Review Article

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Current status of Fusarium wilt resistance research in watermelon (*Citrullus lanatus*): A review

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ABSTRACT

Fusarium wilt is one of the widespread and important fungal diseases of cucurbit crops in the United States and worldwide. It is caused by the fungal pathogen *Fusarium oxysporum* f. sp. *niveum* (*Fon*) that causes significant yield loss in watermelon. There are many commercial cultivars of watermelon that are resistant to *Fon* races 0 and 1. However, no edible cultivars resistant to races 2 or 3 have been developed. There is a necessity to understand the genetic and molecular basis of Fusarium wilt resistance for crop improvement. Therefore, exploring genetic and molecular factors to determine the resistance to virulent *Fon* races 2 and 3 in diverse watermelon genotypes will be useful. The recent advances in marker-assisted selection, genomic selection, whole-genome sequencing, gene-editing tools, and genome-wide association studies provide broader insights for improvement of watermelon varieties. In this review paper, we discuss the biology of the Fusarium wilt pathogen (*Fon*) of watermelon, the history of the Fusarium wilt disease, inheritance studies on Fusarium wilt, quantitative trait loci mapping, bottlenecks in Fusarium wilt resistance breeding, marker-assisted selection and novel plant breeding tools to improve Fusarium wilt resistance in watermelon. This review paper aims to explore the current efforts, challenges, and suggest potential future research for the management of Fusarium wilt using traditional and molecular breeding tools.

Introduction

Watermelon (*Citrullus lanatus* [Thunb.] Matsum. and Nakai) is a widely cultivated cucurbit crop within the family Cucurbitaceae. Watermelon is a good source of biologically active compounds, such as lycopene, vitamins A and C, β-carotene, potassium, and total phenolic compounds. These compounds contain anti-inflammatory, anticancer, and antioxidant properties (Maoto *et al.*, 2019). In addition to these nutrients, it contains specific amino acids, notably arginine and citrulline. Citrulline is predominantly found in the peel, rind and flesh of fruit (Perkins-Veazie *et al.*, 2007). Along with nutritional importance, watermelon has substantial economic value. In 2024, it is estimated that the total production of watermelon was 3.69 billion pounds and it was grown on 110,900 acres of land in the United States (USDA, 2025). The leading watermelon-producing states in the United States (U.S.) are Florida, Texas, Georgia, and California, as the crop requires a warm growing season for optimal production (Wehner, 2008).

Fusarium wilt, caused by a soil-borne, ascomycete fungus *Fusarium oxysporum* f. sp. (forma specialis) *niveum* (*Fon*),

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is one of the most serious diseases of watermelon. It is one of the top five fungi of scientific importance (Dean *et al.*, 2012). Fusarium wilt has been designated as number one constraint in watermelon production in the U.S. (Kousik *et al.*, 2016). It is a major limiting factor in watermelon production worldwide, with yield loss ranging from 30% to 80% (Rahman *et al.*, 2021). Yield losses ranging up to 80-85% have also been reported (Zhang *et al.*, 2012; Hall & Holloway, 2000). Similarly, around 50% disease incidence has been observed in the commercial watermelon fields of southeastern US (Petkar & Ji, 2017). Individual vines or the entire watermelon plant is killed by the pathogen, thereby reducing the yield (Keinath *et al.*, 2010). There are several direct and indirect losses incurred due to Fusarium wilt disease. Direct losses include the loss of the marketable yield. Indirect losses arise due to costs associated with replanting, roguing, soil treatment, labor, keeping land fallow, etc. (Egel & Martyn, 2007).

