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Review

Biology, Diversity, Detection and Management of *Fusarium oxysporum* f. sp. *niveum* Causing Vascular Wilt Disease of Watermelon (*Citrullus lanatus*): A Review

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Abstract: *Fusarium oxysporum* f. sp. *niveum* (Fon) is the causative agent of *Fusarium* wilt disease of watermelon; it is the most serious soil-borne pathogen around the globe. The yield loss is around 30–80% or even more, and is presently a major hindrance to watermelon cultivation worldwide. Initially, the infected watermelon plant shows symptoms like loss of turgor pressure of the leaves and vines that can be recovered at night. The progress of the disease in contaminated transplants turns into dull green to yellow and finally necrotic. When the fungus continues to colonize the xylem vessel, it usually forms more tyloses, finally limiting water movement and causing wilt. The correct identification of the pathogen is necessary for proper disease control. As such, the selection of a molecular marker could serve as an effective means of screening the pathogen. Additionally, different methods have also been reported for the identification of Fon. Therefore, this review focused on the comprehensive description of the biology, diversity, detection, aggressiveness, mycotoxin production, and eco-friendly management strategies of the Fusarium wilt disease of watermelon.

Keywords: biology; disease control; *Fusarium oxysporum*; molecular markers; variability

1. Introduction

Fusarium is a complex genus and the most diverged species in the Eumycota for its worldwide distribution, causing diseases in plants, animals, and humans as well as the profuse presence of non-pathogenic *Fusarium* in the natural ecosystem [1]. *Fusarium oxysporum* species complex (Fosc) is an economically devastating species of *Fusarium* and is globally-dispersed in various habitats, along with indoors, soil, and marine environments [2,3]. It is a crucial ubiquitous soil-borne phylogenetic diversified fungus with a vast host range including horticultural and grain crops that cause diseases like wilt, rot, and damping-off [4,5]. Members of this species complex are not the only source of uncontrollable vascular wilt diseases in various plants, but also the source of contagious diseases in humans and create a serious challenge to food security and public health [6]. In terms of the economic importance of the fungus, the pathogen was ranked fifth among the top 10 plant pathogenic fungi [7]. This seed and soil-borne plant pathogen causes serious detrimental effects on contaminated transplants showing symptoms like chlorosis, necrosis, immature

leaf fall, vascular system browning, and finally wilting, which causes tremendous yield reduction. Additionally, if infection occurs earlier or during the harvesting period, some of them can produce mycotoxins in agricultural products [8,9]. Cereals and other food grains can be contaminated by *Fusarium* toxins and causes many diseases like feed refusal syndromes in mammals, moldy sweet potato toxicity, and poisoning in bean hulls and different other living organisms [10].

In the early 1880s, Smith identified the wilt disease of watermelon from South Carolina and Georgia after that of cotton [11,12]. Watermelon wilt fungus was termed *Fusarium niveum* by Smith (1899) and also suggested that it was a variety of *Neocosmospora vasinfectum* var. *nivea.* After that, Wollenweber and Reinking (1935) gave a new name of watermelon *Fusarium* wilt of *F. bulbigenum* var. *niveum* Woll [13]. Based on this classification, Leach and Currence (1938) reflected that the *Fusarium* of watermelon and melon are various forms of *F. bulbigenum* var. *niveum* (form 1 and 2, correspondingly). Previously, Hansford (1926) first proposed that all species in the *Fusarium* unit assembled as a sole species, *F. oxysporum* [13]. Due to extreme host specificity exhibited by numerous pathogenic isolates of *F. oxysporum,* finally, Snyder and Hansen (1940) restated that all species within the unit Elegans be reflected a sole species, *F. oxysporum*, and furthermore proposed specialized forms (i.e., *forma specialis* (f. sp.)) that can distinguish particular virulence to one host or another [13]. Accordingly, *F. oxysporum* f. sp. *niveum* is named from the watermelon wilt form 1 and *F. oxysporum* f. sp. *melonis* from the melon wilt form 2. After that, the concept of formae speciales achieved widespread recognition, which led to the grouping of 10 species into a single unit with many pathogenic formae speciales, and further physiological races were derived from F. sp. [14,15].

Fusarium wilt pathogen is one of the most widely studied and devastating soil-borne pathogens around the world with both saprophytic and pathogenic members [13,16]. Non-pathogenic and pathogenic *F. oxysporum* strains remain in the soil, but the pathogenic strain causes severe vascular wilt disease in more than 150 economically major agricultural crop species. The most important crops that are likely to be infected by vascular wilt disease are banana, tomato, melon, watermelon, and cotton [17]. In the Cucurbitaceae family, eight various f. sp. have been identified; among them, *F. oxysporum* f. sp. *cucumerium* (Foc; cucumber), *F. oxysporum* f. sp. *niveum* (Fon; watermelon), and *F. oxysporum* f. sp. *melonis* (Fom; melon) are enormously important. Out of this, Fon is the most destructive pathogen of watermelon around the world [18]. The pathogen is responsible for yield losses of around 30–80% or even more [19,20], and presently is a major hindrance in watermelon cultivation. Moreover, difficulties faced by plant pathologists include reliable identification of the causal agents of the disease according to the epidemiological related parameters such as severity, levels of species, formae speciales, pathovars, biovars, and races. The properties of these characteristics would foster appropriate and relevant control measures [21]. This review aimed to summarize the current understanding of the biology, diversity, detection, aggressiveness, mycotoxin production, and novel disease management strategies of Fusarium wilt disease of watermelon.

2. Disease Cycle and Epidemiology

The Fon is a predominant, monocyclic, soil-borne, worldwide diversified fungus including saprophytic and pathogenic entities [4,5]. Fon is a host-specific to watermelon, it even cannot intimately infect concomitant cucurbits crops such as cucumber and cantaloupe with some limitation, which has been studied in greenhouse conditions [22]. The microorganism dispersing media are soil, plant debris, farm machinery [23], and seeds [24] and are known to survive more than 15 years without host plants [25]. Water and contaminated farm equipment can spread the pathogens over short distances, and for extensive areas, the spread of the disease has to be through contaminated soil, seeds, or seedlings. Normally, once a region becomes contaminated, it persists so emphatically [26]. The fungus infects the tissues of the plants as a germinating spore, and the growing hyphae penetrate the plant tissue through the wounds or openings near the site of elongation for

root hair [27]. The fungal hyphae eventually penetrate the vascular tissue and produce microconidia [27]. The microconidia are then released into the xylem, which travel upward with the water and begin to colonize the watermelon's vascular tissue of the plant [28]. Afterward, there is an infection of the watermelon plant by the pathogen frequently inhabited within it, which remains until death or decay, as presented in Figure 1. At the last stages of the disease, the fungus produces thick mats of white mycelia and plenteous macroconidia. Under stressful environmental situations, chlamydospores develop from the aforementioned pathogen structures and conjoin into the soil. *Fusarium* wilt disease is achieved generally by spreading chlamydospores, which is the primary way for the survival of the pathogen [26]. Chlamydospores are the lowest manageable attribute of *Fusarium* wilt infection and can live for more than 10–15 years. *Fusarium* wilt does not spread from plant to plant within season due to the absence of spore production above the ground in the field. Martyn and Vakalounakis (2017) indicated that it could also be spread by seeds [29]. Fon was first isolated from infected seed in 1928. Since then, several researchers have confirmed the seed-borne nature of Fon. Still, the spreading mechanism of the seed-borne nature of watermelon seed is mostly undiscovered and infection rates are normally less than 5% [13]. Another mechanism for the survival of pathogens is in the plant debris and the establishment of non-host plants that are alive [26]. At the advanced stages of infection, permanent wilt and yield losses on watermelon could reach around 30–80% or even more [19,20]. It becomes worse in sandy soils with a temperature range of 77° to 80 °F and a pH range of 5.5–6.5. At the anatomical level, the colonization procedure of pathogenic *Fusarium* species has been described by several researchers [30,31]. Disease development and symptom expression of host plants depend on the colonization of vessels by the pathogen [32].

