Pathogenicity and management of Fusarium wilt of chickpea, Cicer arietinum L. – A review

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ABSTRACT

Wilt is one of the major constraints in production and productivity of chickpea. The causative organism, Fusarium oxysporum f. sp. ciceris is widespread in chickpea growing areas resulting in considerable economic losses. The importance, symptomatology, pathogenicity and management is hereunder reviewed briefly.

KEY WORDS: Chickpea, Fusarium, management, pathogenicity

Chickpea (Cicer arietinum L.) is the world’s third most important pulse crop, after dry beans (Phaseolus vulgaris L.) and dry peas (Pisum sativum L.) – (Vishwadhar and Gurha, 1998). Chickpea (Cicer arietinum L.) is a vital source of plant-derived edible protein in many countries. Chickpea also has advantages in the management of soil fertility, particularly in dry lands and the semiarid tropics. Indian subcontinent accounts for 90% of the total world chickpea production (Juan et al., 2000). Chickpea is contributing nearly 42 to 47 per cent of total pulse production in India. Nearly 90 per cent of the area and production is from six states viz., Madhya Pradesh, Rajasthan, Maharashtra, Uttar Pradesh, Karnataka and Andhra Pradesh. In India, it is grown over an area of 8.56 million ha with an annual production and productivity of 7.35 million tonnes and 858 kg ha⁻¹ respectively (Chickpea research highlights 2009-2010). In Andhra Pradesh, it is grown over an area of 5.34 lakh ha with an annual production and productivity of 6.93 lakh tonnes and 1298 kg ha⁻¹ respectively (Quarterly statistical news letter 2011-2012).

Fusarium oxysporum f. sp. ciceris is a wilt fungus causing severe damage wherever this crop is grown (Rangaswami et al., 1999). It is more prevalent in lower latitudes (0-30°N) where growing season is relatively dryer and warmer than in the higher latitudes (30-40°N). Fusarium wilt is one of the major diseases of chickpea and at national level the yield losses encountered was reported to the tune of 60 per cent (Singh et al., 2007). It causes complete loss in grain yield if the disease occurs in the vegetative and reproductive stages of the crop (Haware and Nene, 1980; Haware et al., 1990; Halila and Strange, 1996; Navas et al., 2000). F. oxysporum f. sp. ciceris infects chickpea at seedling as well as at flowering and pod forming stage (Grewal, 1969), with more incidence at flowering and podding stage if the crop is subjected to sudden temperature rise and water stress (Chaudhry et al., 2007).

The pathogen is mainly soil borne as well as seed borne and also it is a facultative saprophyte. It can survive in the soil upto six years in the absence of susceptible host (Haware et al., 1978). Considering the nature of damage and survival ability of the pathogen, use of resistant varieties is the only economical and practical solution. Most of the resistant varieties have been found to be susceptible after some years because of breakdown of
their resistance due to evolution of variability in the pathogen. The pathogen with high saprophytic ability can survive in soil for a pretty long period during which it may have to go through different environmental stresses and biological competition which may lead to the development of physiological races. Therefore, integrated management strategies are the possible solutions to maintain plant health mainly for soil borne plant pathogens.

These strategies include modification of cultural practices, growing of resistant varieties with minimum application of chemicals (Bendre and Barhate, 1998), and encouragement of beneficial microbial population to reduce pathogen inoculums.

MORPHOLOGY OF *Fusarium oxysporum* f. sp. ciceris

The pathogen is a common soil inhabitant with taxonomic nomenclature *Fusarium oxysporum* f. sp. *ciceris* (Padwick) Matuo and Sato (Snyder and Hansen, 1940). Saxena and Singh (1987) reported that *F. oxysporum* is septate, profusely branched growing on potato sucrose/dextrose agar at 25°C initially white turning light buff or deep brown later, fluffy or submerged. The growth becomes felted or wrinkled in old cultures. Various types of pigmentation (yellow, brown, crimson) can be observed in culture. Couteaudier and Alabouvette (1990) reported that the macroconidia are straight to slightly curved, slender, thin walled usually with three or four septa, a foot-shaped basal cell and curved apical cell. They are generally produced from phialides on conidiophores by basipetal division. The microconidia are ellipsoidal and either have no septum or a single one. Both are formed from phialides in false heads by basipetal division. They are important in secondary infection. The chlamydospores are globose and have thick walls. They are formed from hyphae or alternatively by the modification of hyphal cells. They are important as endurance organs in soils where they act as inocula in primary infection. The teleomorph or sexual reproductive stage, of *Fusarium oxysporum* is unknown (Leslie and Summerell, 2006).

