

## FUSARIUM OXYSPORUM F. SP. LYCOPERSICI: BIOLOGY, PATHOGENICITY, AND CONTROL STRATEGIES

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### Abstract

*Fusarium oxysporum* f. sp. *lycopersici* is a devastating fungal pathogen responsible for Fusarium wilt in tomatoes, leading to significant yield losses worldwide. This review provides a comprehensive examination of the biology, pathogenicity, and control strategies for this pathogen. The biology section explores the pathogen's life cycle, genetic diversity, and the mechanisms underlying its ability to infect and colonize tomato plants. In discussing pathogenicity, we delve into the virulence factors, host-pathogen interactions, and environmental conditions that exacerbate disease progression. Control strategies encompass an array of methods including cultural practices, resistant cultivars, chemical treatments, and biological control agents. Advances in molecular techniques and genetic engineering offer new avenues for developing disease-resistant tomato varieties and effective biocontrol methods. Integrated disease management approaches, combining multiple strategies, are emphasized as the most sustainable solution for managing Fusarium wilt. This review highlights the need for ongoing research and collaboration among scientists, breeders, and growers to develop and implement effective control measures against *F. oxysporum* f. sp. *lycopersici*.

### Introduction

#### The Pathogen: *Fusarium oxysporum* f. sp. *lycopersici*

The causal agent of Fusarium wilt in tomato is *Fusarium oxysporum* f. sp. *lycopersici* (Sacc) Snyder & Hans (Chambers and Corden, 1963). Henceforth the pathogen is referred to as *Fol*. This devastating pathogen belongs to Deuteromycetes and is soilborne with high level of host specificity. There are more than 120 described formae speciales and races within the species. Together they cause diseases on a wide range of agricultural crops (Correll, 1991; Agrios, 2005). The mycelium of the fungus is colourless at first but with age it becomes cream coloured, pale pink or somewhat purplish. *Fusarium oxysporum* produces three kinds of asexual spores: microconidia, macroconidia and chlamydospores. Microconidia, which have one or two cells, are the most frequently and abundantly produced spores under all conditions, even inside the vessels of infected host plants. Macroconidia are the typical “Fusarium” spores; they are three to five celled, have gradually pointed and curved ends, and appear in sporodochia-like groups on the surface of plants killed by the pathogen. Chlamydospores are one- or two celled, thick-walled, round spores produced within or terminally on older mycelium or in macroconidia. All three

types of spores are produced in cultures of the fungus and probably in the soil, although only chlamydospores can survive in the soil for long.

## Symptomology

Fusarium wilt is a warm climate disease caused by the fungus *Fusarium oxysporum* f. sp. *lycopersici*. *F. oxysporum* is a phytopathogenic fungus causing vascular wilt disease on tomato, known as fusariosis (Di Pietro et al., 2003). The first indication of disease in small plants is a drooping and wilting of lower leaves with a loss of green color followed by wilting and death of the plant (Arjunan, 2005). At first the disease appears as slight vein clearing on the outer, younger leaflets and subsequently, the older leaves show epinasty caused by drooping of the petioles (Agrios, 2005). Plants infected at the seedling stage usually wilt and die soon after appearance of the first symptoms. More commonly, however, in older plants, vein clearing and leaf epinasty are followed by stunting of the plants, yellowing of the lower leaves, occasional formation of adventitious roots, wilting of leaves and young stems, defoliation, marginal necrosis of the remaining leaves, and finally death of the plant (Agrios, 2005).

Woltz and Jones (1981) reported that the wilt appeared when plants were around 45 days old and wilted leaves were mildly chlorotic and later turned brown. Tomato plants exhibited symptoms of swelling at the crown with some orange and brown lesions, root rot and interior decay of the lower stem. The lesions progressed towards for 10 – 15 cm and where the lower parts of the stem withered and fungal sporulation was visible. Such plants were wilted within 2 – 3 weeks. These symptoms were typical of *F. oxysporum* f. sp. *radicis lycopersici* and different from the vascular wilt caused by *F. oxysporum* f. sp. *lycopersici* (Can et al., 2004).

The first symptom of fusarium wilt in gardens and fields is usually the golden yellowing of a single leaflet or shoot, or a slight wilting and drooping of the lower leaves on a single stem. Leaves or whole branches will turn yellow, then brown and die still attached to the plant described as a yellow-flagging appearance (Alexander and Tucker, 1945). Leaves of only one side of the stem turn golden yellow at first, sometimes half of a leaf or branch will be affected, with the other half seemingly unaffected.

The fungus can be observed as brown discoloration in the vascular tissue of affected branches (Agrios, 2005). The stem of wilted plants shows no soft decay, but when cut lengthwise, the woody part shows a dark brown discoloration of the water-conducting vessels (Cai et al., 2003). As the fungus develops inside the stem, plants show progressive yellowing, wilting, and withering starting generally with the lowermost foliage. Yellowed and wilted leaflets drop early. Pernezny et al., (2003) reported that the wilted plants shows darkening of stem base and fruits from infected plant, wrinkle and rot.

