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A review on fusarium wilt disease of chickpea and different strategies for its management

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Abstract
Chickpea (*Cicer arietinum* L.), contributes 18% of the global production of grain legume as dietary proteins. Fusarium wilt caused by *Fusarium oxysporum* f. sp. *Cicero* (*Foc*) was first reported from India in 1918. Currently the disease is predominant in major growing areas in the world. Fusarium wilt is a seed and soil borne disease, the pathogen is highly mutable in its cultural characteristics and pathogenicity. Eight pathogenic races of *Foc* (races 0, 1A, 1B/C, 2, 3, 4, 5 and 6) have been reported worldwide. An important decrease in cropping area and production has been recorded during the last two decades. Several biotic and abiotic constraints underlie this decrease. Fusarium wilt epidemics can devastate crops and cause 10 % to 100% loss in extremely infested fields and under favourable conditions. Despite the efforts deployed in breeding and selection of several chickpea varieties with high yield potential that are tolerant to diseases, the situation has remained the same for the last decade. The disease was first occurring 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, xylem and cortex. The phytotoxin produced by the pathogen causes wilting and leaf burning. There exists a correlation between pathogen produced pectate lyase with pathogenicity or virulence. Development of resistant cultivars against fusarium wilt in different breeding programs is principally based on conventional selection. This method is time-consuming and depends on inoculum load and specific environmental factors that influence disease development. The use of molecular tools offers great potential for chickpea development, specifically by identifying molecular markers closely linked to genes/QTLs controlling fusarium wilt. This review summarises the current status of fusarium wilt exploitation and different strategies used for its management.

Keywords: Chickpea, Fusarium Wilt Disease, Management Straitgey

Introduction
Chickpea, also called garbanzo bean or Bengal gram, is a self-pollinated, annual diploid (2n = 2x = 16) species [16] with a genome size of 738 Mb [90], While this size is slightly larger than that of the model legume, *Medicago truncacula Gaertn.,* (530 Mb), it is much smaller than other major legume crops such as soybean, peanut, garden pea, alfalfa, and lentil [52]. The *Cicer* genus belongs to the family Leguminoseae, sub-family Papilionaceae and tribe *Cicereae.* It is composed of 9 annual and about 34 perennial wild species. Among the 9 annual species, chickpea (*C. arietinum* L.) is the only cultivated species [79]. Chickpea has the ability to increase the soil fertility, particularly in dry lands, by fixing atmospheric nitrogen (N). It is mainly used for human consumption and is an essential constituent of the Mediterranean diet and abasic food in India as well as world. The chickpea grain international market is very active due to the crop's nutritional value. Chickpea is a good potential and cheap source of protein for people in developing countries (especially in South Asia), who are mostly vegetarian, either by choice or because of financial reasons [24]. Chickpea is mean source of protein, low in fat and sodium, cholesterol free and is an excellent source of both soluble and insoluble fiber, as well as complex carbohydrates, vitamins, folate, and minerals, especially calcium, phosphorous, iron, and magnesium [66]. It's used as flood purification in case of human.

There are two different types of cultivated chickpea: "Desi and Kabuli". The Desi (Microsperma) types have pink flowers, anthocyanin pigmentation present on stems, and a colored and thick seed coat while, Kabuli (Macrosperma) types have white flowers, lack anthocyanin pigmentation on stems and have white or beige-colored seeds with a ram's head shape, a thin seed coat and a smooth seed surface [41]. The geographic distribution differs for these two types, Kabuli chickpea seeds are grown mainly in temperate regions, whereas

Desitype is grown in the semi-aridtropics [54]. Kabuli-type chick pea is considered Biological. More economically important as it obtains higher market worth than Desi-type. However, most of the genomic resources have been generated for Desi-type chickpea so far [3]. The two types represent different genetic background, differing in important agronomic trait, such as disease resistance, cold tolerance and growth habit [12].

Fluctuations in the acreage of cultivated area of chickpea and annual production were observed this last decade. Average global chickpea yield enhanced during the past decades, increasing from 683 kg/ha in 1990 to 966 kg/ha in 2014 20], but it is still considered below the yield potential of the crop. This increase is attributed to the development of new resistant cultivars against biotic and abiotic stresses. 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.

