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Defence response in plants and animals against a common fungal pathogen, *Fusarium oxysporum*

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SUMMARY

Plant pathogens emerging as threat to human and animal health has been a matter of concern within the scientific community. *Fusarium oxysporum*, predominantly a phytopathogen, can infect both plants and animals. As a plant pathogen, *F. oxysporum* is one of the most economically damaging pathogen. In humans, *F. oxysporum* can infect immunocompromised individuals and is increasingly being considered as a problematic pathogen. Mycotoxins produced by *F. oxysporum* supress the innate immune pathways in both plants and animals. Hence, *F. oxysporum* is the perfect example for studying similarities and differences between defence strategies adopted by plants and animals. In this review we will discuss the innate immune response of plant and animal hosts for protecting against *F. oxysporum* infection. Such studies will be helpful for identifying genes, protein and metabolites with antifungal properties suitable for protecting humans.

1. Introduction

Fusarium oxysporum is an important phytopathogen, infecting many crop plants (Edel-Hermann and Lecomte, 2019). Based on its host specificity in plants, *Fusarium oxysporum* species complex (FOSC) has been divided into several groups or *formae specialis* (f. sp.). *F. oxysporum* infecting tomato are classified as f. sp. *lycopersici* (*Fol*), those infecting pea are classified as f. sp. *pisi* (*Fop*), isolates which infect chickpea as f. sp. *ciceri* (*Foc*), f. sp. *conglutinans* infects plants within the *Brassica oleracea* species and *F. oxysporum* f. sp. *cubense* infecting banana. In addition to plants, some of the members of FOSC can infect both invertebrate and vertebrate animals including human beings. *F. oxysporum* are frequently isolated from clinical samples (Debourgogne et al., 2016), which often leading to severe infection and sometimes death in human. *F. oxysporum* f. sp. *lycopersici* (Fol) can also infect immunodepressed mice (Ortoneda et al., 2004; López-Díaz et al., 2018), *F. oxypsorum* f. sp. *ciceri* (Foc) can infect *C. elegans* (Nag et al., 2017). Several human isolates can also colonize tomato and cucumber (Wang et al., 2020).

In human, *F. oxysporum* is an opportunistic pathogen infecting individuals with compromised immunity. One of the most severe eye infection, ocular keratosis, is caused by either *Aspergillus* or *Fusarium*. Any disruption in the integrity of the corneal epithelium may predispose the individual to ocular keratitis. With the increase in the use of contact lenses, *F. oxysporum* has become one of the major causes of ocular keratitis (Ananthi et al., 2008; Sun et al., 2010). *Fusarium* infection of the skin and nail are common in the developing countries (Nucci and Anaissie, 2002,2007). In deeply invasive infections, *F. oxysporum* can cause 100% lethality (Nucci and Anaissie, 2002). Several cases of pulmonary infection by *F. oxysporum* has also been recorded (Sander et al., 2009). *F. oxysporum* infecting human or animals has not been designated any *formae specialis*. However, the human pathogenic isolates have also been found to be carrying a unique set of lineage specific chromosomes with characteristic genes which are known to help in pathogenicity (Zhang et al., 2020).

The *Fusarium* genome is compartmentalised into two regions- core genome (containing genes for primary metabolism and reproduction) and the dispensable adaptive genome (containing genes for pathogen virulence and host specialisation). The Adaptive genome is located in the supernumerary or lineage-specific (LS) or accessory chromosomes. Along with other proteins required for host-specific interaction, LS chromosomes code for the rapidly evolving effector proteins which are required for the (full) pathogenicity towards a specific host (De Vries et al., 2020; Zhang et al., 2020). LS chromosomes are also considered to be important for adaptation and speciation (Ma et al., 2010; Spersch-neider et al., 2015; Li et al., 2020). Several studies has shown that transfer of pathogenicity related traits can convert non-pathogenic *F. oxysporum* f. sp. *lycopersici* to pathogenic *F. oxysporum* f. sp. *lycopersici*, but pathogencity transfer did not occur between different *formae*

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specialis (Ma et al., 2010; Fokkens et al., 2020; Li et al., 2020). Similarly, Van Dam et al. showed that *F. oxysporum* f.sp. *radicis-cucumerinum* can transfer its disease causing traits to a non-pathogenic strain of the same *f. sp.* by horizontal chromosome transfer (HCT) (van Dam et al., 2017). Based on the studies of HCT of LS or accessory chromosomes in *F. oxysporum*, it is apparent that the transfer of pathogenicity occurs only between strain having high similarities in their core genome. However, the ability of some plant pathogenic FOSC infecting animals including humans warrants for an appraisal of what is known about the host-*F. oxysporum* interaction both in plants and animals (Ortoneda et al., 2004; Nag et al., 2017; Wang et al., 2020).

The parallels in plant and animal innate immunity start with the ability to sense the pathogen using receptors at the point of contact and the ability to transmit this signal downstream via calcium signalling and reactive oxygen species (ROS) mediated pathways, activation of defence-related genes by specific transcription factors (TFs), production of antimicrobials and evasion at the organismal level (Sexton and Howlett, 2006). However, comparative studies on mechanism of host responses at molecular level to pathogens infecting both plants and animals are still limited to few, viz., in *Pseudomonas syringae, Burkholderia cepacia, Fusarium oxysporum, Fusarium solani, Alternaria solani*. In this review we discuss the innate immune response of plants and animals to the ubiquitous *F. oxysporum* species complex (FOSC).

2. *F. oxysporum* infection in plants

Genome analysis of plant pathogenic *Fusarium* revealed the presence of several enzymes which can degrade the cell wall of plants and help in the direct entry of the pathogens (Table 1). The presence of naturally occurring chickpea and garden pea land races with varying degrees of resistance has facilitated studies of *F. oxysporum* infection. In chickpea, after penetration into the root epidermis, the fungus moves towards the vascular bundle through the intercellular spaces. In the susceptible genotype of Chickpea (JG-62 and P-2245), *Foc* race 1 can easily penetrate the epidermis and reach the intercellular spaces within 2 days after infection (dai) and can cause wilting; whereas, in the resistant genotype (WR-315), it takes 8 days to reach the cortex and is restricted before reaching the vascular bundles (Jiménez-Fernández et al., 2013; Upasani et al., 2016). In tomato, entry is through the root tip or elongation zone during hydroponic culture and root hairs or grooves between the root epidermal cells of the collar region during soil culture (Lagopodi et al., 2002; Nahalkova et al., 2008). Like *Foc, Fop* race 2 could not reach the vascular tissues in resistant pea accessions. Lignification and suberization of cell walls and papillae like structures were noticed at the site of penetration in the resistant genotype of pea (Bani et al., 2018). In banana *F. oxysporum* f. sp. *cubense* has been shown to invade the host by penetrating the epidermis and growing through the intracellular spaces to reach the vascular bundles (Li et al., 2017). *F. oxysporum* f. sp. *cubense* infection outbreak in banana cultivar, Cavendish, is a serious threat to this crop as it is grown in monoculture (Dita et al., 2018). However, till date none of the members of this *formae speciales* has been reported to infect animals.

2.1. Pathogen perception and signalling

One of the first reactions to pathogen- or damage-associated molecular patterns (PAMPs and DAMPs) recognition by host receptors is eliciting reactive oxygen species (ROS) in the apoplastic spaces. This is done via the induction of NADPH oxidases (respiratory burst oxidase homologues, RBOHs), peroxidases and polyamine oxidases in a temporal manner (Daudi et al., 2012; Kadota et al., 2015; Waszczak et al., 2015; Gupta et al., 2013). During *Foc* infection in Chickpea, production of ROS and scavenging proteins were upregulated in the resistant genotype WR-315 compared to susceptible JG-62 (Gupta et al., 2013). Consequently, the ROS-scavenging proteins were also upregulated in the resistant genotype (Chatterjee et al., 2014). RBOHF and RBOH, NADH cytochrome b5 reductase, cationic peroxidase 3 (OCP3), flavodoxin-like quinone reductase 1 (FQR1) and iron superoxide dismutase 1 were deregulated in an opposing manner in the resistant genotype WR-315 and susceptible genotype JG-62, showing the importance of the H_2O_2 signalling (Gupta et al., 2013). The initial up-regulation of iron superoxide dismutase in both resistant (WR-315) and susceptible (JG-62) genotypes is stalled in WR-315 as recovery is initiated but continues in JG-62 causing further damage and collapse of vascular tissues in the susceptible genotype. In *Arabidopsis thaliana* two class III peroxidase, At3g49120 (AtPCb) and At3g49110 (AtPCa), generate H_2O_2 in response to elicitors from *F. oxysporum* (Bindschedler et al., 2006). The ROS production in the extracellular matrix may play an important role in cell wall reprogramming by promoting cell wall loosening or crosslinking (Schmidt et al., 2016). Activation of calcium channels located in the plasma membrane leads to increase in Ca^+ concentration in the cytoplasm, activating the calcium-dependent protein kinases (CDPKs), which in turn, relays the signal to the cell nucleus. This leads to a transcriptional, translational and metabolic change in the host plant for initiation of structural or biochemical immune response. Ashraf et al. (2018) could identify several components of calcium signalling in both resistant and susceptible genotypes during *Foc* infection. Calmodulin, which is part of the Ca^+ signalling system, also plays an important role in

Table 1
Comparison between plant pathogenic and human opportunistic strains of FOSC.