Fruit may occasionally become infected and then it rots and drops off without becoming spotted. Roots also become infected; after an initial period of stunting, the smaller side roots rot.

## Biology of *Fusarium oxysporum*

### Taxonomy

Based on the structure in or on which conidiogenous hyphae are borne, *Fusarium* spp. are classified under the Hyphomycetidae subclass of the Deuteromycetes. *Fusarium oxysporum*, as emended by Snyder & Hansen (1940), comprises all the species, varieties and forms recognised by Wollenweber & Reinking (1935) within an intragenic grouping called section *Elegans*. Booth (1971) described *F. oxysporum* as a cosmopolitan soil-borne filamentous fungus. It is an anamorphic species that includes numerous plant pathogenic strains causing wilt diseases of a broad range of agricultural and ornamental host plant species (Appel & Gordon 1996).

*Fusarium oxysporum* produces three types of asexual spores: microconidia, macroconidia and chlamydospores (Nelson et al. 1983). Conidia are produced on monophialides and in sporodochia, and are scattered loosely over the surface of a mycelium (Griffin 1994). Microconidia are predominantly uninucleate and germinate poorly and variably, with germination efficiency ranging from 1 - 20% (Ebbole & Sachs 1990). The macroconidia are produced abundantly, are multinucleate, and germinate rapidly, thereby reproducing the fungus efficiently. Chlamydospores are viable, asexually produced accessory spores resulting from the structural modification of a vegetative hyphal segment(s) or conidial cell possessing a thick wall, mainly consisting of newly synthesized cell wall material (Schippers & van Eck 1981). Its function is primarily survival in soil. Morphological characterization of *F. oxysporum* is based on the shape of macroconidia, the structure of microconidiophores, and the formation and disposition of chlamydospores (Beckman 1987). Asexual reproduction in *F. oxysporum* is accomplished by macroconidia and microconidia, while a sexual state of the fungus has never been observed (Booth 1971).

### Life cycle

The life cycle of *F. oxysporum* commences with a saprophytic phase when the fungus survives in soil as chlamydospores (Beckman & Roberts 1995). Chlamydospores remain dormant and immobile in the remains of decayed plant tissue until stimulated to germinate by utilising nutrients that are released from extending roots of a variety of plants (Stover 1962 a,b, Beckman & Roberts 1995). Following germination, a thallus is produced from which conidia form in 6-8 hours, and chlamydospores in 2-3 days if conditions are favourable. Invasion of the roots is followed by the penetration of the epidermal cells of a host or a non-host (Beckman & Roberts 1995) and the development of a systemic vascular disease in host plants (Stover 1970). In the advanced stages of the disease, the fungus grows out of the vascular system into adjacent parenchyma cells,

producing vast quantities of conidia and chlamydospores. The pathogen survives in infected plant debris in the soil as mycelium and in all its spore forms, but most commonly as chlamydospores in the cooler temperate regions (Agrios 1997).

### Formation and germination of spores

Chlamydospore formation in pathogenic *Fusarium* species commonly takes place in hyphae in the infected and decaying host tissue (Nash et al. 1961, Christou & Snyder 1962). They may also be formed abundantly from macroconidia that originate from sporodochia on lesions at the soil level (Nash et al. 1961, Christou & Snyder 1962). Schippers & van Eck (1981) proposed that chlamydospore formation depends on the nutrient status of the inoculum. Under field conditions, fungal inoculum may be subjected to much lower nutrient levels when compared to the 'well-fed' macroconidia produced on rich agar media. Once carbohydrates are released from decaying plant tissue or from roots, chlamydospore germination is stimulated (Schippers & van Eck 1981). Qureshi & Page (1970) further suggested that chlamydospores are formed with the addition of organic or inorganic carbon sources. From the close resemblance of chlamydospore formation in weak salt solutions to that on soil and in soil extracts, Hsu & Lockwood (1973) concluded that an environment deficient in energy, but with an appropriate weak salt solution, may be required for chlamydospore formation. Chlamydospore germination in nature appears to be dependent on exogenous energy sources (e.g. carbon and nitrogen) (Cook & Schroth 1965, Griffin 1969). Spore density is the single most important factor affecting the nutritional requirements for germination of conidia and chlamydospores in pure culture (Griffin 1981). Exogenous carbon and nitrogen were required for high or complete chlamydospore germination at high spore densities in axenic culture (but not at low spore density) and in soil (Cook & Schroth 1965, Griffin 1969, Griffin 1970). At high conidial densities macroconidia do not germinate, but every conidium is converted into a chlamydospore. At low conidial densities, the conidia germinate but do not convert into chlamydospores (Schneider & Seaman 1974). According to Griffin (1970, 1981), the inability of macroconidia to germinate at high conidial densities resulted from the presence of a self-inhibitor. Self-inhibitors are substances accumulating in growth medium suppressing germination of macroconidia at higher spore densities in the soil (Robinson & Park 1966, Griffin 1969, Robinson & Garrett 1969, Griffin 1970).

