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Fusarium wilt disease of chili: pathogen, its mechanism of infection, eradication, and impacts

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Fusarium wilt is a devastating fungal disease. It is a threat to major fruits and vegetables including chili (*Capsicum annum* L.), reduces about 50% yield of major vegetables, and sometimes leads to complete crop losses. Fusarium wilt of chili, causing fungus *Fusarium oxysporum* f. sp. *capsici*, can survive in the soil for several years. The infected plant can be recognized by the yellowing of older leaves and downward curling of apical shoots, followed by plant wilting and ultimately the death of the plant. The pathogen can survive for many years in the field without a host. Some important factors contribute to the epidemic nature of the disease development include warm soil temperatures, inadequate soil moisture, susceptible host plants, the presence of a virulent pathogen, and particular pH levels. The infection or disease development by pathogen is also assisted by some cell wall degrading enzymes (CWDEs) and certain infectious proteins. These losses by disease and pathogen could be avoided by adopting certain physical, chemical, and biological approaches. The aim of the present study is to shed light on the pathogen profile, its mechanism of infection, and fruitful strategies for disease management. Although an adequate method to completely eradicate fusarium wilt has not been introduced so far, many new methods are under research to eradicate the disease.

Keyword: *Fusarium oxysporum*, *Fusarium oxysporum* f. sp. *capsici*, chili, Fusarium wilt of chili and epidemiology.

INTRODUCTION

The emergence of aggressive and invasive plant pathogens is one of the main threats to sustainable crop production and food security. They significantly lower the crop's yield as well as its quality. In many countries, the agriculture sector is largely supported by the production of fruits and vegetables. Chili (*Capsicum annum* L.) is a member of the genus *Capsicum* of the *Solanaceae* family. Chili is a precious spice cum vegetable widely cultivated all over the world in both tropical and subtropical regions. Many pathogens attack it, reducing its growth and yield. Chili fusarium wilt, one of the most damaging biotic stresses to chili, has a major influence on the vegetable market, limiting export and yield. It affects several commercial chili varieties and species of the capsicum genus globally. This review aims to highlight recent advancements in research concerning the pathogen's profile in chili wilt, the mechanism of its infection and management approaches. It also provides an overview of prior research and highlights areas that still need to be further explored to successful management of this disease.

***Pathogen Profile*:** Fusarium wilt is a well-known disease of chili around the world. There is some disagreement about where it came from. It is believed to have been found for the first time in New Mexico (Leonian, 1919). Many countries around the world have reported it, including Avery Island, Louisiana (Black and Rivelli, 1990), Central Java (Agrios, 2005), Pakistan (Kamal and Moghal, 1968), and Spain (Pérez-Hernández *et al.*, 2014). In Egypt, fusarium wilt is the most important devastating and damaging disease affecting yield of chili (Black *et al.*, 1991; KA, 1994). In India, Fusarium wilt in chili has been reported in many regions and causes around 25% yield losses (Najar *et al.*, 2006). In the USA (Roberts *et al.*, 2004) recorded a 35% occurrence in various states, and it is also observed that after 30 days of soil infection, *Fusarium oxysporum* kills 56% of chili seedlings (Ragab *et al.*, 2012). Globally, 10-80% yield reduction has been recorded due to this disease (Loganathan *et al.*, 2013). *Fusarium oxysporum*

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caused $65.3 billion revenue loss (Akash *et al.*, 2022), and in Pakistan, the fusarium wilt affected 16.6% of hot chili and 21.9% of vegetables, whereas there are 48% more diseases in the rest of the world. From 1999 to 2005, there were yield losses of about 115.5 thousand tons (Hussain and Abid, 2011). Fusarium wilt causes 10 to 50% yield losses in Pakistan under optimal conditions (Irum, 2007).

Plant growth parameters like weight, yield, and length of plant all decrease as a result of spore multiplication (Abdel-Monaim, 2010). According to Baba *et al.* (2014), in Kashmir Valley the incidence of fusarium wilt was 40% during the fruiting/flowering stage and 6% during transplanting. According to a survey, about 80% of Thailand's chili plant crop losses were attributable to fusarium wilt (Wongpia and Lomthaisong, 2010). In Egypt, *Fusarium oxysporum* destroyed 56% of chili seedlings after 30 days of its infestation in soil (Ragab *et al.*, 2012). Moist soil and high temperatures play an important role in enhancing pathogen and disease development. It can also cause the death of leaves, followed by rolling of leaves upward as well as downward (Hussain and Abid, 2011). Although this disease can be seen all year, it is reported to occure more frequently in November and December than in the other months (Bai *et al.*, 2018).

Fusarium oxysporum f. sp. *capsici* (FOC), is a soil-borne and necrotrophic (kill the host organism after establishing on it) fungi. It can survive in the field for more than ten years without a host and is a causal organism that causes chili wilt (Di Primo *et al.*, 2001). Differentiated from those other *Fusarium* species by asexual structures like phialides, microconidia, and macroconidia, as well as unique features such as cultural aroma and colony texture on media culture (Edel *et al.*, 2000). *FOC* has aseptate (not divided into cells or sections by septa or non-septate) microconidia (uninucleate, and size is also smaller than macro-conidia) that are formed on short, false-headed monophialids, while septate macroconidia with three septa are found on monophialides in

Table 1. Major Forma specialis of *Fusarium oxysporum* and their host.