DISTRIBUTION AND ECONOMIC IMPORTANCE OF THE DISEASE

DISTRIBUTION

Occurrence of chickpea wilt has been reported almost all over the world but it was first described in India (Butler, 1918). *Fusarium* wilt is a serious disease of chickpea in India, Iran, Pakistan, Nepal, Burma, Spain, Tunisia and Mexico. It has been observed in Morocco, Algeria, and Syria. An estimated annual loss of 12 million rupees was reported from Pakistan (Sattar et al., 1953). *Fusarium oxysporum* is an ubiquitous, asexual species complex. Isolates of *Fusarium oxysporum* can cause vascular wilt or cortical rot diseases in many agricultural crops and have been classified into *Formae specialis* based on their host specificity (Nelson et al., 1981). Nene and Reddy (1987) recorded the distribution of *Fusarium oxysporum* f. sp. *ciceris* covering North America, Europe, Middle East, Asia and South East Asia. Different pathotypes of the pathogen have distinct geographical distributions. Races 2, 3 and 4 have only been described from India (Haware and Nene, 1982), whereas races 0, 1B/C, 5 and 6 are found mainly in the Mediterranean region as well as in the USA (California) (Jiménez-Díaz et al., 1993; Halila and Strange, 1996). Race 1A has been reported in India (Haware and Nene, 1982), California and the Mediterranean region (Jiménez-Díaz et al., 1993). The chickpea wilt caused by *Fusarium oxysporum* f. sp. *ciceris* was reported to be widely distributed in near about 32 countries of the world (Nene et al., 1996).
ECONOMIC IMPORTANCE

Haware et al. (1978) reported that the fungus *Fusarium oxysporum* f. sp. *ciceris* is a primarily soil borne pathogen, however, few reports indicated that it can be transmitted through seeds. Attempts were made to estimate loss in yield on a per plant basis. Early wilting caused more loss than late wilting. Seeds harvested from wilted plants were lighter than those from healthy plants (Haware and Nene, 1980). Singh and Reddy (1991); Jimenez-Diaz et al. (1989) reported that the average annual yield losses due to wilt have been estimated to be 10 to 90 per cent in wilt affected soils. Annual chickpea yield losses from *Fusarium* wilt vary from 10 to 15 per cent (Jalali and Chand, 1992 and Trapero-casas et al., 1985) but can result in total loss of the crop under specific conditions (Halila and Strange, 1996; Haware and Nene, 1980). Zote et al. (1996); Sugha et al. (1994); Bhatti and Kraft (1992) observed that in recent years, incidence of wilt in the farmer’s fields is increasing considerably every year and its severity is directly related to the increasing density of the pathogen inoculum in the soil. Hanif et al. (1999) reported that in Pakistan the area under chickpea cultivation on irrigated land has reduced from 50 to 10 per cent. It causes complete loss in grain yield if the disease occurs in the vegetative and reproductive stages of the crop (Navas et al., 2000; Halila and Strange, 1996; Haware et al., 1990; Haware and Nene, 1980). *Fusarium* wilt is prevalent in almost all chickpea-growing areas of the world, and its incidence varied from 14 to 32 per cent in different states of India as reported by Dubey et al. (2010).

SYMPTOMATOLOGY

The disease occurs at seedling and flowering stage of plant growth. The symptoms which can be observed are drooping of petioles and rachis, yellowing and drying of leaves from base to upward, browning of vascular bundles, improper branching, withering of plants and finally death of plants (Westerlund et al., 1974; Prasad and Padwick, et al., 1939). Erwin (1958) reported that the foliage of the *Fusarium oxysporum* f. sp. *ciceris* infected plant turns yellow before wilting and the xylem tissue shows light brown discoloration. Saxena and Singh (1987) reported that internal discoloration of pith and xylem can be seen if stem and root of the wilted plants split vertically.