	Plant pathogenic FOSC	Human pathogenic FOSC
Size of adaptive genome	*F. oxysporum* f. sp. *lycopersici* 4287: 40% of genome (Ma et al., 2010)	*F. oxysporum* NRRL 3293: 147.7% of genome (Zhang et al., 2020)
Synteny between NRRL 32931 and Fol4287 (bp)	Core genome: 23,470,972; LS genome: 56,181	
Strains/ *formae specialis* known to cause disease	Some of the best studied and discussed strains are: *F. oxysporum* f. sp. *cicer, F. oxysporum* f. sp. *lycopersici, F. oxysporum* f. sp. *cubense, F. oxysporum* f. sp. *pisi, F. oxysporum* f. sp. *matthioli*, etc..	*F. oxysporum* (NRRL 32931), *F. oxysporum* (NRRL 47514)
	Enzymes produced by FOSC against host plant (Chang et al., 2016; Kwon et al., 2007; Williams et al., 2016)	**Enzymes produced by FOSC against Human (** Zhang et al., 2020; DeIulio et al., 2018**)**
Host entry	Cracks in the epidermis, point of lateral root, root hair, intact epidermis by dissolving cell wall	Airways, wounds and burns, as nosocomial infection
Physical Barrier	Laccases, β-xylosidases, β-glucosidase, protease arylsulfatase, α-L fucosidase, polygalacturonase (PG), pectate lyase (PL), and xylanase	Not known
Pathogen perception and signalling	Chitin, other cell wall components	Chitin, other cell wall components
Host specific adaptations	Effectors: Secreted in xylem genes (SIX genes), expansion of histidine kinase family, the TOR kinase family	Enrichment of metal ion binding transporters, chemical stimuli response proteins, *pacC* genes for alkaline adaptation, Duplication of the ergosterol biosynthesis pathway,expansion of HAL, serine/arginine protein kinase-like (SRPKL) Kinases, SRPKL kinases
Secondary metabolite/ Toxin	Fusaric acid (FA; 5-butylpicolinic acid)	Fusaric acid (FA; 5-butylpicolinic acid)

chickpea during *Fusarium* infection (Gupta et al., 2013). Cell suspension culture of *Arabidopsis thaliana* produces an oxidative burst in presence of *F. oxysporum* elicitors, which is enhanced by addition of Ca^{2+}, may play a role in basal resistance (Davies et al., 2006).

The effector proteins of the pathogen are recognized by receptors like the membrane-anchored *I* (Immunity) gene, first identified from *Solanum pimpinellifolium* (Catanzariti et al., 2017; Bohn and Tucker, 1939). Consequently several receptors have been identified: *I* and *I7*gene encode for a membrane bound leucine-rich repeat receptor-like proteins (LRR-RLP) (Gonzalez-Cendales et al., 2016; Catanzariti et al., 2017), *I2* encodes for an intracellular coiled-coil nucleotide-binding leucine-rich repeat (CC-NB-LRR) protein (Simons et al., 1998) and *I3* for a membrane bound S-receptor-like kinase (Catanzariti et al., 2015). ~~Many~~ Receptor proteins of tomato interact with kinase-containing proteins for relaying the signal further downstream. The Avr1 recognising *I2* interacts with LRR-receptor-like kinase (RLK), suppressor of BAK1-interacting RLK1 (SOBIR1), and Somatic embryogenesis receptor kinase 3/ brassinosteroid insensitive 1 associated kinase 1 (SERK3/-BAK1) (Catanzariti et al., 2017) (Fig. 1A). Several LRRs (IRF1259, IRF731 and IRF314) and the subsequent MAP kinases have been implicated to be differentially expressed in chickpea during *Foc* infection (Catanzariti et al., 2015; Kumar et al., 2016; Chatterjee et al., 2014; Chakraborty et al., 2019). In banana, *F. oxysporum* f. sp. *cubense* infection upregulates the Flagellin-sensitive 2 (FLS2) LRR transcripts (Li et al., 2012; Bai et al., 2013). In *Arabidopsis*, a recently identified leucine-rich repeat receptor-like kinase, MDIS1-INTERACTING RECEPTOR-LIKE KINASE 2 (MIK2), can bind SERINE-RICH ENDOGENOUS PEPTIDE 12 (SCOOP12) like peptides of *Brassica* and SCOOP-LIKE peptides of *F. oxysporum* (Coleman et al., 2021). Perception of *F. oxysporum* SCOOP12-like peptides by the extra-cellular domain of MIK2 induces its association with and the co-receptors SERK3 and SERK4 in *Arabidopsis*. This in turn relays the signalling through the cytosolic receptor-like kinases BOTRYTIS-INDUCED KINASE 1 (BIK1) and AVRPPHB SUSCEPTIBLE1 (PBS1)-LIKE 1 (PBL1) (Hou et al., 2021). The BAK1 homologue in banana and soybean was also upregulated following *F. oxysporum* f. sp. *cubense* infection (Li et al., 2012; Bai et al., 2013) and *F. oxysporum* f. sp. *pisi* infection, respectively (Lanubile et al., 2015).

In *Arabidopsis*, three loci have been identified which induces varying degrees of resistance to *Fusarium oxysporum* f. sp. *matthioli* (*Fom*). The cell wall-associated kinase-like 22 (WAK/WAKL), RFO, confers enhanced protection against different *Fusarium oxysporun* f. sp. *matthioli* (*Fom*) and *Verticillium longisporum* isolates (Berrocal-Lobo and Molina, 2007). The RFO2 locus, with extracellular leucine-rich repeats and one receptor-like protein (RLP) domain, provide modest resistance to *Fom* by inducing Tyrosine-Sulfated peptide signalling in *Arabidopsis* (Shen and Diener, 2013). Another locus in *Arabidopsis* Col-0, RFO3, with a receptor-like kinase (RLK) can confer resistance to independent isolates of *Fom* race 2 (Cole and Diener, 2013).

2.2. *Strengthening of cell wall, a means of defence response to F. oxysporum*

Transcriptional reprogramming as a result of *F. oxysporum* f. sp. *ciceri* (*Foc*) infection induces changes in lignification, phytoalexins, pathogenesis-related proteins of the host plant and yang cycle involved in ethylene biosynthesis (Kumar et al., 2016; Bindschedler et al., 2006). TFs of the families MYB, bHLH and WRKY families are also deregulated and may play important roles during *Fusarium*-plant interaction (Son et al., 2012; Chen et al., 2014; Lanubile et al., 2015). Lignification and formation of cross-linked matrix of carbohydrate and protein biopolymers pathway proteins may be correlated with the ability to exclude the pathogen from the vascular tissues in chickpea (Elagamey et al., 2017), soybean (Lanubile et al., 2015) and banana (Dong et al., 2020). During *Fusarium* infection in *Arabidopsis*, monolignols, the building block of lignins, are synthesized from phenylalanine via the phenylpropanoid pathway (Kostyn et al., 2012; Xie et al., 2018) and the salicylic acid (Gallego –Giraldo et al., 2011) pathways. In plants, methyl-transferase reactions are important in the lignin biosynthesis and ethylene production pathway. S-adenosyl-L-methionine (AdoMet) from the methane salvage pathway or Yang cycle serves as the major methyl-group donor for numerous highly specific methyl-transferase reactions (Eudes et al., 2016). AdoHcy hydrolase convert AdoMet to S-adenosyl-L-homocysteine (AdoHcy), which is a potent inhibitor of methyltransferases. Hence, AdoHcy hydrolase is known to have a role in defence against pathogens by influencing the lignin as well as the ethylene biosynthesis pathway (Kawalleck et al., 1992).

2.3. *Pathogenesis-related proteins and host secondary metabolites with antimicrobial properties against F. oxysporum*

The pathogenesis-related (PR) proteins are induced in the host plant

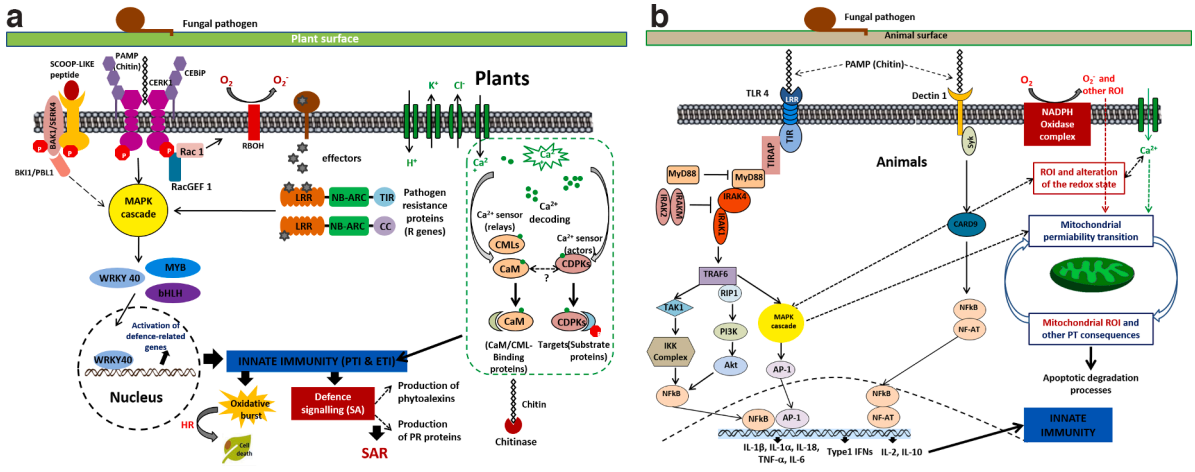

Fig. 1. Comparison of innate immune response in plant and animal to FOSC infection. **A**: Detection of *F. oxysporum* by host and subsequent innate immune response in plants. Chitins, produced by fungi are recognised by the host CERK present on the host cell membrane, SERK3/ BAK1 recognises the effector Avr1 and relays the signal through MAPKs, production of ROS by RBOHs and calcium signalling via CDPKs to the nucleus. Activation of TFs like WRKY40 lead to activation of defence related gene expression. **B**: Detection of *F. oxysporum* by host and subsequent innate immune response in animals. TLR4 and IL-1R (not shown here) and the downstream MyD88 can detect the *F. oxysporum* to relay signals downstream to produce innate immune response. Dectin-1 is also activated during *F. oxysporum* infection.

as a result of pathogen attack and at least few are known to have antifungal and antibacterial properties. PR proteins are induced as a result of systemic acquired resistance after pathogen infection (Van Loon et al., 2006). The antimicrobial peptides (AMP) in plants are small cationic peptides of 45–54 amino acids held together by four disulphide bonds (Parisi et al., 2018). AMPs have been detected in the epidermis of leaves, roots, pods, tubers, fruit, and floral organs, seeds and other possible targets of pathogen contact. In chickpea PR proteins are induced in both the resistant and susceptible varieties during *Fusarium* infection (Kumar et al., 2016; Ashraf et al., 2009). Induction of PR-1 (a and b isoforms), PR-5x, PR-2, PR-4, glucanases, chitinases, peroxidases and xyloglucan-specific endoglucanase inhibitor protein (XEGIP) reported for the xylem sap proteome of *Fol*-infected tomato plants (Houterman et al., 2007; Rep et al., 2002). The PR1 family, identified initially in tobacco, is represented in every plant species studied to date and homologues have been found in animals, fungi and insects. Lincon et al.

