

Jiménez-Díaz, R.M., Trapero-Casas, A., Cabrera de la Colina, J., 1989b. Races of *Fusarium oxysporum* f. sp. *ciceris* infecting chickpeas in southern Spain. In: Tjamos, E.C., Beckman, C. (Eds.), Vascular Wilt Diseases of Plants, Vol. H28. Springer-Verlag, Berlin, Germany, pp. 515–520.

Jiménez-Díaz, R.M., Trapero-Casas, A., Trapero-Casas, J.L., 1992. The use of host resistance to control Fusarium wilt of kabuli chickpeas. In: Tjamos, E.C., Cook, R.J., Papavizas, G.C. (Eds.), Biological Control of Plant Diseases, Progress and Challenges for the Future, NATO ASI Series a: Life Sciences, vol. 230. Plenum Press, New York, USA, pp. 359–362.

Jiménez-Fernández, D., Landa, B.B., Kang, S., Jiménez-Díaz, R.M., Navas-Cortés, J.A., 2013. Quantitative and microscopic assessment of compatible and incompatible interactions between chickpea cultivars and *Fusarium oxysporum* f. sp. *ciceris* races. PLoS One 8 (4), e61360. http://dx.doi.org/10.1371/journal.pone.0061360.

Jiménez-Fernández, D., Montes-Borrego, M., Jiménez-Díaz, R.M., Navas-Cortés, J.A., Landa, B.B., 2011a. In planta and soil quantification of *Fusarium oxysporum* f. sp. *ciceris* and evaluation of Fusarium wilt resistance in chickpea with a newly developed quantitative polymerase chain reaction assay. Phytopathology 101, 250–262.

Jiménez-Fernández, D., Montes-Borrego, M., Navas-Cortés, J.A., Jiménez- Díaz, R.M., Landa, B.B., 2010. Identification and quantification of *Fusarium oxysporum in planta* and soil by means of an improved specific and quantitative PCR assay. Appl. Soil Ecol. 46, 372–382.

Jiménez-Fernández, D., Navas-Cortés, J.A., Montes-Borrego, M., Jiménez-Díaz, R.M., Landa, B.B., 2011b. Molecular and pathogenic characterization of *Fusarium redolens*, a new causal agent of Fusarium yellows in chickpea. Plant Dis. 95, 860–870.

Jiménez-Gasco, M.M., Jiménez-Díaz, R.M., 2003. Development of a specific polymerase chain reaction-based assay for the identification of *Fusarium oxysporum* f. sp. *ciceris* and its pathogenic races 0, 1A, 5, and 6. Phytopathology 93, 200–209.

Jiménez-Gasco, M.M., Milgroom, M.G., Jiménez-Díaz, R.M., 2002. Gene genealogies support *Fusarium oxysporum* f. sp. *ciceris* as a monophyletic group. Plant Pathol. 51, 72–77.

Jiménez-Gasco, M.M., Milgroom, M.G., Jiménez-Díaz, R.M., 2004a. Stepwise evolution of races in *Fusarium oxysporum* f. sp. *ciceris* inferred from fingerprinting with repetitive DNA sequence. Phytopathology 94, 228–235.

Jiménez-Gasco, M.M., Navas-Cortés, J.A., Jiménez-Díaz, R.M., 2004b. The *Fusarium oxysporum* f. sp. *ciceris*/*Cicer arietinum* pathosystem: a case study of the evolution of plant-pathogenic fungi into races and pathotypes. Intern. Microbiol. 7, 95–104.

Jiménez-Gasco, M.M., Pérez-Artés, E., Jiménez-Díaz, R.M., 2001. Identification of pathogenic races 0, 1B/C, 5, and 6 of *Fusarium oxysporum* f. sp. *ciceris* with random amplified polymorphic DNA (RAPD). Eur. J. Plant Pathol. 107, 237–248.

Jukanti, A.K., Gaur, P.M., Gowda, C.L., Chibbar, R.N., 2012. Nutritional quality and health benefits of chickpea (*Cicer arietinum* L.): a review. Br. J. Nutr. 108, S11–S26.

