

Fungi offering resistance against plant pathogens, fusarium oxysporum

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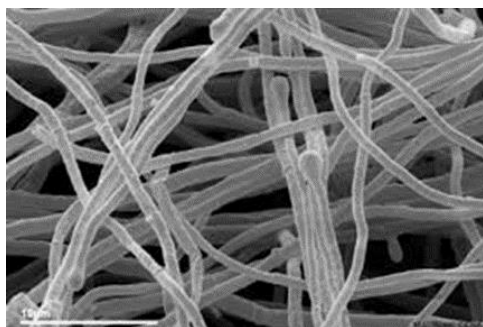
Abstract

Any host and the symbiont, commensal or pathogen interaction may be beneficial, mutual or disease-causing form. As the focus is on *Fusarium oxysporum* (Fo) in the present paper. Fo is known for causing diseases like Wilt syndrome in plant roots, apart from this Fo can also act as root endophyte and protecting plant roots from vascular pathogens like *Verticillium dahliae* and other Fo strains. Endophytes maintain balance between pathogen and host either through antagonising the microbes directly or by intervening in their interaction with the host. Fo provides protection against *Pythium ultimum*. The endophytic strains of Fo with the differ from pathogenic strains in gene loci, colonization mechanism in host, habitat, and host-response. The endophytic and pathogenic strains interact and the induced resistance, antibiosis, mycoparasitism, competition towards the plant pathogens is been discussed in the present paper.

Keywords: fusarium oxysporum, pathogens, fungal resistance

1. Introduction

The genetic and phenotypic populations of *Fusarium Oxysporum* (Fo) are present in a number of habitats ^[1]. In more than 120 plants imported into agriculture and horticulture, most of the Fo complex studies focused on disease pathogenic strains of plants ^[2]. In a recent international survey of fungal pathogens, Fo ranked fifth in the list of top 10 fungal plant pathogens based on scientific and economic importance ^[3]. Unlike the broad group of host species in the FOSC, each strain generally involves host species that are responsible for only one or some of the infectious symptoms of the medicinal plants ^[4]. Pathogenic isolates (f. spp.; plural; form is dependent on its host spectrum) are classified into different groups. Many FOSC members also infect species, including humans and insects ^[5]. The fungus has a haploid genome that has a wide range of karyotypes and is capable of segmental replication ^[6]. The horizontal movement of chromosomes within the complex species and probably across species boundaries appears to have led to the development of new pathogenic variants. Apart from possible interactions between homology TEs distributed across the genome, there tend to be new variants ^[8-10], transposable elements (TE) movement, and chromosome reorganization.



Source: [http:// www.promusa. org/ Fusarium+ oxysporum+ f.+ sp+cubense](http://www.promusa.org/Fusarium+oxysporum+f.+sp+cubense)

Fig 1: Fusarium oxysporum f. sp. Cubens.

Good phylogeny is crucial for the study of evolution, population biology, and reproduction of FOSC members. Clear answers to questions such as whether or not sexual reproduction is possible and whether the host specimen has been developed vertically or horizontally should support the development of effective disease management strategies and quarantine regulations ^[11]. While the phylogenetic relationship between FOSC and other fusaria is well established, the limits of species within the FOSC must be clearly addressed if the concept of 'species' is applied. Phylogenetic FOSC resolution ^[13-18] was evaluated for markers such as IGS ^[12] (intergenic spacer) of the RNA (rDNA) and EF-1a genes, polygalacturonases, mitochondrial small rDNA substrates, phosphate permeases, b-tubulins, nitrate reductions, and mating forms. Phylogenetic signs between markers tend to be sufficient for EF 1a and IGS rDNA only. 850 isolates reflecting the phylogenetic width of the FOSC are analyzed for the development of the Multilocus Sequence Type (MLST) database for the identification of human and pathogenic species. In one study such as this, 101 ^[21] EF-1a, 203 IGS rDNA, and 256 2-locus type (ST) FOSC were found to have a high genetic diversity. The ability to produce mycotoxins such as moniliformin, fumonisin, and enniatin was also evaluated in the resulting phylogenetic system. Conversely, not all *formae specialis* and VCGs-were distinguished by the phylogenetic resolution provided by this MLST dataset ^[19-25].

Classification

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Reproduction

The asexually reproductive essence of *Fusarium oxysporum* is usually considered because a teleomorph has never been detected or laboratory-injected. However, there can be no chance of a mysterious sex cycle. Both mating genes were found among the FOSC participants. While some studies supported Fo's clonality on the basis of all association, re-analyzing the data shows that there could be no exclusion from the possibility of recombination [26]. There are three types of asexual spores available. One or two of the cells are oval or elliptical microconidia. They are produced under a variety of conditions, such as liquid and solid growing media, within the rhizosphere and the vascular system of infected plants. Macroconidia is three or five cells, gradually pointing and bending to the ends. Macro-nidi is also present on the surface of degraded plants and sporogonia [27]. Long-term survival is ensured by thick-walled chlamydospores. The manufacture of chlamydospore in the form of older mycelium or macroconidia is terminal or intercalar and has been possible in the field for several years, making it a permanent restriction in pre-infested areas. The molecular mechanism behind the production of Fo spores is not understood. The application of genome-compatible methods (e.g. genetic and protein profiling of systemic mutagenesis of candidate genes) can therefore easily overcome the deficiency. Given the importance of fo spores in the reproduction of asexual diseases, new perspectives can be given to new forms of fo disease [28-29].

Eco-friendly roles

FOSC members are omnipresent in soil, distributed in various habitats, ranging from the tropics to the Arctic, including the grasslands, forests, and deserts. Although generally known as plant pathogens, saprophytes, or endophytes, asymptomatic colonizing plants [19, 13, 21] may not be pathogenic in survival. The study by Kul-dau and Yates showed that Fo endophytes are closely connected with much more plant species based on a small number of trials, in almost 100 plant species. Some apparently

nonpathogenic strains were successfully used to avoid soil pathogens like Fo itself as biocontrol agents [3]. Fortunately, it is difficult to test the zero hypothesis that saprophytic, endophytic or biopsy strains are not pathogenic to plants due to a large number of potential hosts [15].

History of *Fusarium oxysporum*

Over 91.3 million years ago O'Donnell *et al* identified diversified terminal *Fusarium* clade based on the two-loci model [30]. Geiser in later grouped the divergent clade into a genus *Fusarium*. *Fusarium* consists of 20 species, among which 16 are endophytes and four are plant pathogens [31]. Among these *Fusarium Oxysporum Species Complex* (FOSC) includes majority of the phytopathogens. Laurence *et al* subdivided this complex of FOSC into two PS1 and PS2. Most of pathogenic strains are in *Fusarium oxysporum* species comes under PS2 [32].