Table 2
Antimicrobials, secondary metabolites and plant extracts reported to inhibit *F. oxysporum* growth.

Antifungal	Source of isolation of antifungal compound	Functions against	Mechanism of action	Refs.
Plant derived compound showing antifungal activity				
PvD1	*Phaseolus vulgaris*			Games et al. (2008)
AfpB	*P. digitatum* CECT 20,796 (PHI26)	*F. oxysporum* 4287, *P. digitatum* CECT 20,796, *Botrytis cinerea* CECT 2100, *A. niger* CBS120.49, *Magnaporthe oryzae* PR9, *Aspergillus flavus* CECT 20,802, *Candida albicans* CECT 1394, *C. glabrata* CECT 1448, and *C. parapsilosis* CECT 1449.	Has the ability to bind fungal membranes	Garrigues et al. (2018)
Pr-1	*Cucurbita moschata*	*F. oxysporum*	not known	Park et al. (2010)
Cm-p1	*Cenchritis muricatus*	*F. oxysporum*	not known	López-Abarrategui et al. (2012)
Thaumatin-like protein, Osmotin, Zeamatin	*Zea mays*	*F. oxysporum*	not known	Van der Weerden et al. (2013)
Plant hormone				
Methyl jasmonate (Me-JA)	Jasmonic acid is produced by all plant; Me-JA is a derivative	*F. oxysporum* f.sp. *lycopersici* Race 3	By inducing increase in the levels phenolic compounds such as salicylic acid (SA), kaempferol and quercetin in the plant. No bioassay was done	Król et al. (2015)
Plant Secondary metabolites				
Desoxyhemigossypol, hemigossypol, desoxyhemigossypol-6-methyl ether, hemigossypol-6-methyl ether	*Gossypium hirsutum*	*F. oxysporum* f. sp. *vasinfectum*	Biassay was done. Mechanism of inhibition not known	Zhang et al. (1993)
Naringenin,morin, quercetin, glycitein, apigenin, luteolin, kaempferol, rutin, myricetin, daidzein, genistein and coumestrol	Plant derived	*F. oxysporum* f. sp. *lycopersici*	Biassay was done. Mechanism of inhibition not known	Steinkellner and Mammerler (2007)
Plant extracts	*Adhatoda vasica, Eucalyptus globulus, Lantana camara, Nerium oleander, Ocimum basilicum, Thymus atlanticus, Datura metel,* and many more	*F. oxysporum* f. sp. *lycopersici* race 3, *F. oxysporum* f.sp. *albedinis, F. oxysporum* f. sp. *cubense*	not known	Isaac and Tahon (2014), Bouhlali et al. (2020), Akila et al. (2011)
Animal derived compound showing antifungal activity				
Cecropin A and CecropinB,	*Hyalopora cecropia*	*A. flavus, A. niger, A. fumigatus, F. moniliforme,* and *F. oxysporum*	Binds to cell wall ergosterol and cholesterol	De Lucca et al. (1997)
Drosomycin	*D. melanogaster*	*F. oxysporum*	not known	Tian et al. (2008)
Metchnikowin	*D. melanogaster*	Pathogenic ascomycota, including *F. graminearum*	not known	Moghaddam et al. (2017)
Thanatin	*Podisus maculiveris*	*Neurospora crassa, Botrytis cinerea, Nectria haematococca, Trichoderma viride, Alternaria brassicola, Fusarium culmorum, Ascochyta pisi, Fusarium oxysporum, A. fumigatus* and *T. mentagrophytes* .	Inhibition of spore germination and formation of hyphae	Fehlbaum et al. (1996)
Dermaseptins	*Phyllomedusa sauvagii*	*F. oxysporum and F. moiliformae, Aspergillus flavus, A. fumigetus, A. niger*	Binds to cell wall ergosterol and cholesterol	De Lucca et al. (1998)
Myticin A and Myticin B	*Mytilus galloprovincialis*	*F. oxysporum* and *Escherichia coli D31*	not known	Mitta et al. (1999)
Anti-fungal compound identified from other sources				
Iturin	*Bacillus velezensis*	*Ralstonia solanacearum* and *Fusarium oxysporum*	not known	Cao et al. (2018)
Syringomycin-E	*Pseudomonas* spp.	*A. flavus, A. niger, A. fumigatus, F. moniliforme,* and *F. oxysporum*	Syringomycin-E can bind chitin, b-1,3- glucan, and mannan	De Lucca et al. (1999)
Myriocin	*Bacillus amyloliquefaciens*	*Fusarium oxysporum* f. sp. *niveum*	Myriocin destroyed membrane integrity	Wang et al. (2021)
Fengycin	*Bacillus amyloliquefaciens*	*F. oxysporum* f. sp. *radicislycopersici*	not known	Kang et al. (2020)
Cepacidines (A1 and A2)	*Burkholderia cepacia*	*F. oxysporum*	not known	De Lucca and Walsh (1999)
Atroviridins (A, B, C)	*Trichoderma atroviride* F80317	*F. oxysporum*	not known	Oh et al. (2002)

demonstrated that tomato roots expressing PR1 from plant, human and microbial sources survive Fumonisin B_1 better than wild type plants (Lincon et al., 2018). In soybean three PR10, one PR9, four PR5 one PR4 and one PR1 were upregulated after *F. oxysporum* f. sp. *pisi* infection (Lanubile et al., 2015).

Phytoalexins are low molecular mass secondary metabolites with antimicrobial activity produced by the plant as a result of pathogen infection or abiotic stress (Ahuja et al., 2012). Phytoalexins have been reviewed at great length by Ahuja et al. (2012) and will not be discussed here in detail. To compensate for the high rates of immune reactions occurring after *Foc* infection in *Cicer*, nitrogen and carbon metabolism of the host plant is reprogrammed (Kumar et al., 2016; Gupta et al., 2010). In addition dysregulation of phytoalexins- genistein, luteolin, clotrimazole and quinone- occur in both the resistant and susceptible genotypes of chickpea during *Foc* infection (Kumar et al., 2016). Table 2 lists some of the antimicrobial and secondary metabolite products from plants which have been tested against *F. oxysporum*.

2.4. Plant immune suppression by F. oxysporum toxins

Like effectors, toxins are also thought to reduce ROS production in the host plant by suppressing the immune reactions (Perincherry et al., 2019). Mycotoxins are also produced during the saprophytic phase of the fungi. Fusaric acid (FA), produced by FOSC, is known to be associated with wilting in plants, even external application of FA results in wilt-like symptoms in tomato (Singh et al., 2017). It is also known to cause hypersensitive reaction in tomato by interfering with the antioxidants like catalase and peroxidase (Singh and Upadhyay, 2014). Plant defence responses are activated by FA application in subtoxic doses (Bouizgarne et al., 2006). López-Díaz et al. (2018) showed that mutants of *F. oxysporum* with reduced capacity of producing FA led to reduced virulence in totamo plants.

3. *F. oxysporum* infection in animals

The major pathways for entry of *Fusarium* inside the human body are airways, damaged skin tissue and mucosal membranes (Nucci and Anaissie, 2007). In immunocompetent individuals, *Fusarium* isolates cause allergic fungal sinusitis (Wicken, 1993), keratitis (cornea opacity leading to blindness, and even loss of the eyeball), onychomycosis (infection of the toenails or fingernails) and locally invasive infections (Nucci and Anaissie, 2007), which may become disseminated infections in immune-compromised patients (Marr et al., 2002). Fusariosis, like many other invasive fungal infections share similar features in its manifestation in patients receiving high doses of corticosteroids and those with neutropenia (Smith and Kauffman, 2012). In order to study Fusariosis, several animal models have been identified, e.g., mice (Mayayo et al., 1999), *Drosophila* (Lamaris et al., 2007), *Caenorhabditis elegans* (Muhammed et al., 2012).

Host innate immunity against FOSC shares defence mechanism which are similar against other fungi (Schäfer et al., 2014; Lionakis et al., 2017). In a broad prespective, the effector cells- neutrophils, monocytes and macrophages have anti-fungal roles and are recruited at the site of fungal infection. Phagocytes are more effective against spores and non-filamentous fungi. Phagocytes release anti-fungal peptides and oxygen-intermediates against fungi. The dendritic cells, which link the innate and adaptive immunity, are also important defence against fungal infections.

3.1. Pathogen perception at the barrier tissues of animals

Fungal infections can occur when abrasions and wounds occur in the epidermal layer, making the animal vulnerable (Coates et al., 2018). FOSC frequently infect the barrier tissues at the cutaneous layer of skin following skin breakdown (Nucci et al., 2002), however, FOSC infection can also occur through an intact skin layer in mice (De Paulo et al., 2013). Once the pathogen crosses the structural barrier provided by the skin, pathogen recognition receptors (PRRs) in the sub-epidermis can identify the pathogen- or damage- associated molecular patterns to initiate innate immunity. *Drosophila* Toll and human interleukin-1 receptors (TIR) are examples of PRRs which can recognize pathogens with the help of an extracellular leucine-rich repeat (LRR) domain on the outer side of the sub-epidermal wall. The cytoplasmic TIR domain helps in transduction of the signal further to the nucleus for induction of innate immunity (De Paulo et al., 2013). Toll-like receptors (TLRs) and interleukin-1 receptor (IL-1R) belongs to the same family of transmembrane receptor family with cytoplasmic TIR domains. However, TLRs contain leucine-rich repeats (LRRs) in its endodomain while IL-1R possess Ig-like domains (Nürnberger et al., 2004). TLR-4 and IL-1R seem to be functional in conjunction with MyD88 during *Fusarium* kratitis (68) (Tarabishy et al., 2008) (Fig. 1B). MyD88 is the common adaptor for most TLRs and IL-l receptors (Krishnan et al., 2007). TLRs, IL-lR and MyD88 are distributed in almost all cells of the immune system and activation of MyD88 leads to the induction of innate immune cells via MAPK pathway (to induce different TFs) or through other kinases (to induce NF-κβ) leading to the production of cytokines, chemokines and other immunomodulatory molecules (Nürnberger et al., 2004). Correia et al. (2020) showed that even the crude extract of *F. oxysporum* induced pro-inflammatory IL-6, IL-17, TNF-α and anti-inflammatory TGF-β1 cytokinines. TLRs are conserved among many phyla including human, *Drosophila* and *C. elegans*. Disease development starts in *C. elegans* with force feeding of microconidia (Muhammed et al., 2012) leading to the induction of a unique set of molecular signatures independent of Tol-1 but dependent on Daf-16, TGF-β (Nag et al., 2017) and MAPK pathways (Muhammed et al., 2012).