Sr.	*Fusarium oxysporum*	Host	Source
1	f. sp. melonis	Melon	(Daami-Remadi *et al.*, 2007)
2	f. sp. pisi	Pea	(Kraft *et al.*, 2000)
3	f. sp. lycopersici	Tomato	(Nirmaladevi *et al.*, 2016)
4	f. sp. radicis-lycopersici	Tomato	(Can *et al.*, 2004)
5	f. sp. phaseoli	Bean	(Henrique *et al.*, 2015)
6	f. sp. cubense	Banana	(Li *et al.*, 2012)
7	f. sp. coglutinans	Cabbage	(Reyes and Chadha, 1972)
8	f. sp. citri	Citrus	(Hannachi *et al.*, 2014)
9	f. sp. ciceris	Chickpea	(Gupta *et al.*, 2013)
10	f. sp. cepae	Onion	(Pennypacker and Nelson, 1972)
11	f. sp. cannabis	Hemp	(McCain and Noviello, 1985)
12	f. sp. canariensis	Date palm	(Husaini *et al.*, 2018)
13	f. sp. batatas	Sweet potatos	(Hedge *et al.*, 2012)
14	f. sp. betae	sugar beet	(Cramer *et al.*, 2003)
15	f. sp. herbemontis	Grapes	(Brown, 2009)
16	f. sp. dianthi	Carnation	(Pennypacker and Nelson, 1972)
17	f. sp. lactucae	Lettuce	(Scott *et al.*, 2010)
18	f. sp. gladioli	Gladious	(Pataky, 1988)
19	f. sp. nicotianae	Tobacco	(Johnson, 1921)
20	f. sp. medicaginis	Alfalafa	(Weimar, 1928)
21	f. sp. lini	Flax	(Mohit *et al.*, 2014)
22	f. sp. niveum	Watermelon	(Dau *et al.*, 2009)
24	f. sp. palmarum	Palm	(Elliott *et al.*, 2010)
25	f. sp. perniciosum	Mimosa tree	(Joy and Sherin, 2012)
26	f. sp. ricini	Castor	(Dange *et al.*, 2006)
27	f. sp. tuberosi	Potato	(Daami-Remadi *et al.*, 2009)
28	f. sp. tulipae	Tulip	(Pataky, 1988)
29	f. sp. vasinfectum	Cotton	(Kim *et al.*, 2005)
30	f. sp. raphanin	Radish	(Du Toit and Pelter, 2003)
31	f. sp. fragariae	Strawberry	(Garrido *et al.*, 2016)
32	f. sp. *lentis*	Lentil	(Stoilova and Chavdarov, 2006)

branched conidiophores. Only in sporodochia do these macroconidia develop, and chlamydospores can be found in single form or also as pair. The walls of these macroconidia are smooth or bumpy. However, because the morphological characteristics of Fusarium species change in response to genetic and environmental factors, it is difficult to distinguish between them (Nelson, 1991; Llorens *et al.*, 2006). *FOC* is the causal orgainsm of Fusarium wilt in chili; it belongs to the genus *Fusarium*, phylum *ascomycota*, class *ascomycetes*, order *hypocreales*, and *nectriaceae* family; is saprophytic, found worldwide as soil borne plant pathogen (Booth, 1971). Fusarium genus was given the name because it contained asexual fusiform spores. Link described fusarium genus by year 1809 on the base asexual conidia with a banana like shape (Leslie and Summerell, 2008). Later on Fries (1821) after the International Botanical Code for Nomenclature's authentication, confirmed the specific spores in Fusarium species. In the first century, nearly 1000 species were described based on cursory observation (Toussoun and Nelson, 1975). Wollenweber and Reinking (1935) reconfigured this genus in their monograph to 65 species, and sixteen sections. These 16 sections of the genus were further simplified by Snyder and Hansen (1945) to 9 species, and other several species in a section Elegans (one of 16 sections) were combined in single species, which named *Fusarium oxysporum.* A new classification system suggested by Nelson *et al.* (1983), replaced Synder and Hansen's system by serving as a connection between the old and new classification systems. A detailed information was provided by Leslie and Summerell (2008) about 70 species of *Fusarium* based on their physical appearance and phylogenetic features. More than 100 *Fusarium* species have been verified and described, according to the Dictionary of Fungi (Kirk, 2008). These differences in Fusarium classification are owing to a lack of extensive literature that might differentiate the species based on morphological traits.

***Major forma specialis of Fusarium oxysporum*:** It has been demonstrated that numerous *Fusarium oxysporum* strains can induce wilt infections on a range of significant agricultural products, vegetables, and even ornamental plants. The same forma specialis includes isolates that target crops with similar characteristics (table 1). Several forma specialis cause disease on a specific crop, such *as Fusarium oxysporum* f. sp. *dianthi* infects carnation; *Fusarium oxysporum* f. sp. *cubense* infects banana, *Fusarium oxysporum* f. sp. *capsici* cause wilt disease in chill, and Fusarium oxysporum f. sp. vasinfectum on cotton, but certain Fusarium species are harmful for many crops (Cafri *et al.*, 2005). The genetic basis for *Fusarium oxysporum* host specificity is still unknown (Baayen *et al.*, 2000). However, not all of the strains belonging to the same forma specialis evolved from the same forefathers (Nirenberg *et al.*, 1998).

***Major types of Chili wilt*:** There are four pathogens that cause wilting in chili: Phytophthora root rot, Rhizoctonia root rot, Verticillium wilt, and Fusarium wilt. Many microorganisms may be responsible for these diseases (table 2).

***Symptoms*:** Both primary and secondary symptoms have been noted for most wilt-causing infections, including fusarium-caused chili wilt (figure 1). FOC infects plants, starting at the roots and moving up to the cortex (Beckman, 1987). Brown

Table 2. Major Types of Chili wilt.