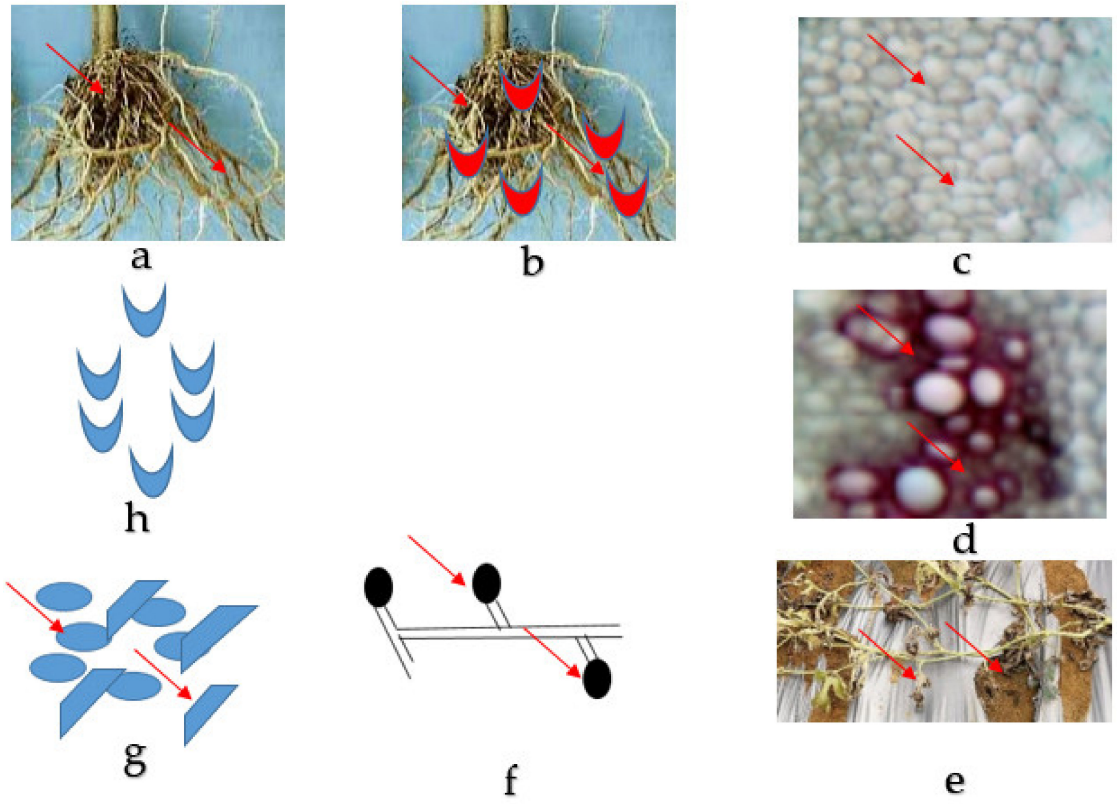

Figure 1. *Fusarium* wilt disease life cycle of watermelon caused by *Fusarium oxysporum* f. sp. *niveum*. (**a**) The healthy root of watermelon. (**b**) Germinating spore in contact with the root for penetration into the tissues. (**c**) Collapsed and distorted vessels in the xylem of the watermelon plant. (**d**) Gum produced by mycelia in the vessels. (**e**) The entire watermelon plant wilts and dies. (**f**) Spore formed by mycelia in the soil. (**g**) Micro and macroconidia present in the soil. (**h**) Germinating spore.

Initially, the infected watermelon plant showed symptoms like loss of pressure (turgor pressure) of vines and leaves, which could be recovered at night; exclusively single or some vines may be affected [33]. The progress of the disease in infected seedlings changed from dull green to yellow and finally necrotic [34]. When the fungus continues to colonize the xylem vessel, the plant forms more tyloses, finally limiting the water movement and the vine begins to wilt [26] (Figure 2).

(**a**) (**b**)

Figure 2. Wilting disease symptoms of watermelon plants caused by *Fusarium oxysporum* f. sp. *niveum* under field condition. (**a**) Showing brown necrotic lesions at the base of the stem. (**b**) Showing a severe form of wilting, laterally the whole plant wilted and died. Pictures are from the personal collection of M. Z. Rahman collected during the field survey.

3. Races and Vegetative Compatibility Groups

Based on the aggressiveness of the pathogen or host cultivar's resistance performance, Fon has been classified into four physiological races (0–3) (Table 1) [16,26]. Crall (1963) first reported Fon race 0 from Florida, USA, and stated that all modern varieties had resistant *F0-1* gene to the race 0, so Fon race 0 loses its economic importance as a pathogen [13]. Fon race 1 is considered to be the widely prevalent race around the commercial watermelon-producing areas in the world. Race 1 was first identified by Smith (1894) from South Carolina, USA, and discrimination between races 0 and 1 could be more quantitative and not qualitative, and race 0 changed to race 1 based on aggressiveness [35]. At present, many diploid (seeded) and some triploid (seedless) cultivars have developed resistance toward Fon races 0 and 1 [13,26]. Later on, Fon race 2 was discovered in Israel and subsequently identified from the United States in 1981 [13]. The prevalence of race 2 was so high in some locations and showed more virulence than Fon race 1, which can infect commercial diploid seeded and triploid seedless varieties [22,36]. After 37 years later, another new race 3 was identified in Maryland, where it was reported to be the highest in terms of aggressiveness than races 0, 1, and 2 [16,37]. The main sources of Fon race 3 could be from contaminated seed or seedlings, selection, or mutation from races 0, 1, and 2 [16].

Traditionally, *F. oxysporum* f. sp. *niveum* can be distinguished from other formae speciales and saprophytic strains of *F. oxysporum* only by its virulence on watermelon. Distinguishing races requires the screening of pathogenic isolates on cultivars of varying levels of resistance. These tests are laborious and often inconsistent or inconclusive. Results can be greatly influenced by environmental factors, host age, inoculum level, and inoculation methods [38]. An alternative approach to the classification of strains of *F. oxysporum* is based on vegetative compatibility [39,40] where isolates are grouped into a specific phenotypic class [41]. Therefore, VCGs are useful for characterizing the genetic diversity within a formae speciales and, in some cases, for distinguishing pathogens from non-pathogens [42]

Table 1. Different watermelon varieties used to separate four races of Fon.

Variety	Response of Disease			
	Race 0	**Race 1**	**Race 2**	**Race 3**
All Sweet	R	R	S	S
Crimson sweet	R	S	S	S
Calhoun Gray	R	R	S	S
Sugar Baby	S	S	S	S
Black Diamond	S	S	S	S
Charleston Gray	R	S	S	S
PI-296341-FR	R	R	R	S

R = Resistant, S = Susceptible.

Genetic exchange and the sexual stage is absent in *F. oxysporum,* therefore it is limited to genetic transformation and the parasexual cycle. Asexual *Fusarium oxysporum* needs heterokaryosis for genetic exchange, which is regulated by a set of heterokaryon loci, and it helps in the formation of vegetative compatibility by the fusion of hypha and cell lysis [43]. *Fusarium oxysporum* with the capability to produce stable heterokaryon is in the same vegetative compatible group (VCG) or more likely genetically the same or clonal lineage [44,45].

