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CHICKPEA WILT AND ITS MANAGEMENT - A REVIEW

Hari Chand and S.K.Khirbat

Department of Plant Pathology
C.C.S. Haryana Agricultural University, Hisar-125004, India

ABSTRACT

Chickpea (*Cicer arietinum* L), wilt caused by *Fusarium oxysporum f.sp. ciceri* was first reported from India in 1918. Currently the disease is prevalent in several countries. The pathogen is highly variable in its cultural characteristics and pathogenicity. Yield losses vary between 10% to 100% depending on varietal susceptibility and agroclimatic conditions. When disease occurs at seedling stage, seedlings collapse and lie flat on soil surface. In case of adult plants, characteristic symptom is brown to black discolouration of xylem vessels. In susceptible plants hyphae are inter and intracellular in pith, xytem and cortex. The phytotoxin produced by the pathogen causes wilting and leaf burning. There exist a correlation between pathogen produced pectate lyase with pathogenicity and/or virulence. The fungus may be seed borne and may survive in soil. The disease is more severe in light sandy soil than heavy clay. High soil temperature and deficiency of moisture appear to have a definite bearing on its incidence. The amount of organic matter is inversely related to wilt incidence. The development of wilt is favoured by increase in nitrogen. The optimum temperature and pH for pathogen are 25°C and 5-6.5 respectively. Delay in sowing helps in minimizing disease. Mixed cropping of chickpea with wheat and berseem gives measurable disease control. Seed treatment with Benlate T (0.15%) destroys seed borne inoculum completely. Biocontrol agents such as *Trichoderma* spp., *Glomus* spp. and fluorescent *Pseudomonas* give measurable reduction in disease. Use of resistant varieties, which are available, is best mean of disease control.

Chickpea (*Cicer arietinum* L), wilt caused by *Fusarium oxysporum* Schlecht and Emnd Snyd. & Hans. f.*sp. ciceri* (Padwick) Snyd. & Hans. was first reported from India by Butler (1918). McKerral (1923) considered this fusarial disease is to be soil-borne. Narasimhan (1929), McRae (1932) and Prasad and Padwick (1939) also reported that it was caused by *Fusarium* species. Padwick (1940) identified *Fusarium orthoceras* var. *ciceri* as the cause of chickpea wilt in India. In the studies of Dastur (1935), *Rhizoctonia bataticola* produced wilted plants and he called the disease 'Rhizoctonia wilt'. Later, the fungus causing typical wilt was named as *F.orthoceras* var. *ciceri* (Padwick, 1940). Association of *Verticillium alboatrum* with chickpea wilt has also been observed (Bhatti *et al.,* 1983; Erwin, 1958a). Erwin (1958b) named the pathogen *F. lateritium* f. sp. *ciceri.* Snyder and Hansen (1940) renamed *F. orthoceras* var. *ciceri F.* oxysporum f. sp. *ciceri* which is now widely accepted.

The disease has been reported from several countries including India, Bangladesh, Burma, Ethopia, Mexico, Pakistan, Syria, Tunisia, Chile, Iran, Nepal, Sudan, the United States, Peru, USSR, Malawi, Spain, Turkey and Italy. However, chickpea cultivation is greatly threatened by this disease in India, Iran, Pakistan, Nepal, Myanmar, Spain and Tunisia.

With regard to crop losses, no definite data are available. However, rough estimates indicate

that losses may hover around 10-15% each year as a regular feature. In the years of severe epidemics, crop losses have gone as high as 60-70%. Nema and Khare (1973) observed damage to be upto 61% at seedling stage and 43% at flowering stage. Similarly, early wilting reduced the seed number/plant and caused more yield losses than late wilting (Haware and Nene, 1980). The seeds harvested from wilted plants are lighter, wrinkled and duller than those from healthy plants. The yield losses vary between 10% and 100% depending on the agroclimatic conditions (Grewal and Pal, 1970). Sattar *et al.* (1953) reported an annual loss of 12 million rupees from Pakistan.

The chief symptoms of the disease are : yellowing and drying of leaves from base upward, drooping of petioles and rachis, improper branching, withering of plants, browing of vascular bundles and finally wilting of plants (Argikar, 1970; Prasad and Padwick, 1939; Westerlund *et al.,* 1974). In observation of Frisullo *et al.* (1989), diseased plants showed stunting also. Chauhan (1962a) reported the initial symptom of the disease to be acropetal vein clearing of leaves. Nene *et al.* (1980) have made detailed symptomatological studies. Murumkar and Chavan (1985) have noted physiological changes taking place in leaves infected by the pathogen. In a similar study the number of chloroplasts and starch formation in the mesophyll cells decreased following infection by the pathogen (Chauhan, 1962a).