Type of wilt	***Phytopathora* wilt**	***Verticillium* wilt**	***Rhizoctonia* wilt**	***Fusarium* wilt**
Common disease name	Phytophthora-root-rot	Verticillium-wilt	Rhizoctonia-root rot	Fusarium-wilt
Pathogen	*Phytophthora capsici*	*Verticillium dahlia* and *V. alboatrum*	*Rhizoctonia solani*	*F. oxysporum*
Phylum	Oomycete	Ascomycota	Basidiomycota	Ascomycota
Class	*Oomycete*	Sordariomycetes	*Basidiomycetes*	Sordariomycetes
Spore type	Zoospores	Conidia are ovoid or ellipsoid and usually single-celled	Basidiospores	Three types of spores: • Microconidia, • Macroconidia, • Chlamydospores
Symptoms	• Plant wilting, caused by infection of the root as well as lower surface of the stem. • Zoospores enable spread of the pathogen from plant to plant.	• Defoliation • Stunted growth • Wilting • Darkening of the vascular bundles.	• Plant withering brought on by infection of the root as well as lower stem. • Pathogens survive in organic matter and soils.	• Chili plants show symptoms such as • Vascular discoloration, • Leaf chlorosis • Wilting.
Parts of plant that may be affected	• Root hairs • Root, • lower stem,	• Root, • Stem, • Twigs, • Fruits	• Root portion, • lower stem	• Root, • Stem, • Fruits, • Twigs • Branches
References	(Leonian, 1922)	(Lazarovits *et al.*, 2000; Subbarao, 2022)	(Muhyi and Bosland, 1995)	(Leonian, 1919)

vascular discolouration is among the primary and initial symptoms of pathogen followed by curling, and finally withering of the plants (Centurión, 1989).

***Symptoms on leaves*:** On leaves, the infection initially appears as moderate clearing of veins on the outside of new leaves, proceeded by older leaves bending downward (epinasty) (Miller *et al.*, 2008; Bashir, 2015). Leaves withering, defoliation, followed by necrosis at the margins of the leaves that survive, and ultimately mortality of entire plant (Agrios, 2005). Discoloration of leaves starting from lower to upper parts of plants. Shriveling and death leave discoloration of the leaf veins, which may turn brown or black. Wilting of leaves that do not recover when water is applied.

***Symptoms on fruits and twigs*:** On fruits and twigs, symptoms include, discoloration of chili, decreased fruit size, reduced fruit yield and early fruit drop. Stunted growth of twigs and branches wilting of twigs and branches, shriveling of twigs and branches, discoloration of twigs and branches. Since the pathogen is soil-borne, it enters via plant's vascular system and causes more disease. The apical branches of dying plants curl downward, and the leaves lose their green color as a result of the chili wilt (Kiran *et al.*, 2006). When an infection affects mature plants, abnormal flower and fruit production results. Wilting can only be seen after the base stem has turned brown and cannot be seen without taking off the top covering of the stem, therefore, it is not a main symptom Ferniah et al., 2014).

Figure 1. Fusarium wilt causes drooping, withering, and mortality of chili pepper plants

***Mechanisms of infection of pathogens*:** Once inside the plant, *Fusarium oxysporum* grows through the root cortical intercellular tissue before entering the arteries via pits of xylem vessels. The ability of plants to absorb water is severely impacted by the growth of fungi in vascular tissues, which leads to stomatal closing on leaves, followed by leaves wilting, and finally death of the whole plant (figure 2). This is when the fungal pathogen begins to infiltrate parenchymatous tissues of the plant, invading until it eventually reaches the surface of dead tissues, where it sporulates abundantly.

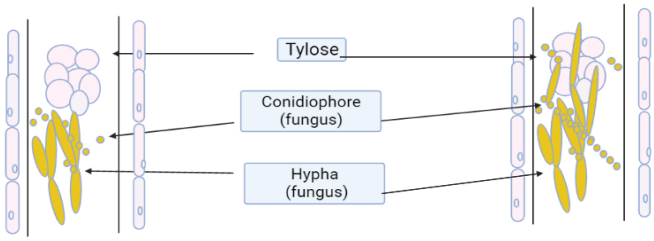

Figure 2. Fusarium wilt pathogen infection mechanism (Yadeta and J. Thomma, 2013)

The spores that come from this act like primary inoculum for more infection and thus fungus's subsequent spread (Agrios, 1988). The pathogens causing wilt efficiently extract nutrients from the sap in xylem tissue to fulfill their nutritional needs. Additionally, they use enzymes to break down host cell walls, invade neighboring cells, and ultimately induce nutrient leakage from the surrounding tissues (Möbius and Hertweck, 2009). For the pathogens that cause vascular wilt, nitrogen is one of the limiting nutrients in the xylem sap Divon et al., 2005). Figure 3 depicts the Fusarium wilt disease cycle in brief.

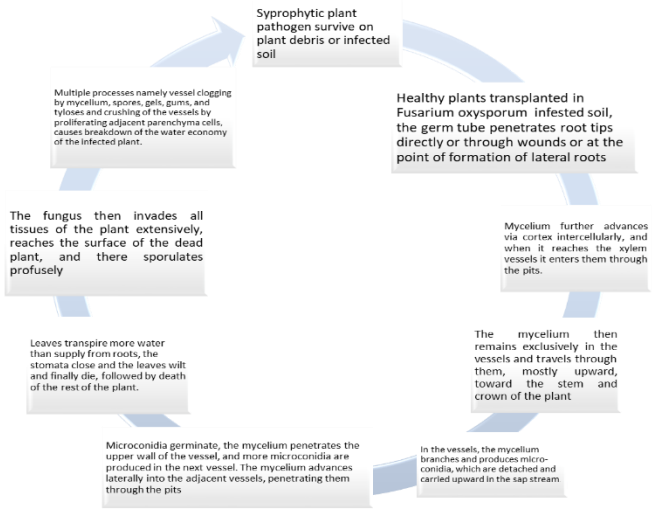

Figure 3. Disease Cycle of Fusarium wilt

***The genetic basis of disease development*:** Similar to other fungi, *Fusarium oxysporum* needs CWDEs (cell wall degrading enzymes) to get into the host plant cells. Different genes are responsible for controlling these enzymes. These genes have previously been discovered, cloned, altered, and silenced to determine whether they contribute to pathogenicity. Given that the fungus has a variety of infection methods, such as cell wall destruction or penetration via a damaged part or other natural opening. Mode of infection determines the genes necessary for pathogenesis (Mendgen *et*

Table 4. The main transcriptional factors in *F. oxysporum* that cause pathogenicity

Sr.	Names	Role	Source
1	Con7-1	Nuclear localization, cell wall biogenesis, and cell division	(Ruiz-Roldán *et al.*, 2015)
2	Ftp	Virulence in the early stages of a disease	(Niño-Sánchez *et al.*, 2016)
3	Sgel	Growth of pathogens, control expression of 6 genes, and took part in colonization	(Michielse *et al.*, 2009)
4	PacC	Virulence negative regulator	(Caracuel *et al.*, 2003)
5	Ste12	Aggressive fungi development	(Rispail and Di Pietro, 2009)
6	XinR	Activators of xylanase gene transcription	(Calero-Nieto *et al.*, 2007)

al., 1996). Idnurm and Howlet in 2001 listed and categorized the 79 genes on the basis of CWDEs, involvement in development of specific structures needed for fungal infection, ability to suppress defense system of host, signal cascades, and the manufacturing of toxin(s) (Idnurm and Howlett, 2001).