Fusarium wilt of chick pea

Fusarium wilt of Chickpea mostly occurs in 32 countries across 6 continents. This disease was first reported in India by Butler in 1918, but its etiology was not correctly determined until 1940 by Padwick [18]. According to Haware and Nene, [31] and Fusarium wilt epidemics can be devastating to individual crops and cause up to 100% loss under favourable conditions.

Symptoms

Symptoms of the disease was observed at any stage of plant growth, and affected plants may be grouped in patches or appear spread across a field [36]. The wilt can be observed in susceptible genotypes within 25 days after sowing in the field (designated "early wilt") [36, 5]. However, symptoms are usually more visible in the early stages of flowering, 6 to 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 [36]. Early wilting causes heavy loss than late wilting. Nevertheless, seeds from late-wilted plants are lighter, rougher and duller than those from healthy plants [59]. The crucial symptoms of the disease are-yellowing and drying of leaves from base of the upward, drooping of petioles and rachis, improper branching, withering of plants, browing of vascular bundles and finally wilting of plants [93]. In observation of Frisullo *et al*. [22], diseased plants showed stunting also. Chauhan [16] reported the initial symptom of the disease to be acrop *et al*. vein clearing of leaves. Murumkar and Chavan [57] have been 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 [14]. Wilting is appearing at two most prominent crop growth stage, seedling and vegetative stage.

Seedling stage

The disease can be observed within 3 weeks after sowing in field. Whole seedlings (3 - 5 weeks after sowing) collapse and lie flat on the ground. These seedlings retain their dull green color. When uprooted seedling, they usually show uneven shrinking of the stem above and below the collar region (soil level). The shrunken portion approximately 2.5 cm. Affected seedlings do not rot on the stem or root surface. However, when stem split open vertically from the collar downwards or cut transversely, dark brown to black discoloration of the inside stem tissues is clearly visible. In seedlings of highly susceptible cultivars, *i.e.,* JG 62, which dies within 10 - 15 days after emergence, the black discoloration may not be clearly visible. However, internal browning from root tip upwards is clearly seen inside the stem [60].

Vegetative stage

The affected plants show typical wilting, *i.e.,* drooping of the petioles, rachis and leaflets. Drooping is clear visible initially in the upper part of the plant but within a day or two, the entire plant droops out. The lower leaves of the plant are showing chlorotic color, but most of the other leaves' droops while still green. Regularly, however, all the leaves turn yellow and then light brown or straw colored. Dried leaflets of infected plants are not shed at maturity. Affected plants, when uprooted and examined before they are completely dry, show no external rotting, drying, or root discoloration. When the stem is split vertically, internal discoloration can be seen. Around the collar region, above and below, the xylem in the central inner portion (pith and part of the wood) is discoloured dark brown or black. In the initial stage of wilting, the discoloration may not be continuous. Discoloration also extends several centimetres above the collar region into the main stem and branches. If the collar region is cut transversely with a sharp razor blade, black discoloration of both pith and xylem can be seen. Sometimes only a few branches are affected, resulting in partial wilt. In certain cultivars (*eg.,* T-3), the lower leaves dry up before the plants wilt [60].

The pathogen

Fusarium wilt of chickpea is caused by *Fusarium oxysporum* f. sp. *Ciceris Schlechtend: Fr.* f sp.*ciceris* (Padwick) T. Matuo & K. Satô [Jimenez-Fernandez *et al.,* 2011]. On potato sucrose agar (PSA) and potato dextrose agar (PDA) and under near-UV light, the aerial mycelium is at first white and cottony, but later it may become creamy or salmonincoloror remain whitish [37]. Fusarium wilt of chickpea produces micro conidia, macro conidia and chlamydospore. The micro conidia (2.5 - 4.5 µm×5 - 11 µm) are oval or cylindrical, straight or curved in shape. Macro conidia (3.5 - 4.5 µm×25 - 65 µm) are produced more sparsely than micro conidia and usually they are three to five septate. Chlamydospore are formed after 15 days old cultures and infected chickpea tissues, formed singly, in pairs or in chains, and are smooth or rough-walled [12]. Hyphae are septate and profusely branched. The fungus can grow at temperatures ranges 7–35 °C and pH 4 - 9.4. Optimal conditions for mycelial growth are 25 - 27 °C and pH 5.1-5.9, depending upon strains. Optimal pH for sporulation is 7.1 - 7.9 [37].