Nene et al. (1991) observed that *Fusarium* wilt infected seedlings collapse and lie flat on the ground retaining their dull green colour. Adult plants show typical wilt symptoms of drooping of petioles, rachis and leaflets. The roots of the wilting plants do not show any external rotting but when split open vertically, dark brown discoloration of internal xylem is seen. Haware (1993) reported that the pathogen gets entry into xylem vessel and invades whole vascular system, inducing symptoms of yellowing and wilting. In the absence of host plant the pathogen can survive upto six years. Brayford (1998), Leslie and Summerell (2006) reported that the fungus enters the vascular system of the infected plant via the roots. It produces enzymes that degrade the cell walls so that gels are formed that block the plant’s transport system. Discolouration of the internal tissues progresses from the roots to the aerial parts of the plant, yellowing and wilting of the foliage occur, and finally there is necrosis.

Singh et al. (2007) reported that following infection of host roots, the fungus crosses the cortex, enters the xylem tissues and then spreads rapidly upward direction through the vascular system, becoming systemic in the host tissues, and may directly infect the seed. Singh et al. (2007) observed that the pathogen is primarily confined to the xylem vessels in which the mycelium branches and produces microconidia. The microconidia detach and are carried upward in the
vascular system until movement is stopped, at which point they germinate and the mycelium penetrates the wall of the adjacent vessel.

**ISOLATION OF THE PATHOGEN**

Fisher *et al.* (1982) reported that highly virulent strain of *Fusarium oxysporum* f. sp. *ciceris* was isolated from infected chickpea plant by using Komada’s medium (Komada, 1975) and confirmation of *Fusarium* was made on carnation leaf agar medium. Patel *et al.* (2001) studied the colony characters, pigmentation, sporulation and conidial characters of *Fusarium oxysporum* f. sp. *ciceris* isolates collected from Annigeri, Bijapur, Bidar and Dharwad (Karnataka), in India and observed similarity among three isolates of *Fusarium oxysporum* f. sp. *ciceris*. Paulkar (2004) studied the 4 isolates of *Fusarium oxysporum* f. sp. *ciceris* from Amaravati (Foc-1), Akola (Foc-2), Buldhana (Foc-3) and Nagpur (Foc-4) and found they were morphologically different.

Honnareddy and Dubey (2007) reported that the isolates of *Fusarium oxysporum* f. sp. *ciceris* had variable pigmentation which varied from normal white to violet, brown, reddish violet, greenish violet, yellowish pink and dark green. Singh *et al.* (2010) collected various isolates of *Fusarium oxysporum* f. sp. *ciceris* from Anand and Arnej (Gujarat), and studied the variability and the virulence among various isolates of *Fusarium oxysporum* f. sp. *ciceris* causing vascular wilt in chickpea under in vitro and found no significant difference in morphology and virulence among the three isolates. Nikam *et al.* (2011) collected wilt infected chickpea plant samples from different locations and isolated *Fusarium oxysporum* f. sp. *ciceris* on potato dextrose agar (PDA) in the laboratory.

**MAINTENANCE OF THE PATHOGEN**

Barnet and Hunter (1972) purified *Fusarium oxysporum* f. sp. *ciceris* by single spore isolation method and maintained on PDA slants throughout the investigation by periodical transfer. Sumitra (2006) reported that *Fusarium oxysporum* f. sp. *ciceris* was sub cultured on PDA slants and allowed to grow at 27 ± 1°C for ten days and such slants were preserved in a refrigerator at 5°C and revived once in 30 days. Pure culture of *Fusarium oxysporum* f. sp. *ciceris* was prepared on Czapekdox agar medium and it was multiplied on Waksman’s agar medium (Glucose 10 g, Peptone 5 g, Potassium dihydrogen phosphate 1 g, Magnesium sulphate 0.5 g, Distilled water 1000 ml) (Muhammad Ansar Ahmad, 2010). *Fusarium* species were maintained on PDA slants and were stored at 4°C till use (Hend *et al.*, 2012).