Four entire chromosomes make up the LS genomic areas. Virulence is a quantitative trait that is regulated by several genes. The transcriptional regulators that these genes control eventually lead to pathogenicity. Table 3 provides a summary of the key transcriptional regulators involved in the pathogenicity of *F. oxysporum*.

The fungal pathogens first colonize the vascular system of plants, which is regulated by MCPs (mitochondrial carrier proteins) regulated by FOW1 gene (Table 4). EndoPGs encoded by the pg1 gene depolymerize the abundantly present homogalacturonan in plant cell walls. Reverse transcription-polymerase (RT-PCR) chain reaction, that exhibited the expression of the pg1 gene in lower side stem as well as root portion of diseased plants, further validated significance of the gene in the development of the disease (Di Pietro and Roncero, 1998). Family-10 xylanases responsible for rupturing the xylan backbone are encoded by the xyl2 and -3 genes of the fungus *F. oxysporum*. These genes were identified and cloned from pathogens by Ruiz-Roldán et al., 1999 who also revealed that significant homology was discovered between the anticipated amino acid sequences of these genes as well as other genes in the xylanase families. In contrast to Xyl3, which was expressed during the whole cycle of infection in the root zone as well as lower stems of infected plants, Xyl2 was only expressed during the disease's terminal phase, according to expression analyses. Another study found that the pathogen's family-11 xylanases were encoded by the gene Xly4, and that this gene was active throughout the full duration of the disease (Gómez-Gómez *et al.*, 2002). Pectate lyase (Pl1) is crucial for the establishment of infection in plants because it reacts with a sizable portion of the middle lamella and plant cell wall.

Vascular tissues that have been exposed to sodium polygalacturonic acid salt exhibit Pl1 gene transcription. Additionally, it was discovered through a study of how it manifested in the roots as well as stem of an infected plant (Huertas-González *et al.*, 1999). Many of the genes associated with CWDE synthesis are observed to express in root or stem regions by expression analysis. The plant's ability to fight this deadly infection may benefit from strengthening this area of the plant.

***Role of cell wall degrading enzymes in pathogenicity*:** Pathogen must be able to bypass genes of host that create defensive proteins to infect any host. Genes in *Fusarium oxysporum* can avoid and detoxifying the host defensive proteins. Pathogenicity and detection of pathogenic genes that code for extracellular enzymes are caused by pathogen biomolecules involving CWDEs. *Fusarium oxysporum* enters the endosperm and then perforates the host cell walls to produce a range of host poisons, or CWDEs (Hammond-Kosack, 2000). By destroying the elements of the cell wall, these enzymes make it easier for fungal infection to enter the plant cell.

Fungal entry into plant cell wells is caused by digesting CWPs (cell wall polymer) to for securing food source, proceeded by cell wall destruction, which then spreads throughout plant

Table 3. Pathogenicity-related gene list

Sr.	Name	Role	Source
1	Fmkl	invasion of the cortex and root systems	(Di Pietro *et al.*, 2001)
2	Xyl2 and Xyl3 (family-10 xylanases)	Xylan backbone breaking	(Ruiz-Roldán *et al.*, 1999)
3	ARG1	Argininosuccinate lyase production	(Namiki *et al.*, 2001)
4	*Pgx4*	Pectinolytic enzyme production	(García-Maceira *et al.*, 2000)
5	FOWI	Plant colonizing	(Inoue *et al.*, 2002)
6	Pl1	Pectate trans-elimination	(Huertas-González *et al.*, 1999)
7	Pg1	homogalacturonan depolymerization	(Di Pietro and Roncero, 1998)
8	Xyl4 (family-11 xylanases)	Xylan backbone breaking	(Gómez-Gómez *et al.*, 2002)

tissue (Gupta *et al.*, 2012). CWDEs, primarily cellulases, proteases, xylanases, pectate lyases, and endo- and exo polygalacturonases (PG). These are necessary for *Fusarium oxysporum* to enter the host cell. These enzymes are synthesized in enzymatic pathways controlled by several genes. These pathways can produce enzymes that can get past the obstacles the plant cell wall creates (García-Maceira *et al.*, 2000). Pectin, a crucial component of lamellae and primary cell walls, is depolymerized by enzymes known as pectate lyases, which is followed by transelimination (Beckman, 1987). Exo polygalacturonases and endo-polygalacturonases, which are further divided into two types, have been effectively identified from a variety of diseases. Since these enzymes cause breakdown of homogalacturonan (HG), whichleads to the maceration of tissues of host plant, endoPGs are concentrated among them (Collmer and Keen, 1986). The endoPG that is most frequently generated by F. oxysporum is PG1 (Di-Pietro and Roncero, 1996).

Plant wilt disease has been connected to xylanase enzymes (Gómez-Gómez *et al.*, 2002), as this xylan is among the most prevailing polysaccharide compounds of plant cell walls, this enzyme may be crucial to infection. Endo 1,4- xylanases are divided into two families, family 10 and family 11, and act on the xylan backbone to depolymerize it (Biely *et al.*, 1997). Christakopoulos (Christakopoulos *et al.*, 1996) revealed that diverse kind of xylanases are secreted by *F. oxysporum*. During the fungus growth on vascular tissue, two xylanases ezymes; one with a basic nature and other having an acidic nature, are secreted (Ruiz *et al.*, 1997).