**Infection:** The process of vascular infection by *F. oxysporum* is complex and requires a series of highly regulated processes:

**Adhesion:** Fungal infection commences when infection hyphae adhere to the host root surface (Bishop & Cooper 1983a). Adhesion of fungi to the host surface is not a specific process, as they can adhere to the surface of both host and non-hosts (Vidhyasekaran 1997). Site-specific binding may be important in anchoring the propagules at the root surface, after which other

processes required for colonization can proceed (Recorbet & Alabouvette 1997). **Penetration:** Penetration is likely to be 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 (Mengden et al. 1996). The means whereby wilt pathogens penetrate roots may differ, but there are two distinct types. Some pathogenic forms penetrate roots directly, whereas others must enter indirectly through wounds (Lucas 1998). The most common sites of direct penetration are located at or near the root tip of both taproots and lateral roots (Lucas 1998). The pathogen enters the apical region of the root where the endodermis is not fully differentiated and fungi are able to grow through and reach the developing protoxylem. *Fusarium oxysporum* has been found to penetrate the root cap and zone of elongation intercellularly in the root of banana (Brandes 1919), china aster (Ullstrup 1937), radish and cabbage (Smith & Walker 1930), while *F. oxysporum* f. sp. *dianthi* probably enters carnation roots through the zone of elongation (Pennypacker & Nelson 1972). The muskmelon wilt organism penetrated a susceptible host variety between cells in the region of elongation (Reid 1958). Although mechanical wounding increases infection it is not essential for lateral root infection (Stover 1962a).

**Colonization:** During colonization, the mycelium advances intercellularly through the root cortex until it reaches the xylem vessels and enters them through the pits (Bishop & Cooper 1983b). The fungus then remains exclusively within the xylem vessels, using them to colonize the host (Bishop & Cooper 1983b). Fungal colonization of the host's vascular system is often rapid and frequently facilitated by the formation of microconidia within the xylem vessel elements (Beckman et al. 1961) that are detached and carried upward in the sap stream (Bishop & Cooper 1983b). Once the perforation plates stop the spores, they eventually germinate and germ tubes penetrate the perforation plates. Hyphae and subsequently conidiophores and conidia are formed (Beckman et al. 1961, Beckman et al. 1962).

### Development of Disease

Agrios (2005) describe the pathogen as a soil inhabitant which survives between the crop on infected plant debris and as mycelium and in all its spore forms in soil. The pathogen is disseminated over short distance by means of contaminated farm equipments and water. However, if once the field is infested with *Fusarium*, it remains so indefinitely. Pathogen penetrates host root with germ tube and mycelium directly or enters through wounds. The mycelium moves forward intercellularly through root cortex and when it reaches the xylem vessels it enters through the pits. Mycelium travels through vessels mostly upward to the stem and crown. In vessels, mycelium produces branches and microconidia, which are finally detached and carried upward in sap system. Later, microconidia starts germinating at the point where movement is stopped, the mycelium penetrates the upper wall of the vessel, and more



microconidia are produced in the next vessel. The mycelium also advances laterally into the adjacent vessels, penetrating them through the pits.

A combination of the processes namely vessel clogging by mycelium, spores, gels, gums, and tyloses and crushing of the vessels by proliferating adjacent parenchyma cells, is responsible for the breakdown of the water economy of the infected plant. When the leaves transpire more water than the roots and stem can transport to them, the stomata close and the leaves wilt and finally die, followed by death of the rest of the plant. The fungus then invades all tissues of the plant extensively, reaches the surface of the dead plant, and there sporulates profusely. The spores may be disseminated to new plants or areas by wind, water, and so on (Agrios, 2005).

### Management practices adapted for Fusarium Wilt

The pathogen occurs throughout most tomato-growing worldwide causing a vascular wilt that can severely affect the crop (Moretti et al. 2008), and the disease is considered as one of the main soil-borne systemic diseases (Schwarz and Grosch 2003). It causes significant losses in tomato production both in greenhouse and field – grown tomatoes (Nusret ozbay and Steven 2004). Several disease management strategies are available e.g. cultural technique, biological control, resistant cultivars, crop rotation and chemical control.

The most effective method of control of diseases caused by *F. oxysporum* is the use of resistant cultivars, when these are available (Fravel et al., 2003). However the onset of new races of the pathogen which overcome the host resistance, by spontaneous random mutation or parasexuality, is possible (Beckman, 1987; Cai et al., 2003; Sidhu and Webster, 1979). Some systemic fungicides may control the disease, but only partially (Amini and Sidovich, 2010; Gullino et al., 2002; Song et al., 2004). Soil fumigation is effective in eradicating the resident inoculum (Miguel et al., 2004) but is expensive and poses environmental and safety concerns (Fravel et al., 2003; Scott et al., 2012). Long-term crop rotation may achieve the same result (Curl, 1963; Hopkins and Elmstrom, 1984; Scott et al., 2012), but the method is economically impracticable where agriculture is intensive and highly specialized (Miguel et al., 2004). For the above reasons, alternative options for management of pathogenic *F. oxysporum* are needed.