Phenology of *Fusarium oxysporum*

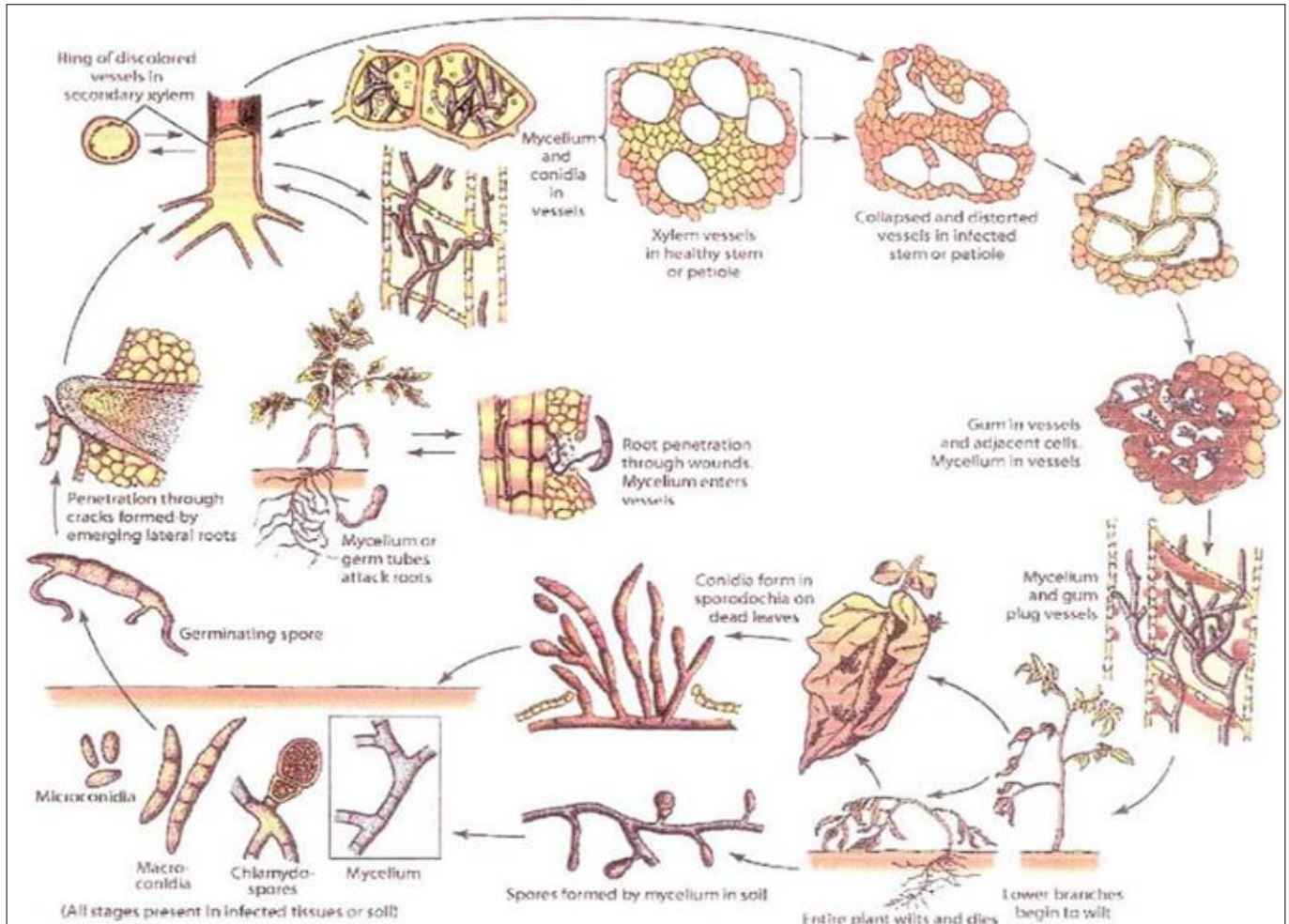
Snyder and Hansen found host specific strains (*FormaeSpeciales*) of Fo and described them as variants of same species [33]. Armstrong and Armstrong in 1981, explored Fo and discovered around hundred *formaespeciales* [34].

Lifecycle of *Fusarium oxysporum*

With the hyphae of Fo able to produce all types of spores like chlamydospores and conidiospores asexually, research predicts the fungal resistance developed through transgenerational diversity helps in the adaptation to the ecological biosystem changes. Fungistasis is the inability of spore to germinate due to bacteria producing fungistatic compounds or withdrawal of nutrients. Fungistatis is also considered as adaptive change of spore to be dormant till environment favourable for germination. Anaerobic soil disinfections and solarizations treatment methods were used to reduce the fungal pathogens in the soil.

Reports of Buxton on differentially affected strains of *Fof.sp.pisi* predicted with less spore germination in exudates from resistant pea plants than in susceptible pea plant [35]. Similarly, Wu *et al* *Fusarium* Wilt of watermelon caused by *Fo.niveum* have higher germination in susceptible cultivars than in resistant watermelon cultivars. A progressive study of this by inoculation of same strain on the root exudate of watermelon grafted on bottle guard, resistant to *Fusarium* wilt germinated at lower percentage than on susceptible watermelon root exudate [36]. Liu *et al* found higher concentrations of sucrose and glucose in the root exudates of cultivars susceptible to *Fusarium* wilt than the resistant cultivars [37]. Rather some strains of *f.sp.melonis* doesn't show differential spore germination on the susceptible and resistant cultivars of *Fusarium* Wilt in Muskmelon.

Hamilton *et al* stated for any parasite, allele outcrossing through periodic reshuffling is essential for changing the target through evolutionary changes of target genes. Till date there is no evidence of reproductive or sexual cycle for Fo under normal environmental conditions. Rather by experimental conditions, the sexual reproductive cycle were assessed by Linkage Disequilibrium and Horizontal Gene Transfer [38].



Source: <https://agronomie.info/en/2017/07/15/fusarium-wilt-tomato-disease-cycle-epidemiology/>

Fig 2: Life cycle of *Fusarium oxysporum*

Diseases caused by *Fusarium oxysporum*:

Fungal infections are more common in plants and some of the important steps in disease development include:

1. Attachment
2. Germination on plant surface
3. Penetration into host,
4. Colonization of the host.

A zone around the root where the effect of the root exudate on the germination capacity of spores is reported as Rhizosphere. It ranges from >1mm near root tip to less than 0.1mm at the subsequent root surface. Studies of Olivain and Stienberg observed germtubes growth towards soil rather than towards site of exudate [39, 40]. Wu F found the *F oxysporum*.sp.linii infection of flax was initiated by colonization of the root tip, whereas *F oxysporum*.sp.lycopersici colonised away from root tip. Fang *et al* *F oxysporum*.sp.fragariae spore adhered to root tips of Strawberry root in causing Fusarium Wilt of Strawberry [41]. With numerous studies and observations by researchers, some *Fo* strains penetration at the root tip was reported. Liu *et al* found that at the site of infection, the *Fosp* produce commonly a swelling on the hyphal surface known as appressoria. Based on the growth of *F oxysporum* on the host, they are of two types: Necrotrophic and Biotrophic. Biotrophes penetrated the host without macroscopic tissue damage and obtain the nutrients from the root cortex [37].

Fo being a soil borne organism found in the plant roots and soil is an endophyte generally. Whereas some strains of *Fo*

are known to cause devastating disease of plant known as Wilt Disease, Dean *et al* survey in 2012 ranks top 10 [42], and considered as major threat to agriculture by Fischer *et al* [43]. Apart from Wilt disease, *Fo* strains also cause foot or root-rot. As the strains produce micro, macro conidiospore and chlamydo-spore that remain viable in the soil for long period of time makes the soil to be infected for decades resulting in the crop yield loss. To overcome this, the cultivation of the fungal resistant plant is considered as economical disease control measure.

Interactions of *Fusarium oxysporum* with plant pathogens

The tripartite interaction of the host with pathogen and endophyte is crucial in the understanding and development of fungal resistance plants. There are four principles in the development of fungal resistance by plants:

1. Competition,
2. Antibiosis,
3. Mycoparasitism, and
4. Induction of Resistance.

All these mechanisms are involved in the development of resistance of plant to fungal pathogens especially *Fospecies* among this research towards induced resistance has been studied extensively to develop fungal resistant plants.

a. Competition
Zabalgogea in 2008, reported competitive exclusion as

determining factor in maintaining the composition of the plant ecological microbiome and inhibition of pathogenic microbe colonization by the endophytes [44]. Boyle *et al* stated intracellular or intercellular either local or systemic colonization of fungal endophytes [45]. Rodriguez *et al* observed fungal endophytes by rapid colonization and scavenging of nutrients available [46]. In 2009, Mohandoss and Suryanarayanan evidenced the colonization of different

fungi on elimination of specific endophyte in mango leaves [47]. However, competitive exclusion is always associated with other mechanism as this is a local phenomenon. To produce systemic effect of the endophyte competition can act as an adjunct for other systemically effective mechanisms. Hence in 2016 Card *et al* suggested, competitive exclusion not an essential mechanism in development the fungal resistance to plant pathogens [48].