***Role of fusaric acid*:** Numerous Fusarium spp. produce fusaric (FA) acid (figure 4), a toxin that is not specific to one particular host. Fusarium oxysporum is the source of this toxin's widespread production (Bacon *et al.*, 1996; Rani *et al.*, 2009). The level of virulence of plant pathogenic Fusarium spp. strains have been linked to high FA production. The tomato plant's stem as well as petioles wilt, develop necrotic spots on the leaves, and shrink and dry out as a result of FA. A rapid transient membrane hyper-polarization is brought on by FA's inhibition of root and root hair growth (Bouizgarne *et al.*, 2006). Many different types of plants exhibit wilt symptoms due to FA. Fusarium infection results in a variety of symptoms, including leaves wilting and necrosis, which suggest that FA may play a part in the onset of the disease (Köhler and Bentrup, 1983; Marrè *et al.*, 1993). FA has an impact on a variety of biochemical pathways at subcellular level, including altered membrane permeability, abnormal mitochondrial activity, as well as downregulation of respiration.

Fusarium wilt induces various effects, such as increased electrolyte leakage, disruption of electrochemical gradients for potassium and hydrogen at the plasma membrane, membrane depolarization leading to decreased intracellular ATP levels, and the inhibition of specific metal-containing enzymes like cytochrome oxidase. These consequences ultimately culminate in respiratory disorders that lead to cellular demise (Marrè *et al.*, 1993). Through an examination conducted using tomato leaves and cultured cells, the impact of toxic concentrations of fusaric acid (FA) on pro- and antioxidant systems was investigated (Kuźniak *et al.*, 1999). Within the same (2006) year, Bouizgarne (Bouizgarne *et al.*, 2006) demonstrated that, in the absence of toxic effects, low levels of FA may cause a variety of protective responses in plant cells and may serve a signaling role in host and pathogen interactions. High FA concentrations (>200 mM) promote necrosis, whereas moderate FA concentrations (50e100 mM) elicit apoptotic characteristics.

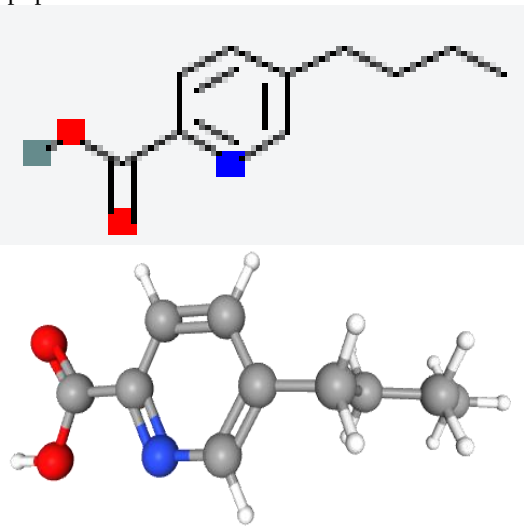

Figure 4. Fusaric acid 2D and 3D structure (retrieved from NCBI) (NCBI., 2023).

Damage to host: The most dangerous plant pathogens, *F. oxysporum* is the main cause of devastating wilt in more than 100 different species of plants (Dean *et al.*, 2012). Plant fungi has developed their own techniques for penetrating the tissues of plant hosts, which is followed by colonizing, growth, and the establishment of pathogen. To react with modifications, such as metabolic and morphological changes necessary for disease, the pathogen needs signals from the host plant. Before invading the host tissue, the pathogen undergoes a number of significant changes, including the secretion of toxins, the development of specialized structures, and hyphal growth (Knogge, 1996).

Effectors are released by pathogenic fungi to suppress the host's defenses and facilitate invasion (Lo Presti *et al.*, 2015). In addition, several morphological and biochemical modifications result from the fungal infection on the plant host (Zeilinger *et al.*, 2016). These modifications are mostly determined by the activity of genes (particular product/protein), as well as signaling pathways involving cAmp signaling (Mitchell and Dean, 1995), MAP Kinase (mitogen-activated-protein kinase) (Xu *et al.*, 1998), as well

as initiation of G proteins (Bölker, 1998). Before infection, the fungus colonizes the host plant's vascular tissue. To enable the pathogen to colonize the tissue, the FOW1 gene produces the MCP (mitochondrial-carrier protein) (Inoue *et al.*, 2002). The soil borne fungi penetrate the stele through the cortex and begin to develop there, which causes the vascular bundles to wilt (Beckman, 1987; Tjamos and Beckman, 2013). It begins to develop in the vascular tissues of stems and roots, which makes it easier for conidia to travel via the transpiration route. Furthermore, only the virulent strain of *Fusarium oxysporum* can enter the vascular system and cause disease, while other strains can do so but cannot cause root rot or tracheomycosis (Fravel *et al.*, 2003). The infection may start at the tip of the root, from air, mechanical injury, or hail-induced wounds, and the pathogen could enter the plant roots using its mycelium or sporangial germ. After pathogens enter the plant, the mycelium begins to grow through the root-cortex as well as the cellular layers. After the mycelium enters the xylem, it occupies the vessels via xylem pits, which significantly reduces the plant's nutrients and water supply. Eventually, the plant dies due to fungal growth in the plant vascular system, which causes the foliage to wilt leading to the stomata to close because of a shortage of water (Agrios, 2005).

F. oxysporum initially infects plants without exhibiting any symptoms, but eventually takes over the vascular system and starts a severe wilting and discoloration of plant's stem sections. The fungus has a systemic reaction inside a plant, which can result in the death of infected plants (Groenewald, 2007). It penetrates and recognizes physical as well as chemical cues from plant species which are resisted with the appropriate morphogenetic as well as metabolic modifications necessary for any virulence bypassing the defense and recognition mechanisms of plants (Woloshuk and Kolattukudy, 1986).