### Chemical control

Control of diseases in tomato cultivation through the use of fungicides and soil fumigants has helped maintain high yields. Some of the commercial chemical fungicides used with higher inhibition rates to eradicate the spread of the microorganism *F. oxysporum* f. sp. *lycopersici* in tomato crops are: carbendazim, propiconazole, benomyl, prochloraz, fuberidazole, thiabendazole and thiophanate which belong to the benzimidazole family and are categorized as systematic fungicides (Bawa, 2016). Myclobutanil, epoxiconazole, difenoconazole, triadimefon, tebuconazole belonging to the family of triazoles and categorized also as systematic fungicides,

among other types of fungicides such as methoxyacrylates and ethyl phosphonates (Khan et al., 2012).

Chemical control of tomato fusarium wilt in vitro and glasshouse was examined repeatedly. Fungicides including benomyl, captafol, imazalil, thiram, and prochloraz Mn, provided inconsistent control of Fusarium crown and root rot on tomatoes, leaving problematic residues in fruit tissues (Marois and Mitchell, 1981; Jarvis, 1988, 1992; Hartman and Fletcher, 1991). Also application of methyl bromide and chloropicrin reduced Fusarium crown and root rot of tomato (Mc Govern and Vavrina, 1998). Mandal and Sinha (1992) found out that such compounds as copper chloride, ferric chloride, manganese sulfate, controlled *F. oxysporum* f. sp. *lycopersici* by inducing resistance in susceptible tomato plants. El-Shami M.A. et al. (1993) reported that Vitavax (carboxin)-thiuram or Vitavax-captan, applied as fungicidal seed treatment, were effective in controlling Fusarium wilt disease so that, Vitavax-captan gave better disease control than Vitavaxthiuram. The effect of mixture of metamidoxime and copper oxychloride on *F. oxysporum* f. sp. *lycopersici* was tested in vitro, and the results showed that these fungicides had a strong synergistic effect and could be used as a basis for a new product to control tomato diseases (Nedelcu and Alexandri, 1995). In addition, it was demonstrated that Thiram and Topsin-M were the most effective at 800 mg/g soil, reducing populations of *F. oxysporum* f. sp. *lycopersici* by 83.4% after 45 days (Dwivedai et al., 1995).

Amini and Sidovich (2010) had evaluated six fungicides; carbendazim, benomyl, fludioxonil, prochloraz, azoxystrobin and bromuconazole for their efficacy against *Fusarium oxysporum* f. sp. *lycopersici*. Seven different concentration i.e. 0.0001, 0.001, 0.01, 0.1, 1, 10, 100 µg/ml were used for estimation of their activities against the pathogen through mycelial growth inhibition on growth media. Four concentrations i.e. 0.1, 1.0, 10 and 100 µg/ml were tested against Fusarium wilt on tomato plants in glasshouse. The result of glasshouse tests revealed a different degree of efficacy of all tested fungicides in reducing disease infestation. Prochloraz and bromuconazole were the most effective fungicides against the pathogen both in vitro and in vivo, followed by benomyl and carbendazim.

Allen et al. (2004) revealed that benomyl at 10 µg/ml (a.s.) completely inhibited fungal growth of *F. solani*, *F. oxysporum* and *F. proliferatum*. Etebarian (1992) reported that, iprodione + carbendazim, benomyl and carbendazim totally inhibited fungal growth at the concentrations of 10 and 100 ppm, after 10 days. Also results indicated that prochloraz and carbendazim were the most effective fungicides in inhibiting mycelial growth of *F. oxysporum* f. sp. *lycopersici* (Song et al. 2004; Weitang et al. 2004).

### Natural products

Natural products present many advantages in term of sustainability, mode of action and toxicity compared to chemical pesticides (Nega, 2014). They decompose more quickly in the environment and are generally less toxic towards non-target species (Thakore, 2006). Plant

extracts and essential oils may have an important role to play in controlling fungi, bacteria, viruses and insect pests (Bowers and Locke, 2000; Isman and Machial, 2006; La Torre et al., 2014; Momin and Nair, 2001; Satish et al., 2007; Varma and Dubey, 2001).

Singha et al. (2011) evaluated the effectiveness of crude chloroform extract of *Piper betle* L. (PbC) against *F. oxysporum* f. sp. *lycopersici*. They observed that 1% (w/w) amendment of the PbC in soil was more efficient in reducing the *Fusarium* population in soil than carbendazim and the combined amendment of carbendazim and PbC. Higher accumulation of total phenolics was observed in the *Fusarium*-infested plants as compared to that of healthy control and PbC-treated plants. Moreover, it was observed that the extract reduced the symptoms and disease development. Electron microscopy studies were also done to observe the *Fusarium* infestation in the vascular bundles and to show the accumulation of total phenolics in the vacuoles of root tissue.