PATHOGENIC VARIABILITY

The pathogen appears to be highly variable. Jimenez-Diaz *et al.* (1993) using differential lines classified 10 isolates from California and 14 from Spain into race group 0, 1, 5 and 6. Dolar (1997) using a set of 10 differential cultivars reported existence of three (0, 2 and 3) of the seven reported races of the fungus in Ankara Province, Turkey. Rao and Krishnappa (1997) categorized isolates colleted from 77 locations of Karnataka into 6 groups on the basis of cultural characters and pathogenicity. Rahman *et al.* (2000) on the basis of reaction on eleven differentials grouped twenty four isolates from 7 states of India into 10. Paulkar and Raut (2004) reported that isolates from Amravati, Akola, Buldhana, and Nagpur (Maharashtra, India) differed in Virulence. Kelly *et al.* (1994) using genetic finger printing and random amplified polymorphic DNA to characterise pathogypes divided 63 isolates into 2 clusters that correlated with the pathotypes causing the yellowing or wilt syndromes. In a further study Kelly *et al.* (1998) could successfully discriminate between a *F.o.* f. sp. *ciceris* which caused wilt and a race which caused yellowing using the polymerase chain reaction primers. Patil *et al.* (2005) on the basis of virulence grouped 6 isolates into two. Jimenez Gasco *et al.* (1998) obtained *F. o.* f.sp. *ciceris* isolates from chickpea representing all pathogenic races and a wide geographical range (India, Israel, Morocco, Spain, Tunisia and USA). DNA bands generated by RAPD-polymerase chain could be used to assign isolates to pathotypes and pathogenic races as well as to discriminate them from non-pathogenic *Fusarium oxysporum*. According to Jimenez Gasco *et al.* (2002) *F.o.* f. sp. *ciceris* consists of two pathogypes (yellowing and wilting) and eight races (race 0, 1 B/C, 1A and 2-6) of diverse geographical distribution. In their studies six isolates, one from each of races 0, 1B/C, 1A, 4, 5, and 6 shared an identical elongation factor lalpha (EFlalpha) gene sequence. *F.o.* f. sp. *ciceris* isolates formed a group distinct from other formae speciales and non-pathogenic isolates. These results indicated that *F. o.* f. sp. *ciceris* is monophyletic. Sivaramakrishnan *et al.* (2002) studied genetic variability among 43 isolates collected from nine states of India using molecular markers, $RAPD_s$ and AFLP. AFLP was found more informative as it differentiated more number of isolates. Khan *et al.* (2002) found no correlation between races and vegetative compatibility groups (VCG). However, a relationship occurred between symptoms produced by the isolates and VCG. They suggested that two distinct VCG's are prevalent in the world. Zamani *et al.* (2004) divided 15 isolates in 3 vegetative compatibility groups. On the basis of virulence also the isolates were grouped in three. Abou-zeid *et al.* (2002) reported that DNA bands generated by RAPD-PCR can be used to assign *F. o.* f. sp. *ciceri* isolates to pathotypes and pathogenic races.

HISTOPATHOLOGY AND PATHOGENESIS

In studies carried out by Kunwar *et al.* (1989) in susceptible plants hyphae were inter and intracellular in pith, xylem and cortex; the epidermis was disintegrated and hypertrophy of cortical and pith cells occurred. A mucilage-like substance was present in the cells of the xylem and cortex. Stevenson *et al.* (1997) observed hyphae in root xylem of wilted plants. In more severe cases, a large portion of stem xylem vessel was also invaded by upto 5 internodes above the points of seed attachment. In studies of Khan *et al.* (2004a) phytotoxin produced by pathogen caused wilting and leaf burning of chickpea cuttings. Perez-Artes *et al.* (2004) observed correlation between pathogen produced pectate lyase with pathogenicity and/or virulence of pathogen. Jorge *et al.* (2005) reported that pathogen produced xylanases on medium which hydrolysed xylan to xylobiose.