Larkin et al. (1990) extensively studied 250 strains of Fon collected from five different states in the USA, Taiwan, and Australia and observed a significant correlation between vegetative compatibility group (VCG) and physiological races or virulence races. He reported three distinct VCGs (0080, 0081, and 0082) for Fon strains, in which race 1 and race 2 belong to VCG 0080 and VCG 0082, respectively [46], whereas VCG 0081 comprises only one Fon strain from Florida. He also demonstrated that the pathogenic strains of Fon were incompatible with non-pathogenic *F. oxysporum*; additionally, within the same race strains were compatible, but incompatibility was observed with the opposite race. Zhou and Everts (2007) described three VCGs: two were alike and earlier identified by Larkin et al. (1990) (i.e., VCG 0080 and VCG 0082), and the other one was distinct *viz.* VCG 0083 [47]. His results differed from those previously described by Larkin et al. (1990) and obtained an insignificant similarity between virulence race and VCG. VCG 0080 and VCG 0082 comprised all three race (0, 1, and 2) strains, and VCG 0083 consisted of only six isolates, which were classified as race 3 [16], but none of the strains were in VCG 0081. The newly identified race 3 is pathogenic to PI 296341-FR, previously stated to be resistant to race 2. Besides, race 3 isolates are compatible vegetatively with one another (VCG 0083) and incompatible with race 1 (VCG 0080) and race 2 (VCG 0082). Among these VCGs, VCG 0080 is considered the main diverged group followed by VCG 0082, VCG 0083, and 0081 distributed in a limited geographic area.

Globally collected Fon isolates were analyzed through mtDNA RFLP and the results indicated that no similarity was reported between geographic origin and race; RFLP was grouped into two common patterns that contained all three Fon races from various regions [48]. Some other forms of formae speciales (Table 2) like *F. oxysporum* f. spp. *melonis* [49], *lycopersici* [50], *cubense* [51], and *asparagi* [52] showed a complex relationship between genetic diversity and virulence. From these findings, it can be concluded that VCG and virulence (race or cross pathogenicity) were in a complex relationship among the strains of Fon [53]. Perhaps VCG cannot be used to differentiate the races of Fon; instead, it only helps to separate pathogenic strains from non-pathogenic strains of Fon and in the characterization of genetic variability among the Fon strains.

Table 2. Vegetative compatibility groups, formae speciales, and several races of Fo responsible for vascular wilt disease in many important crops.

Host	Forma Specialis	VCG	Described Races	References
Watermelon	*Niveum*	008-	0, 1, 2, 3	[11]
Melon	*Melonis*	013-	0, 1, 2, 1.2 Y. 1.2 W	[54]
Cucumber	*Cucumerinum*	018-	1, 2, 3	[55]
	Radicis-cucumerinum	026-	-	
Bitter gourd	*Momordicae*	-	-	[56]
Bottle gourd	*lagenariae*	041-	-	[57]
Vegetable sponge	*Luffae*	-	-	[58]
Wax gourd	*Benincasae*	-	-	[13]
Tomato	*lycopersici*	0030-	1, 2, 3	[59]
	Radicis-lycopersici	009-		
Pisum sp	*pisi*	007-	1, 2, 5, 6	[60]
Radish	*raphani*	022-	-	[61]
Cotton	*vasinfectum*	011-	1, 2, 3, 4, 5, 6, 7, 8	[62]
Banana	*Cubense*	012-	1, 2, 3, 4	[63]
Bean	*Phaseoli*	016-	1, 2, 3, 4, 5, 6, 7	[64]
Cabbage	*conglutinans*	010-	1, 2	[65]
Chickpea	*Ciceris*	028-	1, 2, 3, 4	[66]
Onion	*Cepae*	0420-	-	[67,68]
Asparagus	*Asparagi*	100-	-	[52]
Sweet potato	*Batatas*	036-	1, 2	[69]
Sugar beet	*Betae*	027-	-	[70]
Carnation	*Dianthi*	002-	1, 2, 3, 4 to 11	[71]
Lettuce	*Lactuace*	030-	1, 2, 3	[72]
Gladiolus	*Gladioli*	034-	2	[73]
Tobacco	*Nicotianae*	037-	0, 1, 2, 3	[74]
Alfalfa	*Medicaginis*	004-	-	[75]
Potato	*Tuberosi*	035-	-	[76]
Cyclamen	*Cyclaminis*	015-	-	[77]
Chrysanthemum	*Chrysanthemi*	005-	1, 2, 3	[78]

4. Evolutionary Relationship between Races of Fon and VCGs

Restriction fragment length polymorphism (RFLP), isozyme analysis, analysis of intergenic spacer (IGS), and random amplified polymorphic DNA (RAPD) analysis were used to study genetic variability among several formae speciales of *Fusarium oxysporum* and demonstrated that isolates in the same VCG were similar in genetic makeup than the isolates in different VCG(s) [79]. The high similarities of DNA profiles among all three different races of Fon have been stated in previous works [48,80]. All these races were similar to mtDNA RFLP as well as some common sequences of chromosomal DNA. Generally, a mutation in the gene could cause a modification in developing a new race and its virulence. It was confirmed that the transformational mutagenesis of race 2 isolates of Fon caused a change to race 0 without a definite change in the VCG trait of that isolate [80]. Additionally, the recent instance of one race coming from the other race in a local community within the evolutionary lineage (VCG) was documented in *Fo* f. sp. *lycopersici* in California (USA) [81]. The spread of different races among the VCGs, especially the presence of race 2 in all three different VCGs, implies the probability of the development of a single VCG from the other because of the mutation in a *vic* gene. This mutation in the *vic* gene led to a modification from incompatibility to compatibility or vice versa and has been confirmed in some other fungi [82]. The mutation that alters the vegetative compatibility was also reported to describe the origin of two VCGs with similar virulence (race) in Fo f. sp. *melonis* [49]. The mtDNA polymorphism similarity between VCGs of *Fo* f. sp. *niveum* was discovered by Kim et al. (1992) and supported this model of evolution [48]. Finally, it can be concluded that the transformational mutagenesis of the Fon isolates could cause a change to race without a definite change in the VCG trait of that isolate.

5. Detection of Fon and Other Members of Formae Speciales

Rapid and precise diagnosis of the pathogen is a prerequisite for controlling disease and its management. Traditionally, for genetic diversity and pathogen identification, researchers mainly depend on morphological study and molecular techniques [83]. Presently, disease assessment persists with the primary technique of discriminating host range and physiological races of an infective Fon isolate [13]. Differentiation of Fo races and formae speciales is habitually tested by using time-consuming and labor-intensive assessment of

disease [84], therefore, formae-speciales-specific DNA sequencing for molecular screening technique is highly preferable [85].

To detect and identify various *Fusarium* wilt pathogens as well as the various studies among them, several reference genomes are available that can help in the diagnosis of plant pathogens rapidly, accurately, and cost-effectively [86]. Methods based on DNA polymorphisms, like amplified fragment length polymorphism (AFLP), simple sequence repeat (SSR), random amplified polymorphic DNA (RAPD), insertion-deletion (InDel), utilization of loop-mediated isothermal amplification (LAMP), single-nucleotide polymorphism (SNP), and using specific gene sequences like an internal transcribed spacer, β-tubulin, and calmodulin gene as well as elongation factor-1 alpha (EF-1α) have been reported to be reliable in this regard (Table 3) [87]. Diagnostic based genes like ribosomal intergenic spacer (IGS) or EF-1α help to differentiate between several species of fungi and sometimes subspecies separation, however, these may prove uncertain due to the presence of one or several clonal lineages of each forma specialis of *F. oxysporum* [86,88].

A close relationship in evolution was observed between the five formae speciales causing wilt disease of Cucurbitaceae based on mtDNA RFLPs and the isolates of a single forma specialis was grouped along with many other isolates not of the same forma speciales, and it was observed that many formae speciales were formed in one branch. Within the five formae speciales, Fon appeared to be the most homogenous, but the most diverged group was *F*. f. sp. *cucumerinum* [48]. No polymorphisms were noticed among the 13 isolates of Fon including races 0, 1, and 2 collected from Israel and the USA using mtDNA RFLP analysis [89]. Additionally, the similarity was observed not only in the mtDNA RFLP map, but also in the estimated size of the mtDNA between f. sp. *melonis* and *F*. f. sp. *niveum* (45.1 kb and 44.5 kb, respectively) [48,49].