In addition to TLRs, fungal recognition also involves C-type lectin receptors (CLRs) (Van de Veerdonk et al., 2008; De Figueiredo et al., 2011) present in many tissues like macrophages, monocytes, neutrophils, mast cells, dendritic cells (DCs), bronchial epithelial cells and pulmonary epithelium of the respiratory system, corneal epithelial cells as transmembrane receptors and body fluids. Dectin-1, Dectin-2, Mincle, mannose receptor (MR), and DC-SIGN are some of the CLRs known to recognize mannan, glucans, and chitin present on the fungal cell wall (Goyal et al., 2018) (Fig. 1B).

Th17 cells organise at the barrier membranes and protect against external pathogens. Th17 cells are important not only for mucosal immunity, it also acts a bridge for adaptive immunity, and often is a double edged sword as its balance can decide the outcome from defence to pathogenesis (Khader et al., 2009). Th17 cells are activated by *F. oxysporum, Aspergillus* and *Candida* infection (Taylor et al., 2014). Th17 cells are also important defence during fungal keratisis in mice (Taylor et al., 2014).

3.2. Small peptides and host secondary metabolites with antimicrobial against F. oxysporum

Small proteins with antimicrobial (AMP) or antifungal activity are secreted by the host cells to inhibit the growth of bacterial, fungal and viral pathogens. AMPs are mostly produced by members belonging to different kingdoms in response to pathogen attack. AMPs are gene encoded and maybe also be produced constitutively expression (Hegedüs and Marx, 2013). β-defensins connect innate immunity with the adaptive immunity by recruiting via Toll pathway (Biragyn et al., 2002). Drosomycin and metchnikowin produced by *Drosophila* are active against *Fusarium* and other filamentous fungi (Tian et al., 2008). Metchnikowin acts selectively against pathogenic ascomycota, including *F. graminearum* (Moghaddam et al., 2017). The *C. elegans* genome encodes for several types of AMP gene clusters and neuropeptide-like proteins (NLPs), caenacin family proteins (CNCs), antibacterial factor (ABF) peptides and caenopores or saposin-like proteins (SPP). However, only SPP-11 has been found to be upregulated during *F. oxysporum* infection compared to uninfected controls (Nag

et al., 2017). In mice, antimicrobials which are either induced upon *F. oxysporum* infection or expressed constitutively, seems to be involved in the destruction of fungus inside macrophages, neutrophils and dendritic cells (Schäfer et al., 2014). Although, not much is known about the recognition and uptake of *F. oxysporum* by macrophages, engulfing more than three spores seems to cause lysis of the macrophage (Schäfer et al., 2014). It has been recently demonstrated that β–1,6-linked Galactofuranose- rich peptidogalactomannan present in the cell wall of *F. oxysporum* is responsible for uptake spores by macrophage (de Oliveira et al., 2019).

3.3. Immune suppression in animals by F. oxysporum toxins

Mycotoxicosis in humans and animals following ingestion of food contaminated by toxin-producing *Fusarium* spp. has been reported a long time ago (Nelson et al., 1994). In the case of *F. oxysporum*, the mycotoxins responsible may be Fusaric acid. It has also been recently demonstrated that *fub1Δ* mutants of *F. oxysporum* with reduced FA production has significantly reduced virulence in immunodepressed mice (López-Díaz et al., 2018). *F. oxysporum* secreted FA has been shown to increase the levels of neurotransmitters like serotonin, 5-hydroxyindoleacetic acid, tyrosine, and dopamine in rat pineal cell cultures (Porter et al., 1995). FA causes inhibition of noradrenaline synthesis, reduces the luteinizing releasing hormone and prolactin from the basal hypothalamus in animals (Tobias et al., 1983). In pigs acute doses of FA causes vomiting and lethargy (Smith and MacDonald, 1991) and narcolepsy in chicks (Bungo et al., 1999). FA is immunotoxic to peripheral blood mononuclear cells (PBMCs) and acute monocytic leukaemic (Thp-1) cell line, inciting cell death in both cases. In Thp-1 cells the extracellular receptor kinase (ERK) proteins are upregulated and the c-Jun N-terminal kinase (JNK) proteins are downregulated, which regulate the mitochondrial apoptosis control protein, Bcl-2. Contrastingly, in PBMCs both ERK and JNK proteins are upregulated and p38 MAPK expression is downregulated inducing paraptosis (Dhani et al., 2017). High doses of FA may be toxic for the cells while controlled doses may be of therapeutic use. FA administration in controlled doses had a hypotensive effect by inhibiting Dopamine-β-hydoxylase, thus, reducing the level of noradrenaline, which controls the blood pressure in humans (Hidaka et al., 1969).

4. Conclusions

Fusarium oxysporum is an opportunistic pathogen of plants and can infect animals only when the structural integrity is damaged. During plant infection, the main defence against *F. oxysporum* is the reinforcement of cell wall by augmenting lignin and cell wall carbohydrates. After the fungus successfully establishes itself inside the plant host, production of antimicrobials and secondary metabolites is initiated. Unlike plants, defence against *F. oxysporum* infection in animals does initiate any reinforcement of cell structures. In animals, production of antimicrobials, destruction of fungus inside the macrophages, neutrophils and dendritic cells are the main defence mechanisms. The commonality in plants and animals against *F. oxysporum* appear to be the production of antimicrobial peptides.

Outlook

Several antimicrobial peptides, phytoalexins and secondary metabolites from plants and invertebrates have been tested against *F. oxysporum* to understand whether these antimicrobials have the potentiality to be used as novel theraputics during human infection. Table 2 lists some of the antimicrobial products from animals which have been tested against *F. oxysporum*. Since the *F. oxysporum* strains capable of infecting human and plant differ mostly in the accessory genomic level; while the core genome is predominantly conserved (Van Dam et al., 2017). It may be useful to test the antimicrobials, phytoalexins and secondary metabolites produced by the plants and animals which target the products of the essential genes which are usually located on the core genome of the strains which can infect humans.

Ethics approval and consent to participate

Not applicable to review article.

Consent for publication

All authors consent to participate in this review article.

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This manuscript does not have any data and material.

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CRediT authorship contribution statement

Papri Nag: Conceptualization, Writing – original draft. **Sathi Paul:** Visualization. **Surbhi Shriti:** Writing – original draft. **Sampa Das:** Conceptualization, Supervision.

Declaration of Competing Interest

None declared.

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References

Ahuja, I., Kissen, R., Bones, A.M., 2012. Phytoalexins in defense against pathogens. Trends Plant Sci. 17, 73–90. https://doi.org/10.1016/j.tplants.2011.11.002.

Akila, R., Rajendran, L., Harish, S., Saveetha, K., Raguchander, T., Samiyappan, R., 2011. Combined application of botanical formulations and biocontrol agents for the management of Fusarium oxysporum f. sp. cubense (Foc) causing Fusarium wilt in banana. Biol. Control 57, 175–183. https://doi.org/10.1016/j.biocontrol.2011.02.010.

Ananthi, S., Chitra, T., Bini, R., Prajna, N.V., Lalitha, P., Dharmalingam, K., 2008. Comparative analysis of the tear protein profile in mycotic keratitis patients. Mol. Vis. 14, 500–507. http://www.molvis.org/molvis/v14/a60.

Ashraf, N., Basu, S., Narula, K., Ghosh, S., Tayal, R., Gangisetty, N., Biswas, S., Aggarwal, P.R., Chakraborty, N., Chakraborty, S., 2018. Integrative network analyses of wilt transcriptome in chickpea reveal genotype dependent regulatory hubs in immunity and susceptibility. Sci. Rep. 8, 6528. https://doi.org/10.1038/s41598-018-19919-5.

Ashraf, N., Ghai, D., Barman, P., Basu, S., Gangisetty, N., Mandal, M.K., Chakraborty, N., Datta, A., Chakraborty, S., 2009. Comparative analyses of genotype dependent expressed sequence tags and stress-responsive transcriptome of chickpea wilt illustrate predicted and unexpected genes and novel regulators of plant immunity. BMC Genom. 10, 415. https://doi.org/10.1186/1471-2164-10-415.

Bai, T.T., Xie, W.B., Zhou, P.P., Wu, Z.L., Xiao, W.C., Zhou, L., Sun, J., Ruan, X.L., Li, H. P., 2013. Transcriptome and expression profile analysis of highly resistant and susceptible banana roots challenged with Fusarium oxysporum f. sp. cubense tropical race 4. PLoS One 8 (9), e73945. https://doi.org/10.1371/journal.pone.0073945. Sep 23.

Bani, M., Pérez-De-Luque, A., Rubiales, D., Rispail, N., 2018. Physical and chemical barriers in root tissues contribute to quantitative resistance to Fusarium oxysporum f. sp. pisi in pea. Front. Plant Sci. 9, 199. https://doi.org/10.3389/fpls.2018.00199.

Berrocal-Lobo, M., Molina, A., 2007. Arabidopsis defense response against Fusarium oxysporum. Trends Plant Sci. 13, 145–150. https://doi.org/10.1016/j.tplants.2007.12.004.