Kaiser, W.J., Alcalá-Jiménez, A.R., Hervás-Vargas, A., Trapero-Casas, J.L., Jiménez-Díaz, R.M., 1994. Screening of wild *Cicer* species for resistance to races 0 and 5 of *Fusarium oxysporum* f. sp. *ciceris*. Plant Dis. 78, 962–967.

Kaiser, W.J., Danesh, D., 1971. Etiology of virus-induced wilt of Cicer arietinum. Phytopathology 61, 453–457.

Kaiser, W.J., Klein, R.E., Larsen, R.C., Wyatt, S.D., 1993. Chickpea wilt incited by *Pea streak carlavirus*. Plant Dis. 77, 922–926.

Katan, J., 1981. Solar heating solarization of soil for control of soilborne pests. Ann. Rev. Phytopathol. 19, 311–336.

Kelly, A.G., Alcalá-Jiménez, A.R., Bainbridge, B.W., Heale, J.B., Pérez-Artés, E., Jiménez-Díaz, R.M., 1994. Use of genetic fingerprinting and random amplified polymorphic DNA to characterize pathotypes of *Fusarium oxysporum* f. sp. *ciceris* infecting chickpea. Phytopathology 84, 1293–1298.

Kelly, A.G., Bainbridge, B.W., Heale, J.B., Pérez-Artés, E., Jiménez-Díaz, R.M., 1998. *planta*-polymerase-chain-reaction detection of the wilt-inducing pathotype of *Fusarium oxysporum* f. sp. *ciceris* in chickpea (*Cicer arietinum* L. Physiol. Mol. Plant Pathol. 52, 397–409.

Kirkegaard, K., 2009. Biofumigation for plant disease control- from the fundamentals to the farming system. In: Wale, D. (Ed.), Disease Control in Crops. Wiley-Blackwell, Chichester, West Sussex, UK, pp. 172–195.

Kistler, H.C., 2001. Evolution of host specificity in *Fusarium oxysporum*. In: Summerell, B.A., Leslie, J.F., Beckhouse, D., Bryden, W.L., Burgess, L.W. (Eds.), Fusarium. Paul E. Nelson Memorial Symposium. APS Press, St Paul, MN., USA, pp. 70–82.

Krishna Rao, V., Krishnappa, K., 1996. Interaction of *Fusarium oxysporum* f. sp. *ciceris* with *Meloidogyne incognita* on chickpea in two soil types. Ind. Phytopathol. 49, 142–147.

Kumar, S., 1998. Inheritance of resistance to Fusarium wilt (race 2) in chickpea. Plant Breed. 117, 139–142.

Kumar, J., Haware, M.P., Smithson, J.B., 1985. Registration of four short duration Fusarium wilt-resistant Kabuli (Garbanzo) chickpea germplasm. Crop Sci. 25, 576–577.

Ladizinsky, G., Adler, A., 1976. The origin of chickpea *Cicer arietinum* L. Euphytica 25, 211–217.

Landa, B.B., Hervás, A., Bettiol, W., Jiménez-Díaz, R.M., 1997. Antagonistic activity of bacteria from chickpea rhizosphere against *Fusarium oxysporum* f. sp. *ciceris*. Phytoparasitica 25, 305–318.

Landa, B.B., Navas-Cortés, J.A., Hervás, A., Jiménez-Díaz, R.M., 2001. Influence of temperature and inoculum density of *Fusarium oxysporum* f. sp. *ciceris* on suppression of Fusarium wilt of chickpea by rhizosphere bacteria. Phytopathology 91, 807–816.

Landa, B.B., Navas-Cortés, J.A., Jiménez-Díaz, R.M., 2004a. Influence of temperature on plant-rhizobacteria interactions related to biocontrol potential for suppression of Fusarium wilt of chickpea. Plant Pathol. 53, 341–352.

Landa, B.B., Navas-Cortés, J.A., Jiménez-Díaz, R.M., 2004b. Integrated management of Fusarium wilt of chickpea combining the use of choice of sowing date, host resistance, and biological control. Phytopathology 94, 946–960.

Landa, B.B., Navas-Cortés, J.A., Jiménez-Gasco, M.M., Katan, Retig, B., Jiménez-Díaz, R.M., 2006. Temperature response of chickpea cultivars to races of *Fusarium oxysporum* f. sp. *ciceris*, the causal agent of Fusarium wilt. Plant Dis. 90, 365–374.

