Review

Diversity, Ecological Characteristics and Identification of Some Problematic Phytopathogenic *Fusarium* in Soil: A Review

Dmitry A. Nikitin [1,*], Ekaterina A. Ivanova [1], Mikhail V. Semenov [1], Alena D. Zhelezova [1,2], Natalya A. Ksenofontova [1], Azida K. Tkhakakhova [1] and Vladimir A. Kholodov [1]

[1] Dokuchaev Soil Science Institute, Pyzhyovskiy Lane 7, 119017 Moscow, Russia
[2] Federal State Budgetary Scientific Institution All-Russian Scientific Research Institute of Phytopathology (VNIIF), Institut Street 5, 143050 Moscow, Russia
* Correspondence: e-mail dimnik90@mail.ru; Tel.: +7-9264707491

Abstract: The genus *Fusarium* includes many pathogenic species causing a wide range of plant diseases that lead to high economic losses. In this review, we describe how the *Fusarium* taxonomy has changed with the development of microbiological methods. We specify the ecological traits of this genus and the methods of its identification in soils, particularly the detection of phytopathogenic representatives of *Fusarium* and the mycotoxins produced by them. The negative effects of soil-borne phytopathogenic *Fusarium* on agricultural plants and current methods for its control are discussed. Due to the high complexity and polymorphism of *Fusarium* species, integrated approaches for the risk assessment of *Fusarium* diseases are necessary.

Keywords: fusariosis; mycotoxins; soil suppressiveness; plant diseases

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1. Systematics and Taxonomic Position of the Genus *Fusarium*

Members of the genus *Fusarium* belong to the family Nectriaceae, order Hypocreales, class *Sordariomycetes*, phylum *Ascomycota*, kingdom Fungi, and domain Eukaryotes. The genus *Fusarium* was first described in 1809 by Heinrich Friedrich Link and listed in the taxonomy by Fries in 1821. To date, there are 300 known species of *Fusarium*, but almost half of them have not been officially described [1–4]. The teleomorphs of some *Fusarium* species are the genera *Haematonectria* and *Gibberella*. Depending on the species concept, the taxonomy of *Fusarium* has been constantly changing over the past 100 years [4,5]. From the 1920s to the 1950s, the number of species within the *Fusarium* genus decreased from one thousand to nine species [4]. In 1971, Colin Booth from the Commonwealth Institute of Mycology in the UK published a book entitled *The Genus Fusarium* that is now considered to be the most important study about the diversity of this genus [4]. In 1982, Gerlach and Nirenberg published an illustrated atlas of the genus Fusarium with descriptions of more than 90 species. Nelson and co-authors published a guide on identifying the members of this genus in 1983 in which they confirmed 41 species and considered the description of 16 species to be incomplete [4]. Since the mid-1980s, three main species concepts have been used for the identification of *Fusarium* species: morphological, biological, and phylogenetic [5]. Since the late 1990s, due to the study of new unique ecosystems and the expansion of the geography of analyzed samples, many new *Fusarium* species have been described. Until 2012, the sexual (perfect/teleomorphic) and asexual (imperfect/anamorphic) stages of the life cycle of pleomorphic fungi, including *Fusarium*, were assigned different names [4]. In 2011, the International Code of Nomenclature for Algae, Fungi and Plants (ICN) was adopted at the International Botanical Congress in Melbourne; the ICN suggested using single names for fungal species and cancelling the use of dual nomenclature [5].

The taxonomy of the genus *Fusarium* is still complex; several species belonging to this genus may have radical differences in morphological, physiological, and ecological characteristics [1,6]. The different morphological features of *Fusarium* species are presented in

Supporting Supplementary Material Figure S1 and Supporting Table S1. Another challenge of the *Fusarium* taxonomy is the constant discovery of species that seem to be new but appear to be indistinguishable from each other, according to several criteria [4]. An outdated species concept based only on morphological and biochemical features is still frequently used in the scientific community, though genetic markers should also be considered [5]. Since many studies have aimed to describe the morphological taxonomy of *Fusarium*, this genus has often served as a testing ground for new species' concepts of fungi [4]. Over the past 20 years, biological taxonomy has significantly changed due to the active use of molecular genetic methods, but this has not solved the problems with the *Fusarium* taxonomy [6]. The definition of *Fusarium* given by Geiser et al. [2] may be considered a rejection of the traditional concept of the genus; it combines traits across divergent lineages that were accepted and used to distinguish genera not only within the *Nectriaceae* family but also in other families and orders of fungi [5]. The new concept of the *Fusarium* genus does not have clear criteria, since the variation of attributes in common with the rest of the *Nectriaceae* family is so vast that it can almost be extended to the entire family.

The current difficulties in defining species criteria have motivated experts to revise the phylogenetic classification of the genus *Fusarium*, which has led to the appearance of the term "species complex" in different studies. A species complex combines several species with a similar morphology, ecology, metabolism, and range of infested crops, e.g., the species complex of *Fusarium graminearum* [7] or of *Gibberella fujikuroi* [8]. This unified approach is more convenient because assigning a strain to a species group gives a broader view of its characteristics and reduces the probability of errors. The most recent *Fusarium* phylogeny is presented in the study of Torres-Cruz et al. [9]. The identification and taxonomic characterization of morphologically similar strains and isolates requires comprehensive study, since they may differ in genetics and produced metabolites. Therefore, molecular genetics and metabolic analyses should be applied for this purpose [10].

2. Ecology of *Fusarium*

The members of the genus *Fusarium* are widely distributed in all geographic regions of the world [4]. They usually live in soil or plant debris, but they are also found in air, water, plants, and insects [11], persisting there for a long time as chlamydospores, sclerocytes, or conidia [12]. Information on the life cycle of fungi of the genus *Fusarium* can be obtained from Figure 1. The abundance of *Fusarium* is especially high in agricultural and pasture soils [1,12–14]. There are some eurytopic (*Fusarium poae* and *F. sporotrichioides*) and stenotopic (*F. graminearum, F. culmorum,* and *F. cerealis*) species that only develop in certain regions [15]. Fusarium species differ in their environmental optima. The taxonomic diversity of Fusarium for different areas is primarily determined by climatic factors, soil physicochemical parameters, and vegetation type [16,17].

The lifecycles of the major *Fusarium* plant pathogens, including their growth and pathogenic and saprophytic phases, are well-studied. However, less is known about the effects of temperature, elevated CO_2, and water availability on host plant colonization by pathogens and their toxin production. Climate is a key factor that determines the level of mycotoxin contamination before and after crop harvest. Climate change has a significant impact on the life cycles of phytopathogenic *Fusarium* due to its influence on abiotic factors: increase in CO_2 concentration and temperature, as well as changes in the soil water regime [1]. It also alters host survival and host–pathogen interaction mechanisms, and possibly contributes to new diseases and changes in the diversity of phytopathogens due to variations in their ecological niches [15–18]. This leads to changes in the species distribution area, temporal activity, and community structure of fungal pathogens, and it has a significant impact on species resistance and mycotoxin production [10]. For example, conditions simulating climate change scenarios can significantly change the growth patterns of *F. graminearum* and *F. verticillioides* in vitro. The *in situ* infestation of ripening maize by *F. verticillioides* was shown to be increased with elevated CO_2 and temperature, though fumonisin B1 (FB1) contamination was not detected [19]. Further studies showed that

drought exposure affected FB1 production and stimulated the contamination of ripening maize cobs due to physiological effects on the corn plant [20]. Temperature and water activity (aw) have significant impacts on the key biosynthetic gene expression and growth of *F. verticillioides*, as well as on toxic secondary metabolite production [21]. An ecological study of *F. langsethiae* compared different strains from northern European countries and found that the optimal conditions for its growth are between 0.980 and 0.995 aw at +25 °C, and the production of T-2 and HT-2 mycotoxins was found to be highest at +20–25 °C and 0.995 aw [22]. Contrasting results were obtained during the colonization of stored oats by *F. langsethiae* under the effect of modeled climate change, especially in growing conditions at +30 °C, intermediate water stress (0.98 aw), and an increase in CO_2 concentration to 1000 ppm. Under these conditions, the stimulation of the expression of the *Tri5*, *Tri6*, and *Tri16* genes and a significant increase in T-2/HT-2 toxin contamination were observed compared with the control (+20 °C, 0.995 aw, and CO_2 concentration of 400 ppm) [23]. However, the resilience of pathogenic *Fusarium* species to abiotic factors associated with climate change and their adaptation to these factors are still to be explored. Further studies on the effects of *Fusarium* on the soil mycobiota are required for a better understanding of the consequences of pathogen dominance in the fungal community. In this regard, the study of the adaptation and resilience of biocontrol strains, which are antagonists, in relation to phytopathogenic *Fusarium* is important for the future sustainable control of mycotoxin pollution in field conditions [1].

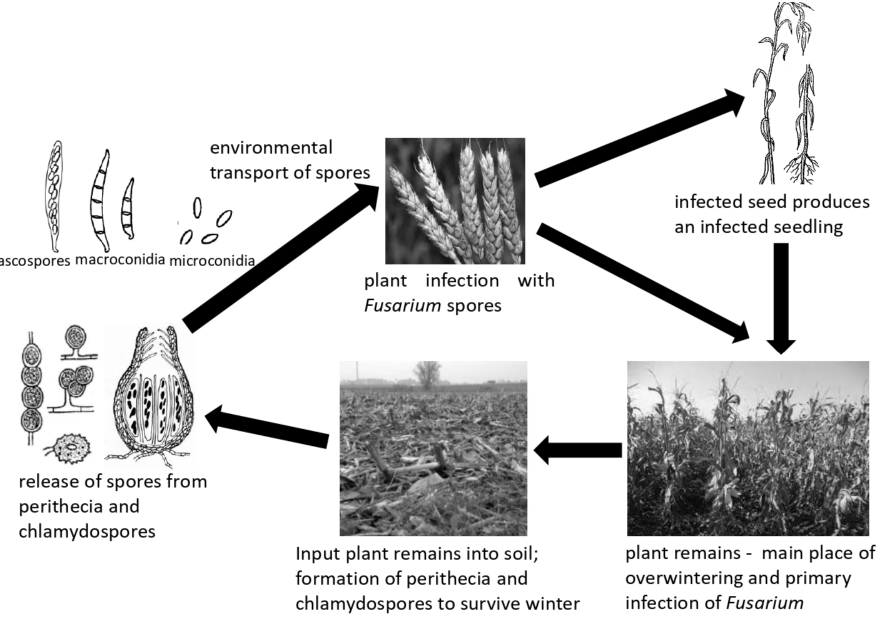

Figure 1. Scheme of distribution, infection, and conservation of *Fusarium*.

