Fusarium wilt's pathogenic studies and disease management: A review

Ch. Muhammad Rafiq[1], Muhammad Tariq Mahmood[2*], Mushtaq Ahmad[2], Imtiaz Ali[3], Sadia Kaukab[1], Muhammad Shafiq[1], Muhammad Saleem[1], Umer Iqbal[4]

[1]Pulses Research Institute, Ayub Agriculture Research Institute (AARI), Faisalabad, Pakistan
[2]Gram Breeding Research Station, Kallurkot, Pakistan
[3]Regional Agricultural Research Institute, Bahawalpur, Pakistan
[4]Crop diseases research institute, national agricultural research centre, Pakistan

Corresponding author: Muhammad Tariq Mahmood
E-mail: taqaisrani@gmail.com
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ABSTRACT. Fusarium wilt caused by *Fusarium oxysporum* f. sp. ciceris is one of the most important fungal diseases of chickpea limiting its productivity in almost all chickpea growing countries across the world. *F. oxysporum* is soil borne and root inhabiting in nature which may survives in soil for longer period (up to six years). Pathogen undergoes asexual reproduction by producing three types of spores; micro-conidia, macro-conidia and chlamydospores. Chlamydospores serve as primary inoculum for the disease occurrence. Prevalence and severity of disease are driven by inoculum populations and susceptibility of cultivar. *F. oxysporum* has high pathogenic variability having eight distinguished races and two known pathotypes. General symptoms of chickpea wilt are wilting, drooping, discoloration, yellowing, browning of xylem vessel and eventual collapse of whole plant. Delayed planting, seed dressing with fungicides, deep ploughing, use of bio-control agents and sowing of certified wilt resistant cultivars have been found helpful to minimize the disease prevalence. Through this review we have attempted to summarize the general aspects of fusarium wilt with an emphasis to pathogenicity and integrated disease management strategies.

Keywords: Chickpea Wilt; Disease Management; *Fusarium oxysporum*

INTRODUCTION

Chickpea (*Cicer arientinum* L.) is an important annual pulse legume crop. It is a member of family Fabaceae, self-pollinated in nature with diploid chromosome number 2n = 16. It is one of the primitive crops of the world originated in Turkish Kurdistan about 8000-9000 years ago from its wild ancestor *C. reticulatus* (Ladizinsky and Adler, 1976; Tekeoglu et al., 2000; and Sunkad et al., 2019). It is being extensively grown as major winter crop in more than 50 tropical and sub-tropical countries in the world (Gaur et al., 2014; Patil et al., 2017 and Rajeev et al., 2019). Various abiotic and biotic factors adversely affect the chickpea productivity around the globe (Tarafdar et al., 2018 and Caballo et al., 2019). Among the biotic stresses, fusarium wilt has been found a devastating fungal disease posing adverse effects on chickpea productivity in most of the chickpea growing countries of world. Chickpea crop in Indian sub-continent, Mediterranean Basin, California and several other countries of the world face severe disease infections causing a significant yield loss (Haware, 1993; Chaudhry et al., 2007 and Cunnington et al., 2007). Fusarium wilt has become a major threat for chickpea productivity and the yield losses range from10-90% depending upon the severity of disease (Haware et al., 1989; Kumar et al., 2012; Sunkad et al., 2019).

Chickpea wilt was reported in Indian Sub-continent for the first time by Buttler in 1918 while, its etiology was further determined by Padwick in 1940 (Padwick, 1940; Kumar et al., 2012; Jimenez-Díaz et al., 2015). Haware and Nene (1980) identified four different physiological races of *F. oxysporum* (1, 2, 3 and 4). Later exploration of pathogenic variability confirmed its eight races 0, 1A, 1B, 2, 3, 4, 5 and 6 (Chobe et al., 2016). Wilt pathogen is root inhabiting, soil borne fungus species which survives in soil between the seasons (Haware et al., 1989 and Patil et al., 2017). Pathogen may also be transmitted through infected seed, farm implements and rain splashes (Gupta, 1991). Haware et al., 1992 reported that the *F. oxysporum* is soil borne and seed borne as well which can survive for 5-6 years in soil. The pathogen spores grow well on 25-30 C^0 temperature and 5-6.5 pH soils (Chauhan, 1963; Agrios, 2005 and Singh et al., 2007).

