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Review

Fusarium Fungi Pathogens, Identification, Adverse Effects, Disease Management, and Global Food Security: A Review of the Latest Research

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Abstract: *Fusarium* pathogens are ubiquitous and mainly associated with diseases in plants. They are the subject of great economic concern in agriculture due to crop losses to contamination of cereal grains with mycotoxins. *Fusarium* species are also considered agents of human and animal mycotic infections, having a wide-ranging spectrum of clinical manifestations in immunocompromised patients. *Fusarium* phytopathogens infect a wide variety of plants and cause symptoms ranging from stunted growth, fruit or seed decay, yellowing, and wilting of the leaves and cankers to root or stem decay. The identification of these fungi is difficult due to their pleomorphic tendency and the presence of both homothallic and heterothallic strains in the same species, and so is identifying them at species level because of variation among isolates. However, molecular tools have so far been very powerful in species identification and phylogeny, as the great diversity of the *Fusarium* genus has compelled scientists to continuously revise previous taxons. Mostly, *Fusarium* diseases are difficult to control, as fungi easily overcome host resistance to various methods of control. We present an overview of the recent research on *Fusarium* fungi, its adverse effects, and its impacts on food security. We further elucidate various methods of identifying them to encourage much-needed research on integrated management of this unavoidable food contaminant to achieve sustainable global food security.

Keywords: *Fusarium*; fungi; pathogens; identification; adverse effects; disease management; food security

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1. Introduction

More than 70% of plant diseases can be traced to fungi or fungus-like pathogens that threaten food security [1]. In addition, many fungal pathogens including *Fusarium* spp. also produce mycotoxins, that further threaten human and livestock health [2,3]. *Fusarium* spp. are also important to food safety in crop production and especially to the production of cereals. The genus *Fusarium* is one of the most economically important fungi, containing many agronomically important plant pathogens, mycotoxin producers, and opportunistic human pathogens [4]. This large and diverse fungi family breeds these mycotoxins mostly in the field before harvest. *Fusarium* species adapt to a variety of habitats and, although they have a notable affinity for moderate climates, they contaminate crops all around the world [5]. Consumption of mycotoxin-contaminated grain by humans or animals can cause acute or chronic illness and, in some cases, death. The most toxic and prevalent *Fusarium* toxins of economic importance include trichothecenes, fumonisins, and zearalenone.

Many *Fusarium* species are pathogenic to several important crops; two of them, namely, *F. graminearum* and *F. oxysporum*, are listed among the ten most important fungal pathogens

in plant pathology [6]. Possible disease symptoms caused by members of Fusarium include root or stem rots, cankers, vascular wilts, fruit or seed rots, and leaf diseases. Most fusarium species can cause a wide range of plant diseases that affect many crops, including major food and cash crops such as wheat, barley, maize, bananas, and cotton, often with devastating socio-economic impact [7].

Cereal crops provide a major food source for humans and animals, yet they are heavily attacked by fungal pathogens. These diseases are often insidious, without obvious symptoms at first, and are extremely difficult to control [7]. Their success as plant pathogens can be attributed to wide host ranges, endophytic infection, and varied modes of survival and dispersal. Representatives occur in virtually all bioclimatic regions of the world in agricultural and natural ecosystems [7]. Also, the emergence of novel fungal pathogens can threaten food production and/or ecological environments. Hence, the successful control of fungal diseases is challenging, as fungi can easily overcome host resistance [8]. Biological control of fungal diseases is one such approach that offers much toward increasing world crop production.

2. *Fusarium*: Overview and Taxonomy

The taxonomy of *Fusarium* started in 1809 when the genus *Fusarium* was first described by Link [9]. It comprises naturally ubiquitous species [10,11]. *Fusarium* is a large group of filamentous fungi that occur predominantly in the air and soil and which usually associate with plants and occasionally with humans. Some of the most important plant pathogenic fungal species known today are members of this genus [12]. Worldwide, it is a concern that a large number of economically important plant species are susceptible to at least one or more *Fusarium* spp. [12]. Fungi now included in the genus *Fusarium* were originally described and defined as *Fusisporium* based on the type *Fusisporium Roseum* described by Link in 1809 [13]. Wollenweber and Reinking [14] reclassified the two *F. roseum* type specimens as *F. sambucinum* and *F. graminearum* and currently accept *F. sambucinum* as the type species for the genus. Although the taxonomy of *Fusarium* continues to undergo major changes, mainly based on molecular classifications, the Wollenweber and Reinking classification system continues to form the foundation on which species are described [12].

Members of the genus *Fusarium* are characterized by having septate, hyaline, delicately curved, and elongate macroconidia [12,15]. Mycelia and spore masses are generally brightly colored [16]. In some species, smaller, 0- to 1-septate microconidia and chlamydospores are common, while some authors recognize a third conidial type known as mesoconidium. The *Fusarium* genus comprises around 70 recognized species, identified using a polyphasic approach, and about 300 putative species. Following phylogenetic species concepts, many putative species do not yet have formal names [17].

3. Identification of Phytopathogenic *Fusarium* Fungal Species

3.1. Morphological Characters for Identifying Phytopathogenic Fusarium Species

Morphological characters are considered the most traditional criteria used to identify any fungal species. *Fusarium* produces a range of mycelia that are cottony with shades of pink, yellow, and purple. Some species produce either macroconidia or microconidia as asexual reproductive structures, while other species produce both macroconidia and microconidia [18]. The morphology of microscopic characteristics, i.e., the general shape and dimensions of the macroconidia, the generation of microconidia, chlamydospores, sclerotia, sexual stages, and pigmentation, are the primary means used for the identification of *Fusarium* species. Members of the genus are variable in cultural characteristics because changes in the environment in which they grow can result in morphological changes both in culture and in conidia [15]. However, the description of these characteristics is necessary under very specific environmental conditions because of the distinctive cultural variability of morphological traits.