Lazarovits, G., 2004. Managing soilborne plant diseases through selective soil disinfestation by a knowledge-based application of soil amendments. Phytoparasitica 32, 427–432.

Leslie, J.F., Summerell, B.A., 2006. The Fusarium Laboratory Manual. Blackwell Publishing, Ames.

Lev-Yadun, S., Gopher, A., Abbo, S., 2000. The cradle of agriculture. Science 288, 1602–1603.

Malcolm, G.M., Kuldau, G.A., Gugino, B.K., Jiménez-Gasco, M.M., 2013. Hidden host plant associations of soilborne fungal pathogens: an ecological perspective. Phytopathology 103, 538–544.

Mani, A., Sethi, C.L., 1987. Interaction of root-knot nematode, *Meloidogyne incognita* with *Fusarium oxysporum* f. sp. *ciceris* and *F. solani* on chickpea. Ind. J. Nematol. 17, 1–6.

Morales, G.J.A., 1986. Chickpea breeding program in Sonora. Intern. Chickpea Newslett. 15, 11–12.

Navas-Cortés, J.A., Alcalá-Jiménez, A.R., Hau, B., Jiménez-Díaz, R.M., 2000a. Influence of inoculum density of races 0 and 5 of *Fusarium oxysporum* f. sp. *ciceris* on development of Fusarium wilt in chickpea cultivars. Eur. J. Plant Pathol. 106, 135–146.

Navas-Cortés, J.A., Hau, B., Jiménez-Díaz, R.M., 1998a. Effect of sowing date, host cultivar, and race of *Fusarium oxysporum* f. sp. *ciceris* on development of Fusarium wilt of chickpea. Phytopathology 88, 1338–1346.

Navas-Cortés, J.A., Hau, B., Jiménez-Díaz, R.M., 2000b. Yield loss in chickpeas in relation to development of Fusarium wilt epidemics. Phytopathology 90, 1269–1278.

Navas Cortés, J.A., Landa, B.B., Méndez-Rodríguez, M.A., Jiménez-Díaz, R.M., 2007. Quantitative modeling of the effects of temperature and inoculum density of *Fusarium oxysporum* f. sp. *ciceris* races 0 and 5 on the development of Fusarium wilt in chickpea cultivars. Phytopathology 97, 564–573.

Navas-Cortes, J.A., Landa, B.B., Rodríguez-López, J., Jiménez-Díaz, R.M., Castillo, P., 2008. Infection by *Meloidogyne artiellia* does not break down resistance to races 0, 1A, and 2 of *Fusarium oxysporum* f. sp. *ciceris* in chickpea genotypes. Phytopathology 98, 709–718.

Navas-Cortés, J.A., Rodríguez-Jurado, D., Jiménez-Díaz, R.M., 1998b. Combined resistance against *Didymella rabiei* and races of *Fusarium oxysporum* f. sp. *ciceris* in kabuli chickpeas, in: AEP (Associated Europeenne des Proteagineux). In: Proc. European Conference on Grain Legumes, 3rd. AEP, Valladolid, Spain, pp. 124–125.

Nene, Y.L., Haware, M.P., Reddy, M.V., 1978. Diagnosis of some wilt disorders of chickpea (*Cicer arietinum* L.). In: Information Bull., vol. 3. International Crops Research Institute for the Semi-Arid Tropics (ICRISAT), Patancheru, A.P., India.

Nene, Y.L., Reddy, M.V., 1987. Chickpea diseases and their control. In: Saxena, M.C., Singh, K.B. (Eds.), The Chickpea. CAB Int., Oxon, UK, pp. 233–270.

Nogales-Moncada, A.N., Jiménez-Díaz, R.M., Pérez-Artés, E., 2009. Vegetative compatibility groups in *Fusarium oxysporum* f. sp. *ciceris* and *F. oxysporum* nonpathogenic to chickpea. J. Phytopathol. 157, 729–735.