Fon is both a seed-and soil-borne plant pathogen, and the infected plant shows symptoms like chlorosis, necrosis, leaf fall, browning of the vascular system, and eventually wilting of plants (Porter, 1928). The loss of turgor pressure in leaves and vines is seen as the early symptom of infected watermelon plants (Kurt *et al.*, 2008). As the condition deteriorates, the plant becomes dull green to yellow and necrotic (Pursley *et al.*, 2010). The continuous colonization of the xylem vessel by *Fon* forms enough tyloses that restricts the water movement, thus causing wilt in plants (Shaban & Abdelssalam, 2009). The optimum temperature for the growth and development of *Fon* is 24 to 32°C (Porter, 1928). *Fon* lacks a known sexual stage and is a root infecting vascular pathogen. It can survive extended periods in the absence of the host, mainly in the form of thick-walled chlamydospores (Pietro *et al.*, 2003). When susceptible watermelon cultivar interacts with *Fon* and develop wilt disease, it indicates a compatible interaction. However, resistant cultivars have an incompatible interaction with *Fon*, hence there is no disease development (Lü *et al.*, 2011).

Management practices such as resistant rootstock, grafting, soil fumigation, chemical treatment, use of cover crops, and biological control agents may help to reduce the incidence of Fusarium wilt in watermelon (Everts & Himmelstein, 2015). In addition, cover crops such as hairy vetch (*Vicia villosa*) are found to be effective in controlling Fusarium wilt in watermelon (Keinath *et al.*, 2010). Furthermore, fungicides like prothioconazole and pydiflumetofen are identified as potential strategies in controlling the Fusarium wilt (Miller *et al.*, 2020). Intercropping watermelon with aerobic rice reduces Fusarium wilt by preventing the *Fon* sporulation (Ren *et al.*, 2008). Late transplanting and selecting the *Fon* race 1 resistant 'Fascination' or the tolerant cultivar 'Melody' can help in controlling Fusarium wilt in watermelon and increase marketable yield in the Southern U.S. (Keinath *et al.*, 2019).

Taxonomy and biology of Fusarium wilt of watermelon

The binomial nomenclature for the genus *Fusarium* was first described in 1809 by Link (Morris & Nutting, 1923). The species was then termed *Fusarium niveum* by Smith in 1899 based upon his observations on wilt disease on watermelon, cotton, and cowpea (Sleeth, 1934). The species was later changed to *Fusarium oxysporum* (Hansford, 1926). In 1935, Wollenweber and Reinking reclassified the fungus isolated from watermelon and gave it the new name *Fusarium bulbigenum* var. *niveum* Woll (Cipolla, 1953). Synder and Hansen further refined this classification and further proposed that the specialized form of *F. oxysporum*, characterized by specific virulence to host plants, should be classified as distinct 'forma specialis' and considered watermelon wilt form 1 as *F. oxysporum* f. sp. *niveum* (*Fon*) (Snyder & Hansen, 1940). Till today, four distinct races (0, 1, 2, 3) of *Fon* have been identified in watermelon genotypes (Zhou *et al.*, 2010). Table 1 shows the responses of diverse watermelon genotypes (differentials) against different races of *Fon* (Egel & Martyn, 2007; Wechter *et al.*, 2016; Dutta & Coolong, 2017). Most of the resistance to Fusarium wilt race 2 was reported in wild relatives, *Citrullus amarus*, such as PI 296341-FR. Most of the sweet, edible watermelon cultivars (*Citrullus lanatus*) are resistant to races 0 and 1 of *Fon* (e.g., Calhoun Gray, Allsweet).

Table 1. Responses of differential watermelon genotypes to four races of Fusarium wilt fungus, *Fusarium oxysporum* f. sp. *niveum*

Genotypes	Taxonomy	Race 0	Race 1	Race 2	Race 3
Sugar Baby	*C. lanatus*	S	S	S	S
Black Diamond	*C. lanatus*	S	S	S	S
Charleston Gray	*C. lanatus*	R	S	S	S
Crimson Sweet	*C. lanatus*	R	S	S	S
Calhoun Gray	*C. lanatus*	R	R	S	S
Allsweet	*C. lanatus*	R	R	S	S
PI 296341-FR	*C. amarus*	R	R	R	S
USVL246-FR2	*C. amarus*	R	R	R	S
USVL252-FR2	*C. amarus*	R	R	R	S