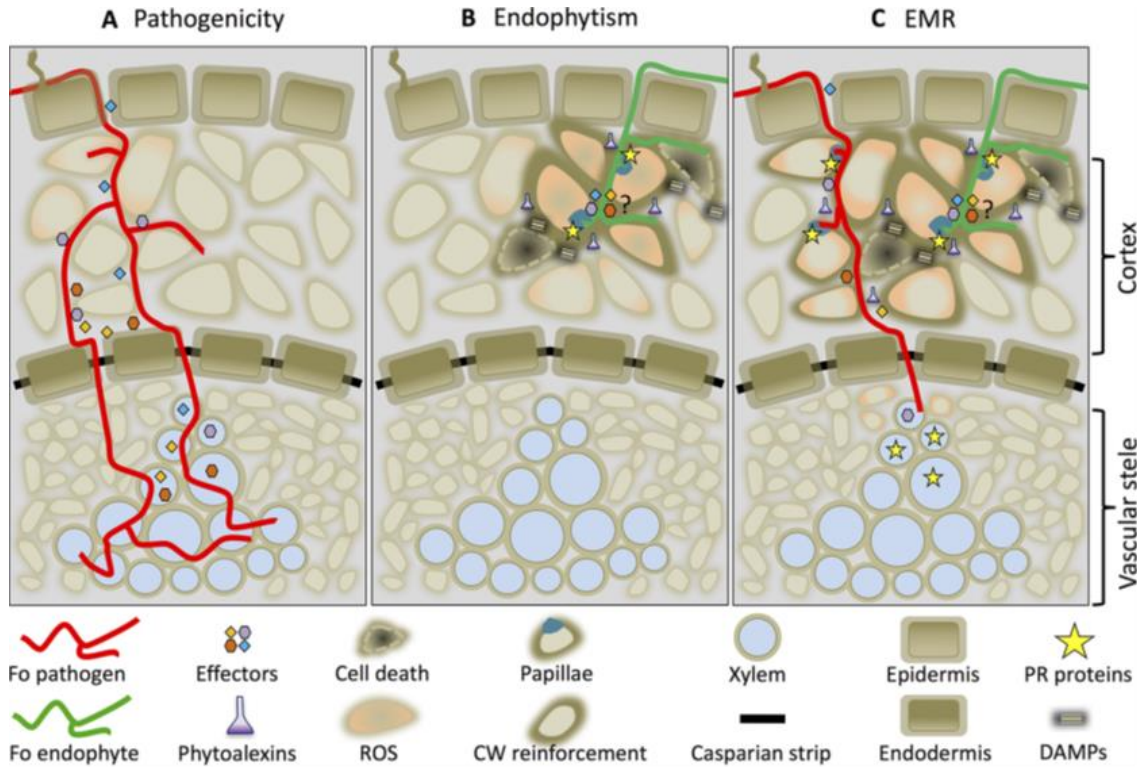


Fig 3

b. Antibiosis

In 1995, Dipterio stated Antibiosis as inhibition of phytopathogens directly by compounds secreted by endophytes [49]. Antibiosis is the process of inhibiting the pathogen by the compounds produced by the endophytes. In 2004 Thines *et al*, reported fungal endophytes as rich sources of metabolites that inhibits the pathogen of plant [50]. Through the extensive research by renowned scholars some natural products with antibiosis properties include volatile organic compounds, steroids, peptides, alkaloids, quinones, terpenoids, flavonoids, and phenols. Kusar *et.al*, in 2012 suggested presence of various microbes on plant trigger production of antibiosis metabolites from the host and endophytes together to suppress the phytopathogens [51]. Heining *et al* in 2013 through his evaluation in his study, concluded endophytes being dependent on host for their production antibiosis metabolites [52].

Authors like Alyet.al,Kusari *et.al* later evidenced the partial production or involvement of endophytes in a specific cycle to produce intermittent product and sharing with host [53, 54]. Antibiosis is very well exemplified by the fungal endophytes producing the Anti-Cancer drugs in *Taxus brevifolia*, the Pacific Yew tree [55]. With growing interest

and need to manage loss of crop yield due to fungal infection, extensive in-vitro trials on effect of individual antibiotic metabolite on controlling the plant pathogen. In-vitro trials by Köhl *et al.*, Deketelaere *et al.*, Laur *et al.*, indicated endophytes production of metabolites even without the presence of host and their inhibition of pathogens [55-57]. Sinha *et al.*,andMathivanan *et al* directly inoculated the metabolites, either by induced resistance or direct intoxication, produced by endophytes into plants and observed resistance exhibited by plants to phytopathogens [58, 59]. Kusari *et al.*, in 2012 tried production of different metabolite by modification of the factors or from the artificial media [60]. Young *et al.*, detected certain metabolites inducing antibiosis can be produced by specific gene expression that cannot be induced through in-vitro conditions like expression of alkaloid lolitrem from *Neotyphodiumlolli* only in cold season-grass [61]. Ideally contact of the anti-microbial compound with the pathogen is essential to demonstrate the antibiosis mechanism induced by metabolite in plants.As the concentration of metabolite is low and translocation of the metabolite to the site of infection occurs, it's difficult to demonstrate antibiosis in plants.