Pathogenic variability

The fusarium wilt of pathogen appears to be highly mutability. [39]. using differential lines classified 10 isolates from California and 14 from Spain into race group 0, 1, 5 and 6. Dolar [19] 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 [65]. Categorized isolates collected from 77 locations of Karnataka into 6 groups on the basis of cultural characters and pathogenicity. Rahman *et al*. [64]. On the basis of reaction on eleven differentials grouped twenty-four isolates from 7 states

of India into 10. Paulkar and Raut [62]. Reported that isolates from Amravati, Akola, Buldhana, and Nagpur (Maharashtra, India) differed in Virulence. Kelly *et al.* (1994) [45]. Using genetic finger printing and random amplified polymorphic DNA to characterise patho types divided 63 isolates into 2 clusters that correlated with the patho types causing the yellowing or wilt syndromes. In a further study Kelly *et al.* [46]. 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.* [61] on the basis of virulence grouped 6 isolates into two and 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 patho types and pathogenic races as well as to discriminate them from non-pathogenic *Fusarium oxysporum.*

According to Jimenez Gasco*et al.* [38] *F. o.* f. sp. *ciceris* consists of two path otypes (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 form aespeciales and non-pathogenic isolates. These results indicated that *F. o.* f. sp. *Ciceris* is monophyletic. Sivarama krishnan *et al.,* (2002) studied genetic variability among 43 isolates collected from nine states of India using molecular markers, RAPDs and AFLP. AFLP was found more informative as it differentiated a greater number of isolates. Khan *et al.* [47] 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.* [94] divided 15 isolates in 3 vegetative compatibility groups. On the basis of virulence also the isolates were grouped in three. Abou-zeid *et al.* [2] reported that DNA bands generated by RAPD-PCR can be used to assign *F. o.* f. sp. *Cicero* isolates to patho types and pathogenic races.

Histopathology

In studies carried out by Kunwar *et al.* [49] 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.* [83] observed hyphae in root xylem of wilted plants, in severe cases, a large portion of stem xylem vessel was also invaded by up to above the 5 internodes points of seed attachment. In studies of Khan *et al.* [47] phytotoxin produced by pathogen caused wilting and leaf burning of chickpea cuttings. Perez-Artes *et al.* [63] observed correlation between pathogen produced pectate lyase with pathogenicity and/or virulence of pathogen. Jorge *et al.* [40] reported that pathogen produced xylanases on medium which hydrolysed xylan to xylobiose.