R=Resistance; S=Susceptible

The *Fon* fungus can spread through soil, plant debris, and farm implements and seeds and can live in soil for almost

a decade (Bruton *et al.*, 2007). As a germinating spore, the fungus initiates the infection process. Eventually, the hyphae enter the plant through wounds or any other natural opening. The hyphae then penetrate the vascular tissue, resulting in the production of microconidia. As microconidia enter the xylem, they infect by spreading into the watermelon's vascular system (Bishop & Cooper, 1983; Di Pietro *et al.*, 2003). These macroconidia form chlamydospores, also known as asexual structures or resting spores, as the fungus does not have a known sexual reproductive stage. Fusarium wilt disease is typically caused by the dissemination of chlamydospores (Egel & Martyn, 2007).

History of Fusarium wilt disease of watermelon

An unknown watermelon wilt disease was inflicting large losses in the Southern United States in the early 1890s, and at the same time, similar wilt symptoms were also seen in okra and cotton in Mississippi, Georgia and Alabama (Martyn, 2014). The Fusarium wilt disease was first reported in 1894 in watermelon fields of Georgia and South Carolina by Erwin. F. Smith, a USDA plant pathologist (Smith, 1894), and further researched upon (Smith, 1899). *Fon* race 2 was first reported in Texas in 1981 (Martyn, 1985), and later in Oklahoma (Bruton *et al.*, 1988), Florida (Martyn, 1989), Maryland and Delaware (Zhou & Everts, 2001), Indiana (Egel *et al.*, 2005), and Georgia (Bruton *et al.*, 2008). Fusarium wilt pathogen (*Fon*) now has four races (0, 1, 2, and 3) that have been identified based on their aggressiveness on different watermelon cultivars (Martyn, 1985, Zhou *et al.*, 2010). The use of watermelon differential lines to determine the race of different *Fon* strains has also been reported (Cirulli, 1972). Cucurbit wilt is caused by six distinct 'forma specialis', the most commercially significant of which are *F. oxysporum* f. sp. *niveum* in watermelon, *F. oxysporum* f. sp. *melonis* in muskmelon, and *F. oxysporum* f. sp. *cucumerinum* in cucumber (Okungbowa & Shittu, 2012). There are more than 100 host-specific strains of *Fusarium oxysporum* (Gordon, 2017).

Inheritance and mapping studies related to Fusarium wilt resistance

Orton developed the first *Fon*-resistant cultivar 'Conqueror' from a cross of *Fon*-resistant citron melon (*C. amarus*) with *Fon*-susceptible 'Eden' variety (Orton, 1907). However, this variety showed resistance against *Fon* in eastern Iowa but was observed to be susceptible in Oregon, showing the environmental effects on the cultivar (Bennett, 1937). The watermelon cultivar 'Summit' was reported to be a completely dominant source of resistance to *Fon* (Henderson *et al.*, 1970). Segregation analysis using different crosses of resistant cultivars (Calhoun Gray & Summit) with the susceptible cultivar (Mallali) along varying generations (F_1, F_2, and Back Cross or BC) showed a single dominant gene was likely conferring resistance to *Fon* race 1 (Netzer & Weintall, 1980). Most of the modern, elite diploid watermelon cultivars show resistance to races 0 and 1 of *Fon* (Martyn & Netzer, 1991; Zhou *et al.*, 2010) and the major resistance QTL (Fo-1.1) for *Fon* race 1 was reported at the end of short arm on chromosome 1 of watermelon genome (Lambel *et al.*, 2014)2014. Among the *Fon* races, race 2 is widely prevalent in the U.S. and is associated with huge yield and financial losses (Hall & Holloway, 2000; Zhang *et al.*, 2012). So, to develop the *Fon* resistant commercial watermelon cultivar, it is necessary to understand the inheritance pattern of race 2 resistance (Martyn, 2014). The resistance of watermelons to virulent *Fon* race 2 is polygenic with moderate heritability (Biswas *et al.*, 2025) and several quantitative trait loci (QTL) studies have reported *C. amarus* germplasm as the source of resistance (Table 2).