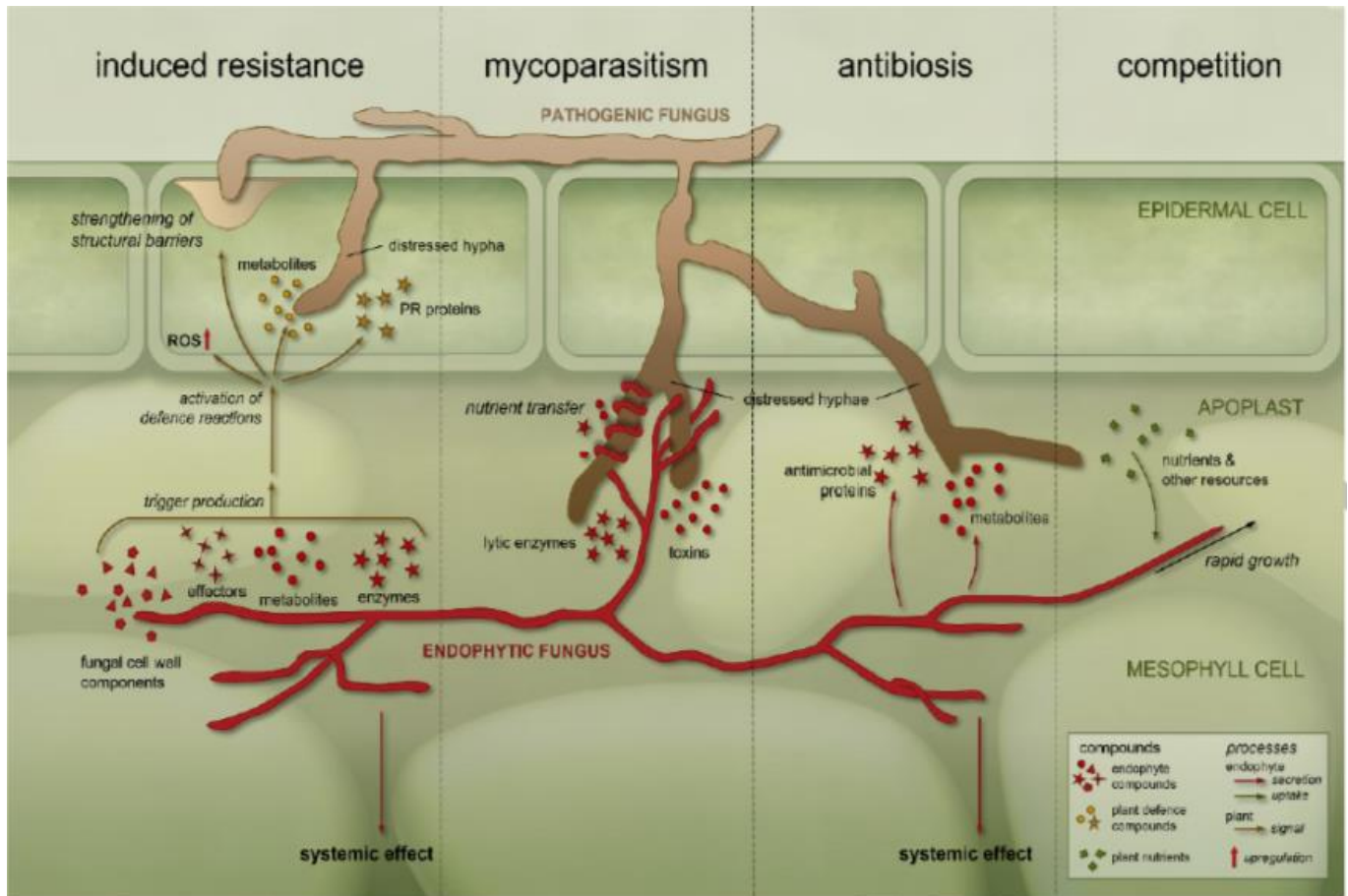


Fig 4

c. Mycoparasitism

Mycoparasitism is a mechanism by which one fungus obtains nutrients from another fungus. As per, Jeffries *et al.*, Horwitz *et al.*, and Kim *et al.*, a Biotrophes obtain nutrients from the host with host being alive in this interaction whereas Necrotrophe leads to cell death in the process of obtaining nutrient from the host [61-63].

In-vivo demonstration of mycoparasitism is quite challenging due to lack of evidence to confirm the nutrient transfer. Hence Jeffries, considered Mycoparasitism as a circumstantial evidence. Close association of two fungi is fungicolous, where nutrient transfer is not reported. Mycoparasitism is always tested in-vitro and evidenced by studies of Donayre and Dailsay [64]. Card *et al* suggested mycoparasitism as not the control principle in the Biocontrol mechanism [48].

d. Induction of Resistance

This is artificial way of inducing resistance to plants by an agent that triggers the gene loci susceptible to disease to initiate the differentiation of the expression of genes, protein synthesis and specific metabolic changes that makes plants resistant to develop disease. In 1992 Kloepper, defined Induced resistance as “the process of active resistance dependent on the host plant’s physical and chemical barriers, activated by biotic and abiotic agent”.The protection of plant from the pathogen by the activation of the defense response of the plant is called Priming” [65]. Cornath in 2012 termed priming as “Induced resistance associated with an enhanced capacity to mobilize infection-induced cellular defense response [66].

Induced resistance by endophytes to pathogenic microbes is an important mechanism employed in Control of infection.

Wani and Xu *et al* in 2015 compared the plant response to plant pathogens and endophytes is similar but differ only in immune mechanism [67, 68]. Nurnberger and Kemmerling in 2009 stated, Pathogen associated molecular Patterns (PAMP) or Microbes Associated Molecular Patterns (MAMP) are recognised by receptors in plants to induce immunity (69). In 2004 Lyon *et al.*, found these MAMP/PAMP in fungal cell wall components like Chitin and β -glucan [70]. Luo *et al* in 2010, evidenced fungal compounds produced by endophytes compete with pathogens to induce resistance to plants [71].

Conventionally, Systemic resistance is categorised into two types: Systemic Acquired Resistance (SAR) and Induced Systemic Resistance(ISR).Pieterse in 2012 enlightened hormones like Salicylic acid in SAR and Ethylene and Jasmonic acid in ISR play prime role. Furthermore, there are studies stating development of resistance independent of these two mechanisms in certain plant species [72].

In this review, we concentrate on the plant pathogens that provide resistance to *Fusarium oxysporum*.

Fusarium oxysporum and fungal resistance to plant pathogens:

Wolfe *et al* in 1989, observed in monocots and dicots around 200 diversified *Fo* strains suggesting fungal resistance as ancient phenomenon [73]. Bao *et al* in 2004 in his study isolated 200 different endophytes of *Fo* from tomato field had resistance to plant pathogens. *Fo* can suppress the oomycete-caused diseases too [74]. In 2009, Le Floch, *Fo* observed resistance to *Pythumoligandrum* in tomato, [75] Benhamau *et al* observed resistance offered by *Fo* to *Pythium ultimum* in cucumber, [76] and similarly

Velsao and Díaz in 2004 found similar resistance to *Phytophthora capsica* in Pepper. All these pathogens are root infecting and Fo inhibits root colonization of these pathogens and protect the vasculature [77].

Biles and Martyn, 1989 induced resistance to *Colletotrichum lagenarium* through pre-inoculation of *Fof.sp.cucumerinum* to watermelon roots and detected reduction in lesion size [78]. Díaz on preinoculation of Fol into pepper showed enhanced resistance to *Botrytis cinerea*. Kroon *et al* induced resistance in tomato plants (*S.lycopersicum*) against the pathogen Fol by inducing *Fof.sp.dianthi* [79]. Fo 47 induced resistance against Fol8/Fol8B in tomato plant evidenced by Fuchs *et al.* [80] in 1997. Blok in 1997, reported Fo 47 offering fungal resistance by competition for the nutrient with Foa offered pathogen resistance in Asparagus [81]. Extensive studies by numerous authors have developed fungal resistance to pathogens in tomato. Nel *et al.*, in 2006 stated resistance to *f.sp.cubense* in banana plant by Fo 47 [82]. Kaur and Singh in 2007 induced resistance against *Fof.sp.ciceri* in chickpea by Fo 52 [83]. In eggplant (*S.melongena*), root pre-inoculated with the endophyte F2 and inoculated with pathogen, *V.dahliae* developed resistance to pathogen. Pu *et al.*, in 2014, in cucumber plant pre-inoculated CS-20 and inoculated Foc in the roots and observed induced resistance [84].

Pathogens breeds

Special shapes also include multiple pathogenic breeds identified by virulence patterns of host plant varieties which are differentially resistant. In a specialized form, virulence-related mutations may lead to new races [12]. The polyphyletic *f.sp.lycopersici* is the best proof of how this could happen. To date, 14 effector genes that encode small proteins secreted by tomatoes have been found in *f.sp.lycopersici*. Three of the genes also identified for AVR are identified, corresponding to three known pathogenic races, i.e. *f.sp.lycopersici*. AVR2 and AVR3 effectors are identified as resistant (R) to full-virulence in tomatoes, respectively I-2 and I-3. I-2 and I-3 are suppressed by host defenses, but I and I-1 proteins are recognized. New breeds have been identified by a mutation in several clonal lines and have apparently been selected for widespread use to contain resistant cultivars I / or I-2 [12, 14, 10, 30]. Race 2 seems to be a result of the loss of AVR1, by the loss of a genomic region, several times from Race 1.