Torre et al. (2016) assessed the use of essential oils in managing fusarium wilt in tomato. Clove oil, thyme oil, rosemary oil and their major components, as well as the two commercial products, Bioxeda and Sporatec were assessed in vitro and in a greenhouse pot trial. In vitro tests consisted of evaluating the development of fungal colonies on agar medium supplemented with the tested products at various concentrations. In addition, the percentage of conidia germination was determined after using the products at different concentrations. In vitro tests showed that almost all the investigated products were able to inhibit mycelial growth and conidial germination. The best results were obtained with clove oil and its major component eugenol. Rosemary oil showed the lowest inhibitory activity. Greenhouse pot experiments indicated that all the tested products were able to reduce *Fusarium* wilt in tomato. The best results were obtained with clove oil, however rosemary oil was also effective in controlling *F. oxysporum* f. sp. *lycopersici*.

Bashir (2015) conducted experiments for the control of *F. oxysporum* f. sp. *lycopersici* using methanolic extracts of radish (*Raphanus sativus* L.). In a laboratory screening bioassay, antifungal activity of different concentrations (1-6%) of methanolic root, leaf and fruit extracts of radish was studied against *F. oxysporum* f. sp. *lycopersici*. Various concentrations of methanolic root, leaf and fruit extracts of radish reduced fungal biomass by 30-39%, 39-52% and 20-35, respectively, over control. Methanolic extracts of leaves and roots were further partitioned using n-hexane, chloroform, ethyl acetate and n-butanol. Antifungal activity of seven concentrations (ranging from 3.125 to 200 mg mL<sup>-1</sup>) of each of the sub-fraction of methanolic extracts was evaluated against the pathogen. Among various sub-fractions of leaf extract, chloroform sub-fraction showed the best antifungal activity causing 52-64% reduction in fungal biomass. Likewise, ethyl acetate and n-butanol sub-fractions of root extract exhibited the best antifungal activity causing 52-96% and 62-95% reduction in fungal biomass over corresponding control treatments, respectively.



Perveen (2015) evaluated the efficacy of aqueous extracts of three plant rhizomes viz., *Curcuma longa* Val., *Allium sativum* L. and *Zingiber officinale* Rosc. @ 20, 40, 60 and 80% concentrations against *F. oxysporum* f. sp. *lycopersici* and *F. solani*. In vitro study revealed that mycelial growth and spore germination was inhibited significantly ( $P < 0.05$ ) with all extracts. *A. sativum* completely reduced the mycelial growth of *F. oxysporum* f. sp. *lycopersici* and *F. solani* at highest concentration. *Z. officinale* showed moderate inhibition ranging from 37.77-48.47% against *F. solani* and 30.33-44.49 % against *F. oxysporum* f. sp. *lycopersici*. *C. longa* exhibited moderate inhibition of *F. oxysporum* f. sp. *lycopersici*, whereas, least inhibition was observed against *F. solani*. Correspondingly, conidial germination of test fungi was almost completely reduced by *A. sativum* extract.

Ben-Jabeur (2015) assessed the potential of thyme essential oil in controlling gray mold and Fusarium wilt and inducing systemic acquired resistance in tomato seedlings grown in hydroponic system. Thyme oil highly reduced 64% of *Botrytis cinerea* colonization on pretreated detached leaves compared to untreated control. Also, it played a significant decrease in wilt incidence and severity especially at 7 days post treatment when it was reduced to 30.76%.

### Biological control

Biocontrol of plant diseases has been promoted as a tool to achieve sustainable and improved crop production systems that are less dependent on agrochemicals. Successful biological control practices usually utilize naturally occurring, antagonistic microorganisms that are able to reduce the activities of plant pathogens. Such antagonists can compete with pathogens for nutrients, inhibit pathogen growth by secreting antibiotics, or reduce pathogen populations through parasitism. In addition, some of these microorganisms induce resistance in host plants, which enhances the plant's ability to defend itself from pathogen attack.

Nonpathogenic strains of *Fusarium* spp. have been evaluated against *F. oxysporum* f. sp. *lycopersici* and suppressed the pathogen under greenhouse and field conditions (Fuchs et al., 1997; Larkin and Fravel, 1999).

Silva and Bettiol (2005) evaluate the efficiency of non-pathogenic *F. oxysporum* isolates (141/3, 233, 233/1, 245, 245/1, 251, 251/2, 251/5, and 257) in controlling vascular wilt caused by *F. oxysporum* f. sp. *lycopersici*, race 2 (isolates C-21A, TO11, and TO245) in tomato. Evaluations were performed 35 days after transplanting, for severity in scale with 1=healthy plant to 6=dead plant or plant showing vessel browning and wilted leaves up to the leader shoot and seedling height. The non-pathogenic *F. oxysporum* isolates were efficient in reducing the severity of the disease and maintaining normal plant development. These results provide evidence of the antagonistic activity of non-pathogenic *F. oxysporum* isolates in controlling vascular wilt caused by *F. oxysporum* f. sp. *lycopersici* race 2 in tomato.