Along with the climate, soil physicochemical parameters also determine the distribution and pathogenic activity of *Fusarium* [17]. *Fusarium* species survive better in soil with a moisture content of 15–25% of the soil field water capacity [24,25]. A negative correlation between survival and soil moisture content was noted for *F. oxysporum* [26,27]. Soil moisture also indirectly affects the physiological activity of *Fusarium*, since an increase in the water availability in soil may stimulate the overall microbiological activity of the microbiome, including the antagonists of *Fusarium*. Model experiments with *Fusarium oxysporum* have demonstrated that moderately acidic soils (pH values of 5–6) are optimal for both spore germination and plant seedling infection by this phytopathogen; the latter may be due to the lower microbial activity in moderately acidic soils compared with

soils with a neutral pH [28]. In acidic soils (pH 4.5), the infestation of winter wheat by phytopathogenic *F. pseudograminearum* and *F. culmorum* was shown to increase fivefold compared with neutral soil (pH 7). An increase in phytopathogenic spore formation on straw placed in acidic soil was also detected [28].

Differences in particle size distribution and soil texture often correlate with the presence of phytopathogenic *Fusarium*. Hoper et al., 1995 found that the effect of clay minerals on fungi depends on soil acidity and the presence of plants [29]. The introduction of kaolinite reduced the abundance of *F. oxysporum* propagules at soil pH values of 4 and 5.2 but increased it at a pH of 6.9 compared with the control. After the germination of wheat, the fungal abundance in the soil with kaolinite was higher than in the control soil at all pH values. The addition of montmorillonite and talc stimulated the development of *F. oxysporum* in sterile soil [30]. Soil particle size distribution also affects *F. oxysporum* populations [31].

Each *Fusarium* species is characterized by a certain tolerance to environmental factors [1]. For example, *F. longipes* and *F. beomiformeare* are mainly found in tropical regions [15]. *F. asiaticum* is distributed in regions with air temperatures above +22 °C, mainly in East Asia [32]. *F. boothii* is more thermophilic and is only found in Africa and Mexico. *F. acuminatum* and *F. culmorum* are mostly identified in temperate climates and do not develop at temperatures above +25 °C [15]. *F. acuminatum*, *F. culmorum*, and *F. cerealis* are non-specific phytopathogens mostly found in temperate regions. *F. graminearum* is a highly aggressive phytopathogen of a local scale in regions with a cold and humid climate, its growth is limited by low temperatures, and it has not been found in polar ecosystems [32]. *F. sporotrichioides* and *F. poae* are eurytopic phytopathogens of cereals. Cotton, potatoes, pumpkin, legumes, and cruciferous vegetables are most susceptible to *Fusarium* wilt. Potato wilt is commonly caused by *F. oxysporum* and *F. bulbigenum*. Different plants are characterized by their own *Fusarium* communities, which do not always act as phytopathogens [1].

Some *Fusarium* species are soil saprotrophs and mutualists with plants (for example, *F. heterosporum* and *F. sambucinum*). These species can form mycorrhiza with many cereals or live inside plant tissues (endophytes) [1,15,33]. At the same time, they do not cause significant damage and increase resistance to phytopathogens [6]. Some *Fusarium* strains can be mutualists of invertebrates [11]. It has been argued that some *Fusarium* species are opportunistic pathogens of invertebrates as these microscopic fungi are occasionally isolated from living and dead insects [11]. However, most members of the *Fusarium* genus are known as phytopathogens of more than 200 crop species, as well as opportunistic animal and human pathogens that cause Fusarium toxicity and dermatitis [4].

3. The Importance of *Fusarium* in Agriculture

Global economic losses in agriculture associated with phytopathogenic strains of *Fusarium* reach billions of USD per year [34]. The prevalence of *Fusarium* infections in plants can be assessed by the fact that two species of *F. graminearum* and *F. oxysporum* are among the five main fungal pathogens of plants, along with smut fungi, rust fungi, and brown and gray rot [11]. *Fusarium* has recently been included in the top ten most economically important phytopathogenic fungi [35]. Even with the advent of new fungicides, these fungi have a high adaptive potential to infect agricultural plants [36]. The main symptoms of plant fusariosis are the wilt and rot of roots and seeds. *Fusarium* head blight (FHB) is one of the most common and dangerous diseases caused by *Fusarium*. This infection leads to the accumulation of mycotoxins in the grain and a significant reduction in yield. FHB is caused by not one but a number of species, the list of which varies depending on geography and climate. For example, in European countries, *F. graminearum*, *F. culmorum*, *F. poae*, and *F. avenaceum* are considered the most common. The direct influence of some risk factors on the development of FHB has been proven. For example, minimal tillage (no-till), excessive moisture during flowering and an excess of nitrogen fertilizers significantly increase the risk of FHB [37].

The outbreaks of fusariosis often occur in years with warm and humid weather and can lead to cereal yield losses of up to 75% [35]. The fusariosis of cereal crops is the most hazardous kind [35,38]. A wide range of *Fusarium* strains may cause fusariosis in cereals, and the infecting strain could be defined by the environmental conditions of crop cultivation. The infestation of grain with *Fusarium* leads to a decrease in its feeding and nutritional quality due to the accumulation of mycotoxins, as well as to a decrease in seed quality [33,35].

F. oxysporum has been shown to be the most important representative of the *Fusarium* genus in terms of agriculture and economy [39]. More than 150 formae speciales of *F. oxysporum* are known, each with a unique range of hosts for a single or closely related set of plant species. *Fusarium* species significantly vary in their confinement to climatic regions and agricultural crops [4,15]. *F. oxysporum* causes wilt in many crops, such as soybean, bananas [39,40], cotton [41], or tomatoes [42], primarily infecting the roots and being asymptomatic in them for a long time. *F. oxysporum* is usually not pathogenic for plants in natural ecosystems despite its high abundance in temperate soils; however, it is a major phytopathogen in agricultural ecosystems [43]. *F. oxysporum* is a race complex of hundreds of genetically isolated populations that can exhibit symbiotic, commensal, or pathogenic relationships with plants depending on a wide range of factors [41,42]. It has been shown that the level of pathogenicity and virulence of *Fusarium* in relation to the plant is largely influenced by the age of the host [40] and the structure of the soil nematode community [41,42].

F. avenaceum prevails in temperate climates and can exist as a saprotroph and phytopathogen, causing the fusariosis of oats and wheat, as well as the root rot of legumes and carnations. *F. moniliforme* is a phytopathogen of cereal crops, causing root rot, plant hypertrophy, and stunting. *F. culmorum* causes the root rot of cereals, stem rot of corn, and dry rot of potatoes and vegetable crops; *F. sporotrichiella* may cause root and stem rot in cereals. *F. sporotrichiella* is widespread in tropical and temperate climates, and it can produce trichothecene mycotoxin (Table 1).

There are many dispersal mechanisms for *Fusarium*, including host seeds, insects, nematodes, soil, and plant debris [41]. Therefore, *Fusarium* diseases are among the most difficult to control. The phytopathogenic strains of *Fusarium* can persist in soil for decades, even in the absence of a host plant. A reliable and accurate method is needed for the early detection of *Fusarium* in seeds and soil [41].

Apart from cereal crops, alfalfa, corn, peas, soybeans, sunflowers, tomatoes, cucumbers, potatoes, and many gymnosperms and rhododendrons are most susceptible to fusariosis [10,34,35]. Fusariosis is the most dangerous and frequent disease of vegetable crops causing root rot, vascular lesions, wilt, and the desiccation of leaves.

The selection of new cultivars resistant to *Fusarium* is one of the obvious approaches to control this threat, but there are currently no durum wheat cultivars resistant to this disease. Different sensitivities to type B trichothecene accumulation were observed in a collection of *Triticum turgidum* subsp. lines infected by *F. culmorum* [47]. The selection of cereal crops with resistance to *Fusarium* and its associated mycotoxins will lead to the emergence of new advanced cultivars [43].

The members of the genus Fusarium can be a useful source for a wide range of bioactive secondary metabolites, such as antibiotics, antioxidants, and anticancer compounds [1]. Some secondary metabolites of *Fusarium* species activate plant resistance mechanisms against phytopathogens. Additionally, the members of *Fusarium* can produce many enzymes that are important for industries. *F. incarnatum* actively synthesizes laccase, and *F. oxysporum* produces chitinases, proteases, cellulases, and beta-glucosidases. Thus, the members of the genus *Fusarium* cause significant damage to agriculture but can also be valuable producers of enzymes and secondary metabolites in the food and cosmetics, biofuel, and pharmaceutical industries.

Table 1. Major phytopathogenic *Fusarium* species and their associated mycotoxins (based on Munkvold et al., 2021; Edel-Hermann and Lecomte 2019; Gálvez and Palmero, 2022; Seefelder et al., 2002).