Table 3. PCR primers used to detect *F. oxysporum*, *F. oxysporum formae speciales*, and their races.

Primers Name	Target Organism	Target Gene	References
β-tubulin	*F. oxysporum*	β-tubulin (TUB2) rDNA region	[90]
EF1 and EF2	*F. oxysporum*	Translation elongation factor1-α coding region	[91,92]
ITS1 and ITS4	*F. oxysporum*	ITS region of rDNA	[93]
NMS1 and NMS2	*F. oxysporum*	The mitochondrial small rRNA subunit (*mtSSU*) region	[94]
FIGS11 and FIGS12	*F. oxysporum*	The intergenic spacer (*IGS*) large rRNA subunit gene region	[95]
Cal228F and CAL2Rd	*F. oxysporum*	calmodulin (*cmdA)*	[96,97]
7cF and 11aR	*F. oxysporum*	RNA polymerase II second largest subunit (rpb2)	[98]
Uni F and UniR	*F. oxysporum*	The endo-polyglacturonase gene (Pg1)	[99]
Fon-1and Fon-2	*F. oxysporum* f. sp. *niveum*	Derived from the random amplified polymorphic DNA (RAPD) fragment	[100]
FONSIX6-F and FONSIX6-R	*F. oxysporum* f. sp. *niveum* race 2	The *SIX6* (secreted in xylemprotein 6)	[101]
FNR3-F and FNR3-R	*F. oxysporum* f. sp. *niveum* race 3	Pathogenicity chromosome	[102]
P12-F2B and P12-R1	*F. oxysporum* f. sp. *Lycopersici*	Secreted in xylem 1 (*SIX1*)	[103,104]
SIX2-F2 and SIX2-R2	*F. oxysporum* f. sp. *Lycopersici*	Secreted in xylem 2 (*SIX2*)	[103]
SIX5-F1 and SIX5-R1	*F. oxysporum* f. sp. *Lycopersici*	Secreted in xylem 5 (*SIX5*)	[88]
SIX4-F1 and SIX4-R1	*F. oxysporum* f. sp. *lycopersici race 1*	Secreted in xylem 4 (*SIX4*)	[88]
SIX3-F1 and SIX3-R2	*F. oxysporum* f. sp. *lycopersici race 2*	Secreted in xylem 3 (*SIX3*)	[103]
SIX6b_210_F and SIX6b_210_R	*F. oxysporum* f. sp. *cubense* (Foc) race 1	Secreted in xylem 6 (*SIX6*)	[105]
SIX8b_206_F and SIX8b_206_R	Foc subtropical race 4	Secreted in xylem 8 (*SIX8*)	[105]
SIX1a_266_F and SIX1a_266_2_R	Foc tropical race 4 (TR4)	Secreted in xylem 1 (*SIX1)*	[105]

The most important point for diagnostics is the genes that are able to encode proteins similar to virulence [106]. In the case of Fo, which attacks the seedlings of a tomato plant, some of the proteins were identified in secreted-in xylem (SIX) and xylem sap [104,107]. The proteins or other sets of molecules (and small RNAs and secondary metabolites) have been reported to be associated with pathogenesis during disease progression and colonization and are widely called effectors [106,108]. *SIX* genes were present across the *formae speciales* and the profile of the genes was used to distinguish the distinct *formae speciales*, isolates, and races [85]. The *SIX* gene profiling was used to differentiate the three races in *F*. f. sp. *lycopersici* [88]. The SIX genes (SIX1, SIX9, SIX4, and SIX8) were also present in another species of *F. oxysporum* [109,110], and SIX6 was present in *F*. f. sp. *vasinfectum* [111], which infects *Brassica* and *Arabidopsis* [112]. Moreover, SIX6 and SIX1 were present in *F*. f. sp.

betae [84]; SIX7, SIX10, and SIX1 were present in *F*. f. sp. *lini* and *canariensis* [113]; and SIX7, SIX8, and SIX1 were reported to be present in *F*. f. sp. *cubense* [114], while SIX5, SIX7, and SIX3 were present in *F.* f. sp. *cepae* [110]. Additionally, SIX4 plays an important role in the virulence of *F*. f. sp. *conglutinans*, which caused a yellowish color in the cabbage plant [115]. A further difference between races 0, 1, 2, and 3 of Fon could be possible because of the recent studies that have that focused specifically on an effector gene (SIX6), which plays a vital role in initiating R-protein-mediated immunity [101,102]. The gene (SIX6) was reported to be effective in Fon-1 isolates that are called FonSIX6, and it could be the reason to initiate a resistance in some genotypes of Fon-1-resistance, whereas Fon race 2 isolates do not have the gene (FonSIX6 effector gene), which resulted in an escape from the higher disease severity and immune system of the plant [101]. Besides, *F*. f. sp. *cubense* and *F*. f. sp. *lycopersici* can be differentiated from other groups of *formae speciales* using PCR primers that are designed to identify the specific SIX effector genes [109].

Species-specific primer Fn-1/Fn-2 were synthesized from ITS sequences for accurate and rapid identification of pathogenic *F.* f. sp. *niveum* [116]. Primer set Fn-1/Fn-2 amplified only a single PCR band around 320 bp from Fon, but the primers were unsuccessful to amplify the DNA of several other fungi and *formae speciales* f. sp. *cucumerinum*. In contrast, primer set FON-1/FON-2 could amplify a single 174 bp DNA fragment, which could differentiate Fon from the other *formae speciales* and *Fusarium* spp, but could not amplify the DNA of other *formae speciales* infecting cucurbits such as *cucumerinum*, *melons*, *momordicae*, and *luffae* [117]. Additionally, secreted in xylem protein 6 (SIX6) (i.e., avirulence gene) was identified in Fon races 0, 1, and 3, but absent in race 2. As a result, Fon race 2 was capable of differentiation by using primer set FONSIX6-F/FONSIX6-R [102]. Finally, Fon race 3 could be distinguished by using primer set FNR3-F/FNR3-R, which amplified the pathogenicity chromosome region (511 bp) of the Fon genome [102], as presented in Figure 3.

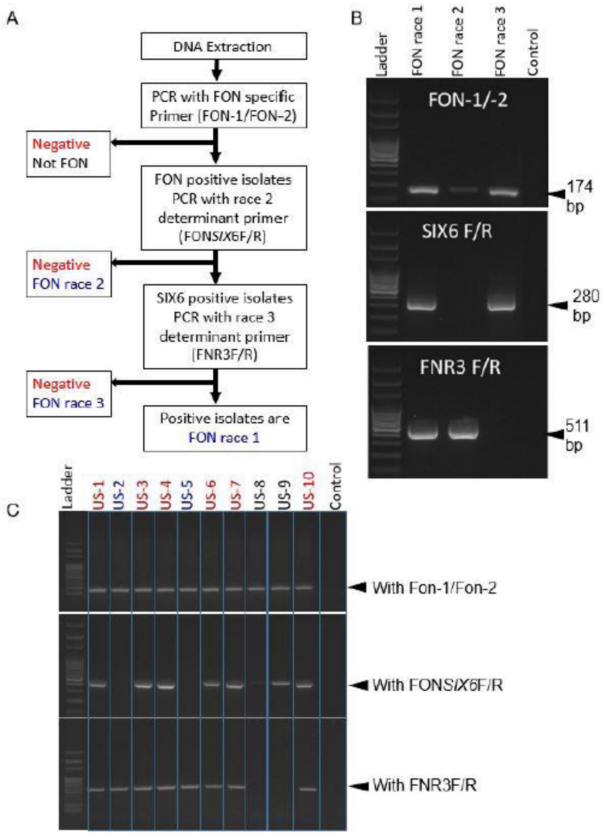

Figure 3. Flow diagram of three marker sets distinguishing Fon races 1, 2, and 3 (**A**); Example of differentiation of known Fon races (**B**); Identification of unknown Fon races 1, 2, and 3 (**C**). Source: [102].