3.1.1. Universally Found Characters

The genus *Fusarium* is categorized by the production of septate, hyaline, delicately curved, elongate macroconidia and chlamydospores along with other secondary characteristics like mycelial growth and pigmentation [15].

(i) The macroconidium is the single most important cultural characteristic for the identification of a culture of *Fusarium* species. The most distinctive characteristic of the macroconidia is its shape, followed by the size and number of septa, and finally, the nature of apical, basal, or foot cells [12]. Regarding shape, most *Fusarium* produce sickle-shaped macroconidia that can be characterized into three types: (1) straight macroconidia, which can appear virtually needle-like if they are thin, e.g., *F. avenacum*; (2) microconidia having dorsivental curvature along all or a portion of the spore (these spores are almost of the same width along their entire length, e.g., *F. equiseti*); and (3) microconidia in which the dorsal side is more curved than the ventral side, e.g., *F. crookwellence* (Figure 1). Macroconidia can be long (*F. armeniacum*) or short (*F. culmorum*), but usually spore size is a relatively constant character and major variations indicate improper culture conditions. Typically, *Fusarium* macroconidia are 3–5-septate. The number of septa ought to be determined depending on the range and the average number of septa per spore [12].

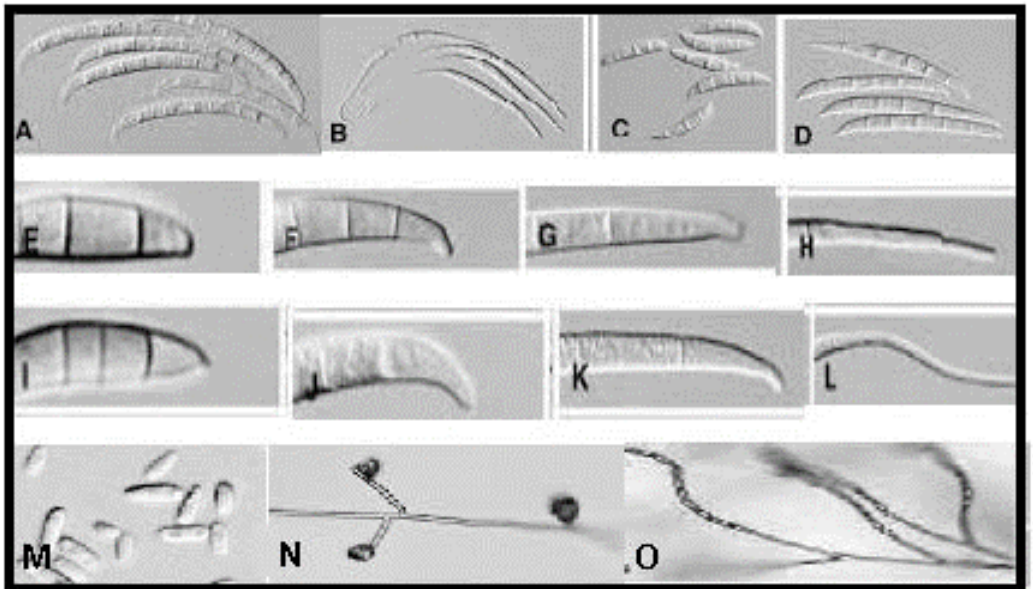

Figure 1. Spore morphological characteristics of *Fusarium* spp. (**A**–**D**): Macroconidia shapes; (**E**–**H**): macroconidia apical cell shapes; (**I**–**L**): macroconidia basal cell shapes; (**M**): microconidia shapes; (**N**): phialide morphology; (**O**): microconidial chains. Adapted from [12].

Another important macroconidium characteristic is the apical and basal cell forms. There are four common forms of apical cells: (1) blunt, e.g., *F. culmorum*; (2) papillate, e.g., *F. sambucinum*; (3) hooked, e.g., *F. lateritium*; and (4) Tapering, e.g., *F. equiseti* (Figure 1).

The apical cell length also can vary amongst species, but it is usually constant within a species. The main diagnostic features of apical cells are the degree of curvature, relative length, and general form. Microconidia are not produced by all *Fusarium* species; therefore, their presence is a potential diagnostic characteristic for *Fusarium* identification. The major characteristics regarding microconidia are the microconidia shape, the endogenous cells where they are born, and their arrangement on or around the conidiogenous cell [12].

(ii) Chlamydospores are verrucose (rough) or smooth-walled structures produced individually, e.g., *F. solani*; as doubles or pairs, e.g., *F. compactum*; as clumps, e.g., *F. scirpi*; or as chains, e.g., *F. compactum*. Chlamydospores are produced rarely and take a longer time (more than 6 weeks) when compared to macro- or microconidia. The

presence of chlamydospores in the aerial mycelia or embedded on the agar surface is another important criterion used in the identification of *Fusarium* species [12].

3.1.2. Other Important Characteristics

The characteristics discussed above are universally found in almost all *Fusarium* species. Some other characteristics are restricted to only a few species of *Fusarium*, which serve as an important delimiting factor in their identification; for example, mesoconidia teleomorphism and other characteristics like circulated (coiled) hyphae in the case of *F. circinatum*. In addition, the formation of sclerotia-like structures (compact masses of hardened mycelium with stored reserves of food material), etc., are some other relevant characteristics that help in the primary identification process [12].