Species	Habitat/Host Plant	Mycotoxins
F. avenaceum	Ubiquitous: cereals, peach, apple, pear, peanut, asparagus, and vegetables (potato, tomato)	Antibiotic Y, aurofusarin, beauvericin, chlamydosporol, chrysogene, enniatins, fusarin C, and moniliformin
F. cerealis	Ubiquitous: cereals and potato	Aurofusarin, butenolide, chrysogene, culmorin, fusarin C, nivalenol, norlichexanthone, rubrofusarin, siccanol, and zearalenone
F. culmorum	Temperate regions: cereals, potato, apple, and sugar beet	Aurofusarin, butenolide, chrysogen, culmorin, deoxynivalenol, fusarin C, 3-acetyldeoxynivalenol, nivalenol, and zearalenone
F. equiseti	Ubiquitous: cereals, fruits, vegetables, nuts, and spices	Chrysogene, diacetoxycirpenol, equisetine, fusarochromanone, nivalenol, and zearalenone
F. graminearum	Ubiquitous: cereals and grasses	Aurofusarin, butenolide, chrysogene, culmorin, deoxynivalenol, fusarin C, nivalenol, and zearalenone
F. oxysporum	Ubiquitous: ornamental plants, cotton, date palm, pear, legumes, nuts, banana, citrus fruits, apple, vegetables (onion, potato), heat-treated juices, spices, and cheese [44]	Fusaric acid, moniliformin, naphthoquinone pigments trichothecenes T-2 toxin, HT-2 toxin, diacetoxyscirpenol, and 3′-OH T-2 (TC-1)
F. poae	Temperate regions: cereals, soybean, sugarcane, and rice	Butenolide, fusarin C, γ-lactones, nivalenol, neosolaniol, iso-neosolaniol, HT-2 toxin, and T-2 toxin
F. proliferatum	Tropical regions: corn, rice, and fruits; Temperate regions: cereals, soybean, and vegetables (garlic and asparagus) [45,46]	Beauvericin, fumonisins, fusaproliferin, fusaric acid, fusarin C, moniliformin, and naphthoquinone pigments
F. sambucinum	Ubiquitous: cereals and potato	Aurofusarin, butenolide, deoxynivalenol, diacetoxycirpenol, and enniatins, T-2 toxin
F. semitectum	Tropical regions: nuts, banana, citrus fruits, melons, vegetables (potato, tomato), and spices	Apicidin, Beauvericin, equisetin, fusapyrone, and zearalenone
F. solani	Ubiquitous: fruits, vegetables, and spices	Anhydrofusarubin, fusaric acid and naphthoquinone pigments, and solaniol
F. sporotrichioides	Ubiquitous: cereals and fruits	Aurofusarin, butenolide, fusarin C, T-2 toxin, diacetoxyscirpenol, neosolaniol, nivalenol, NT-1 toxin, NT-2 toxin, HT-2 toxin
F. subglutinans	Ubiquitous: corn, pineapple, banana, spices, and sorghum	Beauvericin, fusaproliferin, fusaric acid, moniliformin, naphthoquinone pigments, and subglutinols
F. tricinctum	Ubiquitous: cereals	Antibiotic Y, aurofusarin, butenolide, chlamydosporol, chrysogene, fusarin C, and visoltricin
F. venenatum	Temperate regions: cereals and potato	Aurofusarin, butenolide, diacetoxycirpenol, isotrichodermin, isotrichodermol, sambucinol, apotrichothecene, culm orin, and culmorone
F. verticillioides	Tropical and subtropical regions: corn, rice, sugarcane, banana, asparagus, spices, and garlic	Fumonisins, fusaric acid, fusarin C, moniliformin, and naphthoquinone pigments

4. *Fusarium* Control and Soil Suppressiveness against *Fusarium* Fungi

4.1. Mitigation Strategies towards Fusarium

4.1.1. *Fusarium* Detection

The traditional way to detect *Fusarium* in cereals involves counting the proportion of infected grains and performing microbiological plating with the further analysis of *Fusarium* isolates. However, this method gives only approximate estimates of phytopathogen abundance in plant tissues. These estimates do not always reliably reflect the number of mycotoxins; depending on several factors (such as plant cultivar, resistance to phytopathogens, and unfavorable weather events), *Fusarium* can penetrate grain tissues at different depths and localize in the pallet, aleurone layer, endosperm, or embryo [48]. This problem is especially common for glumaceous plants, such as oats and barley, where film can be abundantly colonized by *Fusarium* representatives despite low seed infection. The generative organs of plants before flowering are absolutely immune to *Fusarium*. However, in the case of early

infection, the ear is partly formed from deformed and light-colored grains. These grains are usually removed during post-harvest processing, resulting in significant crop losses [49].

4.1.2. *Fusarium* Control and Its Limitation

Fusarium control should be comprehensively carried out [50]. Three types of approach are usually used for this purpose: agrotechnical (crop rotation, use of disease-resistant plant varieties, weed control, and use of phosphorus–potassium fertilizers), chemical (pre-sowing treatment of seeds with fungicides and fungicide spraying during the growing season), and biological (treatment of seeds and adult plants with biological preparations based on antagonist microorganisms) [12]. However, these methods are often not very effective, since fusariosis is usually caused by several *Fusarium* species that often differ in their physiology and ecology [12]. The main challenge of fusariosis elimination in agricultural practice is the constant presence of *Fusarium* propagules on all organs of living plants, on plant residues, and in the soil [33]. Plants are most susceptible to *Fusarium* during the moist seasons and the flowering phase, but the probability of infection persists throughout the growing season. A dangerous feature of fusariosis is the possible absence of external symptoms, which does not always mean that the pathogen and mycotoxins are absent [12]. Each *Fusarium* is able to produce its own spectrum of mycotoxins, which can be analyzed to determine the species composition of fungi in plant organs or in soil [51].

4.1.3. Agrotechnical Approach—*Fusarium*-Resistant Plant Cultivars

The breeding of *Fusarium*-resistant plants is extremely challenging because many genes control the quantitative traits of resistance to the disease [36]. Moreover, plant resistance to *Fusarium* depends on environmental factors. Although there is no complete immunity to *Fusarium* in any cereal species, five types of physiological resistance have been distinguished [35,49]: (1) resistance to the penetration of *Fusarium* propagules into the plant; (2) resistance of the cereal plant to the spread of *Fusarium* through the ear; (3) resistance of grains to the penetration of *Fusarium*; (4) general tolerance of the plant to the presence of *Fusarium*; and (5) ability of cereal plants to accumulate or degrade *Fusarium* mycotoxins. Unfortunately, plant resistance to *Fusarium* is non-specific, so resistance to one *Fusarium* species does not guarantee this trait in relation to other representatives of the genus. The genes of resistance to *Fusarium* spp. found in cotton, tomato, melon, pea, banana, etc. [52–54]. Although *Fusarium* is able to bypass the plant defense induced by these genes, it enables the identification of races within the special forms and the use of more complex selection strategies, such as gene pyramiding.

Overall, two types of plant resistance to phytopathogens have been described: polygenic (horizontal or resistance to minor genes) and monogenic (vertical or resistance to major genes). Polygenic resistance does not recognize the races of the pathogen and provides a low level of resistance, usually based on multiple genes that act to create physical and/or chemical barriers to pathogen invasion [42]. Polygenic resistance is generally considered to be longer lasting than resistance arising from single genes. Monogenic resistance is based on individual resistance genes in the host that recognize the specific races of pathogens and provide a high level of resistance against specific diseases [42]. In addition, plants can activate latent defense mechanisms in response to infection by pathogens. Induced resistance in plants refers to a state of increased defense capability created by a prior stimulus. Two different types of induced resistance have been extensively studied: systemic acquired resistance (SAR) and induced resistance (IR). SAR is caused by plant exposure to abiotic or biotic elicitors [42].

4.1.4. Chemical Approach—Fungicides

The application of fungicides is the most widely used practice to control *Fusarium*. However, due to the rapid emergence of resistant strains, the effectiveness of these plant protection agents can only be achieved if new effective chemicals are constantly being developed. Another way to control *Fusarium* activity is the use of mixed fungicides with

multiple active ingredients [55]. However, since the application of fungicides is limited to a narrow time interval from the end of earing to the beginning of flowering, precipitation may interrupt the crop treatment or make it ineffective [56]. Therefore, the main emphasis in the control of phytopathogenic *Fusarium* in recent years has been the development of plant cultivars that are resistant to *Fusarium*. There is also evidence that the use of some soil invertebrates (e.g., earthworms, nematodes, and springtails) can reduce the quantity of *Fusarium* phytopathogenic strains in agroecosystems [12].

4.1.5. Biocontrol of *Fusarium*

The use of biocontrol agents could be an environmentally friendly approach. Some bacteria of the genus *Pseudomonas* are promising *Fusarium* biocontrol agents. Antagonistic bacteria could be stimulated by exudates of previously sown crops: *Fusarium* wilt was suppressed via *Pseudomonas* stimulation by the compound produced by *Allium* [57]. *P. putida* reduces the incidence of *Fusarium* wilt in flax, radish, cucumber, and *Dianthus* sp. [27,57]. The biocontrol mechanism appears to be due to competition for iron ions between the pathogen and siderophores produced by *P. putida*. The same effect of *Fusarium* wilt suppression was also observed after the addition of EDDCA (chelate for Fe^{3+} with a high binding constant) and additive suppression when applied together with *P. putida*. The application of EDDCA to soil can confer a selective advantage on siderophore-producing antagonists [58]. The combined application of two strains of *P. putida* (WCS358 and RE8, which implement different mechanisms of *F. oxysporum* control) demonstrated a cumulative effect in the suppression of *Fusarium* rot on radish, exceeding the suppressive effect under the influence of each strain separately by 50% [55]. The presence of *P. fluorescens* suppressed the development of *F. culmorum* mycelium in the soil but stimulated the formation of chlamydospores [59,60]. The decrease in mycelium density in the presence of *P. fluorescens* was significantly higher in the soil without additives and less pronounced when glucose or cellulose were added. Thus, the communities of several beneficial microbial taxa would be more effective as biocontrol agents of phytopathogens than individual species. This also suggests that higher soil microbial diversity is beneficial for plant disease control [42].

4.1.6. *Fusarium*–Plant Interactions on Molecular Level

During fusariosis, the polysaccharide component of *Fusarium* cell walls (e.g., chitin) is usually recognized by the plant quickly, and then the immune response attends to suppressing the pathogen [36]. The siRNA molecules of host plant are used to suppress pathogen target gene expression; this defense mechanism is used in host-induced gene silencing (HIGS) technology. The efficiency of RNAi vector blocking varies with the size and location of the target regions within the TRI6 genetic cluster of trichothecene biosynthesis. Insight into this mechanism has enabled HIGS technology to become a tool that is successfully used to protect plants against fungal pathogens, including *Fusarium* [36]. Plants can export both exogenous artificial siRNAs (small interfering RNAs) and endogenously produced miRNAs (microRNAs) to mycopathogen-infected cells targeting fungal transcripts.