6. Molecular Diversity among Fon Inferred from Markers and DNA Fingerprints

The genetic variability of 50 different isolates of Fon including the three known races (0, 1, and 2) was obtained from many areas using RFLP and stated that no relationship exists between races or geographical areas and RFLP haplotypes [48]. Besides, further researchers observed that all these races shared a common sequence of cDNA and were closely related [80]. Petkar et al. (2019) performed a discriminant analysis of principal components (DAPC) of 99 different isolates with 15 SSR markers, and grouped all the isolates in eight different clusters with two main clusters (clusters 8 and 1). Most of these isolates were either found in cluster 8 and cluster 1 from the two races, race 3 (45.8%) or race 2 (35.6%). Moreover, there was no relationship between the physiological races of the isolates and genetic cluster alignment was noticed, but a strong similarity was observed for Fon genotypes in different geographical areas [36]. The phylogenetic study of some forms of Cucurbitaceae based on RAPD-PCR [118] showed that *F.* f. sp. *niveum* and *F.* f. sp. *cucumerinum* may be polyphyletic, while *F.* f. sp. *melonis* and *F.* f. sp. *radicis-cucumerinum* could be monophyletic. Up to now, ISSR, AFLP, SSR, RAPD, and RFLP-IGS markers have been utilized in PCR form of the molecular identification of Foc (Table 4) [119–121].

The effector gene SIX was studied to expand the molecular diagnostic toolbox for the pathogen (Foc) [105]. About 27 Foc-TR4 isolates from Peninsular Malaysia were studied based on their phylogenetic analysis of SIX sequences (SIX7, SIX8, and SIX1) and their variability. Among the three different SIX genes, SIX8 and SIX1 genes were presented in the 27 Foc-TR4 isolates studied, and the phylogenetic analysis of SIX8 and SIX1 sequences showed that they are similar to TR4 isolates (VCG 16/01213) from Indonesian and Australia, having bootstrap figures of 100% and 94%. No diversity was noticed in the SIX8 and SIX1 sequences since all 27 different isolates were clustered in the same clade. Moreover, there was no correlation with regard to the virulence level with the existence or absence of the genes [122]. A variability was noticed between the intraspecies tree and phylogenetic trees based on *SIX* genes, which suggested horizontal gene transfer (HGT) activities in the *SIX* genes of *Foc*. Czislowski et al. (2018) and Liu et al. (2019) worked on the features of the 12 different *Foc* FA biosynthetic genes (*FUB*), three housekeeping genes, translation elongation factor-1α/RNA polymerase II subunit I/RNA polymerase II subunit II (*EF-1α/RPB1/RPB2*), and coding sequences of the 12 *FUB* genes using genetic variability analysis, selective pressure analysis, phylogenetic analysis, and recombination detection [123,124]. Recombination detection and intraspecies phylogeny analysis showed that more HGT events (normalized) were present in the *FUB* genes than within the three different housekeeping genes. Additionally, several of these events or activities involve the outgroup isolates and have increased the genetic variability of *FUB* genes in *Foc*. Finally, the results showed that *FUB* genes in Fo have benefited from horizontal gene transfer (HGT) to obtain a high genetic variability to respond to many environments and hosts, despite being subjected to negative selection.

Table 4. Genetic variability studies on important formae speciales of *Fusarium oxysporum*.

Formae Speciales	Methods of Analysis	Variability	References
Niveum	VCG, RAPD, RFLP, SSR	Very low	[36,44]
Melonis	VCG, RFLP, SEQ, RAPD, ISSR, SSR	Low	[44,125]
Lycopersici	VCG, RAPD, RFLP, ISSR, SSR	High genetic diversity	[125,126]
radicis-lycopersici	VCG, RFLP, ISSR	High genetic diversity	[44,127]
Cubense	VCG, IS, RFLP, RAPD, ISSR, SSR, SIX genes, FUB genes	High genetic diversity	[122,128]
Cepae	VCG, AFLP, RAPD, RFLP, ISSR	Low levels of genetic diversity	[129,130]
Cucumerium	VCG, RAPD, SSR	Most diverse	[118,125]
radicis-cucumerinum	VCG, RAPD	Low genetic diversity	[118]

Table 4. *Cont.*

Formae Speciales	Methods of Analysis	Variability	References
Ciceris	VCG, RFLP, SSR, ISSR	Low genetic diversity	[131,132]
conglutinans	VCG, IS, RFLP, InDel,	Low genetic diversity	[44,65]
Pisi	VCG, RAPD, ISSR	Very low	[133,134]
Lentis	RAPD, ISSR, SSR	Very low	[135,136]
dianthi	VCG, RAPD, RFLP	Very low	[71,137]
Gladioli	VCG, RAPD, RFLP, cpSSR	High genetic diversity	[138,139]
Vasinfectum	VCG, RAPD, AFLP, RFLP	More genetically diverse	[140,141]
momordicae	VCG, AFLP, ISSR	Relatively low	[142]

7. Mycotoxins Produced during Fon and Watermelon Interactions

The genus *Fusarium* produces harmful secondary metabolites known as mycotoxins [143]; these mycotoxins are universally distributed and have been found to have economic importance due to their toxicity to animals, humans, plant pathogens, and also in food and feeds [144]. Among the *Fusarium* mycotoxins, of primary concern are the trichothecenes, fumonisins, and zearalenone [145]. As a typical vascular wilt fungus, *F. oxysporum* produces the characteristic xylem vessel clogging and wilting of infected plants. Colonization and clogging of vessels in addition to the secretion of several toxins by the fungus including fusaric acid, lycomarasmin, dehydrofusaric acid, etc. play a major role in the development and progression of wilt symptom [143]. Lakshminarayanan and Subramanian (1955) first detected fusaric acid (in-vivo) in wilted cotton plants and suggested that it was involved in the production of wilt symptoms [146]. Gäuman et al. (1957) suggested that fusaric is an important wilt toxin in *Fusarium* wilt of tomato and cotton [147]. Apart from fusarins, fusaric acid (FA) is well-known for its phytotoxicity and has been studied for its role in the pathogenesis of *Fusarium* wilts [148]. Fusaric acid is produced when Fon invades watermelon as an important pathogenic factor that may contribute to plant wilting, and it causes wilt diseases in different varieties of plants such as watermelon, cucumber, tomato, beans, and cotton [147]. It is also a wilt toxin on tomato plants infected with *F. oxysporum* f. sp. *lycopersici*, and the toxic concentration needed to cause wilting is 150 mg L^1 [149]. In 1957, Nishimura showed that several formae speciales of *F. oxysporum* including f. sp. *niveum* produced fusaric acid in culture and infected plants [150,151]. Moderate fusaric acid (a fusarial mycotoxin) doses induce apoptosis in saffron while high fusaric acid doses stimulate necrosis [152]. However, FA produced by Fon mainly disturbs the metabolism of growing plants [153]. Toxins are primary determinants of pathogenesis when they act as the key elements in infection initiation and symptom development and are secondary determinants when they only modify the symptoms intensively [153]. Davis (1969) inoculated several plants with different formae speciales and showed that in most cases, there was little fusaric acid detected in planta. An exception, however, was noted for watermelons inoculated with f. sp. *niveum*. In this case, *Fusarium*-infected plants contained appreciable amounts of fusaric acid and he correlated the pathogenicity of six isolates of f. sp. *niveum* with fusaric acid content in the plants [149]. Wu et al. (2009) reported that the fusaric acid (FA) strongly reduced the chlorophyll content of watermelon seedling leaves, resulting in heavy suppression of leaf photosynthesis, which therefore affected the seedlings' growth and led to leaf wilting and necrosis [154]. Although the toxin does not play any role in the initial infection stage, it significantly contributes to the pathogenesis process during the subsequent stages of the infection [155].