***Biofilm formation*:** The development of biofilms is essential for the relationship between the pathogen and its host. As a result, it is essential for a pathogenic microbes' attack to succeed, for it to survive, and for the epiphyte to be fit. However, it does not effect on the pathogen's infectivity (Dunger *et al.*, 2007). There have been reports of *F.oxysporum* producing biofilms (Peiqian *et al.*, 2014). A tentative model for the creation of filamentous fungi biofilms governs biofilm growth (Harding *et al.*, 2009). Under the right external circumstances, such as temperature, pH, and source of carbon, *F.oxysporum* has the ability to create biofilms. Susceptibility testing indicates that *F.oxysporum* biofilms have advantages for surviving in harsh environments and are resistant to biocides, which could provide a great explanation for why drugs to treat Fusarium wilt are ineffective (Peiqian *et al.*, 2014).

***Eradication and control measures*:** The rise in global trade and travel has significantly increased the risk of introducing invasive plant pathogens and pests into crops (Scheffer, 1991). Various strategies are being employed to halt the spread of the disease, preempt its onset, and eliminate it altogether. The following list includes a few effective ways to prevent Fusarium wilt.

By physical tactics

***Plant removal*:** Removing sick or infected plants from the field usually works when the infection is more localized and only affects a handful of plants or a small area of the field. Remove infected plants or parts of plants if at all possible, to lessen the likelihood of inoculum establishment and disease dissemination during the early stages of a disease.

***By crop rotation*:** Growing a variety of crops that are dissimilar to one another in the same area year after year is known as crop rotation. It is a simple method for reducing or eliminating the cost of controlling insects and diseases. For instance, planting the same crops year after year in the same farm area, such as cauliflower, tomatoes, and cabbage, actually encourages the growth of pathogens. The soil should not be used for the cultivation of the affected plant type for at least 4-5 years after diseased plants have been removed and destroyed. Replace the infested soil with new soil from another area of the garden. Additionally, soil solarization or steaming can be used to disinfect soil particularly in greenhouses or pot to help lower the pathogen load. The number of pathogens in the soil decreases with crop rotation. The pathogen must be completely eradicated from the cropland alongside the plant debris for it to successfully control a soil borne pathogen (Labrada, 2008a; Ajilogba and Babalola, 2013).

***Soil solarization*:** Fusarium oxysporum was eliminated up to 30 cm below the surface of the soil following soil solarization, according to Sharma and his coworkers 2004 report (Sharma *et al.*, 2004). FOC population can be reduced by soil solarization by 76.3 percent (Ajilogba and Babalola, 2013).

***Use of mulching*:** To control weeds, improve soil moisture, and keep the soil below 80°F, mulching involves adding a thick layer of mulch to the soil's surface. By acting as a barrier between parts of the plant above ground and plant pathogens in the soil, it aids in disease management. Since it aids in weed control, it also aids in changing the environment for these pathogens, thereby making conditions unfavorable for them in addition to preventing disease. The plant should be mulched to prevent soil-borne pathogens from splashing on plants when it is being watered (Francis *et al.*, 2010). The severity of disease and pathogen is reduced when two layers of mulch are applied on top of solarized soil (Garibaldi and Gullino, 1991).

***Avoiding over irrigation*:** Due to the ease with which pathogenic bacteria and fungi can spread when the foliage is wet, overhead irrigation exacerbates disease issues. When leaves are humid, for instance, leaf spot diseases spread quickly. As soil-borne fungi diseases like root rots and wilts are more common in overly wet conditions, overwatering should also be avoided. It is advised to drain using drips (Labrada, 2008b).

***Regular inspection of fields*:** Regularly visiting the field and conducting thorough inspections of crops for signs of disease, pathogens, or symptoms is essential. Consistent crop inspections facilitate the prompt identification of diseases, thereby expediting their eradication.

***Soil and environmental factors*:** Soil is necessary for the growth, survival, multiplication, and dissemination of soil-borne pathogens because it provides an environment for their development and growth. One of these pathogens is *F. oxysporum* f. sp. *capsici* (FOC) a thermophilic, prevalent soil-borne pathogen that is greatly influenced by the soil type (Özer *et al.*, 2009) such as clay, sandy loam, and sandy soils. These soils vary in terms of their electrical properties (EC), pH (alkaline versus acid character), organic (OM) manure, soil structure (sand, silt, and clay particles' ratio), and restricting volatile fungicidal chemicals which significantly change the activity of soil-borne plant pathogens (Ma *et al.*, 2001). Because sandy loam type soil contains an adequate amount of soil particles (sand, silt, as well as clay) with distinct acid and alkaline natures compared to sandy and clay soils, it produced notable results with low disease incidence. Because the development of microconidia and macroconidia as well as chlamydospores, of *FOC* was inhibited (Attitalla *et al.*, 2004) by altering the FOC's virulent and aggressive behaviors and genetic traits (Cramer, 2000). One more element that contributes to the reduced disease occurrence is the moderate water retaining ability of sandy loam soil, which is inappropriate for fungi growth (Latiffah *et al.*, 2009).

***Organic manuring*:** According to studies, adding organic matter to soil improves its physical, chemical, and physical-chemical (water holding capacity, aeration, and nutrient uptake) qualities, It additionally encourages plant growth but also prevents diseases caused by soil borne pathogens such as *Fusarium oxysporum* (Bonanomi *et al.*, 2010). In addition to accelerating growth, organic manure (OM) also increases plant cell wall rigidity and resistance to soil-borne diseases (Basak *et al.*, 2002). OM was used against Fusarium wilt disease and showed encouraging results (Randall *et al.*, 2000). The supply of organic matter, the technique of application, the rate, the timing, and the climatic conditions all affect the rate of disease inhibition. The utilization of organic materials to regulate Fusarium wilt by modifying soil pH revealed a reduction in the incidence of Fusarium by 50-80% (Ha and Huang, 2007).