Post-recognition changes in AVR2 have subsequently led to I-2 losses, resulting in the emergence of race 3. Race 3 seems to have also evolved several times independently, considering that there have been three separate point mutations in the AVR2 gene and that the isolates from race 3 can be closely associated in the same region to race 2 isolates. AVR genes in other form specialties have not been identified yet and it has not been demonstrated that pathogenic races for other form specials match in the gene-for-gene host model [17, 26].

Pathogenicity and Host Specificity Models for functional Study

In view of the intimate and complex relationship between Fo and its hosts throughout the disease cycle, the molecular mechanisms underlying colonization and disease progression are important for understanding. The power to control genetically the host. *Arabidopsis thaliana* and

tomato have been widely used as experimental hosts since their rich genetic resources and ability to treat [19]. Our standard for protection, sensitivity, and disease spread mechanisms for various disease-causing species, such as viruses, fungi, nematodes, and insects, has significantly improved with the use of *A.thaliana* as a model [12].

A.thaliana has been used by a variety of studies to study plant reactions to Fo infection in host and pathogen, and the genetic conditions for Fo colonization. Many *A.thaliana* ecotypes were demonstrated to interact differently with various FOSC strains, providing materials for identification and mapping (24). The first of these genes is RFO1, the receptor-like kinase (RLK), that provides non-race resistance to Fo. All RFO2 and RFO3 encode RLP and RLK with *Fo f.sp.matthioli* resistance. The use of its large mutant tools has been used to investigate functions and signaling pathways for the specific defense or fo genes for *A.thaliana* [16]. As mentioned, the small size of *A.thaliana* has made it possible to grow and infect Fo in cameras using a glass base, which causes the colonization and penetration of *A.thaliana* roots by Fo without damaging the physical roots. The association of Tomato *Fof.sp.lycopersici* (Fol) was used primarily to classify the causes of FO pathogenicity and molecular resistance to tomatoes in the Fo region [10]. The I-2 resistance gene is a class of NB-LRR proteins that is predominantly present in the xylene tissue of the vessel. The I-2 gene encodes the NB-LRR class. Tissue I-2 is the most commonly used resistance gene. In cytoplasmic dances with NB-LRR proteins, co-spendig avirulence proteins from Fol, AVR2, have been associated with intracellular receptors due to the detection of Xylem sap from fol-infected tomato plants [16].

In addition to the observation that the role of hormones and the various signaling pathways were scarcely explored in the interaction of Tomato-Fol, unripe (e.g. xylene-insensitive) tomato plants showed reduced symptoms following Fol infection [22].

The pathogen in Human or animals

In addition to infecting different species, certain FOSC memory bodies often contribute to localized or highly invasive human infections, resulting in very high immunodeficiency mortality in patients. With Fusarium keratitis outbreaks in Asia and the U.S.A. [16], Fo may also affect immune-trained, often blinding humans.

A detailed phylogenetic analysis of 850 isolates shows various special forms and human pathogens that, in the three main clades of FOSC phylogenetics, genetically diverse isolates are nested associated with an opportunistic infection of the human nail corresponding to different sequences (STs) [12]. Insect-related Fusaria comprises the phylogenetic respiration of 10 species complexes, including FOSC. The relationship to human and plant diseases, as well as strains, has been investigated by multilocus phylogenetic testing [5] in order to investigate the use of insecticide fusaria as biological control agents in insect pests and to minimize the possibility of accidental use of plant and/or animal pathogens as biological control agents.

Structure of the genome and advancement Structure and remarkable characteristics

Annotations of 11 Fo strains genome sequence are available publicly in the platform of the Broad Institute Fusarium Comparative Genomics, and additional strains are currently

sequenced [5, 17]. The first to be decoded was the 4287 genome, which was pathogenic to the tomato, and was achieved through the San-ger-method whole genome (6X coverage). Physical fittings were created on the optical map of its chromosomes, which led to the adjacent anchor series mounting of the genome. The large platform explains the sequence and mount strategy of this and other Fo strains and provides genome and gene-associated statistics [19, 7, 12].

Structure of the genome

The most remarkable feature of Fo genomes, when compared to *F. graminearum* (Fg) and *F. verticillioides* (Fv) genomes [13, 8, 22], was the presence of large "accessory" chromosomes and chromosomal zones (also known as line-specific (LS) chromosomes and zones). The regions do not synthesize to Fg and Fv genome sequences and are typically specific to evolutionary histo-Ries core genomes. In chromosome [11] size, gene size (* 14,200), and sequence (91 percent mean ornithological identity), Fo's core genome is the same as Fv's genome. [3, 7] of them. For example, transposon-rich accessory genomes have been identified in *Fusarium solani* (= *Nectria haematococca*), *Alternaria dahliae*, and TE-rich chromosomal subregions such as leptosphaeriaculans and *Verticillium dahliae*.

Transfer of Chromosome Interstrain as a Genome Innovation Mechanism

Apart from the location of the genome, the horizontal transfer is the driving force behind the dynamics of the genome of Fo, duplicating and deleting it within the genome. The sex cycle Fo was not observed either in nature or in the laboratory, but there were two strains mixed on the plaster resulting in one or more LS chromosomes [7, 19, 20]. Translations to the genome are possible. The chromosome strain of the recipient may be more virulent to the host. With the availability of medical resistance markers in the laboratory to identify these unusual events and to evaluate the resultant strains, this method would significantly lead to major genetic variations in the soil/plant tissue strain of Fo strain and to the creation of new pathogenic clonal lines for different plant species [30].

It is uncertain how it operates. Following hyphal fusion, chromosome transmission may involve either nuclear fusion or may be triggered either by missing the majority of the parent genome or by taking one or more nucleus colors from 'Parents' or by analyzing the horizontal transmission of LS chromosomes and/or small chromosomes [23].

Identification of strains and phylogenetic analysis

The incorrect and misleading use of the names of organisms was an important obstacle to this research. It's a *Fusarium*. Documentation of *Fusarium's* global diversity and identification of old and new problems caused by *Fusarium* remain fragmented without a robust phylogenetic framework that guides the identification of species and strains, creating uncertainty rather than a taxonomy [27]. Large molecular phylogenetic studies have been conducted and relations will continue to be clarified on the basis of the above-mentioned public cultures at different taxon levels.

On the basis of the findings, web-based community-based networks have been established to enable researchers around the world to identify *fusarium* quickly and accurately. In the early 2000s, *Fusarium* ID v.1.0 was launched and became the first generation of *Fusarium* Identifiers online databases

via EF-1a [18].

Technology advancement and Fluorescence microscope

New advances in the production of microscopic and fluorescence microscopy instruments provide an enormous opportunity for research into the biological structure – functional connections between different molecules and cells and tissues [9]. These methods, combined with increasingly genomic data and various fluorescent proteins (FP) and FP sensors, are of invaluable value for understanding biological cessation at the cellular level and help to investigate the functioning and dynamics of individual gene-products linked to organic and organism-environmental interactions. In this context, a number of these resources for the analysis of Fo are available to researchers [7, 15, 17]. The marking of molecules and species requires a wide range of natural and manufactured fibers with different biochemical and physical properties. FPs are excellent markers for the multiphoton/confocal imaging dynamics of individual proteins, organs and species since they do not require any fluorescence substrates or co-factors [29].

Applications in Fusarium oxysporium:

Eventhough *Fusarium oxysporium* is known to cause the infections in Tomato, Potatoes, Pepper, Eggplants, Banana, Watermelon, Strawberries, Sugarcane, Lettuce, Palm oil plants.