Despite the issues outlined above, *Fusarium* is able to bypass plant immunity. One such pathway includes the proteins produced by some species of phytopathogenic *Fusarium* that are secreted into plant cells to suppress the immune response and enable infection. *F. oxysporum* uses functional homologues of alkalizing peptides (e.g., rapid alkalinization factor, RALF). These metabolites have been found in tomato and *Arabidopsis* plants infected with *F. oxysporum*, and they possibly increase pathogenicity and suppress host immunity [42,52,58]. Increasing extracellular pH promotes the infectious growth of *Fusarium* by stimulating the phosphorylation of a conserved mitogen-activated protein kinase that is essential for pathogenicity [61]. Additionally, one of the possible positive modulators of *Fusarium* pathogenesis is the production of fusaric and abscisic acid [62]. Abscisic acid is a transmitter of environmental stress signals, such as drought, cold, and salinity, and it contributes to the acceleration of plant aging processes, the inhibition of seed germination, the suppression of DNA and RNA, the synthesis of some enzymes, etc. [63,64].

Thus, the abscisic acid produced by *Fusarium* sp. can indirectly reduce the overall physiological status of a plant and contribute to the susceptibility of plants to the negative effects of phytopathogenic *Fusarium*.

4.2. Soil Suppressiveness to Fusarium diseases

In general, soil suppressiveness characterizes the degree of the suppression of phytopathogenic taxa and is determined by the physicochemical and biological properties of soil [65–67]. Highly suppressive soils are characterized by low levels of plant disease, even in the presence of a virulent pathogen and susceptible crops [68,69]. Soil suppressiveness can be defined as general or specific, natural, or induced [70]. General soil suppressiveness is associated with microbial biomass activity in a soil or a plant at a critical moment in the pathogen life cycle; specific soil suppressiveness is linked to the activity of an individual or selective group of microorganisms that are antagonists in relation to certain plant pathogens. Natural soil suppressiveness is governed by the general microbial content inherent in the soil, which is often associated with the physical properties of the soil and is relatively independent of crop rotations. Induced soil suppressiveness could be managed using various agricultural practices that activate soil microbial communities and increase microbial diversity [71]. A decrease in soil suppressiveness combined with the insufficient phytosanitary optimization of crop rotations and cultivation technologies usually lead to an imbalance in soil microbial systems and an increase in the incidence of *Fusarium* diseases in plants. The specific suppressiveness is explained by the convergent activity of certain members of the soil microbial community that intervene in the pathogen's disease cycle [72]. For example, *Pseudomonas* is able to produce pyoverdins and iron-chelating siderophores limiting iron availability for *F. oxysporum*.

The mechanisms of soil suppressiveness can be divided into physicochemical, biological, and anthropogenic. Soil physicochemical parameters (pH, organic matter content, sand, clay, etc.) indirectly affect suppressiveness [69]. Particle size distribution, organic matter content, and soil structure determine water-holding capacity, nutrient availability, gas exchange, and root growth [69]. Low root-zone aeration, caused by poor soil structure or waterlogging, can lead to *Fusarium* blight, and soil compaction significantly increases the probability of root rot disease. Suppressiveness is inversely proportional to soil moisture, and it is minimal in water-saturated soil [69]. Soil suppressiveness is also determined by biological parameters, mainly by the activity of soil microorganisms and the phytopathogen:antagonist ratio [71].

The biological mechanisms of soil suppressiveness include the synthesis of antibiotics, competition for macro- and microelements, and the production of toxins and enzymes that destroy the cell walls of phytopathogens. The comparative analysis has shown that *Fusarium* wilt-suppressive soils harbored some unique bacterial and fungal species [72] while the abundance of *Fusarium oxysporum* inoculums was very low [73]. Some strains of *Sporothrix flocculosa* and *Sporothrix rugulosa* are able to synthesize antibiotics of heptadecenoic and methylheptadecenoic acids with general antimycotic and antibacterial activity [74]. Th fungi of the genus *Trichoderma* are known as biocontrol agents that inhibit or control *Fusarium* blight through the mycoparasitism, consumption, and production of various lytic enzymes and other antimicrobial compounds along with enhanced host plant growth through the production of phytohormones. In soils, *Trichoderma* secrete various antimycoparasitic proteins, enzymes, volatile and non-volatile compounds, and other secondary metabolites that contribute to the dissolution of nutrients and protect plants from pathogens [68]. Micromycetes (*Trichoderma viride*, *Botrytis cinerea*, *Clonostachys rosea*, *Penicillium expansum*, *Rhizoctonia stolonifer*, *Sphaerotheca fuliginea*, and *Puccinia xanthii*, as well as yeasts of the genera *Pichia, Rhodotorula, Cryptococcus, Aureobasidium*, and *Tilletiopsis*) are capable of producing β-1,3-glucanase and chitinase enzymes that dissolve the cell walls of plant pathogens [58]. The actinomycetes of the genus *Streptomyces*, typical representatives of the soil microbial community, also exhibit chitinolytic activity and produce a wide range of antibiotics of a diverse nature (including peptides, beta-lactones, and polyketides)

that help to limit the growth and development of *Fusarium oxysporum f. sp. cubense* [50]. Polyoxins B and D are a special class of antifungal antibiotics isolated from *Streptomyces cacaoi* that specifically inhibit chitin synthase and are used against phytopathogenic fungi in fruit, vegetables, and ornamental plants [56]. When applied to fungal cells, Validamycin A is converted to validoxylamine A, which inhibits the synthesis of trehalose [58].

Anthropogenic activity has a significant impact on soil suppressiveness [75,76]. Crop rotation and intensive tillage increase pathogen suppression, while long-term monoculture and no-till cultivation in most cases lead to the accumulation of phytopathogenic microorganisms [68]. Along with the use of crop rotations, the incorporation of crop residues in soil reduces the stress caused by soil pathogens due to temporal changes in the biological and physicochemical soil properties [77]. The use of organic fertilizers increases soil microbial diversity and stimulates general soil suppressiveness more than mineral fertilizer systems [14,78].

The introduction of arbuscular mycorrhizal fungi into the soil could be the way to increase soil suppressiveness [79]. Arbuscular mycorrhizal fungi increase plant resistance to phytopathogens by improving phosphorus and nitrogen nutrition, thereby increasing their non-specific resistance to microorganisms [79]. Furthermore, the synthesis of antimicrobial metabolites by the symbiotic fungi of arbuscular mycorrhiza has been reported. *Glomus intraradices* synthesizes an unidentified antimicrobial agent that enables the control of the conidial germination of the phytopathogenic *Fusarium oxysporum* [80]. Although arbuscular mycorrhizal fungi are important for the control of phytopathogens, there are limitations to their use in field conditions [79]. Firstly, a sufficiently large number of fungal propagules should be introduced; otherwise, they may not survive in soil. Secondly, possible competition between native and introduced mycorrhizal fungi may weaken the suppressive activity of the soil. The high contents of phosphorus and nitrogen in the soil also hinder the process of mycorrhization. One option is the simultaneous introduction of several different taxa of mycorrhizal fungi into soil to improve their survival in field conditions.

5. Research and Diagnostics of Phytopathogenic *Fusarium*

5.1. Model Laboratory Experiments for Phytopathogenic Fusarium Research

The ecological features of phytopathogens and the investigation of pathogen control methods and mechanisms are usually studied in laboratory experiments due to many difficulties appearing in field research [12,50]. The virulence of *Fusarium* strains and a range of environmental factors (seasonal change or global climate change, soil structure, and microbial community composition and diversity) could be sources of uncertainty. Research on agricultural techniques for *Fusarium* control, the selection of sensitive/resistant crop cultivars, and the selection of antagonistic microorganisms in the first stage of research should also be carried out as part of greenhouse or pot experiments.

A key point in the preparation of such experiments is the creation of an artificial infectious background—the formation of a pathogen inoculum in the soil (or experimental soil, depending on the research task) [50]. The direct "infection" of a soil (or a model substrate that mimics a soil) can be carried out both by introducing a suspension of the pathogen and by introducing an "infection powder": cereals (millet and barley) treated with an infectious material used as a carrier and a certain amount of a homogenizing agent (gypsum/chalk in an amount of 1% of the final volume) [12].

To test the resistance of a new plant cultivar to phytopathogenic *Fusarium*, an artificial infectious background is usually created. To do this, phytopathogenic *Fusarium* strains are cultivated on agar media until sporulation occurs. Then, an aqueous suspension of spores of the studied strain is prepared, and the tested cultivar is infected with it [49]. A similar methodology is used for the evaluation of fungicidal effectiveness [81]. A crop is infected with the studied *Fusarium* strain and treated with fungicides. After that, a visual and laboratory assessment of the damage to the plant in the experiment and control is carried out.

The presence of a sufficient amount of infectious material with high aggressiveness and viability is necessary for the experiment conduction. Usually, an inoculum is prepared in advance, 10–30 days before the experiment [12,38]. An inoculum can be made by growing a large number of conidia (or ascospores, depending on whether the fungus has a teleomorphic stage of its life cycle) under optimal temperature conditions (at +25 °C) and lighting on plant debris or culture media. The following substrates can be used: corn stalk nodes [82], decoctions of bran [83], agar-based acid potato glucose or corn culture media [84], potato sucrose agar [85], or a liquid Czapek–Dox medium in aerated Erlenmeyer flasks [86].

5.2. Molecular Genetic Techniques for Fusarium Identification

The genus *Fusarium* is very heterogeneous in terms of morphological, physiological, and genetic criteria; therefore, the identification of its species requires the simultaneous use of several methods. Until recently, the main identification method was microbiological plating on specific nutrient media [1,6]. The qPCR is also proposed as a promising and effective method for the detection of *Fusaium* sp. [73]. As an alternative to the microbiological plating method, molecular genetic approaches using high-throughput sequencing have been applied to identify the species composition of *Fusarium* in natural substrates: PacBio SMRT [87,88] and Illumina MiSeq technology [89]. At the beginning of the genomic era, the scientific community relied on a single "reference" genome for *Fusarium* due to the significantly high cost of whole-genome sequencing. However, a significant reduction in the cost of sequencing enabled the faster analysis of genomes at a greater depth and with increased sensitivity [36].

Recent sequencing technologies are better adapted to work with short spans (up to 550 base pairs (bp) for MiSeq 300 Pair-End sequencing and up to 400 bp for Ion Torrent PGM). The choice of primer pairs is also important as they should amplify a region of the gene with a sufficient resolution to correctly identify *Fusarium* species while being specific to that genus. Some of the most sequenced *Fusarium* genes include translation elongation factor-1α (tef-1α), RNA polymerase 1 and 2 (RPB1 and RPB2), β-tubulin (tub), and histone (his) [4]. The translation elongation factor (EF-1α) identification is used the most because it facilitates quantitative comparisons between species [90]. Determination by tef-1α provides the correct identification of the following species: *F. verticillioides*, *F. proliferatum*, *F. subglutinans*, *F. thapsinum*, *F. temperatum*, *F. nygamai*, *F. brachygibbosum*, *F. redolens*, and others [90–92].