Disease can occur in almost all stages of plant growth and diseased plants may be found in groups or patches across the fields (Nene and Reddy, 1987; Gomez, 2004; Jimenez-Díaz et al., 2015). Initially disease symptoms appear on upper leaves, flowers and twigs. Drooping, loss of turgidity and discoloration of leaves followed by yellowing and drying of affected plant parts may occur while in later stages it results in collapse of whole plant (Luthra et al., 1943; Murumkar and Chavan, 1985; Gupta, 1991). Fungus enters tap roots, then into the vascular bundle resulting in histological distortion of vascular tissue and brownish discoloration of xylem vessel appears. In a short time, lateral roots wither off, drooping can be observed in upper plant parts and eventually whole plant is collapsed. Wilting may also be partial; affecting some plant parts. Seeds in wilted plants are inferior in quality usually smaller in size, discolored and wrinkled. In early wilt the symptoms may appear in 25-30 days of sowing while in late wilt symptoms usually appear during flowering to podding stage (Trapero-Casas and Jimenez-Díaz, 1985).

LITERATURE REVIEW

Chickpea wilt is the most serious threat to chickpea crop especially in Pakistan, India, Turkey Mexico, Spain and Ethiopia (Nene et al., 1989; Gomez, 2004 and Patil et al., 2017). Chickpea wilt, despite being considered a disastrous and widespread chickpea disease a little headway for its genetics, early diagnosis and management has been made so far. This review is an attempt to generate updated information on fusarium wilt with an emphasis to pathogenicity and integrated disease management strategies.

Disease symptoms

Wilting, necrosis, damping off, withering, drooping, dull-green discoloration, yellowing, loss of turgidity in leaves, browning of vascular system and eventual collapse of whole plant are major symptoms of chickpea wilt (Westerlund et al., 1974; Jalali and Chand, 1992; Jimenez-Díaz et al., 2015). Generally, wilted plants may be spread across the fields or grouped in patches (Figure 1). Pathogen penetrates the host plant through root apices or wounds

and multiplies to plug the vascular bundles. Initially symptoms appear on upper leaves, flowers and twigs which show discoloration, desiccation, drooping of leaflets, rachis and petioles. After few days whole plants wither and collapse (Luthra et al., 1943; Murumkar and Chavan, 1985; Gupta, 1991). Early wilt symptoms observed after 20-30 days of sowing is termed as early wilt and during flowering to podding stage is known as late wilt. Early wilt causes 77-90% yield decline and the late wilt cause 24-65% (Gupta, 1991; Jimenez-Díaz et al., 2015). If few plant parts are affected by wilt, disease is called partial wilt.

Figure 1. Fusarium wilts in chickpea field.

Dark brown discoloration appears on roots of affected plants and can be observed by splitting the roots cross-sectional or vertically (Haware, 1990; Hanif et al., 1999; Jalali and Chand, 1992) (Figure 2). Histological distortion in vascular bundle occurs which results in retarded vascular flow of water as well as nutrients to aerial plant parts which lead to development of disease symptoms and eventual death of plants. Affected seeds are lighter in weight, inferior in quality usually discolored and wrinkled (Haware and Nene, 1980; Navas-Cortes et al., 2000; Jimenez-Díaz et al., 2015).

Figure 2. Cross section of stem; Discoloration of xylem vessel in chickpea.

Disease cycle

Asexual reproduction occurs in *Fusarium oxysporum*. It forms three types of spores; micro-conidia, macro-conidia and chlamydospores. Micro-conidia are ellipsoidal in shape with 0-1 septa and macro-conidia are cylindrical, thin walled, slightly curved with 3-4 septa. Pathogen survives in soil or infected seed between the seasons in the form of free chlamydospores or embedded in plant cells or tissues (Jimenez-Díaz et al., 1989; Agrios, 2005). Disease becomes more epidemic under favorable environmental conditions. Optimum temperature for mycelium growth is 25-30 C_0 and 5-6.5 PH soils (Agrios, 2005 and Singh et al., 2007).