Almeda suggested *Fusarium* like other fungi, bacteria and yeast secrete enzymes like pectinase making them used in food industry for ripening of fruits like grapes to produce wine, extract pulp from tomatoes, fermentation of tea and chocolate and the degumming of fibre [85]. Isabel, Soares *et al.*, enhanced the application of enzyme pectinase for concentrating anti-oxidants in virgin oil. Enzyme Cellulase produced by *Fusarium*, a hydrolytic compound was used in detergent, textile, paper, cosmetic, food, and drug industries [86]. For the production and clarification of orange vinegar and citrus juice respectively. McPartland John used *Fusarium* as biocontrol agent against the *Cannabis sativa* to control the cultivation of Marijuana [87]. Biocontrol mechanism of Fo against striga weeds has been controversial between Avedi *et al.*, and Ciotola and Watson *et al* [88, 89].

Conclusion

With very low application of Fo as biocontrol agent with the focus on the aggressive disease, Wilt syndrome, caused by Fo an extensive *in planta* trial and research for the application of endophytic strains of Fo in controlling the plant pathogens by offering fungal resistance induced by Competition, Antibiosis, Mycoparasitism and Induced Resistance mechanism and developing disease resistant Plants will be effective, economical and biocompatible way to enhance the production of Crop overall.

Summary

Fusarium oxysporium well known for causing devastating Wilt Syndrome leading to wide range of crop loss has also diversified endophytic strains that provide fungal resistance towards plant pathogens. The development of Biocontrol agents and inducing them in susceptible plants gained attention of Fo endophytes and their application in inducing fungal resistance in plant towards phytopathogens. Future research on application of *Fusarium oxysporium* endophytic

strains is need of the hour in combating the plant pathogenic microbes.

References

- Dara SK, Dara SS, Dara SS, Anderson T. First report of three entomopathogenic fungi offering protection against the plant pathogen, *Fusarium oxysporum* f. sp. *vasinfectum*. UC ANR eJournal Strawberries and Vegetables. EJ. Entomol. Biol, 2016.
- Khang CH, Park SY, Rho HS, Lee YH, Kang S. Filamentous fungi (*Magnaporthe grisea* and *Fusarium oxysporum*). In *Agrobacterium Protocols*. Humana Press. 2006; 2:403-420.
- Trapero Casas A, Jiménez Díaz RM. Fungal wilt and root rot diseases of chickpea in southern Spain.
- Parkhi V, Kumar V, Campbell LM, Bell AA, Shah J, Rathore KS, *et al.* Resistance against various fungal pathogens and reniform nematode in transgenic cotton plants expressing *Arabidopsis* NPR1. *Transgenic research*. 2010; 19(6):959-75.
- Kistler HC, Benny UK. Genetic transformation of the fungal plant wilt pathogen, *Fusarium oxysporum*. *Current Genetics*. 1988; 13(2):145-9.
- Boyette CD, Abbas HK, Connick WJ. Evaluation of *Fusarium oxysporum* as a potential bioherbicide for sicklepod (*Cassia obtusifolia*), coffee senna (*C. occidentalis*), and hemp sesbania (*Sesbania exaltata*). *Weed Science*. 1993; 41(4):678-81.
- Jongedijk E, Tigelaar H, Van Roekel JS, Bres-Vloemans SA, Dekker I, van den Elzen PJ, *et al.* Synergistic activity of chitinases and β -1, 3-glucanases enhances fungal resistance in transgenic tomato plants. *Euphytica*. 1995; 85(1-3):173-80.
- Larkin RP, Fravel DR. Efficacy of various fungal and bacterial biocontrol organisms for control of *Fusarium* wilt of tomato. *Plant disease*. 1998; 82(9):1022-8.
- Mullins ED, Chen X, Romaine P, Raina R, Geiser DM, Kang S, *et al.* *Agrobacterium*-mediated transformation of *Fusarium oxysporum*: an efficient tool for insertional mutagenesis and gene transfer. *Phytopathology*. 2001; 91(2):173-80.
- Inoue I, Namiki F, Tsuge T. Plant colonization by the vascular wilt fungus *Fusarium oxysporum* requires FOW1, a gene encoding a mitochondrial protein. *The Plant Cell*. 2002; 14(8):1869-83.
- Peschen D, Li HP, Fischer R, Kreuzaler F, Liao YC. Fusion proteins comprising a *Fusarium*-specific antibody linked to antifungal peptides protect plants against a fungal pathogen. *Nature biotechnology*. 2004; 22(6):732-8.
- Dean R, Van Kan JA, Pretorius ZA, Hammond-Kosack KE, Di Pietro A, Spanu PD, *et al.* The Top 10 fungal pathogens in molecular plant pathology. *Molecular plant pathology*. 2012; 13(4):414-30.
- Newsham KK, Fitter AH, Watkinson AR. Arbuscular mycorrhiza protect an annual grass from root pathogenic fungi in the field. *Journal of ecology*, 1995, 991-1000.
- El-Khallal SM. Induction and modulation of resistance in tomato plants against *Fusarium* wilt disease by bioagent fungi (arbuscular mycorrhiza) and/or hormonal elicitors (jasmonic acid & salicylic acid): 2-changes in the antioxidant enzymes, phenolic compounds and pathogen related-proteins. *Aust J Basic Appl Sci*. 2007; 1(4):717-32.
- Hao Z, Christie P, Qin L, Wang C, Li X. Control of fusarium wilt of cucumber seedlings by inoculation with an arbuscular mycorrhizal fungus. *Journal of Plant Nutrition*. 2005; 28(11):1961-74.
- Woo SL, Zoina A, Del Sorbo G, Lorito M, Nanni B, Scala F, Noviello C, *et al.* Characterization of *Fusarium oxysporum* f. sp. *phaseoti* by Pathogenic Races, vcgs, rflps, and RAPD. *Phytopathology-New York and Baltimore Then St Paul*. 1996; 86:966-73.
- Imazaki I, Kurahashi M, Iida Y, Tsuge T. Fow 2, a Zn (II) 2Cys6-type transcription regulator, controls plant infection of the vascular wilt fungus *Fusarium oxysporum*. *Molecular microbiology*. 2007; 63(3):737-53.
- Zizzerini A, Tosi L. Antagonistic activity of fungi isolated from sclerotia of *Sclerotinia sclerotiorum*. *Plant Pathology*. 1985; 34(3):415-21.
- Daboussi MJ, Langin T. Transposable elements in the fungal plant pathogen *Fusarium oxysporum*. *Genetica*. 1994; 93(1-3):49-59.
- Ghag SB, Shekhawat UK, Ganapathi TR. Host-induced post-transcriptional hairpin RNA-mediated gene silencing of vital fungal genes confers efficient resistance against *Fusarium* wilt in banana. *Plant biotechnology journal*. 2014; 12(5):541-53.
- Arfaoui A, Sifi B, Boudabous A, Hadrami IE, Cherif M. Identification of *Rhizobium* isolates possessing antagonistic activity against *Fusarium oxysporum* f. sp. *ciceris*, the causal agent of *Fusarium* wilt of chickpea. *Journal of Plant Pathology*, 2006, 67-75.
- Kang S, Demers J, del Mar Jimenez-Gasco M, Rep M. *Fusarium oxysporum*. In *Genomics of Plant-Associated Fungi and Oomycetes: Dicot Pathogens*, 2014, 99-119. Springer, Berlin, Heidelberg.
- Girhepuje PV, Shinde GB. Transgenic tomato plants expressing a wheat endochitinase gene demonstrate enhanced resistance to *Fusarium oxysporum* f. sp. *lycopersici*. *Plant Cell, Tissue and Organ Culture (PCTOC)*. 2011; 105(2):243-51.
- Combès A, Ndoye I, Bance C, Bruzaud J, Djediat C, Dupont J, *et al.* Chemical communication between the endophytic fungus *Paraconiothyrium variabile* and the phytopathogen *Fusarium oxysporum*. *PLoS One*, 2012, 7(10).
- Utkhede R. Increased growth and yield of hydroponically grown greenhouse tomato plants inoculated with arbuscular mycorrhizal fungi and *Fusarium oxysporum* f. sp. *radicis-lycopersici*. *Biocontrol*. 2006; 51(3):393-400.
- Maria das Graças MF, Gomes VM, Corsini RE, Machado OL, De Simone SG, Novello JC, *et al.* Isolation and partial characterization of a novel lectin from *Talisia esculenta* seeds that interferes with fungal growth. *Plant Physiology and Biochemistry*. 2002; 40(1):61-8.
- Liu S, Ruan W, Li J, Xu H, Wang J, Gao Y, Wang J. Biological control of phytopathogenic fungi by fatty acids. *Mycopathologia*. 2008; 166(2):93-102.
- del Mar Jiménez-Gasco M, Milgroom MG, Jiménez-Díaz RM. Stepwise evolution of races in *Fusarium oxysporum* f. sp. *ciceris* inferred from fingerprinting with repetitive DNA sequences. *Phytopathology*. 2004; 94(3):228-35.