Disease cycle and epidemiology

Thefunguscanbetransmittedbyseedandmaysurviveinplantdebrisinsoil.Itwasdemonstratedthat the fungus, chlamydospore was found free in soil [29], hilum of the seed, cotyledon and axis [72]. The primary infection is start through chlamydospores or mycelia. The conidia of the fungus are short lived; however, the chlamydospores can remain viable up to the next crop season. Chlamydospore formation depends on the nutrient status of the inoculum. Under field conditions, fungal inoculum may be exposed to much lower nutrient levels compared to the "well-fed" macroconidia produced on rich agar media [81]. The pathogen survives well inside roots and stems, even in appear entry healthylookingplantsgrowingamongdiseasedonesharboringsufficientfungus. The fungus remains dormant in off season as chlamydospores in plant debris until stimulated to germinate, once carbohydrates are released from decaying plant tissue or from roots [18 and 88]. The stimulus for germination may be host or nonhost plant roots, or contact with pieces of fresh (not colonized) plant debris. After the chlamydospores germinate, conidia and new chlamydospores may be formed as well as hyphae. Following germination, if conditions are favourable then a thallus is produced from which conidia form in 6-8 hrs and chlamydospores in 2-3 days. Invasion mechanism of the roots is followed by the penetration of the epidermal cells of the host or the non-host [8] and the development of a systemic vascular disease in host plants [84]. Pen etration occur seethe rthrough wound so redirect. The most common sites of direct penetration are located at or near the root tip of both taproots and lateral roots [53]. Penetration is controlled by a combination of different factors that is included fungal compounds, plant surface structures, activators or inhibitors of fungal spore germination, and germ tube formation [56]. During colonization of roots, the mycelium advances intra cellularly through the root cortex until it reaches the xylem vessels and enters them through the pits. The fungus then remains exclusively within the xylem vessels using them to colonize the host [11].Chlamydospore formation depends upon the nutrient status of the inoculum. Under field conditions, fungal inoculum may be subjected to much lower nutrient levels compared to the "wellfed" macroconidia produced on rich agar media [71]. The pathogen survives in roots and stems, even in apparently healooking plants growing among diseased ones harboring sufficient fungus [88].The fungus remains dormant as chlamydospores in plant debris until stimulated to germinate, once carbohydrates are released from decaying plant tissue or from roots. The stimulus for germination may be host or nonhost plant roots, or contact with pieces of fresh (not colonized) plant debris. After the chlamydospores germinate, conidia and new chlamydospores may be formed as well as hyphae. Following germination, a thallus is produced from which conidia form in 68 h, and chlamydospores in 23 days if conditions are favorable. Invasion of the roots is followed by the penetration of the epidermal cells of the host or the nonhost [8]
and the development of a systemic vascular disease in host plants. Penetration occurs either through wounds or directly [84].The most common sites of direct penetration are located at or near the root tip of both taproots and lateral roots [53].Penetration is controlled by a combination of different factors that include fungal compounds, plant surface structures, activators or inhibitors of fungal spore germination, and germ tube formation [56]. During colonization, the mycelium advances intracellularly through the root cortex until it reaches the xylem vessels and enters them through the pits. The fungus then remainsexclusively within the xylem vessels, using them to colonize the host [Bishopt, 1983].

Wilting is most likely caused by a combination of pathogensactivities. These include accumulation of fungal mycelium in the xylem and/or toxin production, host defence responses, including production of gels, gums and tyloses and vessel crushing by proliferation of adjacent parenchyma cells.

residues, roots and stem tissue buried in the soil for more than 6 years, even in the absence of the host[79, 12, 85 and 30].
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 thedevelopment of Fus arium wilt [5].

Management of fusarium wilt

Management of Fusarium wilt of chickpea is very contrast to achieve and no single control measure is fully effective [30]. 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 [37]. For such a goal, measure of control should include, 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.

Use free pathogen seeds and avoid sowing into high risk soils

The fungus can be transmitted by infected seeds and plant debris [35]. Use of infected propagating material can lead to introducing the pathogen into pathogen-free soils or production areas. Therefore, the importance of checking the health of that material through certification programs, phytosanitary inspection and quarantine legislation should be considered. Proper selection of the planting site optimizes the use of *F. oxysporum* spp.-free planting material in non-infested soils [35]. Disease risk assessment based on inoculum density and disease incidence relationships would be useful. Indeed, the inoculum density in soil at planting sites could be estimated to avoid those with high risk for severe disease. Recently, Jiménez-Fernández *et al.*, [35] developed a real-time quantitative polymerase chain reaction (q-PCR) protocol that allows quantifying *Foc* DNA down to 1pg in soil as well as in root and stems of infected a symptomatic chickpea plants that may be of use for the detection and identification of the pathogen in certification programs, phytosanitary inspections and quarantine legislation. Seed-borne inoculum can be eradicated by seed dressing with Benlate [31].