A pair of primers for detecting *Fusarium* species was recently developed for Illumina sequencing [90]. However, the forward and reverse reads obtained using MiSeq Pair-End technology with read sizes of up to 2 × 300 bp did not overlap due to long amplicons (640 bp). Therefore, the sequence length used for taxonomic assignment is being significantly reduced (up to 250–300 bp), and, consequently, its resolution is also being reduced.

Two databases of *Fusarium* sequences are currently available: *Fusarium*ID (https://www.fusarium.org/ (accessed on 9 September 2022)) and *Fusarium* MLST (https://fusarium.mycobank.org (accessed on 2 October 2022)). These databases contain information in addition to the *Fusarium* sequences available in the GenBank database. The data from these databases can be traced back to controls held in culture collections [4]. Additionally, it is recommended that the tef-1α gene is used as a marker to identify *Fusarium* and to sequence RPB1 and RPB2 genes to confirm this identification. CYP51 genes, which are probably species-specific, are also unique to *Fusarium* [36]. The standard DNA identification for fungi by the internal transcribed spacer region (ITS) of the ribosomal gene is not informative for a large number of Fusarium species and, therefore, should not be used for its species identification [4].

It is important to understand that one reference genome is not enough to identify representatives of *Fusarium* in natural substrates. The pangenome analysis, i.e., the comparative sequence analysis of different collections of genomes, of *Fusarium* isolates is preferable [36]. The pathogenomic approach is a high-resolution technique, which refers to the creation

and analysis of complete genomic sequences of various pathogens. It is performed for the identification of genes and their regulators associated with virulence and pathogenicity, primary and secondary metabolism, and potential genetic targets for the chemical control of pathogens [4]. Pathogenomics helps us to better understand the complex dynamics of host–microbe interactions that lead to disease [36].

It is necessary to distinguish between the concepts of pathogenicity and virulence. Pathogenicity refers to the ability of a pathogen to cause a disease, while virulence is a measure of the ability (probability) of a pathogen to cause a disease (measure of pathogenicity) [36]. Van de Wouw and Howlett [93] first introduced the concept of pathogenicity genes. They have been defined as genes encoding specific proteins in host plants that exhibit a "feedback" relationship between pathogen and host genes, the interaction of which leads to disease. Pathogenicity genes are divided into two classes: basic (common for *Fusarium* and other pathogenic fungi) and specialized (specific for individual *Fusarium* species—for example, the "SIX" and "FTF" gene families). In the PHI database (http://www.phi-base.org/ (accessed on 1 November 2022)), genes associated with pathogenicity and virulence are classified according to mutant phenotypes (loss of pathogenicity, unchanged pathogenicity, increased virulence, and reduced virulence). The functional characterization of putative pathogenicity genes has shown that they are involved in the following processes: the synthesis of enzymes that destroy the cell wall; the regulation of carbon, nitrogen, amino acid, and lipid metabolism; the cell wall formation of a host plant; and the translocation and degradation of proteins [36]. It is important to note that pathogenicity genes can be transmitted by horizontal transfer from strain to strain. A list of the main *Fusarium* pathogenicity genes is presented in Table 2.

However, it is very difficult to establish direct evidence for an association of specific genes with pathogenicity and virulence. There is a principle of "genetic redundancy" when two or more genes can code the same function [36]. Redundancy promotes significant flexibility in gene regulation. For example, *Fusarium* pathogenic genes encode a wide range of cell-wall-degrading enzymes. Gene redundancy for these enzymes increases the adaptability of *Fusarium* to the use of different nutrient sources depending on their availability.

From an agricultural perspective, the ultimate goal of *Fusarium* molecular biology research is to reduce the mycotoxin content in cereals. Therefore, the main efforts of molecular phytopathologists are now focused on identifying aspects of the biosynthesis and regulation of mycotoxins that can be used to control the content of these substances [94]. New information about fungal and plant genomes and their gene expressions is needed for a better understanding of host–microbe interactions. This will help with the development of approaches for the breeding and engineering of crops resistant to Fusarium and mycotoxin contamination. Research on the *Fusarium* genomes has revealed the presence of dozens of genes that are preliminarily responsible for the synthesis of polyketide synthases, nonribosomal peptide synthetases, terpene cyclases, and other types of enzymes that synthesize mycotoxins and other biologically active metabolites [95]. Unfortunately, the comparisons of DNA sequences alone cannot provide detailed information about mycotoxin biosynthetic pathways; this information must be obtained through appropriate experiments.

Table 2. *Fusarium* pathogenicity genes according to Rampersad, 2020 [36].

Species	Target Gene	Gene Function	Host Plant	Reference
F. culmorum	FcFgl1	Secreted lipase	Wheat	[95]
	FcFmk1	Mitogen-activated protein (MAP) kinase	Wheat	[95]
	FcGls1	Beta-1,3-glucan synthase	Wheat	[95]
	FcChsV	Chitin synthase	Wheat	[95]
	FcChsV	Chitin synthase V, myosin motor domain	Wheat	[95]

Table 2. *Cont.*

Species	Target Gene	Gene Function	Host Plant	Reference
F. graminearum	CYP51A	Cytochrome P450 lanosterol C-14-alpha demethylase	*Arabidopsis thaliana*; Barley	[96]
	FgCYP51A; FgCYP51; FgCYP51C	Cytochrome P450lanosterol C-14-alpha demethylase	Barley	[96]
	Chs3b	Chitin synthase	Wheat	[95]
F. graminearum	AGO; DCL	RNA interference (Argonaute, Dicer-like)	Barley	[96]
F. graminearum	FGSG_03101	Alpha/beta hydrolase	Wheat	[95]
F. graminearum	Fg00677; Fg08731	Protein kinase	*Brachypodium distachyon*	[97]
	FgCYP51A; FgCYP51; FgCYP51C	Cytochrome P450lanosterol C-14-alpha demethylase	*Brachypodium distachyon*	[97]
F. graminearum	FgCYP51A; FgCYP51; FgCYP51C	Cytochrome P450lanosterol C-14-alpha demethylase	*Arabidopsis thaliana*	[96]
F. graminearum	FgDCL1, FgDCL2	RNA interference Dicer-like proteins	Wheat	[95]
	FgAGO1, FgAGO2	RNA interference Argonaute 1 and 2	Wheat	[95]
	FgQDE3	RecQ helicase	Wheat	[95]
	FgQIP	AGO interactive protein	Wheat	[95]
	FgRdRP1, FgRdRP2, FgRdRP3, FgRdRP4	RNA-dependent RNA polymerases	Wheat	[95]
F. oxysporum f. sp. *cubense*	Velvet	Transcription factor	Banana	[98]
	ftf1	*Fusarium* transcription factor 1	Banana	[98]
F. oxysporum f. sp. *cubense*	SGE1	SIX (Secreted In Xylem) Gene Expression 1	Banana	[98]
F. oxysporum f. sp. *conglutinan*	FRP1	F-box protein	*Arabidopsis thaliana*	[96]
F. oxysporum f. sp. *conglutinan*	ERG6/11	Ergosterol biosynthesis genes	Banana	[98]

5.3. Fusarium Identification by Mycotoxins

Certain *Fusarium* species can be effectively detected by the presence of their main mycotoxins (for example, fumonisin, fusaric acid, trichothecenes, and enniatins) in the environment [90]. However, due to the high selectivity of molecular recognition mechanisms, it is difficult to simultaneously analyze different compounds or detect new mycotoxins [99]. The most common *Fusarium* mycotoxins are deoxynivalenol (DON), 3-acetyl-deoxynivalenol (3-ADON), 15-acetyl-deoxynivalenol (15-ADON), nivalenol (NIV), fusarenone-X (Fus-X), T-2 toxin, HT-2 toxin, neosolaniol (NEO), diacetoxyscirpenol (DAS), zearalenone (ZEN), fumonisin B1 (FB1), fumonisin B2 (FB2), and fusaric acid [99]. It is suggested that *F. verticillioides*, *F. fujikuroi*, *F. solani*, *F. temperatum*, *F. subglutinans*, *F. musae*, *F. tricinctum*, *F. oxysporum*, *F. equiseti*, *F. sacchari*, *F. concentricum*, and *F. andiyazi* only synthesize fusaric acid, while *F. langsethiae*, *F. sporotrichioides*, and *F. polyphialidicum* only synthesize trichothecenes.

Trichothece nemycotoxins are one of the virulence factors in *Fusarium*. Trichothecene profiles are species- and strain-specific, so they can be used to identify representatives of *Fusarium*. *Fusarium* trichothecene toxin (TRI) genes can be used for this purpose (Table 3).

Acute and chronic exposure to mycotoxins has a range of toxic effects on plants and animals and could be a potential hazard to human health. To date, the risks of combined toxicity have been poorly understood. However, it can be suggested that combined exposure to several different mycotoxins often leads to synergistic effects [99]. Therefore, it is critical to investigate mycotoxin profiles and identify the mycotoxin potential of different *Fusarium* species in various substrates [99].

Table 3. *Fusarium* trichothecene toxin (TRI) genes.