29. Srivastava R, Khalid A, Singh US, Sharma AK. Evaluation of arbuscular mycorrhizal fungus, fluorescent *Pseudomonas* and *Trichoderma harzianum* formulation against *Fusarium oxysporum* f. sp. *lycopersici* for the management of tomato wilt. *Biological control*. 2010; 53(1):24-31.
30. O'Donnell K, Kistler HC, Cignelnik E, Ploetz RC. Multiple evolutionary origins of the fungus causing Panama disease of banana: concordant evidence from nuclear and mitochondrial gene genealogies. *PNAS*. 1998; 95(5):2044-49.
31. Geiser DM, Aoki T, Bacon CW, Baker SE, Bhattacharyya MK. One fungus, one name: defining the genus *Fusarium* in a scientifically robust way that preserves longstanding use. *Phytopathology*. 2013; 103:400-8.
32. Laurence MH, Summerell BA, Burgess LW, Liew EY. Genealogical concordance phylogenetic species recognition in the *Fusarium oxysporum* species complex. *Fungal Biol*. 2014; 118:374-84.
33. Snyder WC, Hansen HN. The species concept in *Fusarium*. *Am. J. Bot.* 1940; 27(2):64-67.
34. Armstrong GM, Armstrong JK. another approach to race classification of *Fusarium oxysporum* f. sp. *pisi*. *Phytopathology*. 1981; 71(5):474-478. doi: 10.1094/Phyto-71-474.
35. Buxton EW. Some effects of pea root exudates on physiologic races of *Fusarium oxysporum* Fr. f. *pisi* (Linf.) Snyder and Hansen. *Trans. Br. Mycol. Soc.* 1957; 40:145-54.
36. Wu F, Liu B, Zhou X. Effects of root exudates of watermelon cultivars differing in resistance to *Fusarium oxysporum* f. sp. *niveum*. *Allelopath. J.* 2010; 25(2):403-14.
37. Liu JJ, Sturrock R, Ekramoddoullah AKM. The superfamily of thaumatin-like proteins: its origin, evolution, and expression towards biological function. *Plant Cell Rep.* 2010; 29(5):419-436. doi: 10.1007/s00299-010-0826-8.
38. Hamilton WD, Axelrod R, Tanese R. Sexual reproduction as an adaptation to resist parasites (areview). *PNAS*. 1990; 87:3566-73.
39. Steinberg C, Whippis JM, Wood D, Fenlon J, Alabouvette C. Mycelial development of *Fusarium oxysporum* in the vicinity of tomato roots. *Mycol. Res.* 1999; 103(6):769-78.
40. Olivain C, Humbert C, Nahalkova J, Fatehi J, L'Haridon F. Colonization of tomato root by pathogenic and nonpathogenic *Fusarium oxysporum* strains inoculated together and separately into the soil. *Appl. Environ. Microbiol.* 2006; 72(2):1523-31.
41. Wu F, Liu B, Zhou X. Effects of root exudates of watermelon cultivars differing in resistance to *Fusarium oxysporum* f. sp. *niveum*. *Allelopath. J.* 2010; 25(2):403-14.
42. Dean R, Van Kan JAL, Pretorius ZA, Hammond-Kosack KE, Di Pietro A, Spanu PD, et al. The top 10 fungal pathogens in molecular plant pathology. *Mol. Plant Pathol.* 2012; 13(4):414-430. doi: 10.1111/j.1364-3703.2011.00783.x
43. Fisher MC, Henk DA, Briggs CJ, Brownstein JS, Madoff LC, McCraw SL, et al. Emerging fungal threats to animal, plant and ecosystem health. *Nature*. 2012; 484(7393):186-194. doi: 10.1038/nature10947.
44. Zabalgoitia Z. Fungal endophytes and their interaction with plant pathogens. *Spanish J Agric Res.* 2008; 6(S1):138-146.
45. Boyle C, Gotz M, Dammann-Tugend U, Schulz B. Endophyte-host interactions III. Local vs. systemic colonization. *Symbiosis*. 2001; 31(4):259-281.
46. Rodriguez RJ, White JF, Arnold aE, Redman RS, White Jr JF, Arnold aE, et al. Fungal endophytes: diversity and functional roles. *New Phytol.* 2009; 182(2):314-330.
47. Mohandoss J and Suryanarayanan TS. Effect of fungicide treatment on foliar fungal endophyte diversity in mango. *Sydowia*. 2009; 61(1):11-24.
48. Card S, Johnson L, Teasdale S, Caradus J. Deciphering endophyte behaviour: The link between endophyte biology and efficacious biological control agents. *FEMS Microbiol Ecol.* 2016; 92(8):1-19.
49. Dipietro A. Fungal antibiosis in biocontrol of plant disease. In: *Allelopathy*, 1995, 271-279.
50. Thines E, Anke H, Weber RWSS. Fungal secondary metabolites as inhibitors of infection-related morphogenesis in phytopathogenic fungi. *Mycol Res.* 2004; 108:14-25.
51. Kusari S, Hertweck C, Spiteller M. Chemical ecology of endophytic fungi: Origins of secondary metabolites. *Chem Biol.* 2012; 19(7):792-798.
52. Heinig U, Scholz S, Jennewein S. Getting to the bottom of taxol biosynthesis by fungi. *Fungal Divers.* 2013; 60(1):161-170.
53. Aly AH, Debbab A, Proksch P. Fungal endophytes - secret producers of bioactive plant metabolites. *Pharmazie*. 2013; 68(7):499-505.
54. Ludwig-Müller J. Plants and endophytes: equal partners in secondary metabolite production? *Biotechnol Lett.* 2015; 37(7):1325-1334.
55. Köhl J, Postma J, Nicot P, Ruocco M, Blum B. Stepwise screening of microorganisms for commercial use in biological control of plant-pathogenic fungi and bacteria. *Biol Control.* 57(1):1-12.
56. Deketelaere S, Tyvaert L, França SC, Hofte M. Desirable traits of a good biocontrol agent against *Verticillium* wilt. *Front Microbiol.* 2017, 8.
57. Laur J, Ramakrishnan GB, Labbé C, Lefebvre F, Spanu PD, Bélanger RR, et al. Effectors involved in fungal-fungal interaction lead to a rare phenomenon of hyperbiotrophy in the tritrophic system biocontrol agent-powdery mildew-plant. *New Phytol.* 2018; 217(2):713-725.
58. Sinha AK Trivedi N. Immunization of rice plants against *Helminthosporium* infection. *Nature*. 1969; 223(5209):963-964.
59. Mathivanan N, Prabavathy VR, Vijayanandraj VR. The effect of fungal secondary metabolites on bacterial and fungal pathogens. Springer, Berlin, Heidelberg, 2008, 129-140.
60. Young CA, Felitti S, Shields K, Spangenberg G, Johnson RD, Bryan GT, et al. A complex gene cluster for indole-diterpene biosynthesis in the grass endophyte *Neotyphodium lolii*. *Fungal Genet Biol.* 2006; 43(10):679-693.
61. Jeffries P. Biology and ecology of mycoparasitism. *Can*