***Planting resistant varieties*:** In order to reduce soil borne diseases, crop resistance is the most effective as well as ecologically beneficial method (Fravel *et al.*, 1998). In addition to minimizing disease related losses, using resistant varieties can also help to reduce the fungicide's harmful effects. In the treatment of soil borne infections globally, particularly against Fusarium wilt of economically significant many crops, genotype/hybrid screening is a crucial component (Naik *et al.*, 2007). The resistance of 33 advanced lines and cultivars of chili to Fusarium wilt was examined. Sixteen varieties/advanced lines indicated a moderately resistant response, compared to two advanced lines that showed a resistant response. Four kinds and advanced lines showed susceptible responses, nine varieties and advanced lines demonstrated a moderately susceptible reaction, while 1776 as well as Desi displayed a highly vulnerable/susceptible reaction in both years (Bashir, 2015).

***Other preventive methods*:** Other safety precautions, including the proper cleaning of instruments as well as staff the careful use of tools to avoid injuries, should also be noticed in fields as well as nurseries.

Chemical approaches

***Copper base sprays*:** Prevost first referred to copper's fungicidal effects on the wheat bunt disease in 1807; however, it wasn't until Millardet's discovered the Bordeaux mixture in France in 1885 that copper's widespread use as a fungicide began. *Plasmopara viticola* caused downy mildew of grapevine and later late blight of potato (Phytophthora infestans) were both successfully controlled by a copper sulphate and lime mixture. The pathogen that causes fusarium wilt has been effectively combated by a variety of copper-based fungicides. The number of fungi can decrease as a result. The majority of fungicides with a copper base are protective types that are applied to seeds. Fusarium wilt was significantly controlled by copper oxychloride in its various concentrations (Fareed *et al.*, 2015; Baloch *et al.*, 2021). A primary limitation associated with the utilization of copper products is that extended use may result in the accumulation of copper in the soil, posing potential harm to the soil, the environment, and plant life.

***Use of systemic fungicides*:** Systemic fungicides enter the plant through the roots or foliage and move throughout it using the vascular system. Systemic fungicides may build up at the leaf margins and typically move upward in the transpiration stream. Fusarium wilt is currently managed among various crops using a variety of systemic fungicides. Carbendazim and benomyl were tested in vitro and in vivo by soaking soil at different depths, which resulted in maximum Fusarium wilt disease inhibition in chili). In laboratory tests against F. oxysporum and other pathogens associated with bell pepper wilt disease, carbendazim (Bavistin), copper oxychloride, captan, and metalaxyl + mancozeb (Ridomil) demonstrated complete inhibition of fungal growth. Fungicides like Ridomil Gold, Carbendazim, Metalaxyl, and Mancozeb are used to prevent Fusarium wilt (Sitara and Hasan, 2011). The use of pesticides has grown over time, which has resulted in damage to the environment, health hazards, resistance to pests, and a decline in the population of beneficial insects. The risk to people and the environment can be decreased by using pesticides safely and responsibly.

***Nano-fungicides*:** Because of their distinct physical and chemical characteristics, which differ greatly from those of their traditional counterparts, nanoparticle (NP) materials have attracted increased attention in recent years (Stoimenov

et al., 2002). Various research has shown the efficacy of silver, zinc, and copper base nanoparticles in preventing fusarium wilt in a variety of plants.

***Post-harvest sanitization*:** Post-harvest sanitization of fruits, vegetables, fields and related material couldhelp minimize the field-to-field and region to region movement of pathogenic inoculum. Because during field operations and movement of goods could become cause of pathogen to infest the field and crops. There is also needed to avoid the cultivation of crop in the field that was previously infected with pathogen, or it should be treated properly to minimize the inoculum. Several chemical sanitizers, including sodium hypochlorite, peracetic acid, chlorine dioxide and calcium oxychloride are employed for this purpose [116].

***By systematic acquired resistance (SAR)*:** By stimulating the defense system with inducers like salicylic acid (SA) and jasmonic acid (JA) systemic induced resistance, or SIR, is a different method of creating resistance in chili against Fusarium oxysporum (Ellis *et al.*, 2002). Several physiological changes, including those in pathogenesis-related proteins (PR), antioxidant enzymes and phenolic compounds were seen in tomato plant leaves as a result of the administration of these plant growth activators and defensive signaling molecules. Furthermore, the application of salicylic acid, coupled with an extended infection duration, resulted in the formation of reactive oxygen species (ROS), notably hydroxide ions (OH-), superoxide ions (O-2), and lipid peroxide. (El-Khallal, 2007).

Biological control

***Using plant breeding and genetics in host plant*:** Any crop plant's ability to be bred to have a specific trait improved depends entirely on the trait's genetic makeup, including whether it is controlled by one gene (mono genic/oligo genic) or several genes (polygenic), as well as how those genes are passed down through the plant's generations (Thakur *et al.*, 2018). A breeder must go through several steps in order to breed against biological stress (such as fusarium wilt), including gathering both resistant and susceptible material, screening germplasms under natural and artificial conditions (like artificial inoculum), and finally transforming these resistant resources through an appropriate breeding method. The most popular techniques for breeding chili are hybridization, single seed descent, backcross breeding, mass selection, and recurrent selection (Padilha and Barbieri, 2016). Backcrossing is thought to be the most effective way to introduce a resistant gene or genes into any cultivated variety out of all the appropriate methods (Thakur *et al.*, 2018).

Resistant resources are essential for a well-balanced agricultural system because they help crop plants survive any type of stress. There are two different types of resistance mechanisms: qualitative (controlled by a single or small number of genes) and quantitative (controlled by many genes). In the first form of inheritance, genes completely resist the pathogen because they interact with its genes and are only active against certain races of the disease. Due to a number of characteristics, epidemic parameters and latency periods are the most significant, the second kind of inheritance process for disease development is comparably slow (Thakur *et al.*, 2018).

SNK P3, KA2 P3, and JAJPUT P3 crosses were examined as part of a study to minimize the cost of fungicides to prevent fusarium wilt, and they discovered a qualitative kind of resistance regulated by just a single gene (Manu *et al.*, 2014). Under both natural and artificial conditions, high-yielding and vulnerable cultivars were crossed with moderately cultivars, and it was discovered that a dominant single gene regulated the pattern of inheritance of fusarium wilt (Jabeen *et al.*, 2009).