Shishido et al. (2005) evaluated the efficacy of non-pathogenic *Fusarium oxysporum* Fo-B2 for the biological control of *Fusarium* wilt of tomato. Inoculation of Fo-B2 onto tomato roots significantly reduced the severity of disease. Relationships between the recovery of Fo-B2 from hypocotyls and the disease severity indicated that the biocontrol agent was most effective when it colonized vascular tissues intensively. In order to evaluate the pathogenicity and colonization ability of non-pathogenic *Fusarium oxysporum* FJAT-9290 on different plants, RongFeng et al. (2015) monitored the invasion and colonization of FJAT-9290. They have also analyzed the effect of FJAT-9290 on tomato growth and its biocontrol efficiency against tomato *Fusarium* wilt. The FJAT-9290 strain showed control potential against tomato *Fusarium* wilt with a control efficiency of 76.70 % and 69.56 % in the pot experiment and in the field respectively.

Sundaramoorthy and Balabaskar (2013) evaluated the efficacy of *Trichoderma* species to manage *Fusarium* wilt under in vitro and in vivo conditions. Fifteen native *Trichoderma* antagonists were isolated from healthy tomato rhizosphere soil in different geographical regions. Under in vitro conditions, the results revealed that *Trichoderma harzianum* (ANR-1) isolate was found to effectively inhibit the radial mycelial growth of the pathogen (by 53%) when compared to all other isolates. Under greenhouse conditions, the application of *Trichoderma harzianum* (ANR-1) exhibited the least disease incidence (by 15.33%). The antagonistic potential of eight *Trichoderma* isolates were evaluated to assess the efficacy of the native isolates of *Trichoderma* species to inhibit the wilt and promote the growth parameters of tomato seedling and to manage the *Fusarium* wilt disease as affected by *Fusarium oxysporum* f.sp. *lycopersici* (Babychan and Simon, 2017). Under in vitro condition the results revealed that *Trichoderma* isolate MiT-4 was found to be effectively inhibiting the radial mycelial growth of the pathogen by 58.4%. Under greenhouse condition application of *Trichoderma* isolates (MiT-1 and MiT-4) exhibit no wilt incidence in tomato seedlings. Application of *Trichoderma* isolates (MiT-3) on tomato seeds, seedlings showed a significant stimulatory effect on shoot and root height by 7.53 cm and 7.1 cm respectively which is higher than the control.

Ojha and Chatterjee (2011) investigated the effect of a soil application of salicylic acid (SA) and a biocontrol agent, *Trichoderma harzianum* on the induction of phenolic accumulation content and defense enzymes in tomato plants infected with *Fusarium oxysporum* f. sp. *lycopersici*. The combined application of SA (1.5 mM) and *T. harzianum* in *Fusarium* infected tomato plants enhanced the activities of both of the enzymes.

## References

- Bawa I (2016) Management strategies of *Fusarium* wilt disease of tomato incited by disease of tomato incited by *Fusarium oxysporum* f. sp. *lycopersici* (Sacc.): A Review. International Journal of Advanced Academic Research 2(5): 32-42. 6.
- Khan A, Dliferoze A, Malik Zia-Ullah, Shoaib A, Khurshid S (2012) In vitro chemical control of *Fusarium oxysporum* f. sp. *lycopersici*. Mycopath 10(2): 57-61.

Singha, I. M., Kakoty, Y., Unni, B. G., Kalita, M. C., Das, J., Naglot, A., ... & Singh, L. (2011). Control of Fusarium wilt of tomato caused by *Fusarium oxysporum* f. sp. *lycopersici* using leaf extract of *Piper betle* L.: a preliminary study. *World Journal of Microbiology and Biotechnology*, 27(11), 2583.

Fuchs, J.-G., Moënne-Loccoz, Y., and Défago, G. 1997. Nonpathogenic *Fusarium oxysporum* strain F047 induces resistance to *Fusarium* wilt in tomato. *Plant Dis.* 81:492-496

Larkin, R. P., and Fravel, D. R. 1999. Mechanisms of action and doseresponse relationships governing biological control of *Fusarium* wilt of tomato by nonpathogenic *Fusarium* spp. *Phytopathology* 89:1152-1161. 11.

Xiao RongFeng, Liu Bo Zhu YuJing Chen YanPing Su MingXing Yang YingYing (2015) Colonization of non-pathogenic *Fusarium oxysporum* FJAT-9290 and its efficiency against tomato *Fusarium* wilt. *Acta Phytophylacica Sinica*; 2015. 42(2):169-175. 29

Silva, J. C. D., & Bettiol, W. (2005). Potential of non-pathogenic *Fusarium oxysporum* isolates for control of *Fusarium* wilt of tomato. *Fitopatologia Brasileira*, 30(4), 409-412.

Shishido, M., Miwa, C., Usami, T., Amemiya, Y., & Johnson, K. B. (2005). Biological control efficiency of *Fusarium* wilt of tomato by nonpathogenic *Fusarium oxysporum* Fo-B2 in different environments. *Phytopathology*, 95(9), 1072-1080.