EPIDEMIOLOGY

The fungus may be seed borne and may survive in plant debris in soil (Brayford, 1998). Haware *et al.* (1982) showed the fungus to be in the hilum of the seed in the form of chlamydospore-like structures. Shakir and Mirza (1994) also studied the location of pathogen in seed and reported it to be present in cotyledons and axis. The primary infection is through chlamydospores or mycelia. The conidia of the fungus are short lived, however, the chlamydospores can remain viable upto next crop season. The pathogen survives well in roots and stem, even in apparently healthy looking plants growing among diseased ones harbouring enough fungus (Padwick, 1941). The fungus, however, did not survive in the roots placed on soil surface. Plant species other than chickpea may serve as symptomless carriers of the disease. Gupta (1991) reported *Vigna radiata, V. mungo, Cajanus cajan, Pisum sativum* and *Lens culinaris* as symptomless carriers of the disease. Haware and Nene (1982) also found *Cajanus cajan, Lens culinaris* and *Pisum sativum* as symptomless carriers of the disease. The pathogen may also parasitize several weeds such as *Cyperus rotendus, Tribulus terrestris, Convolvulus arvensis* and *Cardiospermum halicacabum* (Nene *et al.,* 1980).

The soil type, reaction, moisture and temperature are known to influence disease development. The greenhouse studies substantiate the fact that the disease is more severe in light sandy soil than heavy clay ones (Kotasthane *et al.,* 1979; Sugha *et al.,* 1994a).Chandra *et al.* (1974) attributed higher disease severity in light sandy soil to its low water retention ability. Rachana *et al.* (2002) reported that black soil support highest wilt incidence (75.5%). Wilt incidence in sandy-loam, red and clay soil was found to be 64.4, 59.9 and 46.6%, respectively. Arora *et al.* (1996) recorded the effect of heat stress on chlamydospores of *Fusarium oxysporum* f.sp. *ciceri*. The chlamydospores lost organic carbon, ability to germinate and pathogenic aggressiveness when exposed to heat stress. According to Sugha *et al.* (1994b) soil temperature in the range of 24.8-28.5°C and soil moisture above 25% within the water holding capacity of soil were most conducive for chickpea wilt. Bhatti and Kraft (1992) examined the effect of soil moisture on disease using three soil metric potential regimes, high (-40 to -20 kPa), medium (-260 to -40 kPa) and low (-1060 to -260 kPa). Wilt increased with decreasing soil matric potential, as did rhizosphere population of pathogen. Patel and Anahosur (2001) observed that infection was pronounced at 50 and 75% soil moisture. High soil temperature and deficiency of moisture appear to have a definite bearing on the incidence of disease. Lower levels of soil moisture (10%) kept the plant mortality low due to the disease, though 12% of the plants were damaged as compared to 83% in soil with moisture at 25% level. Soil temperature relation showed that the disease was optimum at 25^{0}C and at a lower ebb at 20°C. Chauhan (1962b) in his studies showed that the disease intensity increased with the lowering of pH, being considerably low at pH 9.2. In another such study, Chauhan (1963) observed that alkaline soils favoured incidence of wilt. However, Saikh (1974) reported that the pathogen tolerated a wide range of pH, with optimum between 5 and 6.5. Sugha *et al.* (1994b) also reported a pH of 5.2 to be optimum. The amount of organic matter (Chauhan, 1965) and humus (Chauhan, 1962c) content of soil were found

CHEMICAL CONTROL, INDUCED RESISTANCE AND BIOLOGICAL CONTROL

The chemical control of chickpea wilt has been summarized in table 1.The control of the disease using non –conventional chemicals (induced resistance),bioagents and incompatible races is presented in table 2, 3 and 4 respectively.

Table 1: Chemical control of chickpea wilt caused by *Fusarium oxysporum f. sp. Ciceris*