Bindschedler, L.V., Dewdney, J., Blee, K.A., Stone, J.M., Asai, T., Plotnikov, J., Denoux, C., Hayes, T., Gerrish, C., Davies, D.R., Ausubel, F.M., Bolwell, P.G., 2006. Peroxidase-dependent apoplastic oxidative burst in Arabidopsis required for pathogen resistance. Plant J. 47, 851–863. https://doi.org/10.1111/j.1365-313X.2006.02837.x.

Biragyn, A., Ruffini, P.A., Leifer, C.A., Klyushnenkova, E., Shakhov, A., Chertov, O., Shirakawa, A.K., Farber, J.M., Segal, D.M., Oppenheim, J.J., Kwak, L.W., 2002. Toll-like receptor 4-dependent activation of dendritic cells by beta-defensin 2. Science 298 (5595), 1025–1029. https://doi.org/10.1126/science.1075565.

Bohn, G.W., Tucker, C.M., 1939. Immunity to Fusarium wilt in the tomato. Science 89 (2322), 603–604. https://doi.org/10.1126/science.89.2322.603.

Bouhlali, E.D.T., Derouich, M., Ben-Amar, H., Meziani, R., Essarioui, A., 2020. Exploring the potential of using bioactive plant products in the management of Fusarium oxysporum f.sp. albedinis: the causal agent of Bayoud disease on date palm (Phoenix dactylifera L.). Beni-Suef Univ. J. Basic Appl. Sci. 9, 46 https://bjbas.springeropen.com/articles/10.1186/s43088-020-00071-x.

Bouizgarne, B., El-Maarouf-Bouteau, H., Frankart, C., Reboutier, D., Madiona, K., Pennarun, A.M., Monestiez, M., Trouverie, J., Amiar, Z., Briand, J., Brault, M., Rona, J.P., Ouhdouch, Y., El Hadrami, I., Bouteau, F., 2006. Early physiological responses of Arabidopsis thaliana cells to fusaric acid: toxic and signalling effects. New Phytol. 169 (1), 209–218. https://doi.org/10.1111/j.1469-8137.2005.01561.x.

Bungo, T., Shimojo, M., Masuda, Y., Choi, Y.H., Denbow, D.M., Furuse, M., 1999. Induction of food intake by a noradrenergic system using clonidine and fusaric acid in the neonatal chick. Brain Res. 826 (2), 313–316. https://doi.org/10.1016/s0006-8993(99)01299-8.

Cao, Y., Pi, H., Chandrangsu, P., Li, Y., Wang, Y., Zhou, H., Xiong, H., Helmann, J.D., Cai, Y., 2018. Antagonism of two plant-growth promoting Bacillus velezensis isolates against Balstonia solanacearum and Fusarium oxysporum. Sci. Rep. 8 (1), 4360. https://doi.org/10.1038/s41598-018-22782-z.

Catanzariti, A.M., Do, H.T., Bru, P., de Sain, M., Thatcher, L.F., Rep, M.J., Jones, D.A., 2017. The tomato I gene for Fusarium wilt resistance encodes an atypical leucine-rich repeat receptor-like protein whose function is nevertheless dependent on SOBIR1 and SERK3/BAK1. Plant J. 89, 1195–1209. https://doi.org/10.1111/tpj.13458.

Catanzariti, A.M., Lim, G.T.T., Jones, D.A., 2015. The tomato I-3 gene: a novel gene for resistance to Fusarium wilt disease. New Phytol. 207, 106–118. https://doi.org/10.1111/nph.13348.

Chakraborty, J., Ghosh, P., Sen, S., Nandi, A.K., Das, S., 2019. CaMPK9 increases the stability of CaWRKY40 transcription factor which triggers defense response in chickpea upon Fusarium oxysporum f. sp. ciceri Race1 infection. Plant Mol. Biol. 100 (4–5), 411–431. https://doi.org/10.1007/s11103-019-00868-0.

Chang, H.X., Yendrek, C.R., Caetano-Anolles, G., Hartman, G.L., 2016. Genomic characterization of plant cell wall degrading enzymes and in silico analysis of xylanses and polygalacturonases of Fusarium virguliforme. BMC Microbiol. 16 (1), 147. https://doi.org/10.1186/s12866-016-0761-0.

Chatterjee, M., Gupta, S., Bhar, A., Chakraborti, D., Basu, D., Das, S., 2014. Analysis of root proteome unravels differential molecular responses during compatible and incompatible interaction between chickpea (Cicer arietinum L.) and Fusarium oxysporum f. sp. ciceri Race1 (Foc1). BMC Genom. 15, 949. https://doi.org/10.1186/1471-2164-15-949.

Chen, Y.C., Wong, C.L., Muzzi, F., Vlaardingerbroek, I., Kidd, B.N., Schenk, P.M., 2014. Root defense analysis against Fusarium oxysporum reveals new regulators to confer resistance. Sci. Rep. 4, 5584. https://doi.org/10.1038/srep05584.

Coates, M., Blanchard, S., MacLeod, A.S., 2018. Innate antimicrobial immunity in the skin: a protective barrier against bacteria, viruses, fungi. PLoS Pathog. 14 (12), e1007353 https://doi.org/10.1371/journal.ppat.1007353.

Cole, S.J., Diener, A.C., 2013. Diversity in receptor-like kinase genes is a major determinant of quantitative resistance to Fusarium oxysporum f. sp. matthioli. New Phytol. 200 (1), 172–184. https://doi.org/10.1111/nph.12368.

Coleman, A.D., Maroschek, J., Raasch, L., Takken, F.L.W., Ranf, S., Hückelhoven, R., 2021. The Arabidopsis leucine-rich repeat receptor-like kinase MIK2 is a crucial component of early immune responses to a fungal-derived elicitor. New Phytol. 229 (6), 3453–3466. https://doi.org/10.1111/nph.17122.

Correia, M.H., Sato, F., Baesso, M.L., Bento, A.C., Gibin, M.S., de Moraes, G.R., Melo, K. S., Svidzinski, T.I.E., Almeida, G.H.D.R., CAB, Amado, Hernandes, L., 2020. Immune response and Raman scattering assessment in rats skin after contact with Fusarium oxysporum metabolites. Spectrochim. Acta A Mol. Biomol. Spectrosc. 234, 118246 https://doi.org/10.1016/j.saa.2020.118246.

Daudi, A., Cheng, Z., O'Brien, J.A., Mammarella, N., Khan, S., Ausubel, F.M., Bolwella, G. P., 2012. The apoplastic oxidative burst peroxidase in Arabidopsis is a major component of pattern-triggered immunity. Plant Cell 24, 275–287. https://doi.org/10.1105/tpc.111.093039 www.plantcell.org/cgi/doi/.

Davies, D.R., Bindschedler, L.V., Strickland, T.S., Bolwell, G.P., 2006. Production of reactive oxygen species in Arabidopsis thaliana cell suspension cultures in response to an elicitor from Fusarium oxysporum: implications for basal resistance. J. Exp. Bot. 7 (8), 1817–1827. https://doi.org/10.1093/jxb/erj216.

De Figueiredo, L.F., Gossmann, T.I., Ziegler, M., Schuster, S., 2011. Pathway analysis of NAD+ metabolism. Biochem. J. 439 (2), 341–348. https://doi.org/10.1042/BJ20110320.

De Lucca, A.J., Bland, J.M., Jacks, T.J., Grimm, C., Walsh, T.J., 1998. Fungicidal and binding properties of the natural peptides cecropin B and dermaseptin. Med. Mycol. 36 (5), 291–298.

De Lucca, A.J., Jacks, T.J., Takemoto, J., Vinyard, B., Peter, J., Navarro, E., Walsh, T.J., 1999. Fungal lethality, binding, and cytotoxicity of syringomycin-E. Antimicrob. Agents Chemother. 43 (2), 371–373. https://doi.org/10.1128/AAC.43.2.371.

De Lucca, A.J., Walsh, T.J., 1999. Antifungal peptides:novel therapeutic compounds against emerging pathogens. Antimicrob. Agents Chemother. 43 (1), 1–11. https://doi.org/10.1128/AAC.43.1.1.

de Oliveira, N.F., Santos, G.R.C., Xisto, M.I.D.S., Pires Dos Santos, G.M., Nucci, M., Haido, R.M.T., Barreto-Bergter, E., 2019. β1,6-linked Galactofuranose- rich peptidogalactomannan of Fusarium oxysporum is important in the activation of macrophage mechanisms and as a potential diagnostic antigen. Med. Mycol. 57 (2), 234–245. https://doi.org/10.1093/mmy/myx167.

De Paulo, L.F., Coelho, A.C., Svidzinski, T.I.E., Sato, F., Rohling, J.H., Natali, M.R.M., Baesso, M.L., Hernandes, L., 2013. Crude extract of Fusarium oxysporum induces apoptosis and structural alterations in the skin of healthy rats. J. Biomed. Opt. 18 (9), 095004 https://doi.org/10.1117/1.jbo.18.9.095004.

De Vries, S., Stukenbrock, E.H., Rose, L.E., 2020. Rapid evolution in plant–microbe interactions – an evolutionary genomics perspective. New Phytol. 226, 1256–1262. https://doi.org/10.1111/nph.16458.

Debourgogne, A., Dorin, J., Machouart, M., 2016. Emerging infections due to filamentous fungi in humans and animals: only the tip of the iceberg? Environ. Microbiol. Rep. 8 (3), 332–342. https://doi.org/10.1111/1758-2229.12404.

DeIulio, G.A., Guo, L., Zhang, Y., Goldberg, J.M., Kistler, H.C., Ma, L.J., 2018. Kinome expansion in the Fusarium oxysporum species complex driven by accessory chromosomes. mSphere 3, e00231–e002318. https://doi.org/10.1186/s12864-016-2486-8.

De Lucca, A.J., Bland, J.M., Jacks, T.J., Grimm, C., Cleveland, T.E., Walsh, T.J., 1997. Fungicidal activity of cecropin A. Antimicrob. Agents Chemother. 41 (2), 481–483. https://doi.org/10.1128/AAC.41.2.481.

Dhani, S., Nagiah, S., Naidoo, D.B., Chuturgoon, A.A., 2017. Fusaric acid immunotoxicity and MAPK activation in normal peripheral blood mononuclear cells and Thp-1 cells. Sci. Rep. 7 (1), 3051. https://doi.org/10.1038/s41598-017-03183-0.