**PATHOGENICITY TESTS**


**POTENTIAL NATIVE ANTAGONIST**

**ISOLATION OF ANTAGONISTIC MYCOFLORA AND BACTERIA FROM RHIZOSPHERE SOIL AND ROOT ENDOPHYES**

King *et al.* (1954) isolated *Pseudomonas* spp. from rhizosphere soil
of chickpea plant using King’s B media. Singh and Mehrotra (1980) isolated Bacillus and actinomycetes from chickpea rhizosphere and were found to be antagonistic to Rhizoctonia bataticola under in vitro conditions. Elad and Chet (1983) collected the rhizosphere soils of vanilla from different locations of Thrissur and Ernakulam districts of Kerala (India) and isolated Trichoderma species by using selective media. Dhingra and Sinclair (1995) isolated dominant rhizosphere fungi of tomato from plants severely affected by wilt disease caused by *F. oxysporum* f. sp. *lycopersici* using potato dextrose agar medium. Nautiyal (1997) isolated 478 chickpea rhizosphere competent bacteria for suppression of chickpea pathogenic fungi *Fusarium oxysporum* f. sp. *ciceris* and found 386 strains that effectively colonize chickpea roots. Parmer and Dadarwal (1997) isolated *Pseudomonas* and *Bacillus* sp. from rhizosphere and rhizoplane of healthy chickpea plants which inhibited the mycelial growth of *Fusarium oxysporum* f. sp. *ciceris*. Rangeshwaran et al. (2002) isolated twenty-five endophytic bacteria from internal tissues of root and stem portions of chickpea, sunflower, niger, chilli, and capsicum plants. Jayalakshmi et al. (2003) isolated *Trichoderma harzianum* from rhizosphere of healthy pigeonpea plants.

Cao Li Xiang et al. (2004) isolated two hundred and forty-two actinomycetes strains from the leaves and roots of healthy and wilting banana plants. Most of them were streptomycetes, *Streptomyces griseorubiginosus*-like strains and were the most frequently isolated strains. Sendhilvel et al. (2005) isolated five different isolates of *Pseudomonas fluorescens* from cowpea rhizosphere region and screened against the *Macrophomina phaseolina* the causal organism of cowpea dry root rot. Ramesh and Korikanthimath (2006) isolated biocontrol agents like *Trichoderma viride*, *T. harzianum*, *Pseudomonas fluorescens* and *Bacillus subtilis* from rhizosphere of various crops and tested their efficacy against the *Macrophomina phaseolina* causing root rot of groundnut.

Zeidan (2006) isolated root endophytes from peanut healthy roots and found that *Bacillus subtilis* abundantly colonized peanut root than *P. fluorescens* and effectively controls the root and pod rot diseases. Tiwari and Thrimurthy (2007) isolated 21 isolates of *Pseudomonas fluorescens* from rhizosphere of rice, maize, wheat, chickpea, mung, urd and soyabean from Raipur and Bastar regions and revealed that seven isolates were found to be effective against *Rhizoctonia solani*, the incitant of rice sheath blight.

**IN VITRO EVALUATION OF THE EFFICACY OF ANTAGONISTS AGAINST *Fusarium oxysporum* f.sp. *ciceris***

Rangeshwaran and Prasad (2000) collected 300 rhizospheric isolates of bacteria from different regions of Karnataka were screened for in vitro antagonism in dual culture on tryptic soya agar (TSA) against *Fusarium oxysporum f. sp. ciceris* and four isolates were selected as potential antagonists viz., *Pseudomonas putida* (PDBCAB 19), *P. fluorescens* (PDBCAB 2), *P. fluorescens* (PDBCAB 29) and *P. fluorescens* (PDBCAB 30) and found *P. fluorescens* (PDBCAB 29) was most effective. Rangeshwaran et al. (2002) screened twenty five endophytes in dual culture on potato dextrose agar (PDA) and tryptic soya agar (TSA) against *Fusarium oxysporum f. sp. ciceris*, *Fusarium udum*, *Rhizoctonia solani*, and *Sclerotium rolfsii* and found ten isolates of endophytes were effective and inhibited the mycelial growth of pathogens.

Cao Li Xiang et al. (2004) screened two hundred and forty two actinomycete strains against *Fusarium oxysporum f. sp. cubense* and revealed that the proportion of antagonistic
actinomycetes in healthy roots was higher than that in wilting roots (P<0.01), but no difference was found between antagonistic strains isolated from healthy and wilting leaves. The antagonistic activity against in vitro growth of Fusarium oxysporum f. sp. ciceris was determined for 90 isolates of fluorescent pseudomonads obtained from the rhizosphere of healthy, partially wilted and completely wilted chickpea (Kaur et al., 2007).