Table 2. Quantitative trait loci (QTL) mapping studies related to Fusarium wilt resistance in watermelon

Cross	Generations	Race	Chromosomes	Logarithm of odds (LOD) score	% Phenotypic variance (PV)	References
'HMw017' × 'HMw013' (R × S)	F_3	1	1, 3, 4, 9, & 10	4.26 to 33.31	11 to 59.9	Lambel *et al.*, 2014
'97103' × 'PI 296341-FR'	F_8 [RILs]	1	1	13.2	48.1	Ren *et al.*, 2015
'97103' × 'PI 296341-FR'	F_8 [RILs]	2	9 & 10	3.1 to 3.3	12.5 to 13.7	Ren *et al.*, 2015
'Calhoun Gray' × 'Sugar Baby' (R×S)	F_3	1	1	Not available	38.4	Meru & McGregor, 2016a

'Charleston Gray' × 'UGA147' (S × R)	$F_{2:3}$	2	11	3.89 to 5.93	9.6 to 16.2	Meru & McGregor, 2016b
'USVL246-FR2' × 'USVL114' (R × S)	$F_{2:3}$	2	2, 5, 8, 9, & 10	7.6 to 40.5	5.0 to 43.2	Branham *et al.*, 2017
'ZXG01478' x '14CB11' (R × S)	F_2	1	1	26.05	80.18	Na *et al.*, 2017
'HMw017' × 'HMw013' (R × S)	F_3	1	1 & 11	27.9	55.2	Branham *et al.*, 20182018
'USVL246-FR2' × 'USVL114' (R × S)	$F_{2:3}$ & RILs	1	9	5.6 to 10.7	14.1 to 24.7	Branham *et al.*, 2019
'USVL252-FR2' × 'PI 244019' (R × S)	F_2 & $F_{2:3}$	2	1	12.8	18.9	Branham *et al.*, 2020
'EC79442'1 x 'BIL-53) (R × S)	$F_{2:3}$	2	1 & 7	Not available	Not available	Pal *et al.*, 20232023

*S=susceptible; R=resistant; RIL=recombinant inbred lines

Marker assisted selection for Fusarium wilt resistance breeding in watermelon

Marker-assisted selection (MAS) can be used as a valuable tool to determine QTL linked to a particular trait (Ren *et al.*, 2015). Dominant, polymerase chain reaction (PCR)-based random amplified polymorphic DNA (RAPD) markers developed for *Fon* races 1 and 2 could not be used because of the large linkage estimate between the trait and the genetic marker (Hawkins *et al.*, 2001). The co-dominant SNP marker 'UGA1_502161' can be used as a useful marker for *Fon* race 1 resistance screening (Fall *et al.*, 2018). Similarly, co-dominant KASP (Kompetitive Allele Specific PCR) markers tightly linked to *Fon* race 1 resistance have also been developed and can be used to screen the watermelon parents and progenies during different early and advanced generations of the breeding cycle (Branham *et al.*, 2018). Further, Ren *et al.* (2015) developed a SNP marker 'Chr1SNP_5202124' that could be used for *Fon* race-1 resistance screening. Microsatellite markers have also been developed to facilitate MAS for *Fon* race 2 resistance in watermelon (Pal *et al.*, 2023).