Manipulation in agronomic practice

A significant linear relationship was found between disease development over time and weather variables at the experimental site, with epidemics developing earlier and faster as mean temperature increased and accumulated rainfall decreased. Under conditions highly conducive for Fusarium wilt development, the degree of disease control depended primarily on choice of sowing date, and to a lesser extent on level of resistance of chickpea genotypes to *F. oxysporum* f. sp. *ciceris*race 5, and the biocontrol treatments. However, that effect was primarily influenced by sowing date, which also determined disease development. Effectiveness of biocontrol treatments in disease management was lowest in January sowings, which were least favourable for fusarium wilt. Sowing in February, which was moderately favourable for wilt development, resulted in the greatest increase in seed yield by the biocontrol agents. In March sowings, which were most conducive for the disease, the biocontrol agents delayed disease onset and increased seedling emergence [51]. Early planted crops usually attract more disease as compare to late planted. Several studies have suggested that higher disease control and yield are obtained when the planting is delayed until the last week of October [13]. 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.* [59] 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 [7]. Planting of seeds at proper depth (10-12 cm) was helpful in reducing the disease incidence, while shallow sown crop seemed to attract more disease [86]. Hanif *et al.* [28] 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 [69] using lower seed rate helped to minimise disease, whereas broadcast method of planting increased wilt incidence [9]. Development of wilt is more prominent under moisture stress conditions [44]. One irrigation before flowering decreases disease incidence and increases yield [69].
However, in studies of Abouzeid *et al.* [1], the disease incidence (root rot/ wilt) increased by 2 to3-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 [69] has given measurable disease control. Agrawal *et al.* [4] 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.* [55] reported that wilt incidence was significantly reduced by amending the soil with 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 [14]. Soils infested with pathogenic forms of *F. oxysporum* can be recovered for agricultural production by reducing the amount of initial inoculum and potential for disease to levels low the beginning for severe disease [35]. This aim can be achieved by means of chemical, physical or biological disease control methods. Fusarium wilt diseases of several crops have been successfully controlled by soil solarization. The heat generated by solarization may not kill a pathogen outright, but the organism may be weakened, resulting in reduction of its aggressiveness for its host and greater susceptibility to attack by other components of the soil microflora [85]. In addition, soil-borne plant pathogen control could be realized by flooding that destroys many soil-borne pathogens [35, 85]. Removal of debris from Fusariumwilt affected chickpea crops and burning or flaming them to achieve thermal killing of *Foc* 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 [36].

Effect of plant extracts/botanicals

Seed treatment with garlic leaf extract [81] and neem oilare reported to produce disease free seedlings and seed treatment with bulb extract of *Allium sativum* reduced wilt from 65.9% in control to 23.6%. Leaf extract of *Azadirachtaindica* at 100% concentration completely inhibited germination of pathogen spores [80].

Biological control

Biocontrol agents is the play vital significant role and ecological were a reduction in the rate of epidemic development over time, a reduction of disease intensity, and an increase in chickpea seedling emergence. Chickpea seed yield was influenced by all three factors in the study. The increase in chickpea seed yield was the most consistent effect of the biocontrol agents. *Bacillus subtilisGB03* and *Pseudomonas fluorescens RG 26*, applied either alone or each in combination with non-pathogenic *F. oxysporum F. o 90105*, were the most effective treatments at overwhelming Fusarium wilt, or delaying disease onset and increasing seed yield, respectively. The use of bio-gents for control exhibitgreat potential. Indeed, a biological control agent colonizes the rhizosphere and leaves not oxicresidues as opposed to chemical. The species of *Trichoderma* have been evaluated against the wilt pathogen and offer great potential in handling chickpea wilt under glasshouse and field conditions [43]. Furthermore, the use of *P. fluorescens* inhibits the growth of *F. oxysporum* f. sp. *ciceris in vitro* and permits significant growth, increases in shoot length, dry weight and grain yield [58] Treatment with *P. fluorrescens* formulation increased chickpea yield in field and can be effectively used as seed treatments to control chickpea wilt[92]. Inaddition, attributeddiseasereductiontoincreasedplantdefensereactionsinr esponse to root colonization by the non-pathogenic strain of *Fusarium* spp. a study was conducted with different isolates of *Bacillus* spp. And *Pseudomona schloro raphis* show headstrong antagonism against three races of *F. oxysporum* f. sp. *Ciceris* [51].