However, the most important and diagnostically relevant secondary characteristic is pigmentation [12]. The different *Fusarium* species produce colors ranging from yellow to orange to carmine red [19]. The pattern of pigmentation is detectable on PDA and a 12:12 h light:dark cycle is usually preferred. Pigments produced by these fungi may be sensitive to light or pH and may be diffusible or non-diffusible into the growth media, and most of the evaluations are carried out a week after incubation. Another important characteristic is the growth rate of the species, usually measured as colony diameter from PDA plates incubated with a single spore culture and incubated at 25 or 30 degrees for 3 days. There are slow-growing species like *F. lateritium* and *F. merismoides* and fast-growing species like *F. culmorum, F. graminearum,* etc. [12]. These characteristics, if not properly analyzed, may not be clear and are not usually preferred for the identification of species. Furthermore, morphological identification is time-consuming and could easily result in misidentifications, especially for phylogenetically related species.

Secondary metabolites and mycotoxins are also characteristic features, which may influence a particular order in the culture and serve as specific secondary characteristics. The chemical background of the metabolites or mycotoxins can be used to primarily group the fungi, which can be further analyzed to finally assign the fungi to particular species. *Fusarium* is known to produce many toxins, which can be effectively used for their specific identification (Table 1).

Table 1. *Fusarium* species and their major mycotoxins.

***Fusarium* Species**	**Major Mycotoxins Produced**
F. cerealis	NIV, FX, ZEA
F. culmorium	DON, 3-AcDON, 15-AcDON, NIV, FX, ZEA
F. equiseti	ZEA, DAS
F. graminearium	DON, 15-AcDON, NIV, FX, ZEA
F. oxysporium	Moniliformin, fusaric acid
F. poae	T-2 toxin, HT-2 toxin, NIV, DAS, FX
F. ploriferatum	Fumonisin, fusarin C, moniliformin
F. solani	Fusaric acid, solaniol
F. sporotrichioides	T-2 toxin, HT-2 toxin, NEO, DAS, FX, ZEA
F. verticiloides	Fumonisin, fusarin C, moniliformin
F. sambucinum	Sambutoxin, DON, DAS, T-2 toxin

Deoxynivalenol (DON), 3-acetyl DON (3-AcDON), 15-acetyl DON (15-AcDON), Nivalenol (NIV), Zearalenone (ZEA), Fusarenon (FX), Neosolaniol (NEO), Diacetoxyscirpinol (DAS).

3.2. Molecular Tools for Identifying Phytopathogenic Fusarium Species Based on Genetic Diversity

Although morphological identification alone might be problematic, it is still helpful in practice and frequently used in combination with molecular methods. Different molecular tools, for example, PCR-based techniques, aid in the description of differences amongst species according to genetic diversity. Some examples such as random amplified

polymorphic DNA (RAPD) [20], restriction fragment length polymorphism (RFLP) [21], amplified fragment length polymorphism (AFLP) [22,23], DNA sequences of intergenic spacers (IGS) [24], β-tubulin (*tub2*) [25], translation elongation factor-1 alpha (TEF-1α) [26], and internal transcribed spacers (ITS) [27] have been used to differentiate and diagnose fungal strains. Analyses of other genes such as calmodulin, topoisomerase II and cell biohydrolase-C have also been used for the identification of *Fusarium* [28,29]. In addition, the MALDI-TOF MS technique is also used in agriculture and food safety for the identification of *Fusarium* species (Wigmann et al., 2019) [30].

(i) Random Amplified Polymorphic DNA (RAPD)

This is a PCR technique where primers (usually 10-20 base-pair (bp) in length) randomly bind to complementary sequences of the genomic DNA of a given organism, which leads to the generation of consensus sequence patterns that serve as fingerprints for the organism [31]. This technique works in such a way that nucleotide sequence variation due to insertions, additions, or base substitutions, inversion of priming site, conformational changes in the template DNA, etc., in the PCR priming regions, especially at the 3′ ends, prevent primer annealing. This results in different-sized PCR fragments that are highly specific for a particular species [32]. The RAPD assays have been effectively used for genome analyses of different bacteria and fungi [33,34]. In addition, RAPD has been used for the identification of other *Fusarium* species such as *F. oxysporum*, *F. avenaceum*, *F. poae*, *F. solani*, and *F. moniliforme* [35–38]. Despite the advantages of RAPD, it has been criticized due to poor reproducibility of results, affecting its use in fungal taxonomy, and there is a need for fastidious PCR conditions [39,40].

(ii) Restriction fragment length polymorphism (RFLP)

This technique is based on restriction enzyme digestion of the pathogen DNA, and afterward, separation of the fragments by electrophoresis in agarose or polyacrylamide gels to identify differences in the sizes of DNA fragments [41]. Polymorphisms within the restriction enzyme cleavage sites are meant to differentiate fungal species. Although the DNA restriction profile can be directly noticed by staining the gels, Southern blot analysis is usually needed. The DNA is transferred to appropriate membranes and hybridized with an appropriate probe [40]. RFLPs have been largely used for the study of the diversity of mycorrhizal and soil fungal populations/communities [21,42,43]. Although meant for the differentiation of pathogenic fungi [44], this early technique has been increasingly supplanted by other fingerprint techniques based on PCR. RFLP combines the amplification of a target region with the further digestion of the PCR products obtained.

(iii) Amplified fragment length polymorphism (AFLP)

The AFLP technology [45] is a modified version of RAPD, which is based on the use of restriction enzymes to digest total genomic DNA followed by ligation of restriction half site-specific adaptors to all restriction fragments. Then, selective amplification of these restriction fragments is achieved with PCR primers that have in their 3′ end the corresponding adaptor sequence and selective bases. The bands of the amplified fragments are visualized on denaturing polyacrylamide gels. The AFLP technology can amplify between 50 and 100 fragments at one time and detect various polymorphisms in different genomic regions simultaneously. It is also very sensitive and reproducible. The disadvantages of AFLPs are that they need high-molecular-weight DNA and more technical expertise than RAPDs (ligations, restriction enzyme digestions, and polyacrylamide gels) and that AFLP analyses suffer the same analytical limitations as RAPDs [46]. AFLP has been used to differentiate fungal isolates at several taxonomic levels, e.g., to differentiate *Monilinia laxa* that infect apple trees from isolates infecting other host plants [47] and to separate non-pathogenic strains of *Fusarium oxysporum* from those of *F. commune* [48]. AFLP profiles have also been widely used for the phylogenetic analysis of the *Fusarium oxysporum* complex [49–51]. Leissner et al. [22] also used AFLP to differentiate between isolates of *F. graminearum*.