Chemical (s)	Rate	Nature of disease control	Reference
Benlate T	0.15% (S.T.)*	Destroys seed borne inoculum completely	Haware *et al*. (1982)
Bavistin or carboxin	0.25% (S.T.)	Protected seedlings upto 30 days	Verma (1976)
Bavisitn or carboxin	0.2% (S.T.)	Reduced wilt in pots and field	Gupta *et al*. (1997)
Bavistin	0.2% (S.T.)	Quite effective under field conditions	Singh *et al*. (1993)
Bavistin	0.5g/kg seed	Improved germination and reduced wilt by 23.7%	Shukla *et al*. (1981)
Bavistin + Thiram	0.5+2g/kg seed	Promising results obtained	Jalali *et al* (1980)
Bavistin or Thiram andBavistin+Thiram	2.5g/kg seed1.25 + 1.25g/kg seed	Highly effective	Sugha *et al*. (1995)
Bavistin+Thiram	2.5g/kg seed	Decreased disease and increased yield under field conditions	Singh and Jha (2003)
Bavistin+*Rhizobium*	0.1% (S.T.)	More effective in reducing wilt and increasing nodulation than bavistin alone	Anonymous (1983)
Benomyl	Soil drench (green house)	Very effective	Illyas *et al*. (1992)
Mildothane	0.1% (S.T.)	Improved seed germination and gave best responses	Shrisat and Kale (1979)
Phytobacteriomycin & trichothecin (Antibiotics)	Dusting	Able to decrease disease	Kuzmina (1966)

*S.T.: Seed Treatment

Table 2: Control of Chickpea wilt by non-conventional chemicals

Chemicals	Nature and mechanism of protection	Reference
Mercuric sulphate cycloheximide Indole acetic acid cycocel	• S.T. At concs. 10-3 to 10-6M • IAA, Cycloheximide and cycocel gave very strong protective effect • Wilt symptoms reduced by 45-57% • Reduced mortality and vascular colonization	Chowdhury (2000)
Salicylic acid and Bion	• Seed soaking at 1.0 and 1.5 mM cons. • Seed soaking at 0.3 and 04 mM concs. • Wilt was significantly reduced in all treatments	Sarwar *et al.* (2005)
Chitosan	• S.T. 0.3 and 1% • Wilt symptoms reduced by 45-59% and prevented plant mortality appreciably • Increase in total and ortho-dihydroxyphenol contents • Enhanced polyphenol oxidase, Peroxidase and phenylalanine ammonia-lyase activities usually associated with defence	Chowdhury and Sinha (2000)
Salicylic acid + *Pseudomonas fluorescens*	• Bacterium induced resistance and reduced wilt by 26-50% • Salicylic acid reduced wilt by52-64% • Reduction in disease more pronounced with combined application	Saikia *et al.* (2003)

inversely related to wilt incidence. In the studies of Sugha *et al.* (1994b) increase in phosphorus and potassium contents did not influence the development of wilt but it was favoured by increase in nitrogen and organic carbon. Laboratory studies of Gupta (1999) showed that zinc inhibited the growth of pathogen. Rao and Krishnappa (1996) observed the relationship between environmental factors and *Meloidogyne-Fusarium* wilt complex of chickpea caused by *Fusarium oxysporum* f.sp. *ciceris*. Maximum and minimum air temperature, soil pH, potassium and nitrogen contents had positive correlation with the disease complex. Rainfall, relative humidity, potassium and organic carbon contents showed negative correlation.

DISEASE MANAGEMENT

Management practices directed toward pathogen for checking the progression of the disease occurrence could be exclusion and eradication of the pathogen and to reduce its inoculum. By the varied nature of pathogen involved, evolving resistant varieties has so far proved to be the best bet, although other conventional chemical, cultural methods and biological control have also yielded good results. Since this crop is grown principally in rainfed areas, many of the known conventional chemical methods have not found wide adoption.

MANIPULATION OF AGRONOMICAL PRACTICES

Early planted crops usually attract more disease. Several studies have suggested that higher disease control and yield are obtained when the planting is delayed until the last week of October (Chandra *et al.,* 1974; Mundkur, 1946; Padwick and Bhagwagar, 1943). The lower disease incidence in late-sown crop was considered to be due to low temperature prevailing during the period of late-sown crop. The studies of Navas-Cortes *et al.* (1998) showed that for each year of experiment epidemic development was related mainly to the date of sowing. Thus, for chickpea crop in southern Spain, advancing the sowing date from early spring to early winter can slow down the development of epidemic, delay the epidemic onset and minimise the final amount of disease.