Dita, M., Barquero, M., Heck, D., Mizubuti, E.S.G, Staver, C.P, 2018. Fusarium Wilt of Banana: current knowledge on epidemiology and research needs toward sustainable disease management. Front Plant Sci 9, 1468. 10.3389/fpls.2018.01468.

Edel-Hermann, V., Lecomte, C., 2019. Current status of Fusarium oxysporum formae speciales and races. Phytopathology 109, 512–530. https://doi.org/10.1094/PHYTO-08-18-0320-RVW.

Elagamey, E., Narula, K., Sinha, A., Ghosh, S., Abdellatef, M.A.E., Chakraborty, N., Chakraborty, S., 2017. Quantitative extracellular matrix proteomics suggests cell wall reprogramming in host-specific immunity during vascular wilt caused by Fusarium oxysporum in chickpea. Proteomics 17, 23–24. https://doi.org/10.1002/pmic.201600374.

Eudes, A., Zhao, N., Sathitsuksanoh, N., Baidoo, E.E.K., Lao, J., Wang, G., Yogiswara, S., Lee, T.S., Singh, S., Mortimer, J.C., Keasling, J.D., Simmons, B.A., Loqué, D., 2016. Expression of S-adenosylmethionine hydrolase in tissues synthesizing secondary cell walls alters specific methylated cell wall fractions and improves biomass digestibility. Front. Bioeng. Biotechnol. 4, 58. https://doi.org/10.3389/fbioe.2016.00058.

Fehlbaum, P., Bulet, P., Chernysh, S., Briand, J.P., Roussel, J.P., Letellier, L., Hetru, C., Hoffmann, J.A., 1996. Structure-activity analysis of thanatin, a 21-residue inducible insect defense peptide with sequence homology to frog skin antimicrobial peptides. Proc. Natl. Acad. Sci. U. S. A. 93 (3), 1221–1225. https://doi.org/10.1073/pnas.93.3.1221.

Fokkens, L., Guo, L., Dora, S., Wang, B., Ye, K., Sánchez-Rodríguez, C., Croll, D., 2020. A chromosome-scale genome assembly for the Fusarium oxysporum strain fo5176 to establish a model Arabidopsis-fungal pathosystem. G3 (Bethesda) 10 (10), 3549–3555. https://doi.org/10.1534/g3.120.401375.

Gallego -Giraldo, L., Escamilla-Trevinob, L., Jackson, L.A., Dixon, R.A., 2011. Salicylic acid mediates the reduced growth of lignin down-regulated plants. Proc. Natl. Acad. Sci. U. S. A. 108, 20814–20819. https://doi.org/10.1073/pnas.1117873108.

Games, P.D., Dos Santos, I.S., Mello, E.O., Diz, M.S., Carvalho, A.O., de Souza-Filho, G.A., Da Cunha, M., Vasconcelos, I.M., Ferreira Bdos, S., Gomes, V.M., 2008. Isolation, characterization and cloning of a cDNA encoding a new antifungal defensin from Phaseolus vulgaris L. seeds. Peptides 29 (12), 2090–2100. https://doi.org/10.1016/j.peptides.2008.08.008.

Garrigues, S., Gandía, M., Castillo, L., Coca, M., Marx, F., Marcos, J.F., Manzanares, P., 2018. Three antifungal proteins from Penicillium expansum: different patterns of production and antifungal activity. Front. Microbiol. 9, 2370. https://doi.org/10.3389/fmicb.2018.02370.

Gonzalez-Cendales, Y., Catanzariti, A.M., Baker, B., McGrath, D.J., Jones, D.A., 2016. Identification of I-7 expands the repertoire of genes for resistance to Fusarium wilt in tomato to three resistance gene classes. Mol. Plant Pathol. 17, 448–463. https://doi.org/10.1111/mpp.12294.

Goyal, S., Castrillón-Betancur, J.C., Klaile, E., Slevogt, H, 2018. The interaction of human pathogenic fungi with c-type lectin receptors. Front. Immunol. 9, 1261. https://doi.org/10.3389/fimmu.2018.01261.

Gupta, S., Bhar, A., Chatterjee, M., Das, S., 2013. Fusarium oxysporum f. sp. ciceri race 1 induced redox state alterations are coupled to downstream defense signaling in root tissues of chickpea (Cicer arietinum L.). PLoS One 8 (9), e73163. https://doi.org/10.1371/journal.pone.0073163.

Gupta, S., Chakraborti, D., Sengupta, A., Basu, D., Das, S., 2010. Primary metabolism of chickpea is the initial target of wound inducing early sensed Fusarium oxysporum f. sp. ciceri race I. PLoS One 5 (2), e9030. https://doi.org/10.1371/journal.pone.0009030.

Hegedüs, N., Marx, F., 2013. Antifungal proteins:more than antimicrobials? Fungal Biol. Rev. 26, 132–145. https://doi.org/10.1016/j.fbr.2012.07.002.

Hidaka, H., Nagatsu, T., Takeya, K., Takeuchi, T., Suda, H., 1969. Fusaric acid, a hypotensive agent produced by fungi. J. Antibiot. 22 (5), 228–230. https://doi.org/10.7164/antibiotics.22.228 (Tokyo).

Hou, S., Liu, D., Huang, S., Luo, D., Liu, Z., Xiang, Q., Wang, P., Mu, R., Han, Z., Chen, S., Chai, J., Shan, L., He, P., 2021. The Arabidopsis MIK2 receptor elicits immunity by sensing a conserved signature from phytocytokines and microbes. Nat. Commun. 12 (1), 5494. https://doi.org/10.1038/s41467-021-25580-w.

Houterman, P.M., Speijer, D., Dekker, H.L., De Koster, C.G., Cornelissen, B.J.C., Rep, M., 2007. The mixed xylem sap proteome of Fusarium oxysporum –infected tomato plants. Mol. Plant Pathol. 8 (2), 215–221. https://doi.org/10.1111/J.1364-3703.2007.00384.X.

Isaac, G.S., Abu-Tahon, M.A., 2014. *In vitro* antifugal activity of medicinal plant extract against Fusarium oxysporum f. sp. lycopersici race 3 the causal agent of tomato wilt. Acta Biol. Hung. 65 (1), 107–118. https://doi.org/10.1556/ABiol.65.2014.1.10.

Jiménez-Fernández, D., Landa, B.B., Kang, S., Jiménez-Díaz, R.M., Navas-Cortés, J.A., 2013. Quantitative and microscopic assessment of compatible and incompatible interactions between chickpea cultivars and Fusarium oxysporum f. sp. ciceris Races. PLoS One 8 (4), e61360. https://doi.org/10.1371/journal.pone.0061360.

Kadota, Y., Shirasu, K., Zipfel, C., 2015. Regulation of the NADPH oxidase RBOHD during plant immunity. Plant Cell Physiol. 56 (8), 1472–1480. https://doi.org/10.1093/pcp/pcv063.

Kang, B.R., Park, J.S., Jung, W.J., 2020. Antifungal evaluation of fengycin isoforms isolated from Bacillus amyloliquefaciens PPL against Fusarium oxysporum f. sp. lycopersici. Microb. Pathog. 149, 104509 https://doi.org/10.1016/j.micpath.2020.104509.

Kawalleck, P., Plesch, G., Hahlbrock, K., Somssich, I.E., 1992. Induction by fungal elicitor of S-adenosyl-L-methionine synthetase and S-adenosyl-L-homocysteine hydrolase mRNAs in cultured cells and leaves of Petroselinum crispum. Proc. Natl. Acad. Sci. U. S. A. 89 (10), 4713–4717. https://doi.org/10.1073/pnas.89.10.4713.

Khader, S.A., Gaffen, S.L., Kolls, J.K., 2009. Th17 cells at the crossroads of innate and adaptive immunity against infectious diseases at the mucosa. Mucosal Immunol. 2 (5), 403–411. https://doi.org/10.1038/mi.2009.100.

Kostyn, K., Czemplik, M., Kulma, A., Bortniczuk, M., Skała, J., Szop, J., 2012. Genes of phenylpropanoid pathway are activated in early response to Fusarium attack in flax plants. Plant Sci. 190, 103–115. https://doi.org/10.1016/j.plantsci.2012.03.011.

Krishnan, J., Selvarajoo, K., Tsuchiya, M., Lee, G., Choi, S., 2007. Toll-like receptor signal transduction. Exp. Mol. Med. 39, 421–438. https://doi.org/10.1101/cshperspect.a011247.

Król, P., Igielski, R., Pollmann, S., Kępczyńska, E., 2015. Priming of seeds with methyl jasmonate induced resistance to hemi-biotroph Fusarium oxysporum f.sp. lycopersici in tomato via 12-oxo-phytodienoic acid, salicylic acid, and flavonol accumulation. J. Plant Physiol. 179, 122–132. https://doi.org/10.1016/j.jplph.2015.01.018.

Kumar, Y., Zhang, L., Panigrahi, P., Dholakia, B.B., Dewangan, V., Chavan, S.G., Kunjir, S.M., Wu, X., Li, N., Rajmohanan, P.R., Kadoo, N.Y., Giri, A.P., Tang, H., Gupta, V.S., 2016. Fusarium oxysporum mediates systems metabolic reprogramming of chickpea roots as revealed by a combination of proteomics and metabolomics. Plant Biotechnol. J. 14, 1589–1603. https://doi.org/10.1111/pbi.12522.

Kwon, H.W., Yoon, J.H., Kim, S.H., Hong, S.B., Cheon, Y., Ko, S.J., 2007. Detection of extracellular enzymes activities in various Fusarium spp. Mycobiology 35 (3), 162–165. https://doi.org/10.4489/myco.2007.35.3.162.

Lagopodi, A.L., Ram, A.F.J., Lamers, G.E.M., Punt, P.J., Van den Hondel, C.A.M.J.J., Lugtenberg, B.J.J., Bloemberg, G.V., 2002. Novel aspects of tomato root colonization and infection by Fusarium oxysporum f. sp. radicis-lycopersici revealed by confocal laser scanning microscopic analysis using the green fluorescent protein as a marker. Mol. Plant Microbe Interact. 15, 172–179. https://doi.org/10.1094/mpmi.2002.15.2.172.