Chlamydospores act as primary inoculum for the disease incidence. They may be found in chains, in pairs and single as well. Chlamydospores are produced in old mycelia and infected plant residues. Initially the fungus attacks the root apices or wounded root parts and penetrates to epidermis, cortex and finds its way to vascular bundle. Fungal growth in xylem vessel develops dark brown discoloration in vascular bundles. These pathogens colonize in vascular bundle and also penetrate to adjacent plant tissues. Colonization in vascular bundle forms dense gels and histological distortion of xylem occurs. These gels and occlusions in vascular tissue plug the normal flow of nutrients and water in xylem vessel. Wilting, drooping, yellowing of leaves occurs and eventually whole plant is collapsed in few days. This pathogen remains in soil, roots, seed and infected plant residues as chlamydospores and mycelium for more than 6 years which serve as inoculum for development of disease in next season (Jalali and Chand, 1992; Singh et al., 2007; Jimenez-Díaz et al., 2015) (Figure 3).

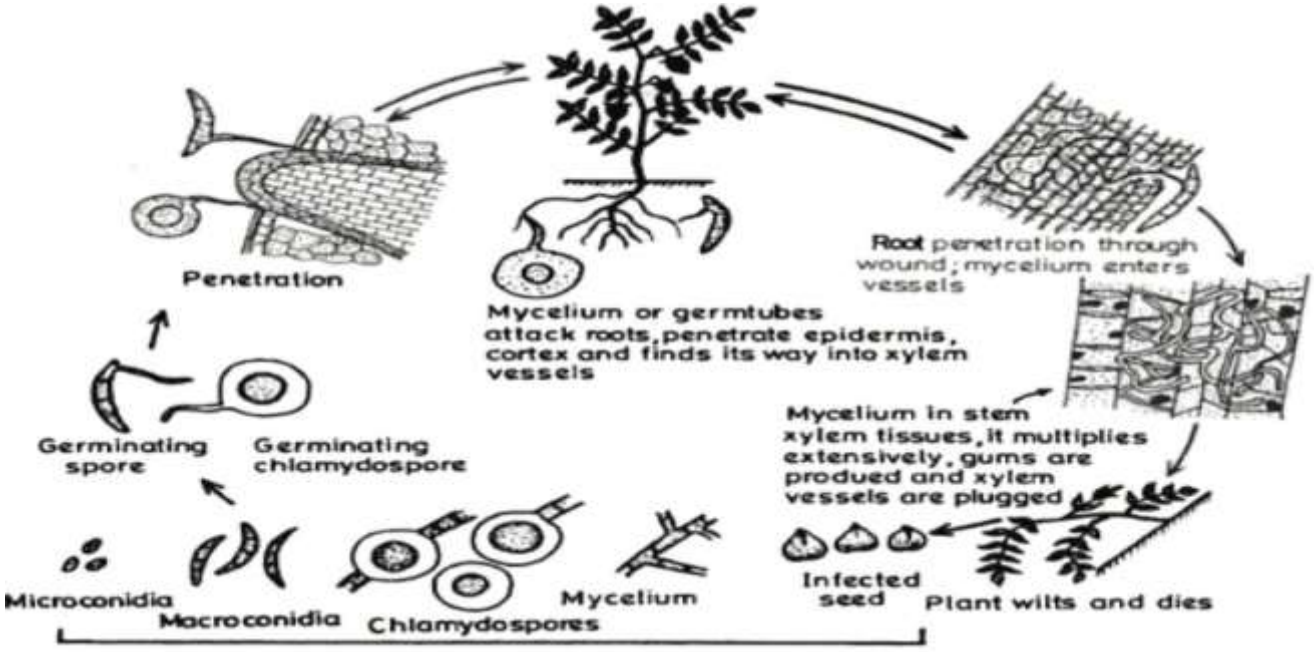

Figure 3. Disease cycle: Slight modification of previous illustration by Jalali and Chand, 1992.