8. Potential Methods of Managing *Fusarium oxysporum* f. sp. *niveum*

Managing *Fusarium* wilt is very difficult because of the persistent nature of chlamydospores (10 to 15 years) and the development of new physiological races [26,117]. However, contemporary management practices that are available for controlling wilt disease of watermelon are discussed below:

8.1. Cultural Practices

8.1.1. Sanitation

This could be achieved by transplanting the seedling in the greenhouse, observing disease symptoms of *Fusarium* wilt, and removing diagnostical trays as well as neighboring trays to avoid the presence of *Fusarium* wilt disease [26]. Expulsion of an infected plant is one of the best ways of disease management. Hence, seeds and seedlings must be acquired from a dependable origin and should be free from pathogens [26]. Movement restriction of infected seedlings will prevent Fon from launching into a virgin area and will restrict the transmission of new races [26]. Escaping pathogens like Fon is the best management strategy, however, many areas are presently occupied by the pathogen, so it is very hard to use contamination-free seed and seedlings for transplanting watermelon [33].

8.1.2. Host Resistance

The introduction of a resistant variety is among the most effective management practices for *Fusarium* wilt disease of watermelon. Breeders have developed many resistant varieties against races of Fon 0, 1, but only a few for race 2 [156]. 'Conquerer' was the first resistant variety against Fon race 1. Later on, 'Calhoun Grey' and 'Summit' were introduced accorded by paramount gene Fo-1 with some modifier genes [13,20]. Watermelon diploid and triploid varieties resistant to Fon race 1 are commercially available in the southern USA [157], but in triploid seedless varieties, Fon race 1 and 2 resistance has not been developed yet [16,158]. Nevertheless, the comparative levels of resistance among triploid varieties have shown some variation [159]. Syngenta developed some cultivars against Fon race 1 such as 'Summer king', 'Distinction', 'Fascination', Seedway ('Indiana'), and Seminis ('Majestic' and 'Cronos'). In contrast, Egel and Martyn (2013) suggested using watermelon pollinizer varieties SP-6 and SP-5 that were found by Syngenta and derived from PI-296341-FR, which have shown resistance to Fon race 2.

The capability to map genes of resistance to be coupled with marker-assisted selection is promoting the breeding of watermelon varieties that are resistant to race 2. Many research groups from the USA and China have been able to identify the quantitative trait loci (QTL), which is situated on many chromosomes and was linked to race 2 or race 1 resistance. Race 1 resistance was mapped to chromosome 1 from the majority of modern cultivars [12,160]. Branham et al. (2019) stated that another resistance gene to race 1 was located on chromosome 9 [161]. The QTL resistance for race 2 was mapped to chromosomes 10, 11, and 9 [12,161] but we still lack complete resistance in commercial watermelon varieties to all races (0–3) of Fon. Several diploid and some triploid varieties have shown superior resistance to Fon races 0 and 1, but resistance lines to Fon races 2 and 3 are unavailable. Even so, under high inoculum density in the soil, this resistance may break down due to the climatic condition or mutation [26].

8.1.3. Crop Rotation

Rotation of different crops (non-host crops) for at least 5–7 years can reduce the amount of pathogen inoculum and lower the *Fusarium* wilt disease of watermelon, however, this rotation is very hard to maintain due to insufficient arable land [162]. Although rotation is followed by the non-host crop for several years, viable chlamydospores can infect the host beyond the inoculum level that is present in low quantity [26]. Zhou and Everts (2004) indicated that as inoculum density increases, wilt percentages also increase, thus managing inoculum density is a key factor in controlling *Fusarium* wilt disease [163]. Even after practicing multiple disease-controlling tools, damages observed from *Fusarium* wilt could still happen [26].

8.1.4. Grafting

Grafting watermelon with other cucurbit species (Table 5) confers many beneficial effects on production such as increased yield, fruit size, number of fruits, and decrease in disease infestation [164,165]. This technology is efficient in decreasing *Fusarium* wilt

mainly because the rootstock is not the host of the pathogen. In countries like China, Japan, and Korea, grafting technology is adopted due to the intensive cultivation of land and the difficulties of crop rotation. This technology has also been adopted in Australia for its multiple benefits [166,167]. In comparison to non-grafted seedlings, grafted watermelon plants reduced *Fusarium* wilt incidence ranges from 88–100%, respectively, as reported in southern USA, Turkey, and China [18,168,169]. It has also been studied that root exudates from non-host rootstock play a significant importance in the reduction of Fon [170]. Rootstocks such as *Lagenaria siceraria* (bottle gourd) and *Cucurbita maxima* (squash hybrid) grafted with susceptible scions protect the plant from *Fusarium* wilt disease and show resistance to Fon races 1 and 2 [18,171]. In contrast, grafted seedlings are very costly compared to non-grafted transplants [18], in addition, many farmers are unwilling to spend money on watermelon production at the early stage of the cropping season.

Table 5. The rootstock used for resistance to Fon in different countries.

S/No	Resistant Root-Stock			References
	Cultivar	Country Name	Species	
1.	'RTX1', 'RS 841', 'Carnivore'	Australia	*Cucurbita maxima* X *Cucurbita moschata*	[167]
2.	Mammoth King, Round and Oblong	Bangladesh	*C. moschata*	[172]
	Benincasa hispida		*Lagenaria leucantha* (*L. siceraria*)	[172]
	wild watermelon		*C. maxima*	[172]
3.	'Marathon'	Chili	*Cucurbita maxima* x *Cucurbita moschata*	[173]
	'Macis'		Bottle gourd (*Lagenaria siceraria*)	[173]
	ChaoFeng F1		Watermelon	[174]
4.	'Wanzhen No. 2	China	Pumpkin (*Cucurbita moschata*) rootstock	[169]
	Dayehuzi		Bottle gourd (*Lagenaria siceraria*)	[170]
5.	F1 hybrid Nun 2001	Italy	Lagenaria	[175]
6.	Strong Tosa	Israel	*Cucurbita moschataxC. maxima*	[176]
7.	Renshi	Japan	Bottle gourd	[177]
	Choseun		*Cucurbita moschata*	[178]
8.	Sintojwa	Korea	*Cucurbita maxima* X *Cucurbita moschata*	[178]
	Vegetable Spaghetti		*C. pepo*	[178]
9.	Super Shintoza	Mexico	Interspecific hybrid squash(*Cucurbita maxima* x *Cucurbita moschata*)	[179]
	Robusta		hybrid of *Citrullus lanatus*	[179]
10.	'Shintoza'	Spain	Interspecific hybrid squash	[180]
11.	Skopje (SKP), Landrace Nun-9075	Turkey	Bottle gourd (*Lagenaria siceraria*) *Cucurbita maxima* × *Cucurbita moschata*	[181,182]
12.	'Shintosa Camel', 'Strong Tosa'	USA	Interspecific hybrid squash(*Cucurbita moschata* X *Cucurbita maxima*)	[18]
	'Emphasis', 'Macis', and 'WMXP 3945'		Bottle gourd (*Lagenaria siceraria*)	[18]
	'Ojakkyo'		Citron (*Citrullus lanatus* var. *citroides*)	[18]

8.1.5. Cover Crops

Recently, several authors have demonstrated that cover crops like hairy vetch (i.e., crimson clover (*Trifolium incarnatum*) and *Vicia villosa*) are used before the cultivation of watermelon to reduce *Fusarium* wilt disease [159]. Several Brassica species such as *Brassica juncea* are rich in glucosinolates, which stimulate antimicrobial compounds to reduce *Fusarium* wilt disease [183]. Zhou et al. (2011) examined nine different *Brassica* spp. to control wilt disease under field conditions; among the species, 'Green Wave' and 'Florida Broadleaf' performed better [184]. Utilization of cover crops as soil amendments ("green manures") can decrease the disease in a specific region, but the importance of the disease reduction could differ based on the region.