(iv) Inter-genic Spacers (IGS)

These are regions that separate nuclear ribosomal DNA repeat units, which consist of highly conserved genes and more variable spacer regions [52]. The number of ribosomal DNA repeat units varies among different species, and this results in variations in the length and restriction sites of IGS [53]. IGS-RFLP has been used for the analysis of genetic variation within and between closely related species or communities [54,55]. Analysis of the IGS region with the RFLP technique has been effectively used for phylogenetic analysis of closely related species of *Fusarium*, such as *F. langsethiae* vs. *F. sporotrichioides* and *F. poae* vs. *F. kyushuense*. The result showed clear differentiation between the two species [24].

(v) β-tubulin

The β-tubulin gene sequences have been widely used for phylogenetic investigations in various fungi [56,57]. It has also been used in the phylogenetic investigations of *F. xylarioides* [58]. Schmidt et al. [23] also used DNA sequences of β-tubulin along with other marker genes for the taxonomic study of *F. langsethiae*, *F. poae*, and *F. sporotrichioides*.

(vi) Translation elongation factors

TEF-1a has been widely used for *Fusarium* classification because it is highly informative at the species level in *Fusarium* [58]. Also, universal primers have been designed that work across the genus [58] to amplify a ~700 bp region of TEF, including three introns. These introns cover over half of the amplicon's length in all known *Fusarium* species [59]. The TEF gene is a single copy in *Fusarium* and its sequence shows high variability among closely related species.

(vii) Internal Transcribed Spacers (ITS)

Internal Transcribed Spacer (ITS) regions are useful tools for the identification of different fungal species [60]. Internal Transcribed Spacers ITS1 and ITS2 undergo more variations even within closely related species and hence are widely used for identification processes and for studying evolutionary events. The highly conserved priming site of the ITS region makes it easy to be amplified from practically all fungal species. The stretches of DNA between 18S, 5.8S, and 28S rRNA regions make up the ITS regions [27]. The growing ITS sequence data is also an added advantage that helps in the identification of various fungi. This information can also be used for the development of species-specific primers for the detection of some fungi in a much-reduced time instead of via the morphology method [29]. For the identification of *Fusarium* species, ITS-RFLPs has been extensively used [61]. Variations occurring in ITS1 and ITS2 sequences have been used to study the genetic relationship between different *Fusarium* species [61].

The ITS region is the perfect region for species identification since even closely related species have sequence variations [27]. In addition, these variable regions are flanked by conserved ribosomal RNAs, which provide the option to use primers for PCR amplification of the variable regions that are recognized by the majority of fungi and fungal-like organisms. For fungal identification, most people use the ITS1/ITS4 primer pair [27] for amplification of the ITS region, which leads to another fact in favor of ITS as a barcode marker: there are huge databases consisting of ITS sequences from the majority of known fungi. Though perfect for species identification, the ITS region is too variable to determine the phylogeny of higher ranks.

This region contains highly conserved areas adequate for genera- or species-consensus primer designing (RNA ribosomal genes), alternate with highly variable areas that allow discrimination over a wide range of taxonomic levels (ITS region) [27]. The ITS region is ubiquitous and found in all eukaryotes. In addition, the high copy numbers of rRNA genes in the fungal genome enable a highly sensitive PCR amplification. Furthermore, large numbers of ribosomal sequences are publicly available in databases, facilitating the validation and reliability of the detection assays. Traditionally, molecular identification of plant pathogenic fungi is accomplished by PCR amplification of the ITS region followed by either restriction analysis [62] or direct sequencing and BLAST searching against GenBank or other databases [27].

(viii) The matrix-assisted laser desorption/ionization time-of-flight mass spectrometry

This technique can also be applied for the identification and differentiation of *fusarium* species, e.g., *F. fujikuroi* and *F. solani*. Besides the medically important fungi, reliable identification at the species level using MALDI-TOF MS has been reported for environmental fungi and other food-spoilage fungi [63,64]. Unlike the PCR-based methods, this technique is an advanced tool for rapid and accurate identification of fungi and other microbes using either intact cells or cell extracts. It is based on the molecular mass spectral readout from an ionized protein mixture. The cell culture assay is carried out based on the mass spectral pattern, which can be taken as the unique fingerprint to identify fungi among closely related species [65].

4. The Pathogen—*Fusarium*

The genus *Fusarium* comprises numerous toxigenic species that are pathogenic to plants and/or humans. They are capable of colonizing various environments on earth [17]. *Fusarium* species as versatile fungi are found everywhere, such as in air, water, soil, plants, and on organic substrates. *Fusarium*'s widespread distribution is attributable to its ability to withstand a wide range of conditions and grow on a broad range of substrates, as well as their efficient mechanisms for dispersal [66]. Often regarded as soil-borne fungi, since they are abundant in soil and frequently associated with plant roots, they are also present in water as parts of water biofilms [67]. *Fusarium* species have been isolated from public swimming pools, shower drainage pipes, and hospital water systems [68].