Pathogenic variability

F. oxysporum has eight races and two known pathotypes. Both pathotypes have distinguished characteristics and mode of action. These pathotypes cause distinct wilting and yellowing syndromes (Patil et al., 2017). Wilting syndrome is faster in action causing early death of plants. Yellowing syndrome takes comparatively more time causing late death of plants (Chobe et al., 2016). In addition, these pathotypes differ genetically and can be differentiated by employing DNA finger printing techniques and RAPD markers. Jimenez-Gasco et al., 2001

concluded that RAPD marker and DNA finger printing placed the isolates from both pathotypes in two distinguished groups.

Haware and Nene (1980) identified four different physiological races of *F. oxysporum* (1, 2, 3 and 4) but later on eight pathogenic races of *Fusarium oxysporum* are 0, 1A, 1B/C, 2, 3, 4, 5 and 6 were identified and characterized (Caballo et al., 2019). These races express distinguished characteristics and symptoms by their differential pathogenic reactions (Haware and Nene, 1982; Jimenez-Díaz et al., 1989). These races not only differ in pathotypes, they also vary in geographic distribution as well. Races 6 and 1A belong to wilting pathotypes while 0, 1B/C are characterized as yellowing pathotypes. Haware and Nene (1982) reported 1A, 2, 3 and 4 in India. 0, 1A, 1B/C, 5 and 6 were reported in Mediterranean region (Jimenez-Gasco et al., 2001). Similarly the races 0, 1B/C, 5 and 6 were reported in northwestern Mexico (Halila and Strange, 1996; Arvayo-Ortiz et al., 2011).

Breeding programs based on development of wilt resistant chickpea genotypes have very few findings and rare selections due to existence of wide genetic diversity of *Fusarium oxysporum* in the form of genetically distinguished races and pathotypes. Pathogen variability has also made the pathotypes more virulent in developing resistance against specific fungicides.

Management of *Fusarium* wilt

Chickpea wilt development is primarily driven by inoculum population therefore disease can be targeted by exclusion, eradication and reduction in efficiency of inoculum. Key disease management options are various agronomic practices, use of chemicals, biological control and use of wilt resistant varieties (Jimenez-Díaz et al., 2015).

Agronomic practices

Chickpea wilt can be managed through various agronomic practices. Following interventions can be helpful in minimizing the disease attack;

- Delayed planting is positively related to disease management and early sown crops are usually more affected (Navas-Cortes et al., 1998).
- Avoidance of dense planting by keeping 15-20 cm plant to plant distance can reduce the disease attack (Haware et al., 1990).
- Deep ploughing is also helpful for reduction of inoculum in soil (Hanif et al., 1999).
- Use of certified pathogen free seed obtained from disease free area are essential for avoidance of wilt disease (Pande et al., 2007)
- Intercropping with other crops has also been found helpful in reduction of disease intensity.
- Crop rotation with non-host crops for consecutive 5-6 years has been found very effective to minimize the pathogen inoculum (Pande et al., 2007).

Use of synthetic chemicals and plant extracts

Various synthetic chemicals have been found effective for management of fusarium wilt. Treatment of seed with Benlate with 30% thiram and 30% benomyl is helpful management of seed borne inoculum (Haware et al., 1978 and Pande et al., 2007). Carbendazim and thiophanate in combination have been very effective to minimize the pathogen populations (Halila and Harrabi, 1990). Topsin-M, Ridomil, Antracol, Captan, Cobox, Sancozeb, Pentacholoro-nitro benzene (PCNB), Trimiltox-forte, Antracol, Daconil and Vitavax have also been reported effective in reduction of disease (Mukhtar, 2007).

Various antifungal substances are produced from plants extracts which inhibit wilt pathogen. Plant extracts are harmless and can easily be prepared by farmers (Okigbo and Nameka, 2005; Mohana and Raveesha, 2007). Seed treatment with Neem oil and garlic leaf extract have been reported very effective for pathogen control (Singh et al.,

2007). Jespers and Ward, 1993 reported that 40% concentration of *Datura metel* L., *Parthenium hysterophorus* L. and *Ocimum sanctum* L were found helpful in controlling the mycelial growth of the pathogens.