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

Chemicals	Nature and m mechanism of protection	Reference
Mercuric sulphate cycloheximide Indole acetic acid cycocel	S.T. at concentration. 10-3 to 10-6 IAA, Cycloheximide and cycocel gave very strong Wilt symptoms reduced by 45-57% Reduced mortality and vascular colonization	Chowdhury and Sinha 2000 [15].
Salicylic acid and Bion	Seed soaking at 1.0 and 1.5 m Mconcs. Seed soaking at 0.3 and 0.4 m Mconcs. Wilt was significantly reduced in all treatments	Sarwan*et al.*,(2005)[70].
Chitosan	S.T. 0.3 and 1 % Wilt symptoms reduced by 45-59% and prevented plant mortality appreciably contents Increase in total and ortho-dihydroxyphenyl contents Enhanced polyphenol oxidase (PPO), Peroxidase (PO) and phenylalanine ammonia-lyase (PAL) activities usually associated with defence	Chowdhury and Sinha (2000)[15].
Salicylic acid + *Pseudomonas fluorescens*	Bacterium induced resistance and reduced wilt by 26-50% Salicylic acid reduced wilt by52-64% • Reduction in disease was more pronounced with combined application	Saikia*et al*., (2003)[68].

Table 2: Chemical control, induced resistance and biological control, Chemical control of chickpea wilt has been summarized in table. The control S.T.- Seed Treatment

Chemical (s)	Rate	Nature of disease control	References
Benlate T	0.15 % (S.T.) *	Destroys seed borne inoculum completely	Haware *et al.* (1982) [31].
Bavistin or carboxin	0.25% (S.T.)	Protected seedlings up to 30 days	Verma (1976) [91].
Bavistin or carboxin	0.2% (S.T.)	Reduced wilt in pots and field	Gupta *et al.*, (1977) [27].
Bavistin	0.2% (S.T.)	Quite effective under field conditions	Singh *et al.*, (1993) [78].
Bavistin or Thiram	0.5 + 2g/kg seed	Promising results obtained	Jalall *et al.*, (1980) [34].
Bavistin or Thiram and Bavistin + Thiram	2.5 g/kg seed, 1.25 + 1.25 g/kg seed	Highly effective	Sugha *et al.*, (1994) [86].
Bavistin + Thiram	2.5 g/kg seed	Decreased disease and increased yield under field conditions	Singh andjha (2003) [76].
Bavistin+ Rhizobium	0.1% (S.T.)	More effective in reducing wilt and increasing nodulation than bavistin alone	Anonymous (1983) [6].
Benomyl	Soil drench (greenhouse)	Very effective	Illyas *et al.*, (1992) [33].
Mildothane	0.1% (S.T)	Improved seed germination and gave best response	Shrisat and kale (1979) [75].
Phytobacterium and Trichothecene (Antibiotics)	Dusting	Able to decrease disease	Kuzmina (1966) [50].

Resistance cultivars

Currently, the use of resistant cultivars appears to be the most practical and economically efficient control measure for management of chickpea Fusarium wilt. Resistant chickpea cultivars represent a key component in integrated disease management (IDM) programs that involve the use of additive or synergistic combinations of biotic, cultural, and chemical control measures [17]. Resistance to *Foc*races had been identified mainly in Desi germplasm and to a lesser extent in Kabuli chickpeas, as well as in wild *Cicer* spp. [36]. The deployment of resistant cultivars has not been extensive because of undesirable agronomic characteristics in some developed materials. Furthermore, the high pathogenic variability in *Foc* populations may limit the effectiveness and extensive use of available resistance.