Plants spaced at 15-20 cm had much higher disease incidence than those spaced at 7.5 cm; this was attributed to the shallower root system in widely spaced plants which were susceptible to wilt when subjected to moisture stress (Bahl, 1976). Planting of seeds at proper depth (10-12

Table 3: Biological control of chickpea wilt

Antagonist (s)	Nature and mechanism(s) of disease control	Reference
Bacillus subtilis *Gliocladium virens* *Trichoderma viride, T. harzianum*	• Seed coating signficnatly reduced wilt by 30-40.8% • Integration of biocontrol agent with carboxin increased yield by 25.4-42.6%	De *et al.* (1996)
Bacillus subtilis *T. harzianum*	Antagonists colonized chickpea roots and suppressed wilt	Hervas *et al.* (1998a)
B. subtilis *T. harzianum*	Protection varied among cultivars	Hervas *et al.* (1998b)
T. viride *T. harzianum* *B. subtilis* *P. fluorescens*	• No reduction in wilt in JG62 but significantly reduction in Ujjan 21. Lowest wilt recorded for *P. fluorescens* • *T. harzianum* + *P. fluorescens* & *B. Subtilis* + *P. Fluorescens* better	Agrawal *et al.* (2002)
B. subtilis *Streptomyces griseus* *Penicillium spp.* *T. harzianum, T. Viride*	• Seed treatment with culture suspension reduced pre-and post emergence losses by 40%. • Lysis of hyphae in vitro conditions observed	Dhedi *et al.* (2002)
T. harzianum + vitavax-200 & thiram	Integration of bioagent with fungicide increased disease control from 29 to 63.3%	Kaur and Mukhopadhyay (1992).
T. harzianum + *T. viride*	• Soil application better than S.T. • *T. harzianum* better than *T. viride*, wilt reduced from 16% to 5.1%.	Prasad *et al.* (2002)
T. harziandum + carbendazim (1.25g/kg)	Very effective than their individual application	Poddar *et al.* (2004)
T. harzianum	Best among many tested	Singh *et al.* (2003)
T. viride, T. harzianum, *Gliocladium virens*	*T. viride* most effective giving 77.8% control	Hari Chand and Surender Singh (2005)
Pseudomonas aeruginosa	Suppressed wilt possibly through production of antibiotic	Anjaiah *et al.* (1998)

Cont...

Fluorescent *Pseudomonas*	S.T. with antagonist reduced wilt in sick plot	Kumar (1998)
Fluorescent *Pseudomonas* and antibiotic producing *B. subtilis* strain	Reduced wilt significantly	Kumar (1999)
Fouorescent *Pseudomonas* strain RBT 13	Seed bacterization reduced wilt by 52% in sick plot	Kumar and Dubey (1992)
Rhizobacteria isolates CRSM8 and CRSNM18	Reduced wilt in sick plot by 48.6 and 31.5%	Khot *et al.* (1996)
T. harzianum	• S.T. (2g/kg seed) with both bioagents suppressed wilt	Khan *et al.* (2004b)
P. fluorescens	• In combination more effective and reduced wilt by 66%	
P. fluorescens	• S.T. and soil application very effective	Landa *et al.* (2001)
RGAF19 & RG26	• Disease control affected by temperature & pathogen inoculum	
Rhizobacteria	• S.T. & soil treat, suppressed wilt	Landa *et al.* (2004)
P. fluorescens	• Temp. affected control	
B. megaterium		
T. viride	• S.T. (4.5g/kg) with commercial formulations of *T. viride* + soil application showed lowest wilt (12.1%)	Jahagirdar *et al.* (2002)
P. fluorescens		
P. fluorescens + Animal manures	Approxmiately 50% reduction in wilt incidence	Inam Ul-Haq *et al.* (2002)
P. aeuginosa	• Controlled wilt & charcoal rot	Saikia *et al.* (2004)
	• Antifungal compound produced	
P. aeruginosa PNAI	• Protected plants from wilt	Anjaiah *et al.* (2003)
	• Antagonist produced antibiotic	
Glomus mossae	Wilt & root rot suppressed by 54 and 62% respectively	Rakesh Kumar *et al.* (2004)
G. fasciculatum + *T. harzianum* + Rhizobium sp.	Lowest wilt index when combiend use was done	Siddiqui and Singh (2004)

Table 4: Induced resistance in chickpea to wilt due to incompatible race/ non-pathogenic *Fusarium oxysporum*