TRI Gene	Encoded Protein	Species	Host Plant
TRI5	Trichodiene synthase	*F. graminearum*	*Secalecereale* (rye), *Triticum* (wheat); *Triticum aestivum* (wheat), and *Glycine max* (soy)
TRI5	Trichodiene synthase	*F. pseudograminearum*	*Triticumaestivum* (wheat)
TRI6	Transcription regulator—Zinc finger superfamily C_2H_2	*F. graminearum*	*Triticum aestivum* (wheat)
TRI10	Transcription regulator—Zinc finger superfamily C_2H_2	*F. graminearum*	*Triticum* (wheat)
TRI12	Trichothecene efflux pump, transmembrane transporter	*F. graminearum*	*T. aestivum* (wheat)
TRI14	Transmembrane transporter	*F. graminearum*	*T. aestivum* (wheat)

6. Conclusions

Over the past few decades, *Fusarium* has become one of the most serious fungal diseases associated with climate change and modern agricultural practices, resulting in huge economic losses worldwide. Current *Fusarium* control strategies are often unsuccessful due to the development of chemical resistance in the pathogen population and the lack of disease-resistant plant cultivars. Due to the active use of pesticides and climate change, the evolution of phytopathogenic fungi seems to have surpassed the evolution of cultivated plants, which has led to an even greater spread of crop diseases. The emergence of strains with new combinations of pathogenicity and virulence factors threatens food security. Obtaining new data on phytopathogenic fungi genomes is an important contribution to maintaining crop health. However, information about genes associated with pathogenicity and virulence must be supported by reliable evidence using several methods. These data are crucial for the development of transgenic approaches in the field of plant disease control. One approach by which to control *Fusarium* in some economically important crops is the host-induced blocking of phytopathogenic genes based on a conservative mechanism for preventing the expression of virulent fungal genes. Fighting *Fusarium* by suppressing gene expression can potentially be achieved bypassing the cost and dangers associated with pesticides. There is no doubt that knowledge about the diversity, ecology, and identification methods of *Fusarium* will help in the fight against fungal plant diseases.

Supplementary Materials: The following supporting information can be downloaded at: https://www.mdpi.com/article/10.3390/d15010049/s1, Figure S1: Fusarium morphology, a–c—polyphialides; d,e—conidiophore, phialids and conidia; Table S1: Morphology features of Fusarium species.

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References

1. Abdel-Azeem, A.M.; Abdel-Azeem, M.A.; Darwish, A.G.; Nafady, N.A.; Ibrahim, N.A. Fusarium: Biodiversity, ecological significances, and industrial applications. In *Recent Advancement in White Biotechnology through Fungi*; Springer: Cham, Switzerland, 2019; pp. 201–261. [CrossRef]
2. Aoki, T.; O'Donnell, K.; Geiser, D.M. Systematics of key phytopathogenic *Fusarium* species: Current status and future challenges. *J. Gen. Plant Pathol.* **2014**, *80*, 189–201. [CrossRef]
3. Cobo-Díaz, J.F.; Baroncelli, R.; Floch, G.L.; Picot, A. A novel metabarcoding approach to investigate *Fusarium* species composition in soil and plant samples. *FEMS Microbiol. Ecol.* **2019**, *95*, 7. [CrossRef]
4. Summerell, B.A. Resolving *Fusarium*: Current status of the genus. *Annu. Rev. Phytopathol.* **2019**, *57*, 323–339. [CrossRef]

5. Crous, P.; Lombard, L.; Sandoval-Denis, M.; Seifert, K.A.; Schroers, H.J.; Chaverri, P.; Thines, M. *Fusarium*: More than a node or a foot-shaped basal cell. *Stud. Mycol.* **2021**, *98*, 100116. [CrossRef]
6. Manganiello, G.; Marra, R.; Staropoli, A.; Lombardi, N.; Vinale, F.; Nicoletti, R. The Shifting Mycotoxin Profiles of Endophytic *Fusarium* Strains: A Case Study. *Agriculture* **2019**, *9*, 143. [CrossRef]
7. Starkey, D.E.; Ward, T.J.; Aoki, T.; Gale, L.R.; Kistler, H.C.; Geiser, D.M.; Suga, H.; Toth, B.; Varga, J.; O'Donnell, K. Global molecular surveillance reveals novel *Fusarium* head blight species and trichothecene toxin diversity. *Fungal Genet. Biol.* **2007**, *44*, 1191–1204. [CrossRef]
8. Gagkaeva, T.Y.; Levitin, M.M. Current state of the taxonomy of fungi of the complex of *Gibberella fuiikuroi*. *Mikol. Fitopatol.* **2005**, *39*, 3–14.
9. Torres-Cruz, T.J.; Whitaker, B.K.; Proctor, R.H.; Broders, K.; Laraba, I.; Kim, H.-S.; Brown, D.W.; O'Donnell, K.; Estrada-Rodríguez, T.L.; Lee, Y.-H.; et al. FUSARIUM-ID v.3.0: An Updated, Downloadable Resource for *Fusarium* Species Identification. *Plant Dis.* **2022**, *106*, 1610–1616. [CrossRef] [PubMed]
10. Moretti, A. Taxonomy of *Fusarium* genus. A continuous fight between lumpers and splitters. *Matica Srp. Proc. Nat. Sci.* **2009**, *117*, 7–13. [CrossRef]
11. Sharma, L.; Marques, G. *Fusarium*, an entomopathogen—A myth or reality? *Pathogens* **2018**, *7*, 93. [CrossRef]
12. Goncharov, A.A.; Glebova, A.A.; Tiunov, A.V. Trophic interactions between *Fusarium* species and soil fauna: A meta-analysis of experimental studies. *Appl. Soil Ecol.* **2020**, *145*, 103302. [CrossRef]
13. Semenov, M.V.; Nikitin, D.A.; Stepanov, A.L.; Semenov, V.M. The Structure of Bacterial and Fungal Communities in the Rhizosphere and Root-Free Loci of Gray Forest Soil. *Eurasian Soil Sci.* **2019**, *52*, 319–332. [CrossRef]
14. Semenov, M.V.; Krasnov, G.S.; Semenov, V.M.; van Bruggen, A. Mineral and Organic Fertilizers Distinctly Affect Fungal Communities in the Crop Rhizosphere. *J. Fungi* **2022**, *8*, 251. [CrossRef] [PubMed]
15. Gagkaeva, T.Y.; Gavrilova, O.P.; Levitin, M.M. Biodiversity and distribution of the main toxigenic *Fusarium* fungi. *Biosfera* **2014**, *6*, 36–45. (In Russian)
16. Du, S.; Trivedi, P.; Wei, Z.; Feng, J.; Hu, H.W.; Bi, L.; Liu, Y.R. The Proportion of Soil-Borne Fungal Pathogens Increases with Elevated Organic Carbon in Agricultural Soils. *Msystems* **2022**, *7*, e01337-21. [CrossRef] [PubMed]
17. Nilsson, R.H.; Anslan, S.; Bahram, M.; Wurzbacher, C.; Baldrian, P.; Tedersoo, L. Mycobiome diversity: High-throughput sequencing and identification of fungi. *Nat. Rev. Microbiol.* **2019**, *17*, 95–109. [CrossRef]
18. Rousk, J.; Bååth, E.; Brookes, P.C.; Lauber, C.L.; Lozupone, C.; Caporaso, J.G.; Knight, R.; Fierer, N. Soil bacterial and fungal communities across a pH gradient in an arable soil. *ISME J.* **2010**, *4*, 40–1351. [CrossRef] [PubMed]
19. Vaughan, M.M.; Huffaker, A.; Schmelz, E.A.; Dafoe, N.J.; Christensen, S.; Sims, J.; Martins, V.F.; Swerbilow, J.; Romero, M.; Alborn, H.T.; et al. Effects of elevated CO_2 on maize defence against mycotoxigenic *Fusarium verticillioides*. *Plant Cell Environ.* **2014**, *37*, 2691–2706. [CrossRef] [PubMed]
20. Vaughan, M.M.; Huffaker, A.; Schmelz, E.A.; Dafoe, N.J.; Christensen, S.A.; McAuslane, H.J.; Alborn, H.T.; Allen, L.H.; Teal, P.E.A. Interactive effects of elevated CO_2 and drought on the maize phytochemical defense response against mycotoxigenic *Fusarium verticillioides*. *PLoS ONE* **2016**, *11*, e0159270. [CrossRef]
21. Medina, A.; Schmidt-Heydt, M.; Cárdenas-Chávez, D.L.; Parra, R.; Geisen, R.; Magan, N. Integrating toxin gene expression, growth and fumonisin B1 and B2 production by a strain of *Fusarium verticillioides* under different environmental factors. *J. R. Soc. Interface* **2013**, *10*, 20130320. [CrossRef]
22. Medina, A.; Magan, N. Comparisons of water activity and temperature impacts on growth of Fusarium langsethiae strains from northern Europe on oat based media. *Int. J. Food Microbiol.* **2010**, *142*, 365–369. [CrossRef]
23. Medina, A.; Magan, N. Temperature and water activity effects on production of T-2 and HT-2 by Fusarium langsethiae strains from north European countries. *Food Microbiol.* **2011**, *28*, 392–398. [CrossRef] [PubMed]
24. Shakhnazarova, V.Y.; Strunnikova, O.K.; Vishnevskaya, N.A. The effect of moisture on the development of *Fusarium culmorum* in soil. *Mikol. Fitopatol.* **1999**, *33*, 53–59. (In Russian)
25. Toropova, E.Y.; Kirichenko, A.A.; Stetsov, G.Y.; Suhomlinov, V.Y. Soil infections of grain crops with the use of the resource-saving technologies in Western Siberia, Russia. *Biosci. Biotechnol. Res. Asia* **2015**, *12*, 1081. [CrossRef]
26. Oritsejafor, J.J.; Adeniji, M.O. Influence of host and non¬host rhisospheres and organic amendments on survival of *Fusarivm oxysporum f.* sp. *elaedis*. *Mycol. Res.* **1990**, *94*, 57–63. [CrossRef]
27. Orr, R.; Nelson, P.N. Impacts of soil abiotic attributes on *Fusarium* wilt, focusing on bananas. *Appl. Soil Ecol.* **2018**, *132*, 20–33. [CrossRef]
28. Gordon, T.R.; Stueven, M.; Pastrana, A.M.; Henry, P.M.; Dennehy, C.M.; Kirkpatrick, S.C.; Daugovish, O. The effect of pH on spore germination, growth, and infection of strawberry roots by *Fusarium oxysporum* f. sp. fragariae, cause of *Fusarium* wilt of strawberry. *Plant Dis.* **2019**, *103*, 691–696. [CrossRef] [PubMed]
29. Hoper, H.; Steinberg, C.; Alabouvette, C. Involvement of clay type and pH in the mechanisms of soil suppressiveness to *Fusarim* wilt of flax. *Soil Biol. Biochem.* **1995**, *27*, 955–967. [CrossRef]
30. Hoper, H.; Steinberg, C.; Alabouvette, O. Modification of the saprophytic behavior of a *Fusarim oxysporm* in sterilised soil supplied with different clay types. In Proceedings of the Sixth International Symposium on Microbial Ecology, Barcelona, Spain, 6–11 September 1992; p. 231.