These field fungi require high moisture levels to colonize and contaminate grain [69,70]. Aside from their ability to act as plant pathogens, *Fusarium* species have been linked to a wide range of diseases and infections, directly or indirectly in humans and animals [71]. *Fusarium* is one of the most economically important fungal genera because of yield loss due to plant pathogenic activity. Mycotoxin contamination of food and feed products, which often renders them unacceptable for marketing, also adds to the huge economic losses of the agricultural industry and poses a great threat to human and animal health due to consumption of mycotoxins [71–73]. Management of plant *Fusarium* diseases is difficult as they are both seed-borne and soil-borne, and many strains have been reported as etiologic instruments of infections in humans [74]. It is therefore important to gain insight into the processes involved in the development of diseases in the host of this transkingdom pathogen.

4.1. Fusarium as a Plant Pathogen

Fusarium is known to contain a range of plant pathogenic fungal species, with a report of devastating infections (Table 2) and has been in existence for the past two centuries [12]. As common invaders of aerial plant parts, they can either be part of the normal mycoflora or act as plant pathogens on horticultural crops and cereal grains, such as maize, where they render them unfit for consumption [75,76]. *Fusarium* spp. can cause seedling, root, and crown rot as well as stalk and ear rot at any stage of plant development [77–79]. For fusarium infection of plants to be successful, different highly regulated processes are involved, starting from initial infection to development of disease in the host [80].

(i) Adhesion

Fungal infection begins after the recognition of roots through unknown host signals, followed by infectious hyphae adhering to the host root surface [81]. This adhesion process of fungi to the host surface is not specific, as they may adhere to the surface of either hosts or non-hosts [82]. Site-specific binding may be vital in the attachment of the propagules at the root surface, after which other processes required for colonization can continue [83].

(ii) Entry

During pathogenesis, the fungus penetrates the complex physical defense barriers of the host plant cell walls [84,85]. Gaining entrance to plant cells requires hydrolytic degradation of physical host barriers such as the cell-wall endodermis, whereby fungi

secrete a mixture of hydrolytic enzymes including cutinases, cellulases, pectinases, and proteases [86]. This is for it to reach the vascular tissues where it lodges.

After penetration in the vascular tissues, it has to adapt to the hostile plant environment and tolerate plant antifungal compounds. The fungus tries suppressing and inactivating host defense responses, usually by secreting toxins or plant-hormone-like compounds that manipulate the plant's physiology to the benefit of the pathogen [86]. This quite often is achieved through the production of phytotoxins with varying degrees of specificity toward different plants [87].

(iii) Colonization, Adaptation, and Propagation

During colonization, the mycelium spreads intracellularly through the root cortex until it reaches the xylem vessels and enters them through the pits. The fungus then remains solely within the xylem vessels, using them to colonize the host [88]. Fungal colonization of the host's vascular system is often fast and frequently facilitated by the formation of microconidia inside the xylem vessel elements [89] that are detached and taken upward in the sap stream [88]. As soon as the perforation plates stop the spores, they ultimately germinate, and germ tubes pierce the perforation plates. Subsequently, hyphae, conidiophores, and conidia are formed [89,90].

(iv) Disease development

Wilting is usually triggered by various pathogen activities. These include a buildup of fungal mycelium in the xylem tissue and/or production of toxins; host defense responses comprising the production of gels, gums, and tyloses; and vessel crushing by multiplication of adjacent parenchyma cells [91]. The wilting symptoms seem to be a result of serious water stress, mainly due to vessel occlusion. Symptoms are somewhat variable but involve combinations of vein clearing, leaf epinasty, wilting, chlorosis, necrosis, and abscission. Harshly infected plants may wilt and die, while plants affected to a lesser extent may become stunted and unproductive [92].

In other words, the parasitic phase in the *Fusarium* disease development involves the fungus' penetration of host plant roots, colonization of the root cortex and endodermis, movement to and colonization of the xylem vessels of stems/leaves, development of the symptoms, and finally, death of the host plant. Meanwhile, the saprophytic stage of *Fusarium* disease development involves the formation of resting structures in the dead host [93]. With fungal penetration of the host, the management of the multiplication rate of the fungus can be very challenging. During the dormant stage of the *Fusarium*, the germination of mycelia, chlamydospores, microconidia, and macroconidia (fungal propagules) that are present in infected soil is suppressed because of microbiostasis or mycostasis [94].

Table 2. Phytopathogenic *Fusarium* fungal species, host plants, and infections caused.

Pathogen	Host Plant	Infection	Reference
F. avenaceum	Wheat	*Fusarium* head blight (FHB)	[95]
F. oxysporium	Oriental lilium plant	Root and bulb disease	[96]
F. oxysporum	Potato	Stem-end rot	[97]
F. oxysporum	Banana	*Fusarium* wilt	[98]
F. oxysporium	Pineapple	Dieback	[99]
F. oxysporium	Avocado	Stem-end rot	[100]
F. fujikuroi	Rice	Bakane	[101]
F. graminearum	Oil palm	*Fusarium* wilt	[102]

Table 2. *Cont.*

Pathogen	Host Plant	Infection	Reference
F. graminearum	Banana	Crown rot	[103]
F. graminearum	Wheat and barley	*Fusarium* head blight (FHB)	[104]
F. proliferatum	Oriental lilium plant	Root and bulb disease	[96]
F. proliferatum	Mango	Leaf spot	[105]
F. proliferatum	Pineapple	Fruitlet core rot	[106]
F. proliferatum	Chilli pepper (*Capsicum annuum* L.)	Fruit rot	[107]
F. solani	Paprika	Fusarium rot	[108]
F. solani	Avocado	Stem-end rot	[109]
F. solani	Papaya	Root rot	[110]
F. verticilloides	Maize	Fusarium ear rot	[111]
F. verticilloides	Mango	Leaf spot	[112]
F. verticilloides	Banana	Crown rot	[113]
F. commune	Chinese water plant (*Eleocharis* dulcis)	Fusarium wilt	[114]
F. equiseti	Papaya	Fruit rot	[115]
F. equiseti	Avocado	Stem-end rot	[116]
F. sporotrichioides	Avocado	Stem-end rot	[117]

4.2. Fusarium as Human and Animal Pathogen

Conventionally, *Fusarium* has been more of an agronomic threat than a medical one, but over the last thirty years, due to a variety of contributing factors, this scenario has undergone a radical change. This made *Fusarium* spp. emerge as a major opportunistic human pathogen, causing an expansive range of infections with high morbidity and mortality [66,118,119]. Infections caused by *Fusarium* species are generally referred to as fusariosis, which is largely dependent on the immune status of the host and the route of entry of the infection [71,120]. Among immunocompetent hosts, the common *Fusarium* infections are keratitis and onychomycosis with other less common conditions such as sinusitis, pneumonia, thrombophlebitis, and fungemia [71].