Bottlenecks in Fusarium wilt resistance breeding in watermelon

Although the consumption of seedless watermelon varieties has increased (Wehner, 2008), most of the seedless triploid cultivars of watermelon are generally more susceptible to all the races of *Fon* (Egel & Martyn, 2007; Keinath *et al.*, 2010). Primers have been developed to distinguish races 1 and 2 of Fon and can detect the presence of pathogen at the earlier stages of wilting (Lin *et al.*, 2010). Many currently available commercial cultivars have resistance to *Fon* races 0 and 1, but until now, no edible cultivar resistant to *Fon* races 2 or 3 has been developed (Ganaparthi *et al.*, 2024). Resistance to race 2 *Fon* has been reported to be polygenic in wild watermelon species (Wehner, 2008). MAS for introgression of *Fon* race 2 resistant genes from PI 296341-FR to develop a resistant commercial cultivar was not successful in developing a sweet, edible cultivar due to a negative linkage drag between the resistant gene and poor-quality traits (Meru & McGregor, 2016b). Thus, there also arises a need to explore alternative technologies to reduce losses in watermelon production, productivity, and quality due to Fusarium wilt.

Novel approaches to enhance Fusarium wilt resistance in watermelon

The domesticated, seeded, sweet watermelon (*C. lanatus*) is a diploid with each of their cells containing two sets of 11 pairs of chromosomes. Reference genomes of pathogenic and non-pathogenic strains of *Fon* are also available (Fulton *et al.*, 2022). Several novel approaches could be utilized to enhance the resistance of watermelon to the virulent *Fon* race 2. For instance, the grafting of rootstock of cucurbit crops (squash, bottle gourd) with watermelon scions inhibited the races 1 & 2 *Fon* and increased the marketable yield of watermelon (Keinath & Hassell, 2014). Since *Fon* race 2 resistance is governed by multiple genes, another breeding strategy – genomic selection (GS) could be more effective to incorporate all the resistance genes at once than trying to incorporate few major QTL (Meuwissen

et al., 2001; Biswas *et al.*, 2025). Fungicide-based disease management practice can be effective to lower Fusarium wilt incidence and effect in watermelon fields (Everts *et al.*, 2014). Previously, methyl bromide – a popular soil fumigant used in controlling Fusarium wilt – had been banned owing to its ozone-depleting characteristics (EPA, 2006). Conventional PCR-based primers to differentiate race 3 from races 1 & 2 of *Fon* have been developed (Hudson *et al.*, 2021). Real-time PCR analysis to detect *Fon* race 1 of watermelon from plants and soil have also been reported (Zhong *et al.*, 2022). Precise genome editing technology (e.g., Clustered Regularly Interspaced Short Palindromic Repeats or CRISPR/Cas9 system) are crucial tools for the improvement of crops such as watermelons (Bhatta & Malla, 2020; Feng *et al.*, 2013). In watermelon, CRISPR/Cas9 has been already used to develop herbicide-resistance (Tian *et al.*, 2018; Tian *et al.*, 2017) and to perform knockout of the *Clpsk1* gene to increase resistance to *Fon* race 1 (Zhang *et al.*, 2020). A recent genome-wide association (GWAS) study using a diversity panel of 120 *C. amarus* accessions found several quantitative trait nucleotides (QTNs) on chromosomes 1, 5, 9, and 10 explaining up to 60% of *Fon* race 2 resistance (Ganaparthi *et al.*, 2023).

Conclusion

Continuous domestication and selective breeding for specific trait have led to a narrow genetic base and susceptibility to diseases such as Fusarium wilt in the cultivated sweet dessert watermelons (Levi *et al.*, 2001). The future studies should study interaction between different *Fon* races and host (watermelon) using molecular, biochemical, and physiological approaches. Further research on identifying *Fon* resistant sources besides *C. amarus*, such as the *C. mucosospermus* and other species, will expand the repertoire of disease resistant alleles to breed for Fusarium wilt-resistant, cultivated watermelons.

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P.B., G.T., & L.J.: *Literature Review, First Draft Preparation*; **B.P.B. & F.M.**: *Review and Editing*; **B.P.B.**: *Conceptualization, Supervision, and Final Review*

Conflict of interest

The authors declare no conflict of interest.

Data Sharing

This review article did not generate any supplementary data.

Informed Consent

All the authors agree with the content of this review article.

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