31. Steinberg, C.; Gautheron, N.; Burtin, G.; Alabouvette, C. Role of soil structure on the interaction between indigenous and introduced populations of *Fusarim*. In Proceedings of the Sixth International Symposium on Microbial Ecology, Barcelona, Spain, 6–11 September 1992; p. 235.
32. Backhouse, D. Global distribution of *Fusarium graminearum, F. asiaticum* and *F. boothii* from wheat in relation to climate. *Eur. J. Plant Pathol.* **2014**, *139*, 161–173. [CrossRef]
33. Gagkaeva, T.Y.; Gavrilova, O.P.; Nikonov, I.N.; Laptev, G.Y. The possibilities of biodegradation of mycotoxins produced by fusarium fungi. *Mycol. Today* **2016**, *3*, 202–238. (In Russian)
34. Bakker, M.G.; Brown, D.W.; Kelly, A.C.; Kim, H.S.; Kurtzman, C.P.; Mccormick, S.P.; Ward, T.J. *Fusarium* mycotoxins: A trans-disciplinary overview. *Can. J. Plant Pathol.* **2018**, *40*, 161–171. [CrossRef]
35. Timmusk, S.; Nevo, E.; Ayele, F.; Noe, S.; Niinemets, Ü. Fighting *Fusarium* pathogens in the era of climate change: A conceptual approach. *Pathogens* **2020**, *9*, 419. [CrossRef] [PubMed]
36. Rampersad, S.N. Pathogenomics and management of *Fusarium* diseases in plants. *Pathogens* **2020**, *9*, 340. [CrossRef] [PubMed]
37. Karlsson, I.; Persson, P.; Friberg, H. Fusarium head blight from a microbiome perspective. *Front. Microbiol.* **2021**, *12*, 628373. [CrossRef] [PubMed]
38. Sokolova, L.M.; Mikhailov, V.V.; Beloshapkina, O.O.; Egorova, A.A. About the method of creating an infectious, background of *Fusarium* for vegetable pea. *Agrar. Nauka* **2020**, *7–8*, 92–98. (In Russian) [CrossRef]
39. Ploetz, R.C. *Fusarium* wilt of banana is caused by several pathogens referred to as *Fusarium oxysporum f.* sp. *cubense*. *Phytopathology* **2006**, *96*, 653–656. [CrossRef]
40. Karangwa, P.; Mostert, D.; Ndayihanzamaso, P.; Dubois, T.; Niere, B.; Zum Felde, A.; Viljoen, A. Genetic diversity of *Fusarium oxysporum* f. sp. cubense in East and Central Africa. *Plant Dis.* **2018**, *102*, 552–560. [CrossRef]
41. Bell, A.A.; Gu, A.; Olvey, J.; Wagner, T.A.; Tashpulatov, J.J.; Prom, S.; Liu, J. Detection and characterization of *Fusarium oxysporum* f. *sp. vasinfectum* VCG0114 (race 4) isolates of diverse geographic origins. *Plant Dis.* **2019**, *103*, 1998–2009. [CrossRef]
42. McGovern, R.J. Management of tomato diseases caused by *Fusarium oxysporum*. *Crop Prot.* **2015**, *73*, 78–92. [CrossRef]
43. Torbati, M.; Arzanlou, M.; da Silva Santos, A.C. Fungicolous *Fusarium* Species: Ecology, Diversity, Isolation, and Identification. *Curr. Microbiol.* **2021**, *78*, 2850–2859. [CrossRef]
44. Edel-Hermann, V.; Lecomte, C. Current status of *Fusarium oxysporum* formae speciales and races. *Phytopathology* **2019**, *109*, 512–530. [CrossRef] [PubMed]
45. Gálvez, L.; Palmero, D. *Fusarium* dry rot of garlic bulbs caused by *Fusarium proliferatum*: A review. *Horticulturae* **2022**, *8*, 628. [CrossRef]
46. Seefelder, W.; Gossmann, M.; Humpf, H.U. Analysis of fumonisin B1 in *Fusarium proliferatum* infected asparagus spears and garlic bulbs from Germany by liquid chromatography− electrospray ionization mass spectrometry. *J. Agric. Food Chem.* **2002**, *50*, 2778–2781. [CrossRef] [PubMed]
47. Boutigny, A.L.; Richard-Forget, F.; Barreau, C. Natural mechanisms for cereal resistance to the accumulation of Fusarium trichothecenes. *Eur. J. Plant Pathol.* **2008**, *121*, 411–423. [CrossRef]
48. Gagkaeva, T.Y.; Gavrilova, O.P.; Orina, A.S.; Kazartsev, I.A.; Gannibal, F.B. Comparison of methods to detect Fusarium toxin-producing fungi in grain. *Mikol. Fitopatol.* **2017**, *51*, 292–298. (In Russian)
49. Gagkaeva, T.Y.; Gavrilova, O.P. Features of lesion of oat by fusariosis (review). *Agric. Biol.* **2011**, *6*, 3–10. (In Russian)
50. Bubici, G.; Kaushal, M.; Prigigallo, M.I.; Gómez-Lama Cabanás, C.; Mercado-Blanco, J. Biological control agents against *Fusarium* wilt of banana. *Front. Microbiol.* **2019**, *10*, 616. [CrossRef]
51. Munkvold, G.P.; Proctor, R.H.; Moretti, A. Mycotoxin production in *Fusarium* according to contemporary species concepts. *Annu. Rev. Phytopathol.* **2021**, *59*, 373–402. [CrossRef]
52. Takken, F.; Rep, M. The arms race between tomato and *Fusarium oxysporum*. *Mol. Plant Pathol.* **2010**, *11*, 309–314. [CrossRef]
53. Murray, J.J.; Hisamutdinova, G.; Sandoya, G.V.; Raid, R.N.; Slinski, S. Genetic Resistance of *Lactuca* spp. against *Fusarium oxysporum* f. sp. lactucae Race 1. *HortScience* **2021**, *56*, 1552–1564. [CrossRef]
54. Epstein, L.; Kaur, S.; Henry, P.M. The Emergence of *Fusarium oxysporum* f. sp. apii Race 4 and *Fusarium oxysporum* f. sp. coriandrii Highlights Major Obstacles Facing Agricultural Production in Coastal California in a Warming Climate: A Case Study. *Front. Plant Sci.* **2022**, *13*. [CrossRef] [PubMed]
55. Buchneva, G.N.; Gusev, I.V.; Korabelskaya, O.I.; Dubrovskaya, N.N.; Chekmarev, V.V. Varietal composition and frequency of fusarium presence on winter wheat varieties in the Tambov region. *Holoprystan Reg. Employ. Cent.* **2019**, *2*, 4–76. (In Russian) [CrossRef]
56. Santra, H.K.; Banerjee, D. Natural products as fungicide and their role in crop protection. In *Natural Bioactive Products in Sustainable Agriculture*; Springer: Singapore, 2020; pp. 131–219. [CrossRef]
57. Nishioka, T.; Suga, H.; Shimizu, M. The Stimulation of Indigenous Bacterial Antagonists by γ-Glutamyl- S -Allyl- l-Cysteine Increases Soil Suppressiveness to *Fusarium* Wilt. *Appl. Environ. Microbiol.* **2022**, *88*, e01554-22. [CrossRef] [PubMed]
58. Abdel-Aziz, M.S.; Ghareeb, M.A.; Hamed, A.A.; Rashad, E.M.; El-Sawy, E.R.; Saad, I.M.; Ghoneem, K.M. Ethyl acetate extract of *Streptomyces* spp. isolated from Egyptian soil for management of *Fusarium oxysporum*: The causing agent of wilt disease of tomato. *Biocatal. Agric. Biotechnol.* **2021**, *37*, 102185. [CrossRef]
59. Scher, F.M. Biological Control of *Fusarium* wilts by *Pseudomonas putida* and its Enhancement by EDDHA. In *Iron, Siderophores, and Plant Diseases*; Swinburne, T.R., Ed.; Springer: Boston, MA, USA, 1986; p. 117. [CrossRef]