Mane and Mahendra Pal (2008) screened the effects of different antagonists, i.e. Acrophialophora sp., fluorescent pseudomonas isolates 1,3,4 and Gliocladium virens, G. catanallium, G. deliguesences, Trichoderma hamatam, T. harzianum and T. koningii and found that T. hamatam inhibited an average of 26.03 per cent of radial growth, followed by fluorescent pseudomonas isolate-4 (25.08%) and fluorescent pseudomonas-3 (25.5%), respectively.

Boureghda and Bouznad (2009) studied the efficiency of the antagonist species Trichoderma atroviride (strains Ta.3, Ta.7 and Ta.13), T. harzianum (Th.6, Th.12, Th.15, Th.16 and Th.18) and T. longibrachiatum (TL.1, TL.2, TL.4, TL.5, TL.8, TL.9, TL.10, TL.11, TL.14 and TL17) against Fusarium oxysporum f. sp. ciceris using in vitro- and in vivo-based bioassay and found significant decrease of both growth and conidia production of the pathogen.

COMPATIBILITY STUDIES BETWEEN THE FUNGAL AND BACTERIAL ANTAGONISTS

Rini and Sulochana (2007) tested Trichoderma isolates and Pseudomonas fluorescens isolates against Fusarium oxysporum diseases in tomato and revealed that the combined application of both Trichoderma and Pseudomonas isolates has given highest disease suppression

IN VITRO EVALUATION OF FUNGICIDES AGAINST PATHOGEN

Ayyub (2001) evaluated eleven fungicides and found Benlate, Follicur and Derosal, as the most effective against mycelial growth of Fusarium wilt. Moderate response was observed in case of Topas-l00 and Tilt, whereas, Daconil, Antracol, Apron and Polyram combi were found least effective. Singh et al. (2003) evaluated seven fungicides, i.e. Thiram, Bavistin (carbendazim), Blitox (copper oxychloride), Captaf (captan), Indofil M-45 (mancozeb+thiophanate-methyl) Ridomil MZ (mancozeb+metalaxyl) and Kitazin (iprobenfos), against chickpea wilt under in vitro (each at 0.1% concentration) and in vivo (as seed treatment each at 2.5 g kg⁻¹ seed, except for Kitazin (1.0 ml kg⁻¹) and as soil drenching each at 0.3%) and found thiram and bavistin as the most suitable fungicides inhibiting the mycelial growth of Fusarium oxysporum f. sp. ciceris under in vitro.

Harender Raj et al. (2005) evaluated the efficacy of Quintal (carbendazim 25% + iprodione 25%), Bavistin (carbendazim), SAAF (carbendazim 12% + mancozeb 63%), Thiram 75 DS (thiram) and Hilnate 70 WP (thiophanate-methyl) against Fusarium oxysporum f. sp. gladioli under in vitro conditions and observed that Quintal recorded the lowest disease incidence followed by carbendazim and SAAF.

Chhata and Jeewa Ram (2006) found that out of 4 fungicides tested, Bavistin (carbendazim; 0.2%) and Topsin-M (thiophanate-methyl; 0.2%) were more effective than other seed dresser fungicides and showed least seedling mortality (2-4 and 4-6%, respectively).

Sunita and Manica (2007) evaluated the efficacy of fungicides, i.e. carbendazim, thiophanate-methyl, thiram , 25% carbendazim + 25% iprodione (Quintal) and 12% carbendazim + 63% mancozeb
(SAAF), as well as biopesticides, i.e. Neemazal (1.0% EC) and Nimbicidine (0.03% EC) against *Fusarium* wilt (*Fusarium oxysporum* f. sp. *gladioli*) of *Gladiolus grandiflorus* and found Quintal as effective fungicide followed by carbendazim and SAAF. Shovan et al. (2008) evaluated five fungicides namely Tilt-250 EC, Vitavax-200 75 per cent WP, Rovral 50 WP, Dithane M-45 80 per cent WP and Cupravit 50 per cent WP at 100, 200 and 400 ppm for their efficacy against the radial colony growth and mycelial dry weight of *Fusarium oxysporum* and observed the complete inhibition of radial growth and hyphal dry weight was obtained with Tilt-250 EC at all the selected concentrations.