Class II chitinase, a protein that is part of the class of protective proteins (PR proteins) and is encoded by the gene CaChi2, is also linked to resistance to fusarium wilt. Enzymes called chitinases (EC 3.3.14) operate on chitin to hydrolyze it (Suo and Leung, 2001). These particular enzymes are stimulated in plants when pathogens attack, gather at infection sites, and act on infection sites to decrease plants' susceptibility (Huang and Backhouse, 2006).

Non-traditional methods can be used on existing germplasms and can yield encouraging outcomes in limited time. Since, *Fusarium oxysporum* targets the base portion of a plant and invades the vascular system through the plant roots; so, hardening this region of the plant may contribute to the development of a resistance response in plants against this deadly disease. Many genes found in plants are connected to defensive processes and may be used to combat pathogens to defend against pathogenic onslaught. Such a gene, Solyc08g075770, has been discovered among mutants of tomato and is expressed in the plant root portion.

***Use of plant extracts*:** Several medicinal and decorative plant extracts were thought to have an inhibiting impact on fungus spores. Together, many botanists and plant pathologists assessed the effects of many extracts to discover their inhibitory effects against diseases of certain crops (Niaz *et al.*, 2008). Similar to this, Moringa leaf extracts, which are thought to have antifungal and antibacterial properties due to their caffeoylquinic acid, quercetin, zeatin, b-sit-sterol, and kaempferol content, had fungicidal activity against well-known soil-born fungi like *fusarium*, *Pythium and Rhizoctonia* (Katayon *et al.*, 2006). The capacity of 6 species of brassica, including *Brassica juncea*, *Brassica carinata*, *Brassica nigra*, and *Brassica* napus, was tested for their ability to suppress the growth of Fusarium spp. It was shown that *Brassica nigra* and *Brassica napus* prevented Fusarium species from growing radially by more than 50% (Mayton *et al.*, 1996).

By using the disc diffusion method, the effect of different plant extracts such as plucao and sabsua on Foc, the disease-causing pathogen of Fusarium wilt of chili, was

assessed. It was also discovered that subsua plant extract inhibited pathogen growth by 52.5 percent when compared to plucao extracts, which inhibited pathogen growth by 50.2 percent. Plant extracts such as groundnut cake, castor cake, neem cake, coconut cake and mustard cake were assessed in vitro against *F. oxysporum* using the poisoned food methodology. Neem cake (59.8) produced the most substantial outcomes than others (Yelmame *et al*., 2010).

***Use of endophyte microbes*:** Internal tissues of the host plants are colonized by beneficial endophyte. Numerous microbial endophytes play a role as biological control and enhance host plant growth (Sturz *et al*., 2000). Several isolates like *Bacillus pseudomycoides* strain NBRC 101232, *Bacillus thuringiensis* strain ATCC 10792 and *Bacillus mycoidesstrain* 273 found to have potential to be used for biocontrol of FOC and *R. syzigii subsp. indonesiensis* in chili pepper (Yanti *et al*., 2018).

***Mycoviruses*:** Mycoviruses belong to a group of viruses that attack, kill, or reproduce in fungi. They thus have enormous potential for use in combating fungus-based plant pathogens. Consequently, they are used to manage plant pathogenic fungi because they are their natural enemies (Xie and Jiang, 2014; Ghabrial *et al*., 2015). These can activate specific targets and, in some cases, to restrict RNA silencing, the fungus's natural response to viruses. Through the suppression of RNA silencing, viruses protect themselves from the fungus's antiviral response. In addition to downregulating genes involved in virulence and growth, mycoviruses also control the host fungus's gene expression.

Sclerotinia sclerotiorum 4 (SsMYR4) virus was used by Wu and his coworkers in 2017 to suppress the host's vital cellular processes and signaling pathways (Wu *et al*., 2017). Furthermore, hypo-virulence in plant pathogenic fungus such as *Fusarium graminearum is also induced* by FgV-ch9 (*Fusarium-graminearum*-virus-China-9)and FgV1(*Fusarium graminearum* viruses1) and FgV2 (*Fusarium graminearum* viruses2) (Darissa *et al*., 2012). As a result, there was a noticeable decrease in conidiation when *F. graminearum* isolate China 9 and dsRNA mycovirus (Fgv-ch9) were compared (Lemus-Minor *et al*., 2018). Lemus-Minor and his colleagues in 2018 used FodV1 (*F. oxysporum* f. sp. *dianthi* virus-1) to activate hypo-virulence in *F. oxysporum*. This reduced mycelial growth, conidiation, and virulence on carnation plants, implying that it functions as a biocontrol agent for Fusarium wilt of carnation (Lemus-Minor *et al*., 2018).

***Conclusions and Future Prospects*:** *Fusarium oxysporum* is a devastating pathogen of many crops especially vegetables including tomatoes, potatoes, peas, and bananas, etc. Chili a very valueable spice cum vegetable, having significance in global market is badly affected by *Fusarium oxysporum* f. sp. *capsici*. Since we live in modern era and population is rapidly growing, especially in Pakistan, food demands will be higher in the near future than they are now. Chili has a key part in supplying food requirements, and this can only be accomplished by expanding production, which is limited by certain factors; Fungal pathogens and diseases are one of the most influential elements restricting the yield of chili in Pakistan as well as around the world. The disease and pathogen are favored by many biotic and abiotic factors like soil and air temperature, soil humidity, soil texture, nutrients, soil pH, host plant virulence, alternative host etc. So, by proper knowledge about pathogen life cycle, alternate host, mode of spreading, surviving, molecular and chemical nature of disease and genetic information, many strategies could be adopted to minimize the impact of disease and pathogen on crop quality, quantity and economics. Seedling phase is the critical in this case, and incidence of disease can be significantly limited by implementing safety measures at this stage. Several methods have been highlighted and studied. This study will be helpful in future to provide advanced information about pathogen profile, mechanism, and eradication of fusarium wilt of chili.

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