D. Fravel, C. Olivain, C. Alabouvette *Fusarium oxysporum* and its biocontrol. *New Phytol.*, 157 (2003), pp. 493-502

C.H. Beckman *The Nature of Wilt Diseases of Plants* APS Press, St. Paul, MN, USA (1987)

G. Cai, L.R. Gale, R.W. Schneider, H.C. Kistler, R.M. Davis, K.S. Elias, E.M. Miyao *Origin of race 3 of *Fusarium oxysporum* f. sp. *lycopersici* at a single site in California.* *Phytopathology*, 93 (2003), pp. 1014-1022

Sldhu, G. S., & Webster, J. M. (1979). A study of heterokaryosis and its influence on virulence in *Fusarium oxysporum lycopersici*. *Canadian Journal of Botany*, 57(5), 548-555.

Amini, J., & Sidovich, D. (2010). The effects of fungicides on *Fusarium oxysporum* f. sp. *lycopersici* associated with *Fusarium* wilt of tomato. *Journal of plant protection research*, 50(2), 172-178.

Gullino, M. L., Minuto, A., Gilardi, G., & Garibaldi, A. (2002). Efficacy of azoxystrobin and other strobilurins against *Fusarium* wilts of carnation, cyclamen and Paris daisy. *Crop Protection*, 21(1), 57-61.

Song, W., Zhou, L., Yang, C., Cao, X., Zhang, L., & Liu, X. (2004). Tomato *Fusarium* wilt and its chemical control strategies in a hydroponic system. *Crop protection*, 23(3), 243-247.

Miguel, A., Maroto, J.V., San Bautista, A., Baixauli, C., Cebolla, V., Pascual, B., Lopez, S. and Guardiola, J.L., 2004. The grafting of triploid watermelon is an advantageous alternative to soil fumigation by methyl bromide for control of *Fusarium* wilt. *Scientia Horticulturae*, 103(1), pp.9-17.

Scott, J., Thomas Gordon, S. Kirkpatrick, S. Koike, M. Matheron, O. Ochoa, M. Truco, and R. Micheltore. "Crop rotation and genetic resistance reduce risk of damage from *Fusarium* wilt in lettuce." *California Agriculture* 66, no. 1 (2012): 20-24.

Curl, E. A. (1963). Control of plant diseases by crop rotation. *The Botanical Review*, 29(4), 413-479.

Hopkins, D. L., & Elmstrom, G. W. (1984). Effect of nonhost crop plants on watermelon *Fusarium* wilt. *Plant disease*, 68(3), 239-241.

Babychan, M., & Simon, S. (2017). Efficacy of *Trichoderma* spp. against *Fusarium oxysporum* f. sp. *lycopersici*.(FOL) infecting pre-and post-seedling of tomato. *Journal of Pharmacognosy and Phytochemistry*, 6(4), 616-619.

Sundaramoorthy, S., & Balabaskar, P. (2013). Biocontrol efficacy of *Trichoderma* spp. against wilt of tomato caused by *Fusarium oxysporum* f. sp. *lycopersici*. *Journal of Applied Biology & Biotechnology*, 1(03), 36-40.

Booth C (1971) *The genus Fusarium*. Eastern Press, London, 237 pp

Appel, D.J. & Gordon, T.R. (1996) Relationships among pathogenic and nonpathogenic isolates of *Fusarium oxysporum* based on the partial sequence of the intergenic spacer region of the ribosomal DNA. *Molecular Plant-Microbe Interactions* 9: 125-138

Nelson, P.E., Toussoun, T.A. & Marassas, W.F.O. (1983) '*Fusarium* species. An illustrated manual for identification.' The Pennsylvania State University Press, USA.

Griffin, D.H. (1994) Introduction to the fungi. In *Fungal physiology* 2nd edition, (D.H. Griffin, eds): 1-20, Wiley-Liss, New York.

Ebbole, D. & Sachs, M.S. (1990) A rapid and simple method for isolation of *Neurospora crassa* homokaryons using microconidia. *Fungal Genetic Newsletter* 37: 17-18.

Schippers, B. & van Eck, W.H. (1981) Formation and survival of chlamydospores in *Fusarium*. In *Fusarium: Diseases, Biology and Taxonomy*, (P.E. Nelson, T.A. Toussoun, R.J. Cook, eds): 250-260. The Pennsylvania State University Press, University Park and London.

Beckman, C.H. (1987) The nature of wilt diseases of plants. American Phytopathological Society, St Paul. MN., USA. 175 pp.

Beckman, C.H. & Roberts, E.M. (1995) On the nature and genetic basis for resistance and tolerance of fungal wilt diseases. *Advances in Botanical Research* 21: 35-77.

Stover, R.H. (1962a) Fusarial wilt (Panama disease) of bananas and other *Musa* species. Commonwealth Mycological Institute. Surrey, UK, 177 pp.

Stover, R.H. (1962b) Studies on *Fusarium* wilt of bananas. IX. Competitive saprophytic ability of *F. oxysporum* f. sp. *cubense*. *Canadian Journal of Botany* 40: 1373-1481.

Stover, R.H. (1970) Banana root diseases caused by *Fusarium oxysporum* f. sp. *cubense*, *Pseudomonas solanacearum*, and *Radopholus similis*: A comparative study of life cycles in relation to control. In *Root diseases and soil-borne pathogens*, (T.A. Toussoun, R.V. Bega, & P.E. Nelson, eds): 197-200. University California Press.