8.1.6. Intercropping

Intercropping is an effective and eco-friendly technology for the management of watermelon *Fusarium* wilt disease [185]. Intercropping of watermelon with aerobic rice (*Oryza sativa*) reduces Fon sporulation and spore development and was reported to decrease *Fusarium* wilt disease [186]. Lv et al. (2018) and Li et al. (2019) demonstrated that wheat intercropping increased the disease resistance against the pathogen by enhancing the articulation of defense sensitive genes of watermelon and restricted *Fusarium* wilt disease [124,187].

8.2. Physical Methods

8.2.1. Soil Solarization

Soil solarization is another method to minimize the quantity of Fon in the infested field. In this method, plastic mulch is used to increase temperature under the plastic cover of soil that reduces the pathogen level [26]. The temperature only cannot minimize the fungal pathogens as it depends on many factors [188,189]. For the inactivation of *F. oxysporum* propagules, the temperature required was 57.5–65 °C for 30 min, and this temperature may even be obtained exclusively from the topmost 5 cm of soil. Transplanting watermelon after mid-April (Temperature > 27 °C) with the Fon race 1 resistance variety as fascination can reduce *Fusarium* wilt disease and increase the yield of watermelon in the USA [190]. Soil solarization with plastic mulch has been used in some regions around the globe efficiently, but the challenge how to dispose of the utilized plastic in the environment. Soil solarization is usually applied to minimize the population of Fon in soil and to interrupt the attack of the disease as well as decrease the disease incidence, however, this method cannot control the disease and its efficiency is minimal due to the dependency on appropriate climatic conditions. Sometimes soil temperature fails to reach an adequate level >30 °C to destroy the pathogen population. Finally, it is very effortful and costly to manage the Fon population [26].

8.2.2. Soil Amendments

Formulation of a soil amendment mixture of rice husks, cornstalks, cow manure, mineral ash, different inorganic nutrients, sugarcane residue (bagasse), and oyster shell powder could reduce Fon severity up to 84% when applied in infested fields in Taiwan [191]. Soil amendment with hairy vetch and seaweeds (*Melanothamnus afaqhusainii, Stokeyia indica*, and *Spatoglossum variabile*) suppressed Fon up to 53 to 87% and produced vigorous plants with abandoned fruits and weight [192,193].

8.3. Biological Control

Biological agents are an effective and sustainable alternative approach to control the growth and reproduction of Fon. Various microorganisms have been found to manage soil-borne pathogens including *Paenibacillus spp., Trichoderma spp., Streptomyces spp. Pseudomonas spp. Bacillus spp*., and non-pathogenic *Fusarium spp.* [194]. Conidial suspension of *Penicillium oxalicum* to watermelon transplants and seeds has reduced the disease incidence in both laboratory and field tests [195]. The utilization of BIO (bioorganic fertilizer + 3×10^9 $CFUg^{-1}$ *Paenibacillus polymyxa* and 5×10^7 $CFUg^{-1}$ *Trichoderma harzianum*) can decrease the Fon of watermelon by 59–73% and 60–100% in the field and pot evaluation, respectively [154]. Among the Bacillus species, *Bacillus subtilis* and *Bacillus amyloliquefaciens* suppressed plant pathogens due to the presence of antimicrobial active compounds [196]. Myriocin from the cell-free supernatant (CFS) of *B. amyloliquefaciens* LZNO1 inhibited the growth and suppressed the reproduction of the Fon by deforming conidial structures and damaging membranes. *B. amyloliquefaciens* LZNO1 has been found to be a promising biocontrol tool against Fon for its synergistic effects among antifungal compounds [194]. For sustainable agricultural development, essential oils have become ecological and substitute bio-products against synthetic pesticides. Antifungal compounds of essential oils from different bio-sourced products including 18 Egyptian plant species, *Metasequoia glyptostroboides*, *Eucalyptus erythrocorys*, *Genista quadriflora*, *Echinophora platyloba* (seed), *Piper chaba*, *Syzygium aromaticum, Eucalyptus globulus*, *Cymbopogon citratus*, *Mentha x piperita*, *Mikania scandens,* and *Salmea scandens* acted against vascular wilt disease pathogens [197]. Essential oils from lavender (*Lavender angustifolia*) and marjoram (*Origanum majorana*) effectively managed the Fusarium wilt of melon by 60% and 23%, respectively, when used as a bio-fumigant [198]. A new effective biological antifungal drug from *Tagetes erecta* L. fungicide (TEF) containing flavonoids, 2,5-dicyclopentylidene cyclopentanone could efficiently manage the pathogen (Fon) through the sterol biosynthesis inhibition process [199]. Several

researchers have investigated the potency of different kinds of biological management strategies of the disease in some crop species (muskmelon and watermelon) [162,200].

8.4. Chemical Management

8.4.1. Soil Fumigation

Fumigation has the highest economic and efficient management against soil-borne wilt disease caused by *Fusarium* spp. [201]. In previous findings, methyl bromide was exploited as soil fumigants for managing various soil-borne pests [13]. As methyl bromide has been banned [159], such researchers have tried to evaluate alternative fumigants viz. chloropicrin, methyl iodide, and metam sodium [13,26]. Until now, no impressive alternate fumigants are in stock that can manage *Fusarium* wilt as demonstrated by methyl bromide [201]. Nevertheless, chemicals like methyl bromide, chloropicrin, and metam sodium fumigate are often entirely promising due to the resistant behavior of the chlamydospores, roots developed under the fumigated region, pathogen re-colonization of the soil, and inappropriate utilization of the fumigants. However, the hazardous effect of fumigation on health and the environment has led to the banning of methyl bromide. Finally, if seeds and seedlings are already contaminated and transplanted into the antecedently fumigated field, the fumigation would have a slight effect in controlling the disease. However, soil fumigation is not a profitable and executable method for pathogen elimination [26].

8.4.2. Fungicide

Soilborne pathogens (i.e., *Fusarium* spp.) are very hard to control with chemical fungicides, but the non-systemic nature of protective fungicides shows a slight efficacy on the Fon pathogen. Application of benomyl (15 ppm a.i) as a soil drenching has performed well in controlling tomato and watermelon *Fusarium* wilt under a glasshouse experiment [202]. However, under the same condition, Topsin-M was used either as a soil drench or a seed dresser in Egypt and showed significant efficacy against watermelon *Fusarium* wilt disease [203]. Miller (2017) conducted studies in Georgia and revealed that Proline (a.i. prothioconazole) and Topsin (a.i. thiophanate-methyl) decreased the Fon pathogen significantly under field conditions [204]. Evaluation of three fungicides such as prothioconazole, acibenzolar-S-methyl, and thiophanate-methyl combined with prothioconazole showed significant cut down of watermelon *Fusarium* wilt disease in the field [47,205]. Pydiflumetofen, a new fungicide that was applied two times, first using soil drenching in the course of transplanting and after two weeks (14 days) as a foliar spray, performed better compared to prothioconazole and increased the weight and number of marketable fruit [205]. Control of *Fusarium* wilt disease of watermelon with fungicides is highly ambitious [13] and fungicidal spray or fumigation with methyl bromide is hazardous to the environment [200,206]. Furthermore, as a result of continuing with these fungicides, Fon has developed resistance [207].