60. Strunnikova, O.K.; Shakhnazarova, V.Y.; Vishnevskaya, N.A.; Chebotar', V.K.; Tikhonovich, I.A. Development and relations of *Fusarium culmorum* and *Pseudomonas fluorescens* in soil. *Microbiology* **2007**, *76*, 596–602. [CrossRef]
61. Masachis, S.; Segorbe, D.; Turrà, D.; Leon-Ruiz, M.; Fürst, U.; El Ghalid, M.; Guy, L.; López-Berges, M.S.; Richards, T.A.; Felix, G.; et al. A fungal pathogen secretes plant alkalinizing peptides to increase infection. *Nat. Microbiol.* **2016**, *1*, 10643. [CrossRef]
62. Syrova, D.S.; Shaposhnikov, A.I.; Makarova, N.M.; Gagkaeva, T.Y.; Khrapalova, I.A.; Emelyanov, V.V.; Gogolev, Y.V.; Gannibal, P.B.; Belimov, A.A. Prevalence of the ability to produce abscisic acid in phytopathogenic fungi. *Mikol. Fitopatol.* **2019**, *53*, 301–310. (In Russian) [CrossRef]
63. Hartung, W. The evolution of abscisic acid (ABA) and ABA function in lower plants, fungi and lichen. *Funct. Plant Biol.* **2010**, *37*, 806–812. [CrossRef]
64. Sokolov, M.S.; Marchenko, A.I.; Sanin, S.S.; Toropova, E.Y.; Chulkina, V.A.; Zakharov, A.F. Soil health of agrocenoses as an attribute of its quality and resistance to biotic and abiotic stressors. *Izv. Timiryazevskoy Sel'skokhozyaystvennoy Akad.* **2009**, *1*, 13–22. (In Russian)
65. Alabouvette, C. *Fusarium*-wilt suppressive soils from the Châteaurenard region: Review of a 10-year study. *Agronomie* **1986**, *6*, 273–284. [CrossRef]
66. Cook, R.J.; Baker, K.F. The soil ecosystem and biological control. In *The Nature and Practice of Biological Control of Plant Pathogens*; Cook, R.J., Baker, K.F., Eds.; American Phytopathological Society: St.Paul, MN, USA, 1983; pp. 233–281.
67. Mazzola, M. Mechanisms of natural soil suppressiveness to soilborne diseases. *Antonie Van Leeuwenhoek* **2002**, *81*, 557–564. [CrossRef] [PubMed]
68. Chandrashekara, C.; Bhatt, J.C.; Kumar, R.; Chandrashekara, K.N. Suppressive soils in plant disease management. In *Eco-Friendly Innovative Approaches in Plant Disease Management*; Singh, A., Ed.; International Book Distributors: Delhi, India, 2012; pp. 241–256. [CrossRef]
69. Janvier, C.; Villeneuve, F.; Alabouvette, C.; Edel-Hermann, V.; Mateille, T.; Steinberg, C. Soil health through soil disease suppression: Which strategy from descriptors to indicators? *Soil Biol. Biochem.* **2007**, *39*, 1–23. [CrossRef]
70. Nikitin, D.A.; Semenov, M.V.; Chernov, T.I.; Ksenofontova, N.A.; Zhelezova, A.D.; Ivanova, E.A.; Khitrov, N.B.; Stepanov, A.L. Microbiological Indicators of Soil Ecological Functions: A Review. *Eurasian Soil Sci.* **2022**, *55*, 221–234. [CrossRef]
71. De Boer, M.; Bom, P.; Kindt, F.; Keurentjes, J.J.; van der Sluis, I.; van Loon, L.C.; Bakker, P.A. Control of *Fusarium* Wilt of Radish by Combining *Pseudomonas putida* Strains that have Different Disease-Suppressive Mechanisms. *Phytopathology* **2003**, *93*, 626–632. [CrossRef] [PubMed]
72. Siegel-Hertz, K.; Edel-Hermann, V.; Chapelle, E.; Terrat, S.; Raaijmakers, J.M.; Steinberg, C. Comparative microbiome analysis of a *Fusarium* wilt suppressive soil and a *Fusarium* wilt conducive soil from the Châteaurenard region. *Front. Microbiol.* **2018**, *9*, 568. [CrossRef]
73. Manikandan, R.; Karthikeyan, G.; Raguchander, T. Soil proteomics for exploitation of microbial diversity in *Fusarium* wilt infected and healthy rhizosphere soils of tomato. *Physiol. Mol. Plant Pathol.* **2017**, *100*, 185–193. [CrossRef]
74. Musheer, N.; Ashraf, S.; Choudhary, A.; Kumar, M.; Saeed, S. Role of Microbiotic Factors Against the Soil-Borne Phytopathogens. In *Phytobiomes: Current Insights and Future Vistas*; Springer: Singapore, 2020; pp. 251–280. [CrossRef]
75. Jayaraman, S.; Naorem, A.K.; Lal, R.; Dalal, R.C.; Sinha, N.K.; Patra, A.K.; Chaudhari, S.K. Disease-Suppressive Soils—Beyond Food Production: A Critical Review. *J. Soil Sci. Plant Nutr.* **2021**, *21*, 1437–1465. [CrossRef]
76. Palojärvi, A.; Kellock, M.; Parikka, P.; Jauhiainen, L.; Alakukku, L. Tillage System and Crop Sequence Affect Soil Disease Suppressiveness and Carbon Status in Boreal Climate. *Front. Microbiol.* **2020**, *11*, 534786. [CrossRef] [PubMed]
77. Yuan, X.; Hong, S.; Xiong, W.; Raza, W.; Shen, Z.; Wang, B.; Li, R.; Ruan, Y.; Shen, Q.; Dini-Andreote, F. Development of fungal-mediated soil suppressiveness against *Fusarium* wilt disease via plant residue manipulation. *Microbiome* **2021**, *9*, 1–15. [CrossRef]
78. Semenov, M.V.; Krasnov, G.S.; Semenov, V.M.; van Bruggen, A.H. Long-term fertilization rather than plant species shapes rhizosphere and bulk soil prokaryotic communities in agroecosystems. *Appl. Soil Ecol.* **2020**, *154*, 103641. [CrossRef]
79. Jain, P.; Pundir, R.K. Biocontrol of soil phytopathogens by arbuscular mycorrhiza—A review. In *Mycorrhizosphere and Pedogenesis*; Varma, A., Choudhary, D., Eds.; Springer: Singapore, 2019; pp. 221–237. [CrossRef]
80. Filion, M.; St-Arnaud, M.; Fortin, J.A. Direct interaction between the arbuscular mycorrhizal fungus *Glomus* intraradices and different rhizosphere microorganisms. *New Phytol.* **1999**, *141*, 525–533. [CrossRef]
81. Buchneva, G.N. Tank compositions of fungicides as one of the factors controlling the development of fungi of the genus *Fusarium*. *Agric. Sci* **2020**, *7*, 37–38. (In Russian)
82. Bennett, A.; Ponder, M.M.; Garcia-Diaz, J. *Phoma* infections: Classification, potential food sources, and their clinical impact. *Microorganisms* **2018**, *6*, 58. [CrossRef]
83. Galich, M.T.V. Fusarium head blight in Argentina. In Fusarium Head Scab: Global Status and Future Prospects, Proceedings of the Workshop Held at CIMMYT, El Batan, Mexico, 13–17 October 1996; de Octubre, Dubin, H.J., Gilchrist, L., Reeves, J., McNab, A., Eds.; International Maize and Wheat Improvement Center (CIMMYT): El Batan, Mexico, 1996; pp. 19–26.
84. McCluskey, K. *Plant Pathology Related Culture Collection Resources*; APSnet Features Online: St. Paul, MN, USA, 2003.
85. Salem, E.A.; Abd El-Shafea, Y.M. Biological control of potato soft rot caused by Erwinia carotovora subsp. *carotovora*. *Egyptian J. Biol. Pest Control.* **2018**, *28*, 94. [CrossRef]

86. Bauermeister, A.; Amador, I.R.; Pretti, C.P.; Giese, E.C.; Oliveira, A.L. β-(1 → 3)-Glucanolytic yeasts from Brazilian grape microbiota: Production and characterization of β-glucanolytic enzymes by *Aureobasidium pullulans* 1WA1 cultivated on fungal Mycelium. *J. Agric. Food Chem.* **2015**, *63*, 269–278. [CrossRef] [PubMed]
87. Karlsson, I.; Edel-Hermann, V.; Gautheron, N.; Durling, M.B.; Kolseth, A.K.; Steinberg, C.; Persson, P.; Friberg, H. Genus-specific primers for study of *Fusarium* communities in field samples. *Appl. Environ. Microbiol.* **2016**, *82*, 491–501. [CrossRef]
88. Walder, F.; Schlaeppi, K.; Wittwer, R.; Held, A.Y.; Vogelgsang, S.; van der Heijden, M.G. Community profiling of *Fusarium* in combination with other plant-associated fungi in different crop species using SMRT sequencing. *Front. Plant Sci.* **2017**, *8*, 2019. [CrossRef]
89. Boutigny, A.-L.; Gautier, A.; Basler, R.; Dauthieux, F.; Leite, S.; Valade, R.; Aguayo, J.; Ioos, R.; Laval, V. Metabarcoding targeting the EF1 alpha region to assess *Fusarium* diversity on cereals. *PLoS ONE* **2019**, *14*, e0207988. [CrossRef]
90. Henry, P.M.; Koehler, S.I.; Kaur, S.; Epstein, L.; Mitchell, J.P.; Gordon, T.R.; Leveau, J.H. Amplicon Sequencing of Fusarium Translation Elongation Factor 1α Reveals that Soil Communities of Fusarium Species Are Resilient to Disturbances Caused by Crop and Tillage Practices. *Phytobiomes J.* **2022**, *6*, 261–274. [CrossRef]
91. Fallahi, M.; Saremi, H.; Javan-Nikkhah, M.; Somma, S.; Haidukowski, M.; Logrieco, A.F.; Moretti, A. Isolation, molecular identification and mycotoxin profile of *Fusarium* species isolated from maize kernels in Iran. *Toxins* **2019**, *11*, 297. [CrossRef]
92. Aslam, S.; Tahir, A.; Aslam, M.F.; Alam, M.W.; Shedayi, A.A.; Sadia, S. Recent advances in molecular techniques for the identification of phytopathogenic fungi—A mini review. *J. Plant Interact.* **2017**, *12*, 493–504. [CrossRef]
93. Van De Wouw, A.P.; Howlett, B.J. Fungal Pathogenicity Genes in the Age of 'Omics'. *Mol. Plant Pathol.* **2011**, *12*, 507–514. [CrossRef]
94. Desjardins, A.E.; Proctor, R.H. Molecular biology of *Fusarium* mycotoxins. *Int. J. Food Microbiol.* **2007**, *119*, 47–50. [CrossRef]
95. Chen, W.; Kastner, C.; Nowara, D.; Oliveira-Garcia, E.; Rutten, T.; Zhao, Y.; Deising, H.B.; Kumlehn, J.; Schweizer, P. Host-Induced Silencing of *Fusarium culmorum* Genes Protects Wheat from Infection. *J. Exp. Bot.* **2016**, *67*, 4979–4991. [CrossRef]
96. Koch, A.; Kumar, N.; Weber, L.; Keller, H.; Imani, J.; Kogel, K.-H. Host-Induced Gene Silencing of Cytochrome P450 Lanosterol C14-Demethylase-Encoding Genes Confers Strong Resistance to *Fusarium* Species. *Proc. Natl. Acad. Sci. USA* **2013**, *110*, 19324–19329. [CrossRef]
97. He, F.; Zhang, R.; Zhao, J.; Qi, T.; Kang, Z.; Guo, J. Host-Induced Silencing of Fusarium graminearum Genes Enhances the Resistance of Brachypodium distachyon to Fusarium Head Blight. *Front. Plant Sci.* **2019**, *10*, 1362. [CrossRef]
98. Ghag, S.B.; Shekhawat, U.K.S.; Ganapathi, T.R. Host-Induced Post-Transcriptional Hairpin RNA-Mediated Gene Silencing of Vital Fungal Genes Confers Efficient Resistance against Fusarium Wilt in Banana. *Plant Biotechnol. J.* **2014**, *12*, 541–553. [CrossRef]
99. Shi, W.; Tan, Y.; Wang, S.; Gardiner, D.M.; De Saeger, S.; Liao, Y.; Wang, C.; Fan, Y.; Wang, Z.; Wu, A. Mycotoxigenic potentials of *Fusarium* species in various culture matrices revealed by mycotoxin profiling. *Toxins* **2017**, *9*, 6. [CrossRef]

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