- J Bot. 1995; 73(S1):1284-1290.
62. Horwitz BA, Viterbo A. Mycoparasitism. In: Cellular and Molecular Biology of Filamentous Fungi. American Society of Microbiology, 2010, 676-693.
 63. Kim SH, Vujanovic V. Relationship between mycoparasites lifestyles and biocontrol behaviors against *Fusarium* spp. and mycotoxins production. Appl Microbiol Biotechnol. 2016; 100(12):5257-5272.
 64. Donayre DKM, Dalisay TU. Identities, characteristics, and assemblages of dematiaceous-endophytic fungi isolated from tissues of barnyard grass weed. Philipp J Sci. 2016; 145(2):153-164.
 65. Kloepper JW, Tuzun S, and Kuć JA. Proposed definitions related to induced disease resistance. Biocontrol Sci Technol. 1992; 2(4):349-351.
 66. Conrath U, Pieterse CMJ, Mauch-Mani B. Priming in plant-pathogen interactions. Trends Plant Sci. 2002; 7(5):210-216.
 67. Wani ZA, Ashraf N, Mohiuddin T, Riyaz-Ul-Hassan S. Plant-endophyte symbiosis, an ecological perspective. ApplMicrobiolBiotechnol. 2015; 99(7):2955-2965.
 68. Xu X, Wang C, Li S, Su Z, Zhou H, Mao L, *et al.* Friend or foe: differential responses of rice to invasion by mutualistic or pathogenic fungi revealed by RNAseq and metabolite profiling. Nat Sci Reports. 2015; 5(13624):1-14.
 69. Nürnberger T, Kemmerling B. Pathogen-associated molecular patterns (PAMP) and PAMP-triggered immunity. In: Annual Plant Reviews. Oxford, UK: Wiley-Blackwell, 2009, 16-47.
 70. Lyon GD. Agents that can elicit induced resistance. In: Induced resistance for plant defense: a sustainable approach to crop protection, 2014, 11-40.
 71. Luo Y, Zhang DD, Dong XW, Zhao PB, Chen LL, Song XY, *et al.* Antimicrobial peptaibols induce defense responses and systemic resistance in tobacco against tobacco mosaic virus. FEMS Microbiol Lett. 2010; 313(2):120-126.
 72. Pieterse CMJ, Van der Does D, Zamioudis C, Leon-Reyes A, Van Wees SCM. Hormonal modulation of plant immunity. Annu Rev Cell Dev Biol. 2012; 28(1):489-521.
 73. Wolfe KH, Gouy ML, Yang YW, Sharp PM, Li WH. Date of the monocot dicot divergence estimated from chloroplast DNA-Sequence Data. PNAS. 1989; 86(16):6201-6205. doi: 10.1073/pnas.86.16.6201.
 74. Bao J, Fravel D, Lazarovits G, Chellemi D, van Berkum P, O'Neill N. Biocontrol genotypes of *Fusarium oxysporum* from tomato fields in Florida. Phytoparasitica. 2004; 32(1):9-20.
 75. Le Floch G, Vallance J, Benhamou N, Rey P. Combining the oomycete *Pythium oligandrum* with two other antagonistic fungi: Root relationships and tomato grey mold biocontrol. Biol. Control. 2009; 50(3):288-298. doi: 10.1016/j.biocontrol.2009.04.013
 76. Benhamou N, Garand C, Goulet A. Ability of nonpathogenic *Fusarium oxysporum* strain fo47 to induce resistance against *Pythium ultimum* infection in cucumber. Appl. Environ. Microbiol. 2002; 68(8):4044-4060. doi: 10.1128/AEM.68.8.4044-4060.2002.
 77. Veloso J, Díaz J. *Fusarium oxysporum* Fo47 confers protection to pepper plants against *Verticillium dahliae* and *Phytophthora capsici*, and induces the expression of defence genes. Plant Pathol. 61, 281-288. doi: 10.1111/j.1365-3059.2011.02516.x
 78. Biles CL, Martyn RD. Local and systemic resistance induced in watermelons by formaespeciales of *Fusarium oxysporum*. Phytopathology. 1989; 79(8):856-860. doi: 10.1094/Phyto-79-856.
 79. Díaz J, Silvar C, Varela MM, Bernal A Merino F. *Fusarium* confers protection against several mycelial pathogens of pepper plants. Plant Pathol. 2005; 54:773-780. doi: 10.1111/j.1365-3059.2005.01285.x
 80. Fuchs JG, Moëgne-Loccoz Y, Défago G. Ability of nonpathogenic *Fusarium oxysporum* Fo47 to protect tomato against *Fusarium* wilt. Biol. Control. 1999; 14(2):105-110. doi: 10.1006/bcon.1998.0664
 81. Blok WJ, Zwankhuizen MJ, Bollen GJ. Biological control of *Fusarium oxysporum*f.sp. *asparagi* by applying non-pathogenic isolates of *F. oxysporum*. Biocontrol Sci. Technol. 1997; 7(4):527-541. doi: 10.1080/09583159730596
 82. Nel B, Steinberg C, Labuschagne N, Viljoen A. The potential of nonpathogenic *Fusarium oxysporum* and other biological control organisms for suppressing fusarium wilt of banana. Plant Pathol. 2006; 55(2):217-223. doi:10.1111/j.1365-3059.2006.01344.x
 83. Kaur R, Singh RS. Study of induced systemic resistance in *Cicer arietinum* L. due to nonpathogenic *Fusarium oxysporum* using a modified split root technique. J. Phytopathol. 2007; 155(11-12):694-698. doi: 10.1111/j.1439-0434.2007.01300.x
 84. Pu X, Xie B, Li P, Mao Z, Ling J, Shen H, *et al.* (2014). Analysis of the defence-related mechanism in cucumber seedlings in relation to rootcolonization by nonpathogenic *Fusarium oxysporum* CS-20. FEMS Microbiol. Lett. 2014; 355(2):142-151. doi: 10.1111/1574-6968.12461
 85. Almeida Catarina. Use of two different carriers in a packed bed reactor for endopolygalacturonase production by a yeast strain. Process Biochemistry. 2005; 40(5):1937-1942.
 86. Soares Izabel. Microorganism-produced enzymes in the food industry. Scientific, Health and Social Aspects of the Food Industry. In Tech, 2012, 83-95.
 87. McPartland John M, Karl Hillig W. Cannabis clinic *Fusarium* Wilt. Journal of Industrial Hemp. 2004; 9(2):67-77
 88. Ciotola M, DiTommaso A, Watson AK. Chlamydospore production, inoculation methods and pathogenicity of *Fusarium oxysporum* M12-4A, a biocontrol for *Striga hermonthica*. Biocontrol Science and Technology. 2000; 10(2):129-145.
 89. Avedi Edith K. *Fusarium oxysporum* f. sp. *strigae* strain Foxy 2 did not achieve biological control of *Striga hermonthica* parasitizing maize in Western Kenya. Biological control. 2014; 77:7-14.