Raju et al. (2008) evaluated carbendazim, captan, Dithane Z-78, thiophanate-methyl and thiram against *F. oxysporum* f. sp. *udum* under *in vitro* and found that carbendazim completely inhibited the growth of the pathogen at all concentrations (100, 250 and 500 ppm). Singh (2009) found that carbendazim (0.1%) and mancozeb (0.25%) was effective against *Fusarium oxysporum* f. sp. *coriandrii* causing coriander wilt. Srivastava et al. (2011) studied the efficacy of carbendazim 50 per cent WP against *Fusarium oxysporum* f. sp. *psidii* associated with rhizosphere soil of guava and found that the radial growth of *Fusarium oxysporum* f. sp. *psidii* was fully inhibited at high concentrations like 100, 1000 and 10,000 ppm of carbendazim 50 per cent WP.

**IN VITRO COMPATIBILITY OF POTENTIAL ANTAGONISTS WITH FUNGICIDES**

Vyas (1987) reported that *Trichoderma* sp. and *B. subtilis* showed high degree of tolerance to thiram and carbendazim. Vidhyasekharan et al. (1995) reported that thiram and carbendazim were not inhibitory to *P. fluorescens* under *in vitro* conditions. Rajeevpanth and Mukhopadhyay (2001) observed that Vitavax was compatible with *G. virens* and *T. harzianum* for management of seed and seedling rot of soybean caused by *M. phaseolina*. Girija and Umamaheswaran (2003) studied the compatibility of *T. virens* with carbendazim under *in vitro* at three concentrations (100,150 and 1000 ppm) and observed that the antagonist *T. virens* was compatible with carbendazim at 100 ppm concentration.

Poddar et al. (2004) found that *T. harzianum* in combination with carbendazim (100 ppm) is effective against wilt (*F. oxysporum* f. sp. *ciceris*) in chickpea (*Cicer arietinum* cv. JG 62). Tiwari and Singh (2004) evaluated the *in vitro* efficacy of different fungicides against *T. harzianum* @ 1500 ppm and found that the mycelial growth of *T. harzianum* was completely inhibited by carbendizam and hexaconazole @ 1500 ppm and the inhibition with copper oxychloride and mancozeb was 90 and 41 per cent. Gupta et al. (2005) reported that carbendazim was incompatible with *Trichoderma viride* TV2 isolate while with carboxin was compatible for integrated treatment.

Naseema Beevi et al. (2005) tested the *in vitro* compatibility of *T. harzianum* with mancozeb, carbendazim and copper oxychloride and found that carbendazim at 0.1 per cent completely inhibited the mycelial growth while mancozeb and copper oxychloride showed compatibility with the antagonist at 0.2 and 0.1 per cent respectively. Dubey et al. (2007) studied the efficacy of *Trichoderma* species in combination with carboxin and found that the integration of *T. harzianum* (106 spores/ml/10 g seed) and carboxin (2 g kg⁻¹ seed) for seed treatment was the best which enhanced seed germination by 12.0 to 14.0 per cent and grain yields by 42.6 to 72.9 per cent and reduced wilt incidence (44.1 - 60.3%). Khan and Gangopadhyay (2008) tested the compatibility of
Pseudomonas fluorescens with the fungicides and revealed that carboxin, and carbendazim were least toxic to P. fluorescens strain PFBC-25 whereas captan was most inhibitory.

Shahida et al. (2010) reported that the common fungicides Bordeaux mixture at 0.5,1.0,1.5 per cent concentration, copper oxychloride (Fytolan 50 WDP) at 0.1, 0.2, 0.3 per cent concentration, potassium phosphonate (Akomin 40) at 0.2, 0.3, 0.4 per cent concentration and mancozeb (Indofil M-45) at 0.1, 0.2, 0.3 per cent concentrations were tested for their compatibility with the effective isolates of T. harzianum and T. viride, and found Bordeaux mixture was not compatible and potassium phosphonate is compatible at all concentrations. Similarly, the above mentioned fungicides were tested for their compatibility with Pseudomonas fluorescens and found potassium phosphonate is compatible at all three different concentrations. Vijay Krishna Kumar et al. (2011) in their investigation found that Bacillus subtilis was compatible to 1000 ppm of hexaconazole, propiconazole, tebuconazole and validamycin.