Incompatible race	Nature and mechanisms of protection	Reference
Race 0 and Non-host isolates of *Fusarium Oxysporum*	• Delayed the onset of symptoms and reduced wilt • Non-host isolates more effective • Inoculation with inducers gave rise to synthesis of phytoalexins • Accumulation of Chitanase, Beta-1,3-glucanase and peroxidase activities	Cachinero *et al.* (2002)
Non-Pathogenic *Fusarium Oxysporum*	• Disease Incidence reduced by 25-30%	Kaur *et al.* (2003)

cm) was helpful in reducing the disease incidence (Singh and Sandhu, 1973), while shallow sown crop seemed to attract more disease (Dahiya *et al.,* 1988; Saikh, 1974; Sugha *et al.,* 1994b). Hanif *et al.* (1999) noted the effect of various sowing depths on wilt incidence in wilt-sick field in Pakistan. Deep sowing had no effect on reduction of *Fusarium oxysporum* wilt incidence in susceptible chickpea variety Aug 424 in field in Faislabad, Pakistan in 1995. The level of infection was similar at the 10 cm sowing depth as it was at the 30 cm depth. Planting the crop with "Pora" method (Saraf *et al.,* 1973) using lower seed rate helped to minimise disease, whereas broadcast method of planting increased wilt incidence (Bedi and Pracer, 1952). Development of wilt is more prominent under moisture stress conditions (Kausar, 1968). One irrigation before flowering decreases disease incidence and increases yield (Saraf *et al.,* 1973; Sekhon, 1952). However, in studies of Abouzeid *et al.* (2003), the disease incidence (root rot/ wilt) increased by 2 to 3 fold as the number of irrigations increased. The pathogen was most frequently isolated from the infected stem and root samples of chickpea receiving one irrigation. Mixed cropping of chickpea with wheat and berseem (Saraf *et al.,* 1973) has given measurable disease control. Agrawal *et al.* (2002) noted effect of wheat, barley, linseed and mustard intercrops/mixed cropping with chickpea on wilt incidence. Intercropping/mixed cropping reduced wilt incidence and increased yield of chickpea. Lowest wilt incidence obtained with intercropping and mixed cropping with linseed. Mayur *et al.* (2003) reported that wilt incidence was significantly reduced by amending the soil with de-oiled mustard cake, groundnut cake and farm yard manure. Soil solarization (covering soil with transparent 100 mm thick polythene sheet for 6-8 weeks from April to May) decreased population of *Fusarium* and plant parasitic nematodes (Chauhan *et al.,* 1998).

EFFECT OF PLANT EXTRACTS

Seed treatment with garlic leaf extract (Singh *et al.,* 1979) and neem oil (Singh *et al.,* 1980) are reported to produce disease free seedlings. Hari Chand and Singh (2005) reported that seed treatment with bulb extract of *Allium sativum* reduced wilt from 65.9% in control to 23.6%. Leaf extract of *Azadirachta indica* at 100% conc. completely inhibited germination of pathogen spores (Singh and Hari Chand, 2004).

DISEASE RESISTANCE

Techniques for screening chickpeas against the disease are available (Nene *et al.,* 1981). A very good correlation has been observed between sick plot screening and screening using spore free culture filtrate of the fungus in laboratory (Bajwa *et al.,* 2000).

Table 5: Chickpea wilt resistant varieties developed recently

Variety/ varieties	Country	Reference
BG1053	India	Sandhu *et a*l. (2002)
PDG4	India	Singh *et al*. (2002)
Kabuli CV ICCV2 and UC 15	Sudan	Ali *et a*l. (2002)
Gujrat Gram 4	India	Pithia *et al.* (2003a)
Gujrat Gram 1	India	Pithia *et al*. (2003b)
Himachal Chana 2	India	Anand Singh *et al*. (2003)
PKV Kabuli 2	India	Zope *e*t al. (2002)
Virat (Kabuli)	India	Deshmukh *et al.* (2004)
Punjab 2000	Pakistan	Ali *et a*l. (2004)
PBG 5	India	Sandhu *et al* . (2004)
JGK 1	India	Gaur *et al*. (2004)
Chefe	Ethiopia	Daba *et a*l. (2005)
Vihar	India	Jamadagni *et al*. (2005)
CA 2954	Spain	Rubio *et al.* (2004)
BGM 547	India	Kharkwal *et al*. (2005)
COG 29-1	India	Sivakumar and Muthiah (2001)
L551	India	Bains *et al*. (2000)

However, a cultivar resistant under field conditions may show susceptibility under laboratory conditions by tissue culture method using culture filtrate (Singh *et al.,* 2003).

Sources of resistance identified have been reviewed (Jalali and Hari Chand, 1992; Hari Chand *et al.,* 2002). Several wilt resistant cultivars have been developed recently (Table 5).

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