Table 3: Genetics of resistance to different races of the chick pea wilt pathogen *F. o*. f. sp.*ciceris*

Fusarium race	Name of the resistance gene	Effect of resistance gene on wilting	Reference
0	*foc*-0_1/ *Foc* 0_b *foc* -0_2/ *Foc*-o_2[a]	Complete resistance[b]	Rubio *et al.*, (2003) [67].
1 A	h_1 (syn *foc*-1),	Late wilting	Singh *et al.*, (1987) [77].
	h_2	Late wilting	
	h_3	Late wilting	
1B/C	-	-	-
2	*foc-2*[c]	Complete resistance	Sharma *et al.*, (2005) [73].
3	*foc -3/ Foc-3*[a]	Complete resistance	Sharma *et al.*, (2004. 2005) [73, 74].
4	*foc-4*	Complete resistance	Sharma *et al.*, (2005) [73].
	Two recessive genes	Complete resistance[b]	Tullu *et al.*, (1999) [89].
5	*foc-5/ Foc-5*[a]	Complete resistance	Takeoglu *et al.*, (2000) [87].

1. Dominant/ recessive nature not known
2. Effect of individual genes in resistance not known
3. Kumar (1998) found it to be governed by three genes, a,b, and c. each of the three genes led to late wilting whereas the first two genes conferred complete resistance

(-) genetics of resistance not known

Chick pea resistance against Fusarium wilt has been reported to be monogenic oroligogenic depending upon the race or resistance source [73]. Early studies on genetic resistance to *Foc* revealed that resistance to race 1A is governed by at least three independent genes namely h1, h2, and H3 [73]. Late wilting resistance is conferred by any one of these three genes, but a combination of any two of the late wilting genes confers complete resistance (h1, h2, or h1 H3 or h2 H3) [36, 12]. A similar genetic system based on two [26]. or three [48]. independent genes were found to confer resistance to race 2. [73]. demonstrate that resistance to *race 2* is governed by a single recessive gene, whereas resistance to *race 3* has been found to be monogenic [25]. Others studies showed that resistance to race 4 is recessive and digenic [89], while race 5 resistance is controlled by a single gene [87]. For *Foc*, race 0 resistance is controlled by two genes which segregate independently: *foc* 01 present in accession JG-62, and foc 02 present in lines CA-1938, CA-2139 and WR-315 [67]. Nevertheless, the genetic basis of resistance to races 1B/C and 6 is still unknown. Genetic resistance to *Foc*races was reviewed by [73]. some genes resistant to races 0, 1, 2, 3, 4 and 5(foc02,foc-1, foc-2, foc-3, foc-4 and foc-5) are located on LG2 of the chickpea map [55]. However, one of the two resistance genes for race 0 (foc01) was found in LG5 [16].

Breeding methods against fusarium wilt

Breedingeffortshavecontributedtosignificantlyreducethefusarium wilteffectonthechickpeacrop. Three main steps or components are generally involved in any chickpea breeding program: 1. genetic variation, which is the base of the breeding program, 2. selection within that variation for desirable plant types and disease resistance and 3. Evaluation of the selected lines for commercial production. Since chickpea is self-pollinated, the development of pure-line cultivars requires fixing genes in breeding lines. Mass or pure-line selection from landraces was the simplest method initially employed. Later, crossing programs and various modifications of pedigree and bulk methods were used in handling segregating generations. Single crosses have been adopted in most chickpea breeding programs, especially in intraspecific hybridization between Desi and Kabuli-types having different genetic backgrounds [10]. Desi parents have been used to transfer important genes to Fusarium wilt resistance into Kabuli-type breeding programs, conversely, Kabuli parents have been used as a source to improve large seed size and seed quality in Desi breeding programs [23]. Efforts have been made to use interspecific crosses for enhancing genetic variability and in trogressing useful genes from wild *cicer* spp. into the cultivated species. Resistance to *Foc* races has been identified mainly in Desi germplasm as well as in wild *cicer* spp. Indeed, combined resistance against race 0 and 5 was identified in accessions of *C. bijigum, C.cuneatum, C. judaicum*, whereas accessions of *C. canariense* and *C. chorassanicum*were resistant to race 0 but susceptible to 5. Moreover, accessions tested of *C. pinnatifidum* were susceptible to race 5, but some were resistant to race 0 [42].