It is not all species of the genus that possess the ability to induce disease or infection; only a few cause infections in humans and animals. *Fusarium* human pathogens of growing importance include *F. oxysporum*, *F. moniliforme*, and *F. solani*, although infections by *F. proliferatum* and *F. napiforme* have also been reported recently [66,121]. According to Sullivan and Moran, as well as Gnat et al. [122,123], the types of diseases found in animals, especially pets, are often the same as those that are found in people. Although not much is known as regards fungal pathogenesis, it involves a complex interplay of many factors [124].

The disease mechanisms of *Fusarium* human/animal pathogens include the following:

(i) Adhesion

Fungal hyphae must adhere to the host surfaces both as a commensal to avoid being washed out of the various niches and during the onset of infections [125]. Fungal pathogens can adhere to host cells by way of specialized cell-wall glycoproteins.

(ii) Entry

A major factor in the pathogenesis of invasive fusariosis involves the disruption of the mucosa or cutaneous barrier of the host cell. Fungi hardly cause disease in healthy, immunocompetent hosts, although humans are constantly exposed to infectious propagules. It is only when fungi accidentally penetrate barriers such as intact skin and mucous

membrane linings, or once immunologic deficiencies and other devastating conditions occur in the host, that fungi can colonize and grow [124]. Hence, the ability of fungi to penetrate host cells is crucial for the progression of infection in the setting of intact skin or gut barriers [125]. *Fusarium* from soil or water enters the body, making contact with minute breaks in the skin or mucous membranes, causing infections. These sites serve these organisms as cutaneous portals of systemic entry during periods of immunosuppression, allowing for the dissemination of infection [126]. Infectious agents may also enter the body because of extensive skin breakdowns, such as in burns and wounds, where even airborne conidia may be the source [127], or due to the presence of foreign bodies, such as keratitis in contact lens wearers or peritonitis in patients receiving continuous ambulatory peritoneal dialysis (CAPD).

(iii) Colonization, Adaptation, and Propagation

To effectively colonize the host, these organisms must be able to survive at the elevated body temperature and either evade phagocytosis, counteract the hostility they come across, or adapt in a way that will make them multiply. Several factors contribute to the infection and pathogenesis of these organisms. The ability to secrete enzymes, e.g., keratinase, their ability to grow at 37 °C, dimorphism, and other yet-undefined factors contribute to fungal pathogenesis, which involves a complex interplay of many fungal and host factors. Fungi often develop both virulence mechanisms that facilitate their multiplication within the host [124].

(iv) Disease development, Dissemination

Propagation of fungi in the body shows a breach or paucity of host defenses. Endocrinopathies or immune conditions may cause such a breach, or it may be by iatrogenic induction [124]. Effective infection may result in disease, defined as an abnormality or interruption of the normal structure or function of body parts, organs, or systems (or combinations thereof) that is marked by a characteristic set of symptoms and signs and whose etiology, pathology, and prognosis are known or unknown [124].

5. Plant Pathologies Caused by *Fusarium* Species

The *Fusarium* species cause most plant diseases, infect all plant parts, and induce cell death. While growing on a host plant, *Fusarium* produces an array of toxins that are both phytotoxic and/or mycotoxic. During infection processes in plants, these toxins modulate plant physiology and favor *Fusarium* colonization and multiplication [128].

These toxins remain on the infected agriculture products and enter the food chain, where they cause mycotoxicosis in animals, livestock, and humans. The *Fusarium* genome is compartmentalized into two regions: the core genome responsible for primary metabolism and reproduction, and the adaptive genome responsible for pathogen virulence, host specialization, and probably other functions [4]. In some hosts, for instance, tomato plants, genes involved in virulence and host specialization are located on pathogenicity chromosomes within pathogenic strains [4]. Genome comparison studies revealed that the transfer of *Fusarium* pathogenicity chromosomes into a nonpathogen transformed the non-pathogen into a plant pathogen. Therefore, horizontal transfer may explain the polyphyletic origins of host specificity within the *Fusarium* genus [4].

6. Host–Pathogen Interaction in *Fusarium* Infections

The interactions between the plant and the fungus are diverse and complex, and can alter the physiology and morphology of both partners. *Fusarium* establishes the colony and destroys plant tissues by overriding the plant defense mechanisms and also by producing host-specific toxins (Figure 2). It is facilitated by the secondary metabolites produced by the fungus, for which the host plant initiates a defense response termed effector-triggered immunity or pathogen-associated molecular pattern (PAMP)-triggered immunity [129]. Penetration into the plant cell wall is achieved by the action of several

cell-wall-degrading enzymes like cellulases, pectinases, lipases, and xylanases [130,131]. So, the fungal pathogenic factors which act as pathogens to plants include the following:

(i) Host-specific toxins in *Fusarium* infections

Most phytopathogenic fungi produce toxins (Table 1) that can cause plant diseases [132]. Notably, many fungal toxins are low-molecular-weight secondary metabolites that show obvious symptoms such as wilting, chlorosis, necrosis, growth inhibition, and leaf spotting [133] in host plants. Fungal toxins can increase the permeability of the host plant cell membrane, cause leakage of cell electrolytes, and damage the membrane system, thereby causing it to lose normal physiological functions and potentially die [134]. Toxins can also damage the inner membrane of the chloroplasts in host plant leaves, and progressively cause the disintegration of the basal lamella, causing the chloroplasts to form vesicles, thus triggering severe poisoning effects or death to the host plants. Toxins that act on the mitochondria of the host plant cause damage to the mitochondrial membrane structure, swelling of the cristae, vacuolization, decrease in the mitochondrial matrix and cristae number, or make the cristae disappear [132].