INTEGRATED MANAGEMENT OF Fusarium WILT OF CHICKPEA

Pratibha Sharma (2000) found that carnation wilt caused by Fusarium oxysporum f. sp. dianthi was effectively managed by the combined use of carbendazim and T. harzianum, when the bioagent was applied 14 to 16 days before transplanting along with the dipping of cuttings in carbendazim. Prasad et al. (2002) reported that the soil application of T. viride and Trichoderma harzianum one week before sowing was more effective in reducing wilt and wet root rot of chickpea.

Kaur (2003) studied on control of Fusarium oxysporum f. sp. ciceris by nonpathogenic Fusarium and fluorescent pseudomonads in growth chambers and in micro plots and the selected isolates of nonpathogenic Fusarium and fluorescent pseudomonads were evaluated singly as well as in combination against Fusarium oxysporum f. sp. ciceris causing chickpea wilt and found that the combination of Fo52 with C7R12 was the best where none of the plants showed disease at 30 days after sowing and only 10 per cent plants showed wilting after 60 days. Seed treatment with T. harzianum mutant UM2R + carbendazim (1.25 g kg\(^{-1}\)) resulted in the maximum seed yield (4.6 g plant\(^{-1}\)) and lowest disease incidence (2.5\%) as reported by Poddar et al. (2004).

Steinberg et al. (2004) reported that integrated use of spent mushroom compost and cattle manure significantly reduced the disease incidence of both Fusarium wilt (Fusarium oxysporum) and dry root rot (Rhizoctonia solani) in chickpea.

Landa (2005) found that B. subtilis GB03 and P. fluorescens RG 26, applied either alone or each in combination with nonpathogenic F. oxysporum Fo 90105, was the most effective treatment in suppressing Fusarium wilt of chickpea. Hari Chand and Surender Singh (2005) reported that all the plant extracts, viz., eucalyptus (Eucalyptus globulus), jatropha (Jatropha multifida), neem (Azadirachta indica), garlic (Allium sativum) tested, except Calotropis procera, were significantly superior in reducing wilt incidence in gram. Seed treatment with T. viride and Allium sativum bulb extract are identified as important components of integrated management of chickpea wilt.

Sharma et al. (2005) reported that the combination of neem cake + carbendazim + T. harzianum provided the highest control of the disease Fusarium yellows caused by Fusarium oxysporum f. sp. gladioli. Raju (2005) reported that the lowest disease (pigeon pea wilt) incidence (6.6\%), and the highest number of nodules
per plant (23.3), fresh weight per plant (6.3 g), and dry weight per plant (2.2 g) were obtained with *T. viride* + carbendazim. Kapoor *et al.* (2006) found that amendment with *Lantana camara* (10 t ha\(^{-1}\)) + bioagent Tricoguard at 2.5 kg or 62 kg FYM ha\(^{-1}\) + spray with carbendazim at pre flowering stage was most effective in managing the root rot-wilt complex disease in pea.

Nikam *et al.* (2007) reported that combined soil application of *T. viride* and ground nut cake followed by neem cake had given good control against chickpea wilt caused by *Fusarium oxysporum* f. sp. *ciceris*. Jayasekhar *et al.* (2008) found that under field conditions soil application of *Pseudomonas fluorescens* Pf\(_{ND}\) followed by carbendazim spray (0.2%) after 30 days of *Pseudomonas* application recorded the lowest disease incidence of 3.77 percent. Singh (2009) reported that the application of two bioagents viz., *Trichoderma harzianum*, *Pseudomonas fluorescens* and two fungicides carbendazim (0.1%) and mancozeb (0.25%) at 45 DAS was found effective for the control of coriander wilt caused by *Fusarium oxysporum* f. sp. *coriandrii*. Rashmi Srivastava *et al.* (2010) found that combined use of fluorescent pseudomonads, *T. harzianum* and Arbuscular mycorrhizal fungus significantly reduced disease incidence and severity by 74 per cent and 67 per cent in pots and field, respectively.

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