There is a need to enhance precision and efficiency of selections in the segregating generations for higher and rapid genetic gains. Effective wilt sick-plot for the field and hot spot location screening, greenhouse and laboratory procedures for resistance screening have been developed for successful breeding programs [42]. Nene *et al.* [60] developed the field technique of screening for a "wilt-sick plot". The technique is outlined as follows: (i) Select a plot of adequate size cropped in the previous year with chickpea and isolated from other chickpea fields to avoid spread of the fungus inoculum from this plot to others; (ii) Collect wilted plants from other fields, chop into small pieces, and incorporate uniformly in the surface soil of the plot; (iii) Plant a highly susceptible cultivar in this plot. This will help increase the level of the inoculum to make the soil "sick". Such plots are available at many research stations around the world and are widely used for screening for fusarium wilt resistance. The methodology consists of planting a highly susceptible check every two test entries with two replications.

The percentage of wilt incidence of each entry is calculated, and the level of resistance and susceptibility of each test entry is determined by using the appropriate rating scale. Screening of over 13,500 Desi germplasm accessions for resistance to fusarium wilt using wilt-sick plots at ICRISAT (International Crops Research Institute for the Semi-Arid Tropics) allowed the identification of 165 sources of resistance [Haware *et al.*, [30]]. Moreover, among 5174 Kabuli germplasm accessions screened for Fusarium wilt resistance at ICARDA (International Centre for Agricultural Research in the Dry Areas), 110 lines were identified as resistant. Some Kabuli accessions with high resistance to fusarium wilt have been identified: 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), and line CA-2954 (races 0 and 5) [67]. Moreover, several cultivars with stable resistance to fusarium wilt have been identified in many countries as USA, India, Israel, Mexico and Tunisia, including cvs. ICCV-2 through ICCV-6 (race 1A) [48]. Amdoum 1, Béja1, Nour(race0), and Gavilan, Surutato-77, Sonora-80, Tubutama, UC-15andUC-27. For these six later cultivars, resistance introgressed from Desi line L- 1186 is effective against races 0, 1A, 1B/C, 5,

and 6 and it has been operative in California, Mexico, and Spain [39].

Several pathogenic *Fusarium*spp. Such as *F. redolens* [35] may cause similar symptoms in chickpea as *Foc.* These features emphasize the importance of assuring accurate identification and discrimination between pathogenic *Fusarium* spp. that show similar symptoms in a determined geographical area for which suitable molecular protocols are available. This approach would facilitate a better understanding of the etiology and epidemiology of the disease caused by the different *Fusarium* spp. and accelerate the development of new resistant chickpea cultivars. The development of efficient molecular markers in plant breeding will continue to be a very dynamic process in the coming years. Indeed, identifying molecular markers closely linked to a gene/QTL controlling a trait is a prerequisite for applying marker-assisted selection (MAS) in a conventional breeding program. Therefore, incorporating MAS for wilt resistance into breeding programs is a valuable tool that would greatly improve the efficiency of selection.

Conclusion

Above the finding of numerous reviewed that should be conclude the management of fusarium wilt pathogen is very contrast yet. These, feature emphasized the importance of assuring accurate identification and discrimination between pathogenic *Fusarium* spp. that show similar symptoms in a determined geographical area for which suitable molecular protocol sare available. This approach would facilitate a better understanding of the etiology and epidemiology of the disease caused by the different *Fusarium* spp. and accelerate the development of new resistant chickpea cultivars, developed new molecules, because old molecules *i.e.*, non-conventional and conventional chemicals are not ecologically to be greatest for the well crop ecosystem as well as environment. Several alternative option should be available such as used of biological agent for soil treatment, used of several plant extract for seed treatment and soil solarization, soil heat sterilization are effectively manage the fusarium wilt but these approaches are not better for the long term because *Foc* is very mutable pathogen, rapidly changes in self (strain). The development of efficient molecular markers in plant breeding will continue to be a very dynamic process in the coming years. Indeed, identifying molecular markers closely linked to a gene/QTL controlling a trait is a prerequisite for applying marker-assisted selection (MAS) in a conventional breeding program and find out resistance sources. Therefore, incorporating MAS for wilt resistance into breeding programs is a valuable tool that would greatly improve the efficiency of selection. So, now days only molecular techniques isone of the greatest potentials which helps in identify resistant sources as R gene and developed new resistant cultivars.

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