(ii) Effector Proteins

Phytopathogenic fungi secrete proteins that can interact with host plants during the infection process. These effector proteins, e.g., Fg62 and Avr^2, play crucial roles in plant cells and therefore affect the interaction between pathogens and their hosts. Research on plant-pathogenic fungal effectors has often focused on model fungi and those that had their whole genome sequenced [135,136]. So far, *Fusarium* spp., e.g., *Fusarium oxysporum* f.sp. *lycopersici* are among the major cloned pathogenic fungal effectors. The virulence effects and transport molecular mechanisms of effector proteins are thus far in the early stages of research. It has not yet been identified which effector proteins can modify plant metabolism to meet the nutritional needs of the infecting fungi, or which plant signal transduction pathways control the expression of effector protein genes [132]. Through comparative analysis of fungal effector molecules, common motifs of fungal effectors may be found. Such knowledge would lead to better explanations of the interactions between pathogenic fungi and host plants and would indicate the pathogenic mechanisms of pathogenic fungi and the disease-resistance mechanisms of host plants [132].

(iii) Cell-Wall-Degrading Enzymes

These enzymes break the cell wall and cuticle of the plant and enhance the invasion, colonization, and proliferation of pathogenic fungi [137,138]. The cell-wall-degrading enzymes of these fungi and their interaction with host plants during the infection process is increasingly being studied using molecular biology and proteomics. The main cell-wall-degrading enzymes are pectinase, chitinase, cellulase, and protease [139]. In living plant tissues, cellulases from pathogens play a role in the softening and disintegration of cell wall material [139,140]. *Fusarium graminearum* secretes cell-wall-degrading enzymes such as cellulase, pectinase, and xylanase during infection. The secreted enzymes cause the decomposition of plant cell-wall components and aid the penetration and growth of the pathogen in the host tissue [141]. Specifically, the cell-wall-degrading enzyme β-galactosidase can promote the degradation of lactose in the cell wall, which precedes the production of galactose and glucose and enhances fruit softening [142]. Ma et al. [143] suggested that there is an abundance of β-galactosidase in the early stages of fruit softening which contributes to the breakdown of cell-wall galactosyl bonds, preceding the reduction of cell-wall integrity. Some enzymes, including protease, hemicellulase, amylase, and phospholipase, which can degrade protein, hemicellulose, starch, and lipids, respectively, also play specific roles in the infective process [142]. Fungal infection of plants and disease production is not only caused by cell-wall-degrading enzymes, but also by hormones, toxins, and other factors. Simultaneously, pathogen infection can also activate plant defense enzyme systems and induce plants to generate antifungal substances to inhibit the pathogens' cell-wall-degrading enzymes and achieve disease resistance. Therefore, the infection of plants by pathogenic fungal species is a complex biochemical process [132].

Fusarium spp., for instance, *F. oxysporum*, exhibit a high degree of host specificity [4]. A complex network comprising interconnected and overlapping signaling pathways is activated once *Fusarium* recognizes a host in its vicinity. These pathways include mitogen-activated protein (MAP) kinase signaling pathways [144], Ras proteins, G-protein signaling components, [145–147] components of the velvet complex [148], and cAMP pathways [146] (Figure 2).

Therefore, effective management of *Fusarium* wilt diseases will require thorough knowledge and understanding of the molecular mechanisms involved in the pathogenesis.