O'Donnell, K., Kistler, H.C., Cigelnik, E., Ploetz, R.C., 1998. Multiple evolutionary origins of the fungus causing Panama disease of banana: Concordant evidence from nuclear and mitochondrial gene genealogies. Proc. Natl. Acad. Ss. U. S. A. 95, 2044–2049.

Palomares-Rius, J.E., Castillo, P., Navas-Cortés, J.A., Jiménez-Díaz, R.M., Tena, M., 2011. A proteomic study of in-root interactions between chickpea pathogens: the root-knot nematode *Meloidogyne artiellia* and the soil-borne fungus *Fusarium oxysporum* f. sp. *ciceris* race 5. J. Proteomics 74, 2034–2051.

Pande, S., Rao, J.N., Sharma, M., 2007. Establishment of the chickpea wilt pathogen *Fusarium oxysporum* f. sp. *ciceris* in the soil through seed transmission. Plant

Please cite this article in press as: Jiménez-Díaz, R.M., et al., Fusarium wilt of chickpeas: Biology, ecology and management, Crop Protection (2015), http://dx.doi.org/10.1016/j.cropro.2015.02.023

Pathol. J. 23, 3–6.
Powelson, M.L., Rowe, R.C., 2008. Managing diseases caused by seedborne and soilborne fungi and fungus-like pathogens. In: Johnson, D.A. (Ed.), Potato Health Management, second ed. APS Press, St. Paul, MN, pp. 183–195.
Pérez-Artés, E., Roncero, M.I.G., Jiménez-Díaz, R.M., 1995. Restriction fragment length polymorphism analysis of the mitochondrial DNA of *Fusarium oxysporum* f. sp. *ciceris*. J. Phytopathol. 143, 105–109.
Pundir, R.P.S., Reddy, K.N., Mengesha, M.H., 1988. ICRISAT chickpea germplasm catalogue: evaluation and analysis. International Crops Research Institute for the Semi-Arid Tropics, Patancheru, A.P., India.
Rubio, J., Martínez, C., Gil, J., Moreno, M.T., 2004. Registration of Ascochyta blight and Fusarium wilt resistant CA2954 kabuli chickpea germplasm. Crop Sci. 44, 1881–1882.
Saraf, C.S., 1974. Agronomic management to reduce gram wilt incidence. Indian J. Genet. Plant Breed. 34, 263–266.
Sharma, K.D., Muehlbauer, F.J., 2007. Fusarium wilt of chickpea: physiological specialization, genetics of resistance and resistance gene tagging. Euphytica 157, 1–14.
Sharma, K.D., Chen, W., Muehlbauer, F.J., 2005. Genetics of chickpea resistance to five races of Fusarium wilt and a concise set of race differentials for *Fusarium oxysporum* f. sp. *ciceris*. Plant Dis. 89, 385–390.
Sharma, M., Nagavardhini, A., Thudi, M., Ghosh, R., Pande, S., Varshney, R.K., 2014. Development of DArT markers and assessment of diversity in *Fusarium oxysporum* f. sp. *ciceris*, wilt pathogen of chickpea (*Cicer arietinum* L.). BMC Genomics 15, 454.
Sharma, M., Varshney, R.K., Rao, J.N., Kannan, S., Hoisington, D., Pande, S., 2009. Genetic diversity in Indian isolates of *Fusarium oxysporum* f. sp. *ciceris*, chickpea wilt pathogen. Afr. J. Biotech. 8, 1016–1023.
Shehabu, M., Ahmed, S., Sakhuja, K., 2008. Pathogenic variability in Ethiopian isolates of *Fusarium oxysporum* f. sp. *ciceris* and reaction of chickpea improved varieties to the isolates. Int. J. Pest Manag. 54, 143–149.
Singh, H., Kumar, J., Smithson, J.B., Haware, M.P., 1987a. Complementation between genes for resistance to race 1 of *Fusarium oxysporum* f. sp. *ciceris* in chickpea. Plant Pathol. 36, 539–543.
Singh, H., Kumar, J., Haware, M.P., Smithson, J.B., 1987b. Genetics of resistance to Fusarium wilt in chickpeas. In: Day, P.R., Jellis, G.J. (Eds.), Genetics and Plant Pathogenesis. Blackwell Scientific Publications, Oxford, UK, pp. 339–342.