Biological control

Biological control is environment friendly and most accepted method used to control *Fusarium oxysporum* (Anjajah et al., 2003). Plant growth promoting rhizobacteria (PGPR) may be effectively exploited as control agents for the wilt pathogen (Schmidt et al., 2004). Pseudomonas and Bacillus (PGPR strains) may be utilized for root inhabiting pathogens (Joseph et al., 2007). Landa et al., 1997 reported that Bacillus and Pseudomonas can be successfully exploited for control of chickpea wilt. These rhizobacteria produce pyrolnintrin, phenazin, phloroglucinol, siderophores that inhibit and suppress the *Fusarium oxysporum* (Fridlender et al., 1993). Wani et al., 2007 and Verma et al., 2014 reported that *Bacillus, Pseudomonas, Trichoderma* and *Burkholderia* show significant inhibition efficient bio-control agents for chickpea wilt. *Trichoderma harzianum* and *Bacillus subtillis* inhibit and suppress the disease by increasing β-1, 3-glucanase enzyme activity and ultimately suppressing the pathogen growth (Anjajah et al., 2003 and Moradi et al., 2012).

Use of wilt resistant chickpea varieties

Host plant resistance is the most suitable and effective measure to avoid the disease incidence. Since the last 3 decades researchers have emphasized to exploit the resistant genetic resources to reduce the chickpea wilt attack. Screening and identification of wilt resistant genetic sources were attempted by large number of researchers (Gurha and Misra, 1983). 13500 chickpea desi accessions were screened at ICRISAT and 165 accessions were identified as wilt resistant (Van Rheenen et al., 1992; Haware et al., 1992). ICARDA (International Center for Agricultural Research in Dry Areas) identified 110 resistant strains in a test of 5174 chickpea kabuli strains (Singh, 1997). Chaudhry et al., 2007 evaluated 196 chickpea accessions and and reported that none of the studied strain was found immune or resistant. Iqbal et al., 2005 screened national and exotic germplasm and identified 14 resistant lines. Likewise, a lot of other researchers screened thousands of chickpea accession and identified resistant genetic sources (Halila and Strange, 1996; Pande et al., 2007 and Jimenez-Díaz et al., 2015).

Various strategies have been developed for screening of wilt resistant genetic resources. A disease rating scale (1-9 scale) suggested by Toker et al., 1999 where, rank 1 is given to Immune (No symptoms on plants), rank 2 is allotted to highly resistant (small tissue depression or spot), rank 3 is given to resistant (elongating spot), rank 4 for moderately resistant (coalescent spot), rank 5 is marked for tolerant (stem girdling), ranked as 6 for moderately susceptible (stem breaking), 7 for susceptible (lesion growth), 8 for highly susceptible (most parts of plant affected) and 9 for highly susceptible (All plants dead).

Nene and Haware, 1980; Thaware et al., 2017 utilized 0-5 scale for screening on a larger scale on the basis of percentage mortality of plants as shown in Table 1 below:

Table 1. 0-5 scale for screening on a larger scale on the basis of percentage mortality of plants.

Grade	Percent mortality	Disease reactions
0	No disease	Highly resistant (HR)
1	0 to 10	Resistant (R)
2	10.1 to 20	Moderately resistant (MR)
3	20.1 to 30	Moderately susceptible (MS)
4	30.1 to 50	Susceptible (S)
5	50 and above	Highly susceptible (HS)

DISCUSSION, CONCLUSION AND FUTURE PROSPECTS

Chickpea wilt is widespread fungal disease in most of the chickpea growing countries around the world. Long survival of pathogen and its existence in diverse pathogenic races favour the development of disease. Prevalence and severity of disease are driven by inoculum density and susceptibility of cultivar. Effective disease management is best attained by quick diagnosis and practicing integrated disease management strategies. Use of quarantine pathogen free seed of wilt resistant cultivars is most successful, practical and cost efficient measure for disease management. Moreover, additional efforts for explorations of genetics and host-pathogen interaction are required to release more high yielding wilt resistant cultivars. Combined use of wilt resistant cultivars and other management measures (delayed planting, seed dressing with fungicides, avoidance from dense planting, deep ploughing and use of bio-control agents) help in reduction of disease incidence. These pre-planting measures require wide extension services, awareness sessions and skilful guidance to the farming community. Sustainable chickpea production through integrated disease management measures is the only possible solution by reducing the disease prevalence.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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