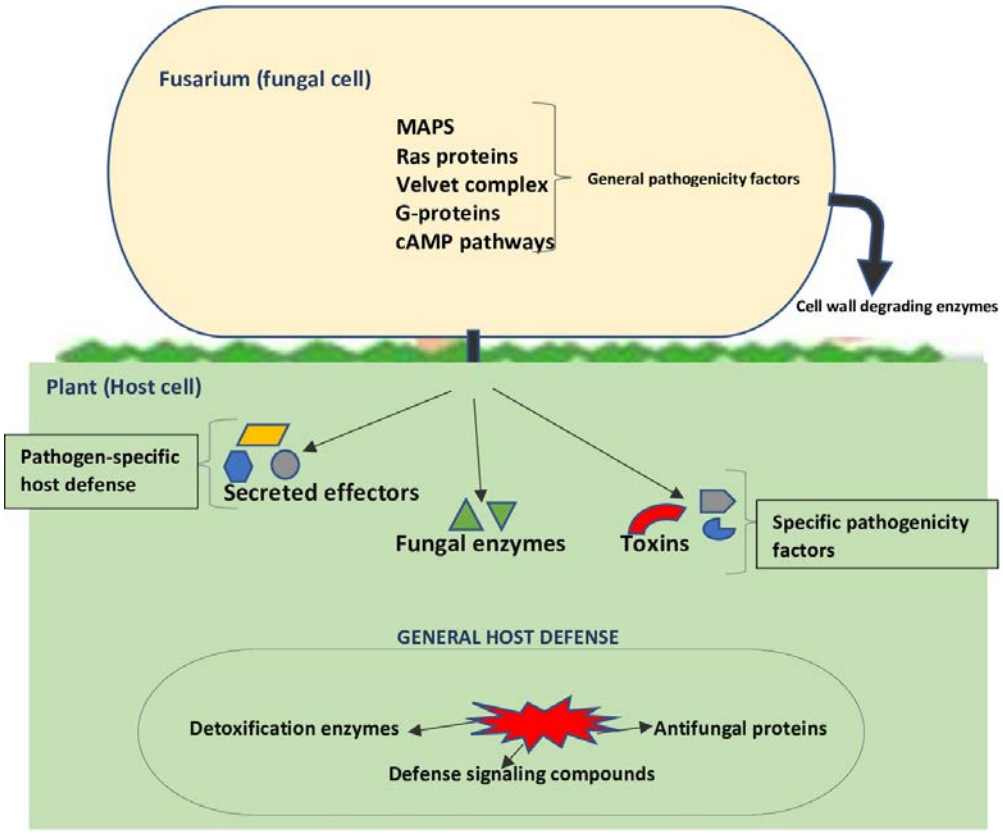

Figure 2. *Fusarium* pathogen and plant host defense mechanisms (molecular interactions). Adapted from Ma et al. [4].

7. Control and Management of Phytopathogenic *Fusarium* spp.

Fusarium is difficult to control in crops, as methods such as the physical, chemical, and cultural ways of control are not only ineffective but also expensive. Some of the reasons why *Fusarium* species are widely distributed include the following: (1) they can grow and develop on a wide variety of substrates and (2) their means of spore dispersal are very efficient [68]. Fusarium is a soilborne phytopathogen; hence, it can survive in soil and plant debris for many years. The infected soil could be redistributed to different places by tools used in the fields and by animals, which can lead to outbreaks in other new areas. This fungus can survive in a wide range of environmental conditions. However, one of the less common routes for fungal transmission include insects acting as vectors for plant pathogens, and recent studies have shown that beetles can be effectual vectors for the *Fusarium* solani species [149]. The amount and distribution of the fungal inoculum in the field and the stage of plant growth at which the *Fusarium* disease occurs determine the extent of yield reduction [150]. Mostly dry and warm growing conditions favor the growth of *Fusarium*. This poses a great problem in parts of the world and in areas where crops are grown in such seasons [151]. One of the challenges of fusarium elimination in agricultural practice is the constant presence of *Fusarium* propagules on all organs of living plants, on

plant residues, and in the soil [152]. A dangerous feature of *Fusarium* infection of plants is the possible absence of external symptoms, which does not always mean that the pathogen and mycotoxins are absent [153].

Wilts caused by *Fusarium* are commonly assumed to be monocyclic, which means that the disease does not spread from one plant to another during the season [154]. This is because the propagules that can disseminate the disease to other plants to cause secondary infections only form very late during the crop season. So, managing *Fusarium*'s dispersal and growing population is a very difficult task at the field level.

Plants are most susceptible to *Fusarium* infection and sporulation during the warm moist seasons and the flowering phase, but the probability of infection continues throughout the growing season [155]. Since *Fusarium* diseases are difficult to control, as fungi easily overcome host resistance to various methods of control, biological control of fungal diseases has increasingly received attention as one of the alternative means of control [1]. Biological control is also usually perceived as natural and thus more environmentally friendly than chemical control since no completely novel molecule is being introduced to the environment.

8. Phytopathogenic *Fusarium* spp. and Global Food Security

Fusarium is regarded as the most devastating fungi to crop plants in most parts of the world, especially in Asia, Africa, the USA, Europe, and Australia [156]. It damages the host plant severely, causing a decrease in quality and productivity and affecting plant development.

Fusarium diseases of crops have been increasing in severity since the last century and now pose a serious threat to global food security [157,158]. Recently, it has become evident that *Fusarium* pathogens pose the greatest biotic challenge to many of the world's staple crops. They destroy both the essential calorie crops, such as rice, wheat, maize, and soybean, and the commodity crops, such as bananas, coffee, and barley [159,160]. Fusarium infection of crops also causes significant socio-economic and international trade implications for global food security by its ability to reduce crop yields and contaminate plant products with mycotoxins. Additionally, the loss of commodity crops to fungal disease destabilizes the economies of developing countries that rely on export revenues generated by the global trade of these crops to import food from elsewhere [160], thereby increasing the dimension of the food insecurity threat.

Moreover, modern agricultural practices of monoculture planting of uniform crops have accelerated the emergence of new virulent fusarium strains [161]. Climate change, on the other hand, also heightens the problem, as we observe altered disease demographics as the pathogens move towards the warmer region [162]. In addition, the trade and transport of plants and plant products have further dispersed plant pathogens to new hosts in previously unaffected areas of the world [160]. However, modern intensive agriculture has become the mainstay of global food security and generates the calories needed to feed the increasing global population. Nevertheless, we face a changing world plagued by fungal pathogens and new crop diseases. Therefore, to protect our future harvests, there is a need for better disease surveillance and the adoption of new disease intervention strategies.

9. Conclusions

The *Fusarium* spp. is one of the most important fungal groups and is significant in the fields of agriculture, medicine, and veterinary science [163]. They have emerged over the past decades as an important cause of diverse plant diseases, opportunistic human and animal pathogens, and mycotoxin producers. Many of the world's staple food crops are prone to fungal plant pathogens [164]. Furthermore, the loss of economically important crops to these fungal diseases can destabilize the economies of developing nations who depend on export revenues generated to import food grown somewhere else in the world. However, successful disease management strategies are challenging, since fungi can easily develop resistance to various methods of control. Therefore, biological control of fungal

diseases has emerged as one of the alternative methods of control and promises much toward increasing world crop production. Thus, we need better disease surveillance, more accurate predictive forecasting, and new disease interventions in order to achieve better disease control and protect our future harvests, thus achieving sustained global food security.

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