Lamaris, G.A., Chamilos, G.R.E., Kontoyiannis, D.P., 2007. Virulence studies of Scedosporium and Fusarium species in Drosophila melanogaster. J. Infect. Dis. 196, 1860–1864. https://doi.org/10.1086/523765.

Lanubile, A., Muppirala, U.K., Severin, A.J., Marocco, A., Munkvold, G.P., 2015. Transcriptome profiling of soybean (Glycine max) roots challenged with pathogenic and non-pathogenic isolates of Fusarium oxysporum. BMC Genom. 16, 1089. https://doi.org/10.1186/s12864-015-2318-2. Dec 21.

Li, C.Y., Deng, G.M., Yang, J., Viljoen, A., Jin, Y., Kuang, R.B., Zuo, C.W., Lv, Z.C., Yang, Q.S., Sheng, O., Wei, Y.R., Hu, C.H., Dong, T., Yi, G.J., 2012. Transcriptome profiling of resistant and susceptible Cavendish banana roots following inoculation with Fusarium oxysporum f. sp. cubense tropical race 4. BMC Genom. 13, 374. https://doi.org/10.1186/1471-2164-13-374.

Li J., Conneely L.J., Rep M. 2020. Partial pathogenicity chromosomes in Fusarium oxysporum are sufficient to cause disease and can be horizontally transferred. Environ. Microbiol. 10.111/1462-2920.15095.

Li, W., Li, C., Sun, J., Peng, M., 2017. Metabolomic, biochemical, and gene expression analyses reveal the underlying responses of resistant and susceptible banana species during early infection with Fusarium oxysporum f. sp. cubense. Plant Dis. 101 (4), 534–543. https://doi.org/10.1094/PDIS-09-16-1245-RE. Epub 2017 Jan 30PMID: 30677364.

Lincon, J.E., Sanchez, J.P., Zumstein, K., Gilchrist, D.G., 2018. Plant and animal PR1 family members inhibit programmed cell death and suppress bacterial pathogens in plant tissues. Mol. Plant Pathol. 19 (9), 2111–2123. https://doi.org/10.1111/mpp.12685.

Lionakis, M.S., Iliev, I.D., Hohl, T.M., 2017. Immunity against fungi. JCI Insight 2 (11), e93156. https://doi.org/10.1172/jci.insight.93156.

López-Abarrategui, C., Alba, A., Silva, O.N., Reyes-Acosta, O., Vasconcelos, I.M., Oliveira, J.T., Migliolo, L., Costa, M.P., Costa, C.R., Silva, M.R., Garay, H.E., Dias, S. C., Franco, O.L., Otero-González, A.J., 2012. Functional characterization of a synthetic hydrophilic antifungal peptide derived from the marine snail Cenchritis muricatus. Biochimie 94 (4), 968–974. https://doi.org/10.1016/j.biochi.2011.12.016.

López-Díaz, C., Rahjoo, V., Sulyok, M., Ghionna, V., Martín-Vicente, A., Capilla, J., Di Pietro, A., López-Berges, M.S., 2018. Fusaric acid contributes to virulence of Fusarium oxysporum on plant and mammalian hosts. Mol. Plant Pathol. 19 (2), 440–453. https://doi.org/10.1111/mpp.12536. Feb.

Ma, L.J., van der Does, H.C., Borkovich, K.A., Coleman, J.J., Daboussi, M.J., Di Pietro, A., Dufresne, M., Freitag, M., Grabherr, M., Henrissat, B., Houterman, P.M., Kang, S., Shim, W.B., Woloshuk, C., Xie, X., Xu, J.R., Antoniw, J., Baker, S.E., Bluhm, B.H., Breakspear, A., Brown, D.W., Butchko, R.A., Chapman, S., Coulson, R., Coutinho, P. M., Danchin, E.G., Diener, A., Gale, L.R., Gardiner, D.M., Goff, S., Hammond-Kosack, K.E., Hilburn, K., Hua-Van, A., Jonkers, W., Kazan, K., Kodira, C.D., Koehrsen, M., Kumar, L., Lee, Y.H., Li, L., Manners, J.M., Miranda-Saavedra, D., Mukherjee, M., Park, G., Park, J., Park, S.Y., Proctor, R.H., Regev, A., Ruiz-Roldan, M.C., Sain, D., Sakthikumar, S., Sykes, S., Schwartz, D.C., Turgeon, B.G., Wapinski, I., Yoder, O., Young, S., Zeng, Q., Zhou, S., Galagan, J., Cuomo, C.A., Kistler, H.C., Rep, M., 2010. Comparative genomics reveals mobile pathogenicity chromosomes in Fusarium. Nature 464 (7287), 367–373. https://doi.org/10.1038/nature08850.

Marr, K.A., Carter, R.A., Crippa, F., Wald, A., Corey, L, 2002. Epidemiology and outcome of mould infections in hematopoietic stem cell transplant recipients. Clin. Infect. Dis. 34 (7), 909–917. https://doi.org/10.1086/339202.

Mayayo, E., Pujol, I., Guarro, J., 1999. Experimental pathogenicity of four opportunist Fusarium species in a murine model. J. Med. Microbiol. 48, 363–366. https://doi.org/10.1099/00222615-48-4-363.

Mitta, G., Hubert, F., Noël, T., Roch, P., 1999. Myticin, a novel cysteine-rich antimicrobial peptide isolated from haemocytes and plasma of the mussel Mytilus galloprovincialis. Eur. J. Biochem. 265 (1), 71–78. https://doi.org/10.1046/j.1432-1327.1999.00654.x.

Moghaddam, M.R.B., Gross, T., Becker, A., Vilcinskas, A., Rahnamaeian, M., 2017. The selective antifungal activity of Drosophila melanogaster metchnikowin reflects the species dependent inhibition of succinate-coenzyme Q reductase. Sci. Rep. 7, 8192. https://doi.org/10.1038/s41598-017-08407-x.

Muhammed, M., Fuchs, B.B., Wu, M.P., Breger, J., Coleman, J.J., Mylonakis, E, 2012. The role of mycelium production and a MAPK-mediated immune response in the C. elegans-Fusarium model system. Med. Mycol. 50, 488–496. https://doi.org/10.3109/13693786.2011.648217.

Nag, P., Aggarwal, P.R., Ghosh, S., Narula, K., Tayal, R., Maheshwari, N., Chakraborty, N., Chakraborty, S., 2017. Interplay of neuronal and non-neuronal genes regulate intestinal DAF-16 mediated immune response during Fusarium infection of Caenorhabditis elegans. Cell Death Discov. 3, 17073 https://200410.1038/cddiscovery.2017.73.

Nahalkova, J., Fatehi, J., Olivain, C., Alabouvette, C., 2008. Tomato root colonization by fuorescent-tagged pathogenic and protective strains of Fusarium oxysporum in hydroponic culture differs from root colonization in soil. FEMS Microbiol. Lett. 286, 152–157. https://doi.org/10.1111/j.1574-6968.2008.01241.x.

Nelson, P.E., Dignani, M.C., Anaissie, E.J., 1994. Taxonomy, biology, clinical aspects of Fusarium species. Clin. Microbiol. Rev. 7 (4), 479–504. https://doi.org/10.1128/cmr.7.4.479.

Nucci, M., Anaissie, E., 2007. Fusarium infections in immunocompromised patients. Clin. Microbiol. Rev. 20 (4), 695–704. https://doi.org/10.1128/CMR.00014-07.

Nucci, M., Anaissie, E., 2002. Cutaneous infection by Fusarium species in healthy and immunocompromised hosts: implications for diagnosis and management. Clin. Inf. Dis. 35, 909–920. https://doi.org/10.1086/342328.

Nürnberger, T., Brunner, F., Kemmerling, B., Piater, L., 2004. Innate immunity in plants and animals:striking similarities and obvious differences. Immunol. Rev. 198, 249–266. https://doi.org/10.1111/j.0105-2896.2004.0119.x.

Oh, S.U., Yun, B.S., Lee, S.J., Kim, J.H., Yoo, I.D., 2002. Atroviridins A-C and neoatroviridins A-D, novel peptaibol antibiotics produced by Trichoderma atroviride F80317. I. Taxonomy, fermentation, isolation and biological activities. J. Antibiot. 55 (6), 557–564. https://doi.org/10.7164/antibiotics.55.557 (Tokyo).

Ortoneda, M., Guarro, J., Madrid, M.P., Caracue, Z., Roncero, M.I.G., Mayayo, E., Di Pietro, A., 2004. Fusarium oxysporum as a multihost model for the genetic dissection of fungal virulence in plants and mammals. Infect. Immun. 72, 1760–1766. https://doi.org/10.1128/iai.72.3.1760-1766.2004.

Parisi, K., Shafee, T.M.A., Quimbar, P., Van der Weerden, N.L., Bleackley, M.R., Anderson, M.A., 2018. The evolution, function and mechanisms of action for plant defensins. Semin. Cell Dev. Biol. https://doi.org/10.1016/j.semcdb.2018.02.004.

Park, S.C., Lee, J.R., Kim, J.Y., Hwang, I., Nah, J.W., Cheong, H., Park, Y., Hahm, K.S., 2010. Pr-1, a novel antifungal protein from pumpkin rinds. Biotechnol. Lett. 32 (1), 125–130. https://doi.org/10.1007/s10529-009-0126-y.

Perincherry, L., Lalak-Kánczugowska, J., Stępién, Ł., 2019. Fusarium-produced mycotoxins in plant-pathogen interactions. Toxins 11, 664. https://doi.org/10.3390/toxins11110664.

Porter, J.K., Bacon, C.W., Wray, E.M., Hagler, W.M., 1995. Fusaric acid in Fusarium moniliforme cultures, corn, feeds toxic to livestock and the neurochemical effects in the brain and pineal gland of rats. Nat. Toxins 3, 91–100. https://doi.org/10.1002/nt.2620030206.