Singh, K.B., 1987. Chickpea breeding. In: Saxena, M.C., Singh, K.K. (Eds.), The Chickpea. CAB International, Wallingford, pp. 127–162.
Singh, K.B., 1997. Chickpea (*Cicer arietinum* L.). Field Crops Res. 53, 161–170.
Singh, K.B., Dahiya, B.S., 1973. Breeding for wilt resistance in chickpea. In: Symposium on Wilt Problem and Breeding for Wilt Resistance in Bengal Gram. Indian Research Institute, New Delhi, India, pp. 13–14.
Singh, K.B., Saxena, M.C. (Eds.), 1993. Breeding for Stress Tolerance in Cool Seasons Food Legumes. John Wiley and Sons, Chichester, UK.
Singh, K.B., Jiménez Díaz, R.M., 1996. Registration of six Fusarium wilt -resistant chickpea germplasm lines. Crop Sci. 36, 817.
Singh, K.B., Saxena, M.C., 1996. Winter Chickpea in Mediterranean-type Environments. International Center for Agricultural Research in Dry Areas, Aleppo, Syria.
Stevenson, P.C., Turner, H.C., Haware, M.P., 1995. Phytoalexin accumulation in the roots of chickpea (*Cicer arietinum* L.) seedlings associated with resistance to Fusarium wilt (*Fusarium oxysporum* f. sp. *ciceris*). Physiol. Mol. Plant Pathol. 50, 167–178.
Stevenson, P.C., Turner, H.C., Haware, M.P., 1997. Phytoalexin accumulation in the roots of chickpea (*Cicer arietinum* L.) seedlings associated with resistance to Fusarium wilt (*Fusarium oxysporum* f. sp. *ciceris*). Physiol. Mol. Plant Pathol. 50, 167–178.
Trapero-Casas, A., Jiménez-Díaz, R.M., 1985. Fungal wilt and root rot diseases of chickpea in southern Spain. Phytopathology 75, 1146–1151.
Tullu, A., Kaiser, W.J., Kraft, J.M., Muehlbauer, F.J., 1999. A second gene for resistance to race 4 of Fusarium wilt in chickpea and linkage with a RAPD marker. Euphytica 109, 43–50.
Uma Maheswari, T., Sharma, S.B., Reddy, D.D.R., Haware, M.P., 1995. Co-infection of wilt-resistant chickpeas by *Fusarium oxysporum* f. sp. *ciceris* and *Meloidogyne javanica*. J. Nematol. 27, 649–653.
Uma Maheswari, T., Sharma, S.B., Reddy, D.D.R., Haware, M.P., 1997. Interaction of *Fusarium oxysporum* f. sp. *ciceris* and *Meloidogyne javanica* on *Cicer arietinum*. J. Nematol. 29, 117–126.
Upadhyaya, H.D., Haware, M.P., Kumar, J., Sminthson, J.B., 1983a. Resistance to wilt in chickpea. I. Inheritance of late-wilting in response to race 1. Euphytica 32, 447–452.
Upadhyaya, H.D., Sminthson, J.B., Haware, M.P., Kumar, J., 1983b. Resistance to wilt in chickpea. II. Further evidence for two genes for resistance to race 1. Euphytica 32, 749–755.
van Rheenen, H.A., Reddy, M.V., Kumar, J., Haware, M.P., 1992. Breeding for resistance to soil-borne diseases of chickpea. In: Singh, K.B., Saxena, M.C. (Eds.), Disease Resistance Breeding in Chickpea, International Center for Research in Dry Areas (ICARDA), Aleppo, Syria, pp. 89–112.
Westerlund Jr., F.V., Campbell, R.N., Kimble, K.A., 1974. Fungal root rots and wilt of chickpea in California. Phytopathology 64, 432–436.
Zohary, D., Hopf, M., 2000. Domestication of Plants in the Old World: the Origin and Spread of Cultivated Plants in West Asia, Europe, and the Nile Valley, third ed. Oxford University Press, Oxford, UK.

Please cite this article in press as: Jiménez-Díaz, R.M., et al., Fusarium wilt of chickpeas: Biology, ecology and management, Crop Protection (2015), http://dx.doi.org/10.1016/j.cropro.2015.02.023