Agrios, G.N. (1997) *Plant Pathology* 4th ed. Academic Press 635pp.

Nash, S.M., Christou, T. & Snyder, W.C. (1961) Existence of *Fusarium solani* f. *cucurbitae* and *F. solani* f. *phaseoli* in soil. *Phytopathology* 55: 963-966.

Christou, T. & Snyder, W.C. (1962) Penetration and host-parasite relationships of *Fusarium solani* f. *phaseoli* in the bean plant. *Phytopathology* 52: 219-226.

Qureshi, A.A. & Page, O.T. (1970) Observations on chlamydospore production by *Fusarium* in a two-salt solution. *Canadian Journal of Microbiology* 16: 29-32.

Hsu, S.C. & Lockwood, J.L. (1973) Chlamydospore formation by *Fusarium* in sterile salt solutions. *Phytopathology* 63: 597-601.

Cook, R.J. & Schroth, M.N. (1965) Carbon and nitrogen compounds and germination of chlamydospores of *Fusarium solani* f. *phaseoli*. *Phytopathology* 55: 254-256.

Griffin, G.J. (1969) *Fusarium oxysporum* and *Aspergillus flavus* spore germination in the rhizosphere of peanut. *Phytopathology* 59: 1214-1218.

Griffin, G.J. (1981) Physiology of conidium and chlamydospore germination in *Fusarium*. In *Fusarium: Diseases, Biology and Taxonomy*, (P.E. Nelson, T.A. Toussoun & R.J. Cook, eds): 331-339, The Pennsylvania State University Press, University Park and London.



- Griffin, G.J. (1970) Exogenous carbon and nitrogen requirements for chlamydospore germination by *Fusarium solani*: dependence on spore density. *Canadian Journal of Microbiology* 12: 1366-1368.
- Schneider, E.F. & Seaman, W.L. (1974) Development of conidial chlamydospores of *Fusarium sulphureum* in distilled water. *Canadian Journal of Microbiology* 23: 763-769.
- Robinson, P.M. & Garrett, M.K. (1969) Identification of volatile sporostatic factors from cultures of *Fusarium oxysporum*. *Transactions of the British Mycological Society* 52: 293-299.
- Robinson, P.M. & Park, D. (1966) Volatile inhibitors of spore germination produced by fungi. *Transactions of the British Mycological Society* 49: 639-649.
- Bishop, C.D. & Cooper, R.M. (1983a) An ultrastructural study of root invasion of three vascular wilt diseases. *Physiological Molecular Plant Pathology* 22: 15-27.
- Vidhyasekaran, P. (1997) Fungal pathogenesis in plants and crops. Molecular biology and host defense mechanisms. Marcel Dekker Inc. New York, 553 pp.
- Recorbet, G. & Alabouvette, C. (1997) Adhesion of *Fusarium oxysporum* conidia to tomato roots. *Letters in Applied Microbiology* 25: 375-379.
- Mendgen, K., Hahn, M. & Deising, H. (1996) Morphogenesis and mechanisms of penetration by plant pathogenic fungi. *Annual Review of Phytopathology* 34: 367- 386.
- Lucas, J.A. (1998) *Plant Pathology and Plant Pathogens* 3rd ed. Blackwell Science. 274 pp.
- Brandes, E.W. (1919) Banana Wilt. *Phytopathology* 9: 339-389.
- Ullstrup, A.J. (1937) Histological studies on wilt of china aster. *Phytopathology* 27: 737-748.
- Smith, R. & Walker, J.C. (1930) A cytological study of cabbage plants in strains susceptible or resistant to yellows. *Journal of Agricultural Research* 41: 17-35.
- Pennypacker, B.W. & Nelson, P.E. (1972) Histopathology of carnation infected with *Fusarium oxysporum* f. sp. *dianthii*. *Phytopathology* 62: 1318-1326.
- Reid, J. (1958) Studies on the fusaria which cause wilt in melons. 1. The occurrence and distribution of races of muskmelon and watermelon Fusaria and a histopathological study of the colonization of muskmelon plants susceptible or resistant to *Fusarium* wilt. *Canadian Journal of Botany* 36: 393-410.
- Stover, R.H. (1962a) Fusarial wilt (Panama disease) of bananas and other *Musa* species. Commonwealth Mycological Institute. Surrey, UK, 177 pp.

Bishop, C.D. & Cooper, R.M. (1983b) An ultrastructural study of root invasion in three vascular wilt diseases 1. Colonization of susceptible cultivars. *Physiological Plant Pathology* 23: 323-343.

Beckman, C.H., Halmos, S. & Mace, M.E. (1962) The interaction of host, pathogen and soil temperature in relation to susceptibility to *Fusarium* wilt of bananas. *Phytopathology* 52: 134-140.

Beckman, C.H., Mace, M.E., Halmos, S. & McGahan, M.W. (1961) Physical barriers associated with resistance in *Fusarium* wilt of bananas. *Phytopathology* 51: 507- 515.