Rep, M., Dekker, H.L., Vossen, J.H., De Boer, A.D., Houterman, P.M., Speijer, D., Back, J. W., De Koster, C.G., Cornelissen, B.J.C., 2002. Mass spectrometric identification of isoforms of PR proteins in xylem sap of fungus-infected tomato. Plant Physiol. 130, 904–917. https://doi.org/10.1104/pp.007427.

Sander, A., Beyer, U., Amberg, R., 2009. Systemic Fusarium oxysporum infection in an immunocompetent patient with an adult respiratory distress syndrome (ARDS) and extracorporal membrane oxygenation (ECMO). Mycoses 41 (3–4), 109–111. https://doi.org/10.1111/j.1439-0507.1998.tb00310.x.

Schäfer, K., Bain, J.M., Di Pietro, A., Gow, N.A.R., Erwig, L.P., 2014. Hyphal growth of phagocytosed Fusarium oxysporum causes cell lysis and death of murine

macrophages. PLoS One 9 (7), e101999. https://doi.org/10.1371/journal.pone.0101999.
Schmidt, R., Kunkowska, A.B., Schippers, J.H., 2016. Role of reactive oxygen species during cell expansion in leaves. Plant Physiol. 172 (4), 2098–2106. https://doi.org/10.1104/pp.16.00426.
Sexton, A.C., Howlett, B.J., 2006. Parallels in fungal pathogenesis on plant and animal hosts. Eukaryotic Cell 5 (12), 1941–1949. https://doi.org/10.1128/EC.00277-06.
Shen, Y., Diener, A.C., 2013. Arabidopsis thaliana resistance to Fusarium oxysporum 2 implicates tyrosine-sulfated peptide signaling in susceptibility and resistance to root infection. PLos Genet. 9 (5), e1003525 https://doi.org/10.1371/journal.pgen.1003525.
Simons, G., Groenendijk, J., Wijbrandi, J., Reijans, M., Groenen, J., Diergaarde, P., Van der Lee, T., Bleeker, M., Onstenk, J., de Both, M., Haring, M., Mes, J., Cornelissen, B., Zabeau, M., Vos, P., 1998. Dissection of the Fusarium I2 gene cluster in tomato reveals six homologs and one active gene copy. Plant Cell 10 (6), 1055–1068. https://doi.org/10.1105/tpc.10.6.1055.
Singh, V.K., Singh, H.B., Upadhyay, R.S., 2017. Role of fusaric acid in the development of 'Fusarium wilt' symptoms in tomato: physiological, biochemical and proteomic perspectives. Plant Physiol. Biochem. 118, 320–332. https://doi.org/10.1016/j.plaphy.2017.06.028.
Singh, V.K., Upadhyay, R.S., 2014. Fusaric acid induced cell death and changes in oxidative metab-olism of Solanum lycopersicum L. Bot. Stud. 55, 66. https://doi.org/10.1186/s40529-014-0066-2.
Smith, J.A., Kauffman, C.A., 2012. Pulmonary fungal infections. Respirology 17 (6), 913–926. https://doi.org/10.1111/j.1440-1843.2012.02150.x.
Smith, T.K., MacDonald, E.J., 1991. Effects of Fusaric acid on brain regional neurochemistry and vomiting behaviour in swine. J. Anim. Sci. 69, 2044–2049. https://doi.org/10.2527/1991.6952044x.
Son, G.H., Wan, J., Kim, H.J., Nguyen, X.C., Chung, W.S., Hong, J.C., Stacey, G., 2012. Ethylene-responsive element-binding factor 5, ERF5, is involved in chitin-induced innate immunity response. Mol. Plant Microbe Interact. 25 (1), 48–60. https://doi.org/10.1094/MPMI-06-11-0165.
Sperschneider, J., Gardiner, D.M., Thatcher, L.F., Lyons, R., Singh, K.B., Manners, J.M., Taylor, J.M., 2015. Genome-wide analysis in three Fusarium pathogens identifies rapidly evolving chromosomes and genes associated with pathogenicity. Genome Biol. Evol. 7 (6), 1613–1627. https://doi.org/10.1093/gbe/evv092, 19.
Steinkellner, S., Mammerler, R., 2007. Effect of flavonoids on the development of Fusarium oxysporum f. sp. lycopersici. J. Plant Interact. 2 (1), 17–23. https://doi.org/10.1080/17429140701409352.
Sun, Y., Chandra, J., Mukherjee, P., Szczotka-Flynn, L., Ghannoum, M.A., Pearlman, E, 2010. A murine model of contact lens–associated Fusarium keratitis. Invest. Ophthalmol. Vis. Sci. 51 (3), 1511–1516.
Tarabishy, A.B., Aldabagh, B., Sun, Y., Imamura, Y., Mukherjee, P.K., Lass, J.H., Ghannoum, M.A., Pearlman, E., 2008. MyD88 regulation of Fusarium keratitis is dependent on TLR4 and IL-1R1 but not TLR2. J. Immunol. 181, 593–600. https://doi.org/10.4049/jimmunol.181.1.593.
Taylor, P.R., Leal, S.M., Sun, Y., Pearlman, E., 2014. Aspergillus and Fusarium corneal infections are regulated by Th17 cells and IL-17-producing neutrophils. J. Immunol. 192 (7), 3319–3327. https://doi.org/10.4049/jimmunol.1302235.
Tian, C., Gao, B., Rodriguez, M.C., Lanz-Mendoza, H., Ma, B., Zhu, S, 2008. Gene expression, antiparasitic activity, functional evolution of the drosomycin family. Mol. Immunol. 45 (15), 3909–3916. https://doi.org/10.1016/j.molimm.2008.06.025.
Tobias, H., Carr, L.A., Voogt, J.L., 1983. Catecholamine mechanisms in the feedback effects of estradiol benzoate on the release of LH and prolactin. Proceedings of the Society for Experimental Biology and Medicine Soc. Exp. Biol. Med. 174 (2), 284–290. https://doi.org/10.3181/00379727-174-41738 (New York, N. Y.).
Upasani, M.L., Gurjar, G.S., Kadoo, N.Y., Gupta, V.S., 2016. Dynamics of colonization and expression of pathogenicity related genes in Fusarium oxysporum f. sp. ciceri during chickpea vascular wilt disease progression. PLoS One 11 (5), e0156490. https://doi.org/10.1371/journal.pone.0156490.
van Dam, P., Fokkens, L., Ayukawa, Y., van der Gragt, M., Ter Horst, A., Brankovics, B., Houterman, P.M., Arie, T., Rep, M, 2017. A mobile pathogenicity chromosome in Fusarium oxysporum for infection of multiple cucurbit species. Sci. Rep. 7 (1), 9042. https://doi.org/10.1038/s41598-017-07995-y.
Van de Veerdonk, F.L., Kullberg, B.J., Van der Meer, J.M.W., Gow, N.A.R., Netea, M.G., 2008. Host–microbe interactions:innate pattern recognition of fungal pathogens. Curr. Opin. Microbiol. 11, 305–312. https://doi.org/10.1016/j.mib.2008.06.002.
Van der Weerden, N.L., Bleackley, M.R., Anderson, M.A., 2013. Properties and mechanisms of action of naturally occurring antifungal peptides. Cell. Mol. Life Sci. 70 (19), 3545–3570. https://doi.org/10.1007/s00018-013-1260-1.
Van Loon, L.C., Rep, M., Pieterse, C.M.J., 2006. Significance of inducible defense-related proteins in infected plants. Annu. Rev. Phytopathol. 44, 135–162. https://doi.org/10.1146/annurev.phyto.44.070505.143425.
Wang, C.J., Thanarut, C., Sun, P.L., Chung, W.H., 2020. Colonization of human opportunistic Fusarium oxysporum (HOFo) isolates in tomato and cucumber tissues assessed by a specific molecular marker. PLoS One 15 (6), e0234517. https://doi.org/10.1371/journal.pone.0234517.
Wang, H., Wang, Z., Liu, Z., Wang, K., Xu, W, 2021. Membrane disruption of Fusarium oxysporum f. sp. niveum induced by myriocin from Bacillus amyloliquefaciens LZN01. Microb. Biotechnol. 14 (2), 517–534. https://doi.org/10.1111/1751-7915.13659.
Waszczak, C., Carmody, M., Kangasjärvi, J, 2018. Reactive oxygen species in plant signaling. Annu. Rev. Plant Biol. 69, 209–236. https://doi.org/10.1146/annurev-arplant-042817-040322.
Wickern, G.M., 1993. Fusarium allergic fungal sinusitis. J. Allergy Clin. Immunol. 92, 624–625. https://doi.org/10.1016/0091-6749(93)90087-v.
Williams, A.H., Sharma, M., Thatcher, L.F., Azam, S., Hane, J.K., Sperschneider, J., Singh, K.B., 2016. Comparative genomics and prediction of conditionally dispensable sequences in legume-infecting Fusarium oxysporum formae speciales facilitates identification of candidate effectors. BMC Genom. 17, 191. https://doi.org/10.1186/s12864-016-2486-8.
Xie, M., Zhang, J., Tschaplinski, T.J., Tuskan, G.A., Chen, J.G., Muchero, W., 2018. Regulation of lignin biosynthesis and its role in growth-defense tradeoffs. Front. Plant Sci. 9 (1427) https://doi.org/10.3389/fpls.2018.01427.
Zhang, J., Mace, M., Stipanovic, R., Bell, A., 1993. Production and fungitoxicity of the terpenoid phytoalexins in Cotton inoculated with Fusarium oxysporum f. sp. vasinfectum. J. Phytopathol. 139, 247–252. https://doi.org/10.1111/j.1439-0434.1993.tb01423.x.
Zhang, Y., Yang, H., Turra, D., Zhou, S., Ayhan, D.H., DeIulio, G.A., Guo, L., Broz, K., Wiederhold, N., Coleman, J.J., Donnell, K.O., Youngster, I., McAdam, A.J., Savinov, S., Shea, T., Young, S., Zeng, Q., Rep, M., Pearlman, E., Schwartz, D.C., Di Pietro, A., Kistler, H.C., Ma, L.J., 2020. The genome of opportunistic fungal pathogen Fusarium oxysporum carries a unique set of lineage-specific chromosomes. Comput. Biol. 3 (1), 50. https://doi.org/10.1038/s42003-020-0770-2.