Though any of these single technologies cannot provide sufficient control of Fon under field conditions, they could be helpful when combined with other methods of control [26]. Still, the use of resistant varieties is the most promising method of controlling widespread *Fusarium* wilt disease of watermelon. Nowadays, this single management option does not exist in many places. This is why, in order to control *Fusarium* wilt of watermelon successfully, there is a need to involve multiple management tactics such as host resistance, cultural practices (soil amendments and solarization), cover crops, application of fungicide through the drip irrigation system, and the use of disease free seedlings. By using nitrate nitrogen, cover crops can enhance the soil organic matter in the watermelon field. Subsequently, cultural management can play a vital role and help to understand an indigenous antagonistic strain of Fo as disease suppression agents of the *Fusarium* wilt of watermelon. The combination of various methods will provide farmers with a heterogeneous and better system of managing watermelon *Fusarium* wilt disease. Thus, sufficient control might be achieved against the Fon pathogen of watermelon.

8.4.3. Fungicides Resistance and Its Mechanism

Several chemicals have been used to control *Fusarium* wilt disease including ethyl mercury phosphate, dazomet, vancide, dimethyl dithiocarbamate, mancozeb, benzimidazole carbamate (MBC) fungicides [FRAC code: 1], 14α-demethylation inhibiting (DMI) fungicides [FRAC code: 3], and succinate dehydrogenase inhibitor fungicides (SDHI; FRAC code: 7) [205,208]. Fungicides currently approved for regulating Fusarium wilt on watermelon production are restricted; however, recent field studies have shown that thiophanate-methyl, prothioconazole, and pydiflumetofen are effective in lowering *Fusarium* wilt on watermelon [205]. The mode of action of prothioconazole and thiophanate-methyl are distinct but both target single sites; as a result, isolates of *F. oxysporum* f. sp. *niveum* are at risk of developing resistance to one or both fungicides [209].

Resistance to thiophanate-methyl has been found in populations of *F. oxysporum* f. sp. *niveum* as well as diseases other than *Fusarium* spp. such as *B. cinerea* and *M. fructicola* [209,210]. Chung et al. (2009) found that 13 isolates of *F. oxysporum* f. sp. *lilii* and six isolates of *F. oxysporum* f. sp. *gladioli* were resistant to benomyl, thiophanate-methyl, carbendazim, and thiabendazole [208]. Isolates of *Alternaria alternata*, *A. tenuissima*, and *A. arborascens* were found to be resistant to azoxystrobin [211]. Isolates of *B. graminis* f. sp. *tritici* and *Mycosphaerella fijiensis* were detected as strobilurin resistant and DMI-resistant isolates were identified in *E. graminis* f. sp. *hordei* [212]. In another investigation, *F. graminearum*'s unique paralogue FgCYP51C was discovered. FgCYP51C acted as a virulence factor and can alter sterol 14-demethylation indirectly, and did not influence azole sensitivity, implying that CYP51C is a neo-functionalized paralogue. In separate research of *F. graminearum* isolates, Liu et al. (2011) found that FgCYP51C mutants were more sensitive to tebuconazole and prochloraz [213]. Other virulence factors that work as specialized genes or as a part of complex pathways can be activated by *Fusarium* [214].

The resistance mechanism is similar among the benzimidazole fungicides carbendazim, benomyl, thiophanate-methyl, thiabendazole as well as fuberidazole, and positive cross-resistance is likely among group members [215]. Fungicide resistance has become a major issue for the triazole or demethylase inhibitor (DMI) class of fungicides, which are important components of disease-control regimens for humans, animals, and plants [216]. According to the Fungicide Resistance Action Committee (FRAC) of Crop Life International, Brussels, the DMI and SDHI fungicides have a medium risk of resistance [217].

Alteration of the biochemical target site increased production of the target protein, development of an alternative metabolic pathway, metabolic breakdown of the fungicide, and exclusion or expulsion of the fungicide through ATPase-dependent transporter protein are all examples of fungicide resistance mechanisms [208]. A change in the amino acid content of the target molecule, β-tubulin, is the most typical route of resistance to MBC fungicides, which is generally imparted by a single point mutation at precise locations in the gene that encodes the protein [215]. Mutations in TUB2 of fungal pathogens can cause MBC fungicide resistance at codons 6, 50, 167, 198, 200, or 240, with codons 198 or 200 being the most common point mutation [218]. Point mutations in the β-tubulin gene are correlated with resistance, with distinct mutations leading to distinct amino acid alterations at the benzimidazole-binding region. Different levels of resistance occur from these varied mutations at different codon positions [218]. Nakaune and Nakano (2007) revealed that the putative leucine zipper protein CaBEN1 may improve target protein production by boosting β-tubulin gene (CaTUB1) countenance in benomyl resistant *C. acutatum* isolates, and that this protein is likely responsible for the benomyl resistant pathogen [219]. Moreover, in *A. nidulans* and *P. digitatum*, toxin-efflux ATP-binding cassette (ABC) transporters are also involved in resistance to benzimidazole fungicides [220].

Petkar et al. (2017) investigated the effects of thiophanate-methyl on mycelium development and spore germination in 100 isolates of *F. oxysporum f. sp. niveum* and discovered that 33 and 4% of the isolates were resistant to the fungicide at 10 and 100 mg/mL, respectively [209]. This resistance was linked to an amino acid change in the β-tubulin gene caused by a point mutation. The β-tubulin gene was sequenced, and it was discovered that

a mutation occurred at codon 200, with tyrosine replacing phenylalanine in the resistant isolates [209]. Suga et al. (2011) identified isolates of *F. asiaticum* that were resistant to thiophanate-methyl, and the resistant isolates exhibited F167Y or F200Y mutations in the β-tubulin gene [221]. A few isolates of *F. oxysporum* f. sp. *gladioli* and *F. oxysporum* f. sp. *lilii* were resistant to thiophanate-methyl, although sequencing a partial β-tubulin gene of the resistant isolates revealed no mutations in either position 198 or 200. According to the findings, other mechanisms might be implicated in the resistance of the isolates [208].

9. Conclusions and Future Perspectives

Fusarium oxysporum f. sp. *niveum* (Fon) is a pathogenic fungus that significantly attacks and destroys watermelon farms. This pathogen is morphologically indistinguishable but phylogenetically unambiguous using different markers like SSR, ISSR, LAMP, and using specific gene sequences like calmodulin (*cmdA*) as well as the elongation factor-1alpha (*tef1α*) that was reported to be reliable. Presently, different disease assays have remained the primary method of differentiating races and host range of a pathogenic strain of Fon. Some techniques using DNA sequences to differentiate the formae speciales have been discovered and could differentiate among not only formae speciales, but also their difference in races. The Fon was reported to produce mycotoxin (fusaric acid), which mainly disturbs the metabolism of growing plants. Some novel disease management strategies like the use of resistant varieties (summer king, seedway, SP-5, and SP-6) and crop rotation have shown promising results. Another safer method includes the use of biological agents that serve as an effective and sustainable alternative approach in controlling the growth and reproduction of Fon. Likewise, essential oils from lavender (*Lavender angustifolia*) and marjoram (*Origanum majorana*) as well as a new effective antifungal botanical drug from *Tagets erecta* L. fungicide (TEF) could efficiently manage the pathogen. The chemical fungicides currently approved for regulating *Fusarium* wilt on watermelon are restricted and are at risk of developing resistance in Fon. The above discussion on the Fon pathogen could be beneficial for watermelon plant breeders to channel their energy in developing a watermelon variety that is resistant to Fon. Moreover, this review will stipulate extensive in and out information of host–pathogen synergy at the heritable level. This paper contributes to the understanding of aggressive patterns of Fon and the development of molecular markers for the evolution of new strategies of Fon management in the future.

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